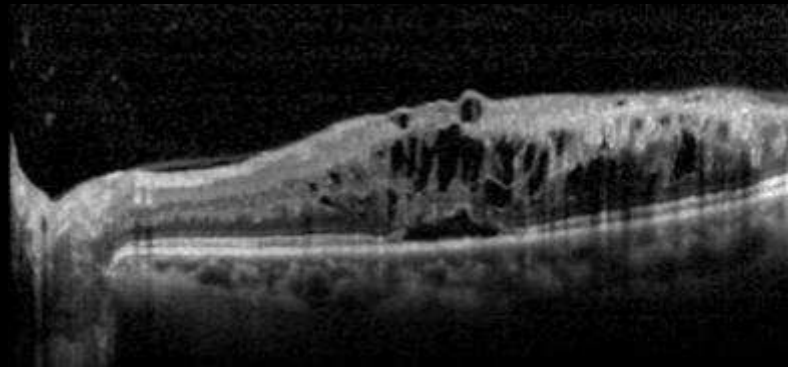


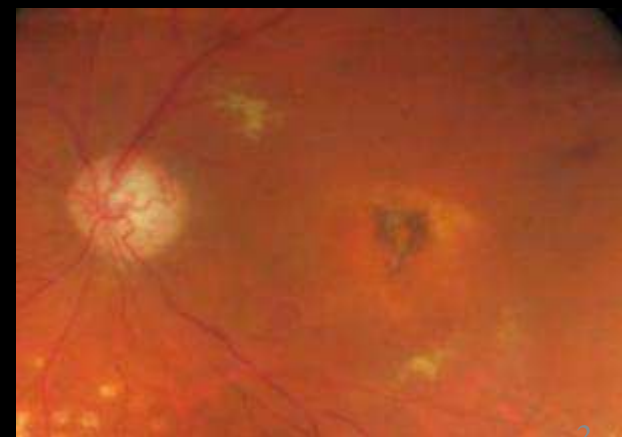
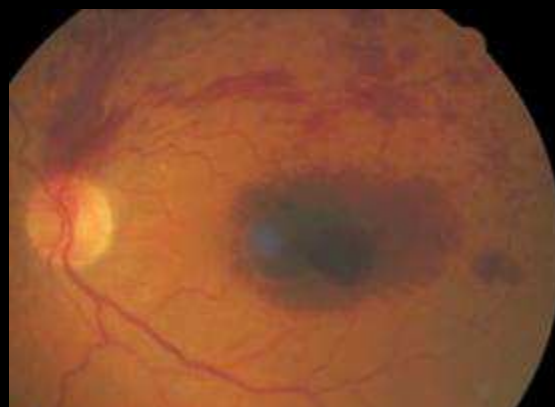
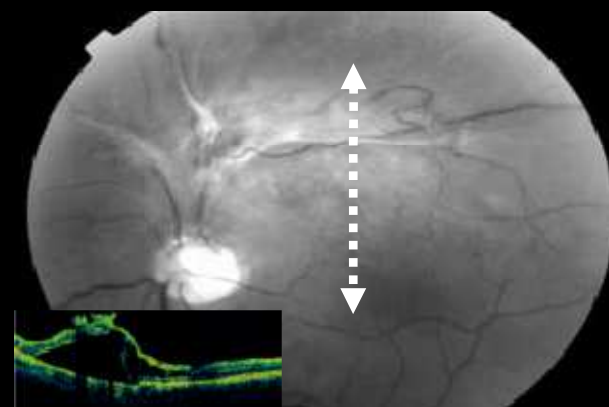
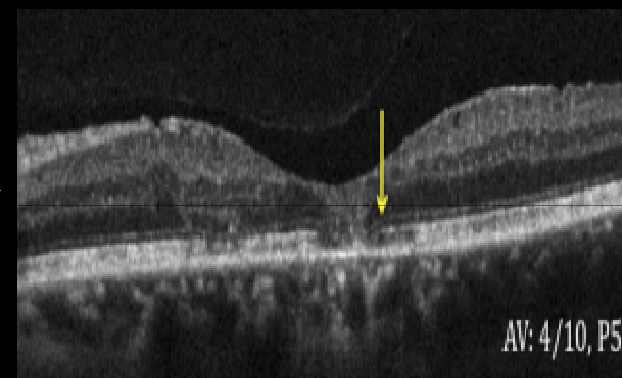
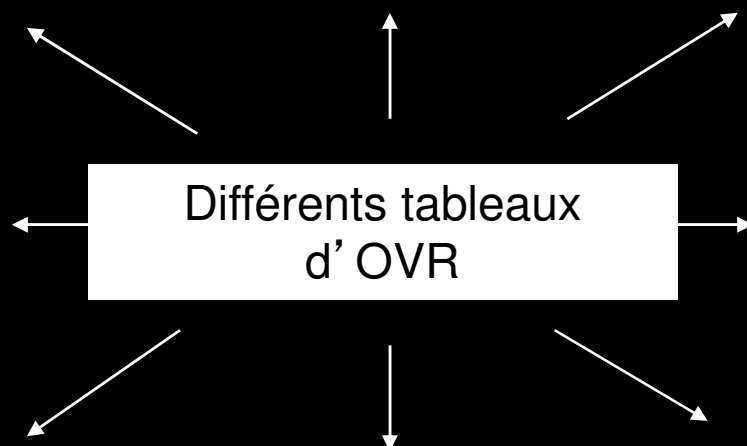
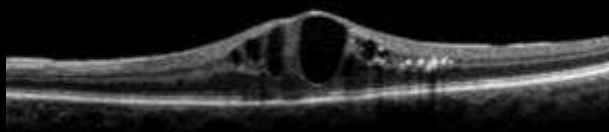
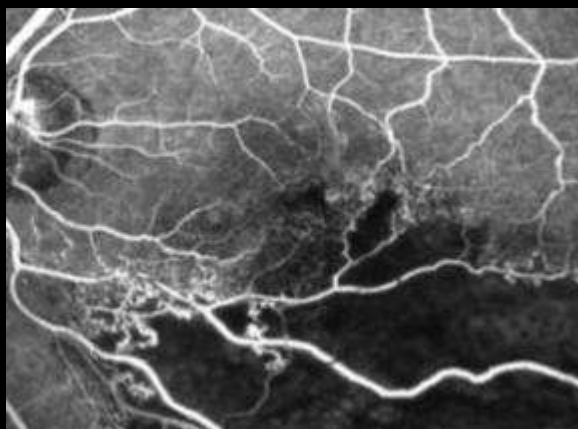
OCCLUSIONS VEINEUSES RETINIENNES



M. Paques, JF Girmens

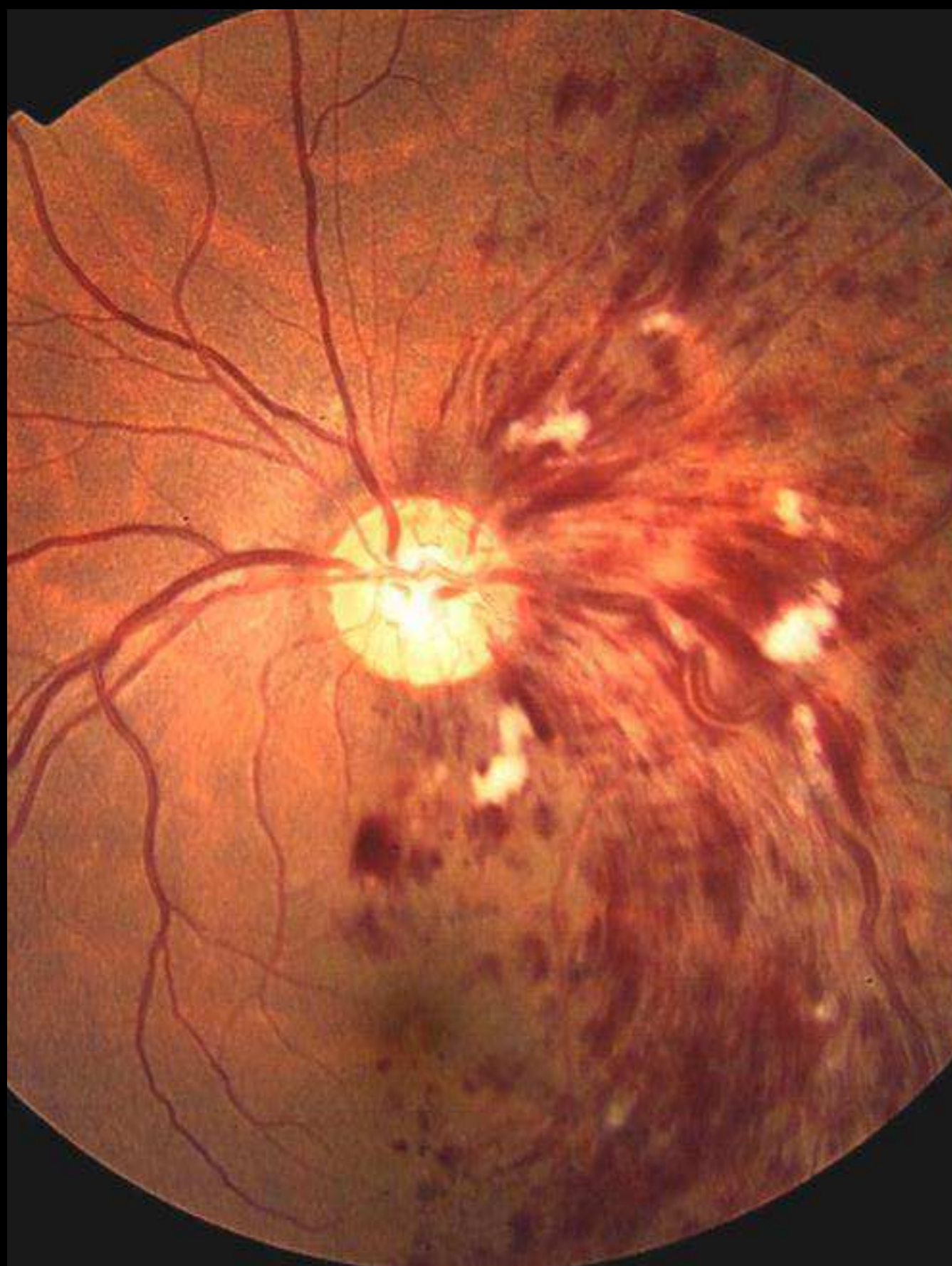
CHNO des Quinze-Vingts

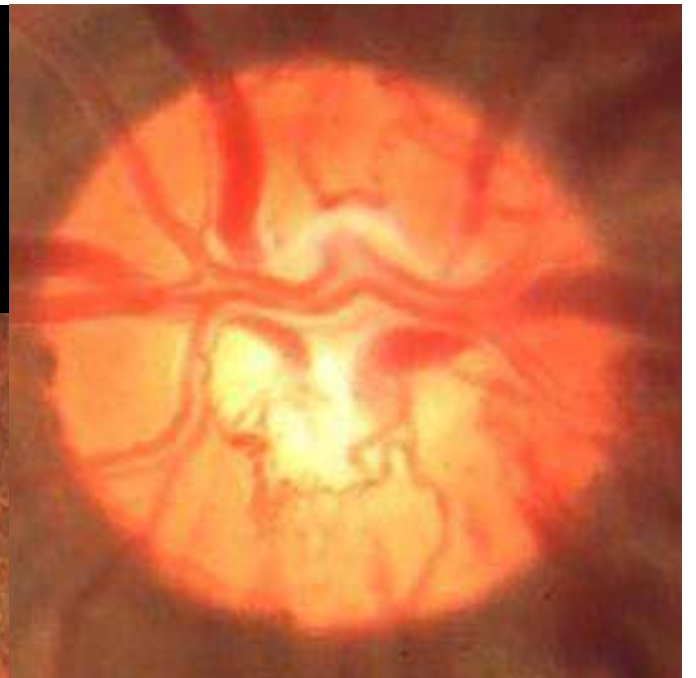
Conflits d'intérêts: aucun

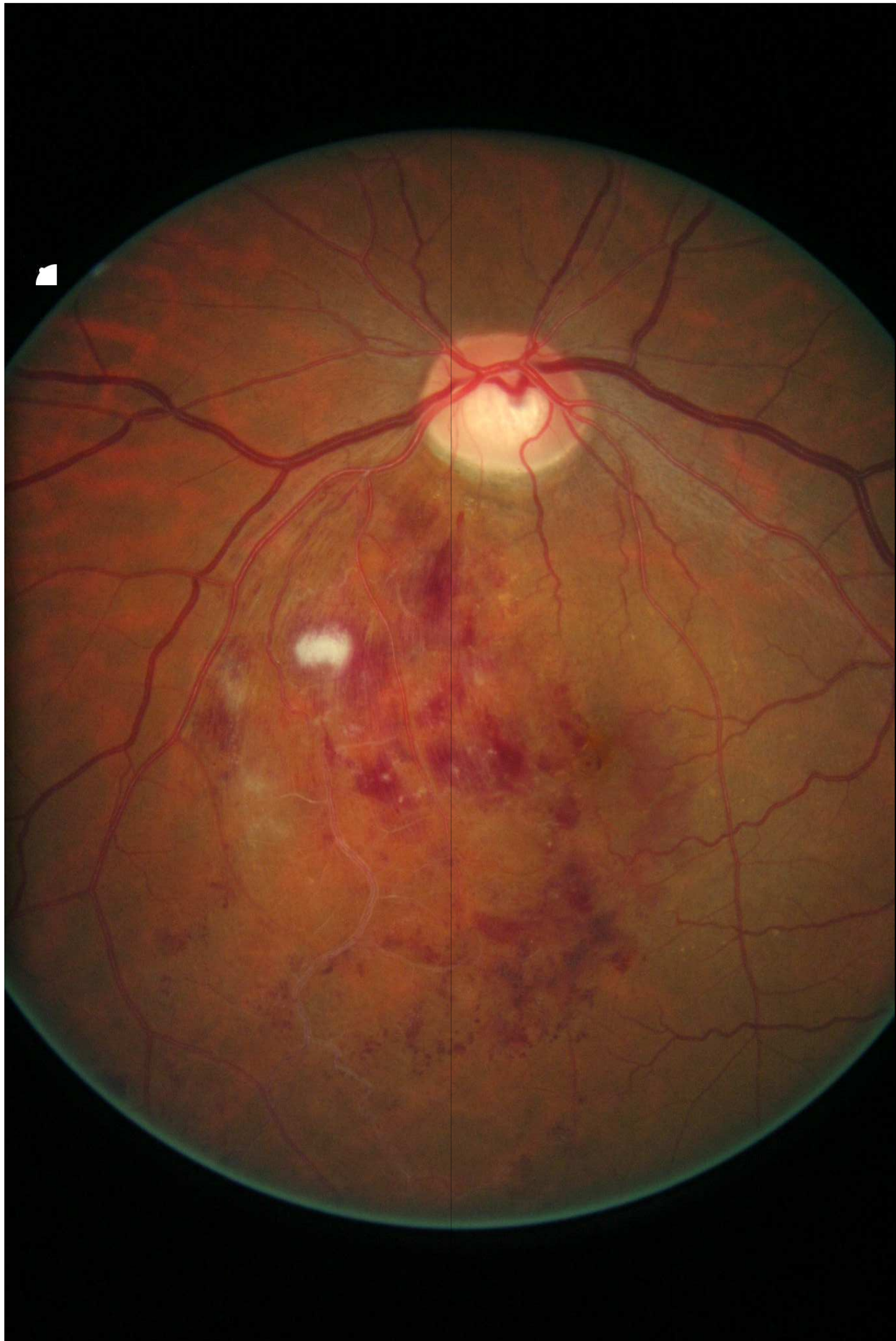


DU IMAGERIE ET PATHOLOGIE RETINIENNES













Occlusions veineuses rétiniennes

4 territoires:

- Veine centrale de la rétine
- Héli-veine centrale
- Branche veineuse
- Excavation papillaire

Occlusions veineuses rétiniennes

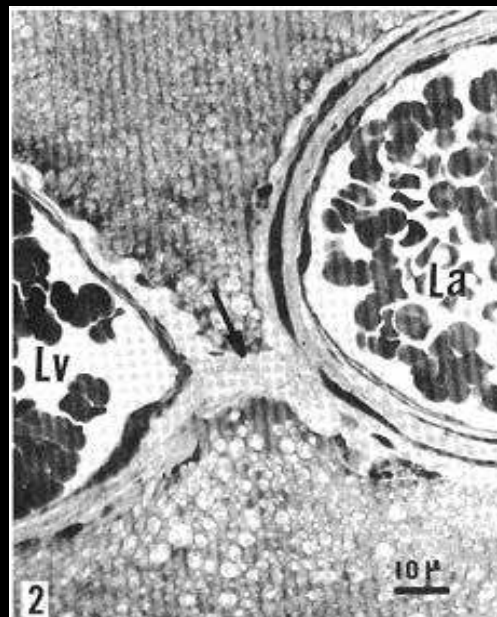
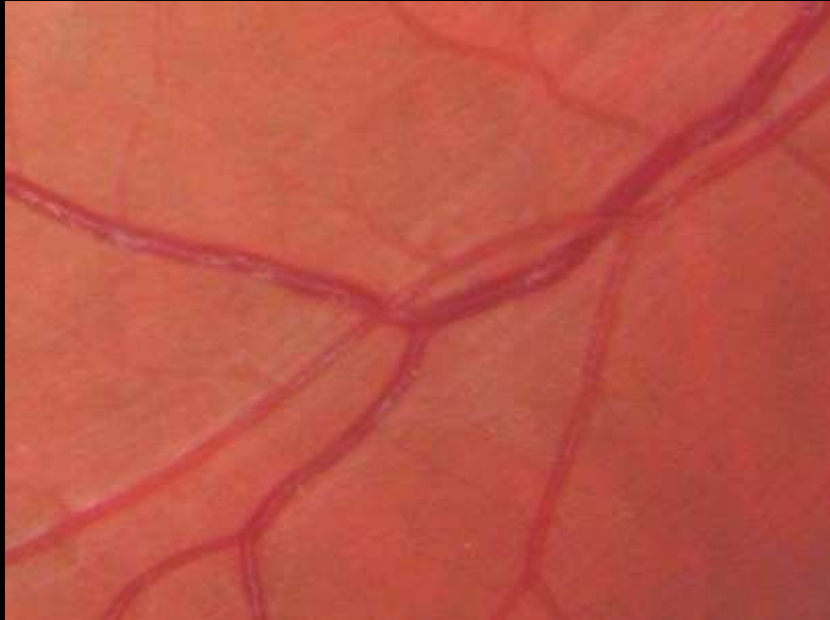
- Pathologie spécifique de l'œil
- Thrombose incertaine
- ≠ Occlusion complète
 - ➔ Maladie obstructive veineuse rétinienne

Quelle est la cause des OVR?

Anatomopathologie des OVCR

- Pas de modèle animal
- Est-ce une pathologie thrombotique?
 - Association OVCR et phlébite est inhabituelle
 - Pas de trouble de coagulation clairement associé
 - Des OVCR peuvent survenir sous anticoagulants
 - Peu de cas très récents examinés en anapath
 - Coats 1903: absence de thrombus initial
 - Verhoeff 1907, Green 1981: thrombus+athérosclérose de l' artère centrale de la rétine
- ➔ Thrombus primitif ou secondaire au ralentissement du flux?
- ➔ Aucun argument anapath formel pour la présence d' un thrombus primitif

Williamson BJO 1997

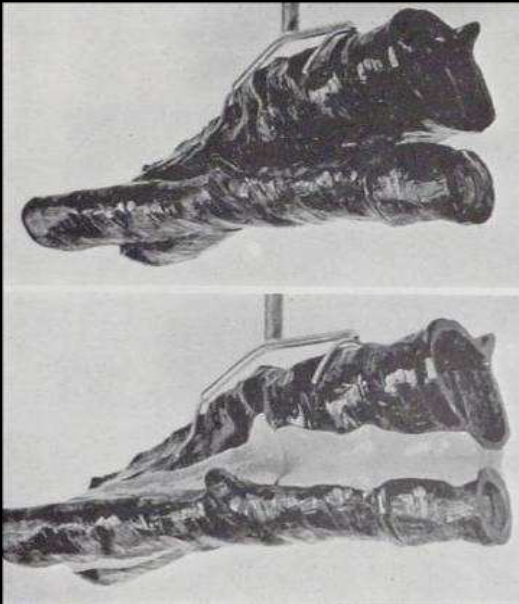


Adventice commune:
Fibres solidarissant l'artère et la veine
aux croisements AV

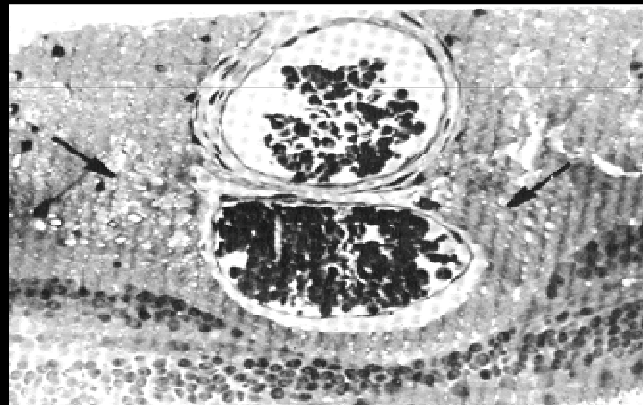
Que se passe-t-il dans un signe du croisement?

L'histologie montre

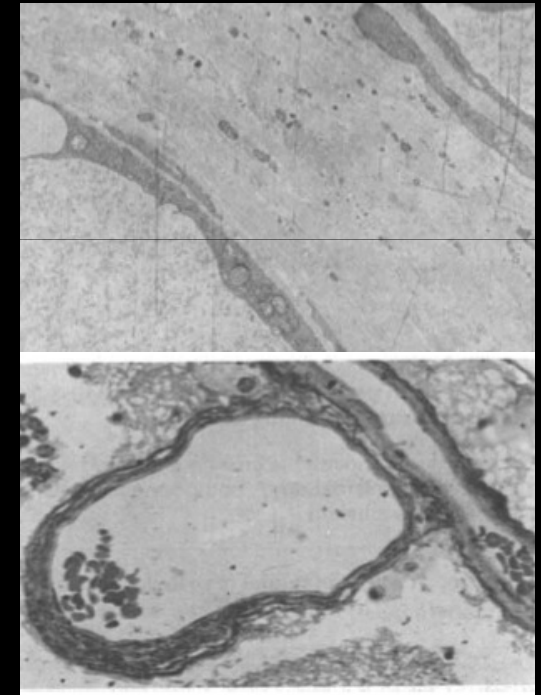
- *des modifications histologiques rétinienne autour du couple artérioveineux*
- *Mais que la veine n'est pas directement atteinte par l'artère*



Seitz et coll. 1964

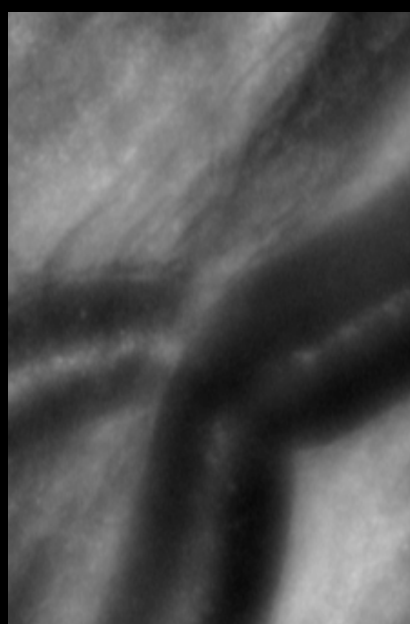
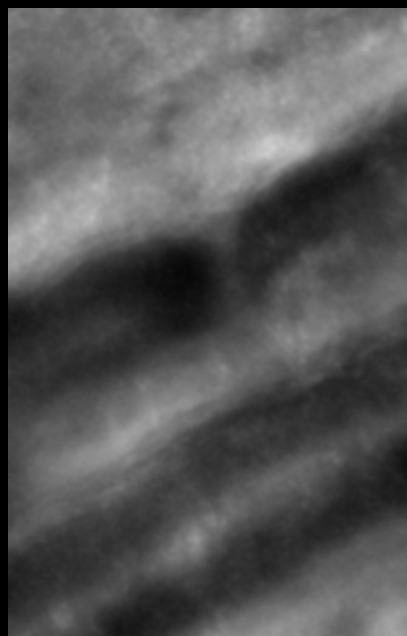
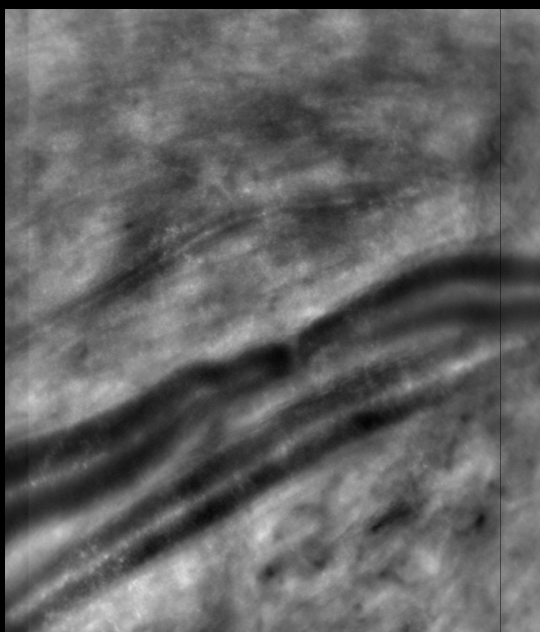


Kimura et al. Int Ophthalmol 2005

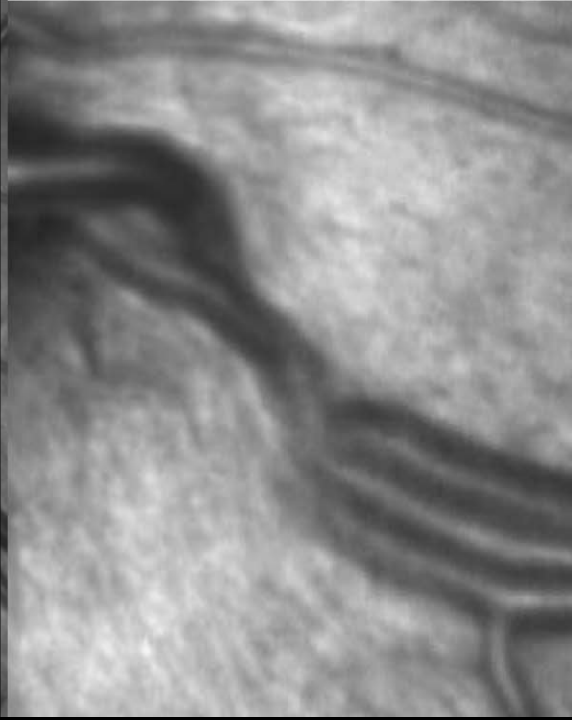
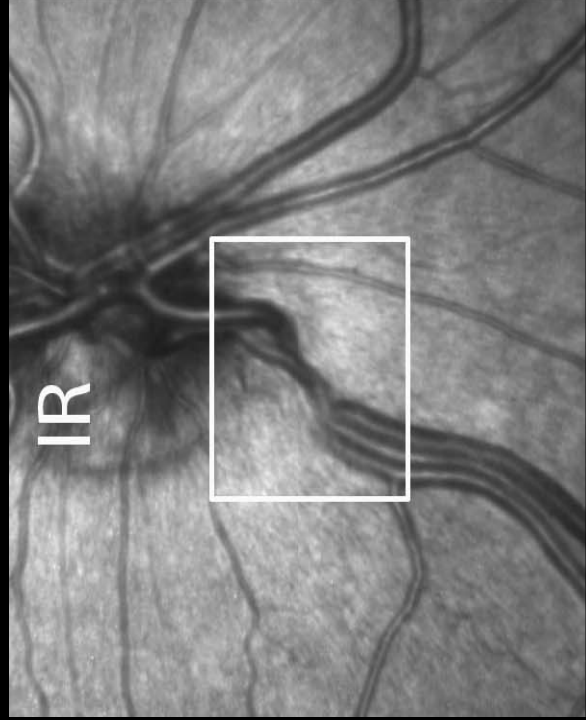
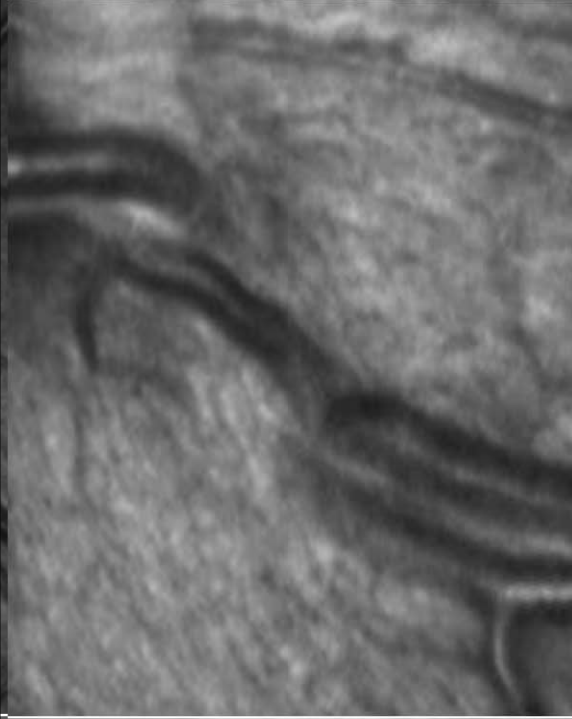
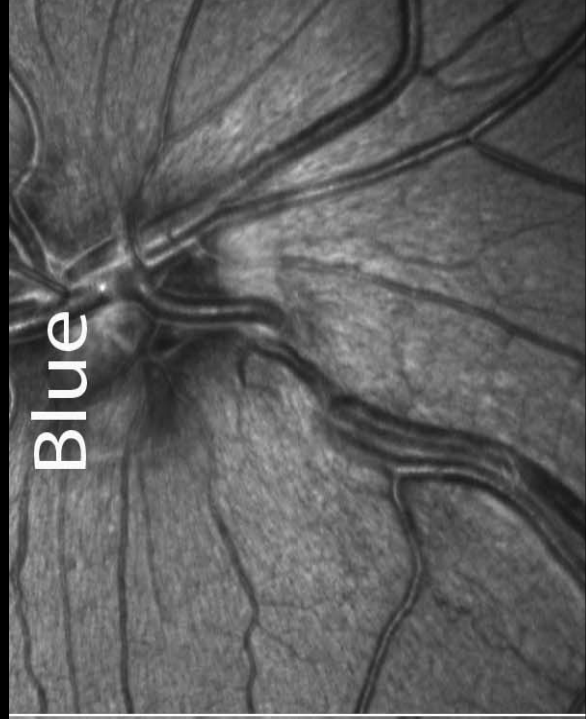
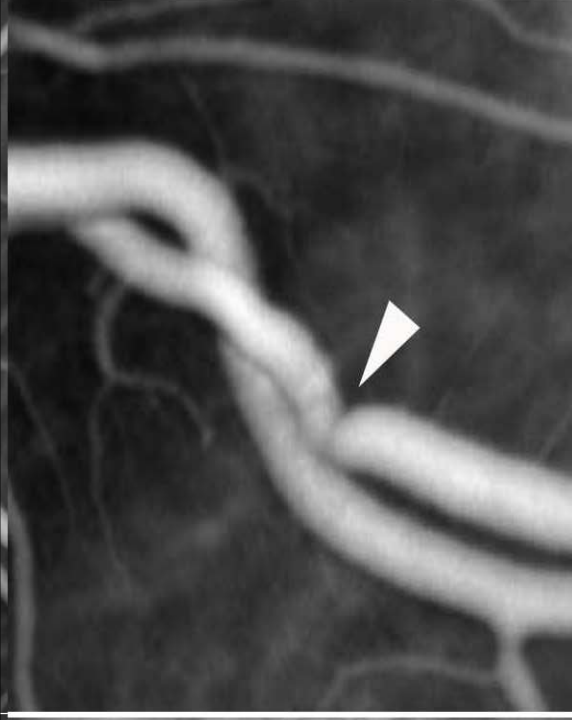
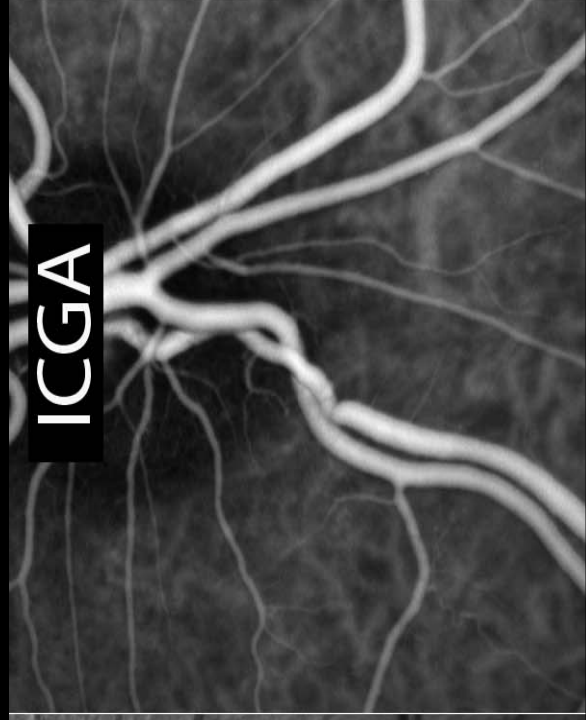


- *Jefferies et al.*
- *Aust NZ J Ophth 1993*

➔ *Fausse évidence de l'écrasement veineux par l'artère*

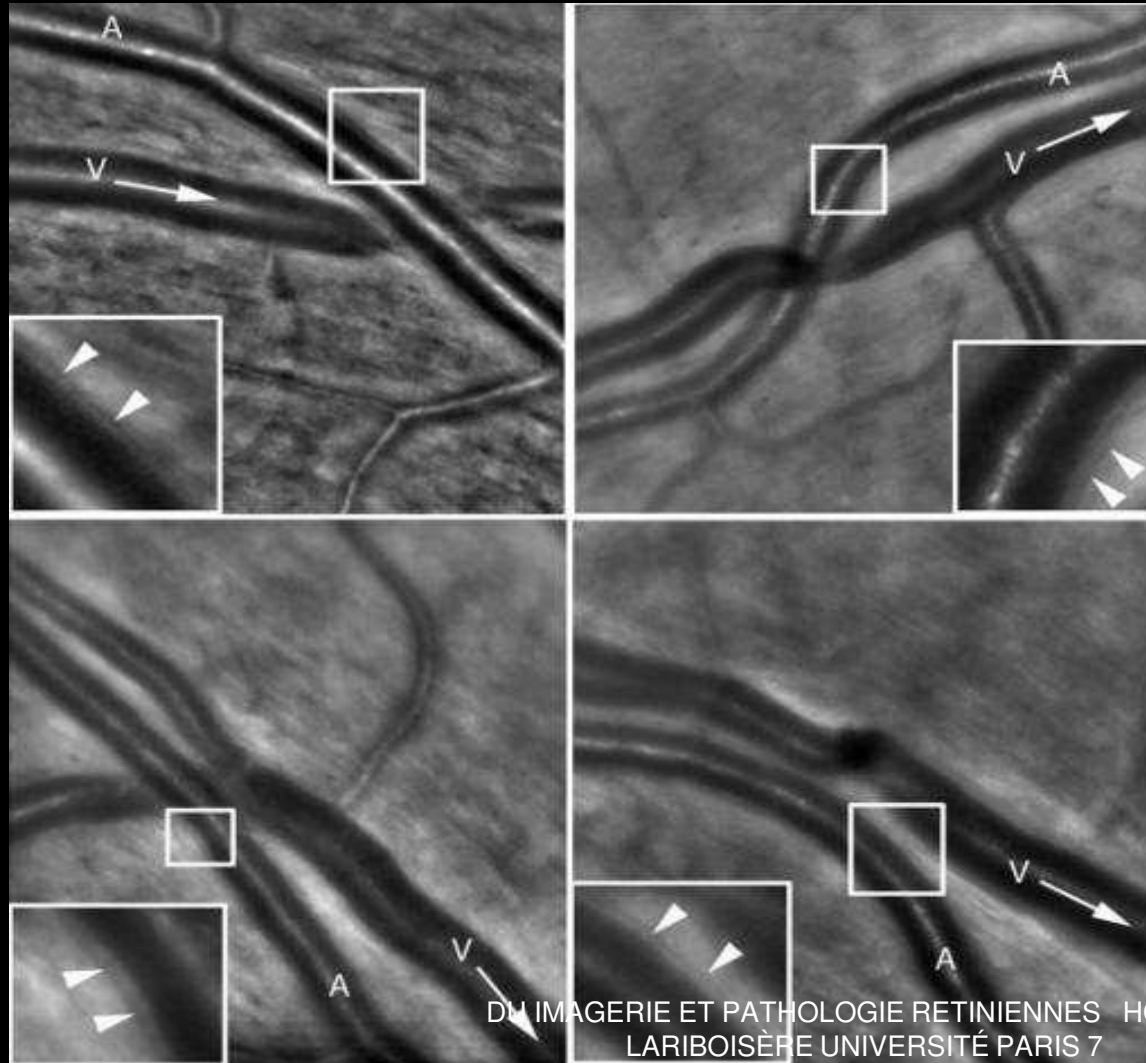


ICGA

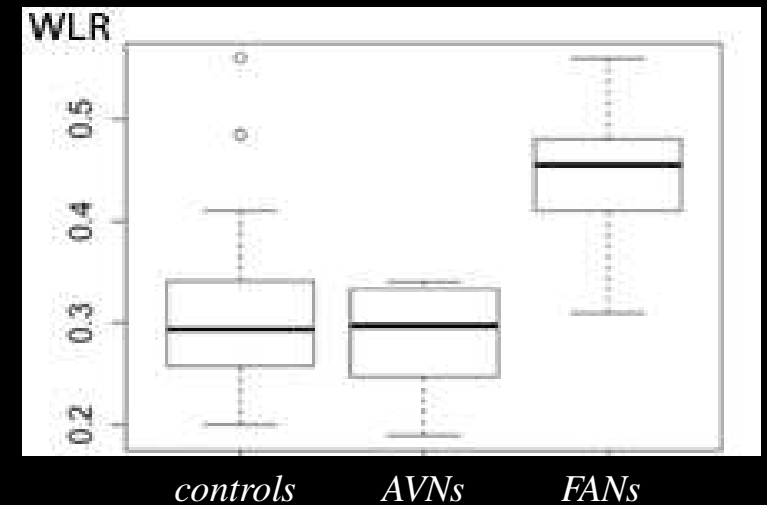


IR

Paroi artériolaire et signe du croisement

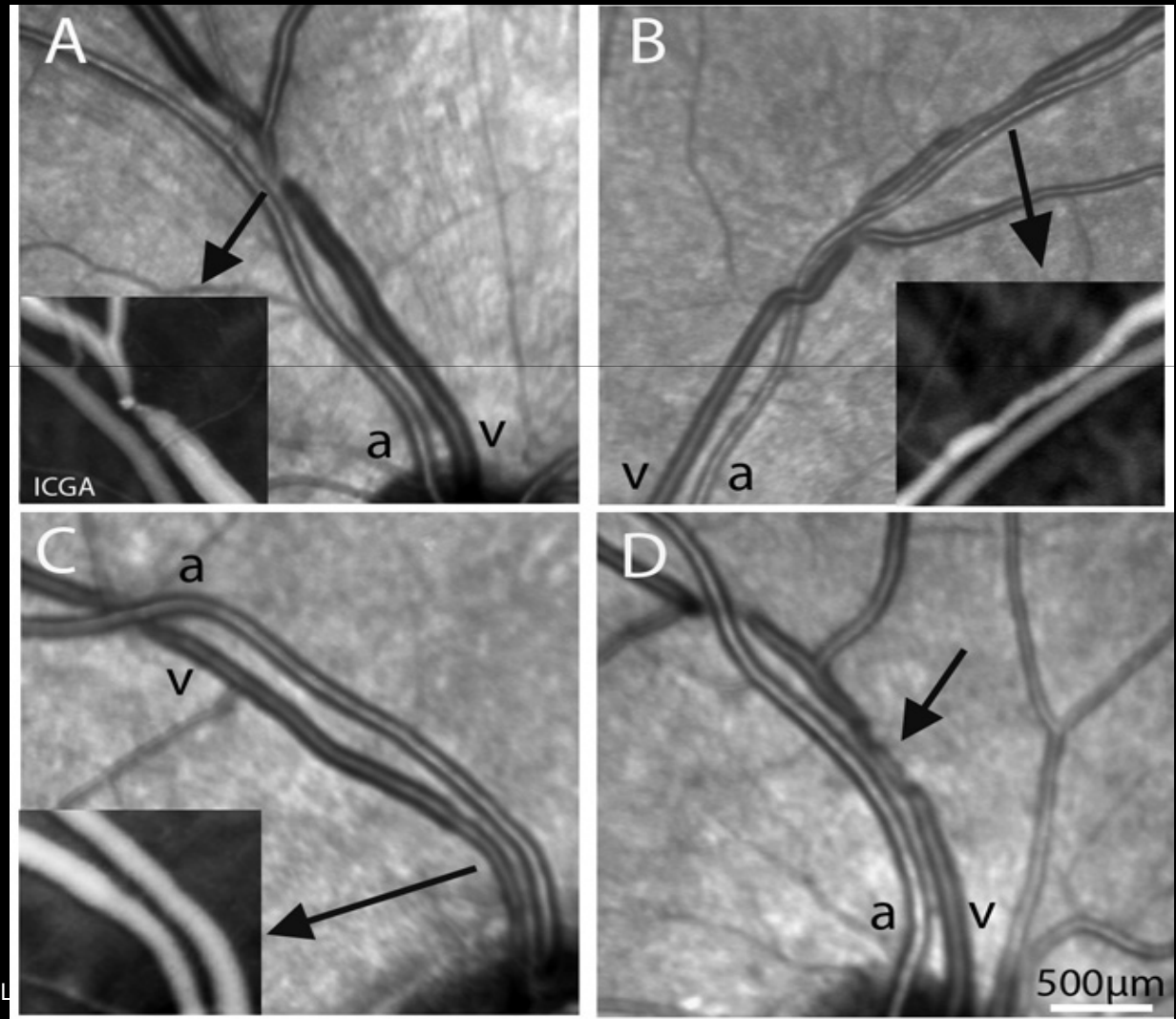


- La paroi artériolaire n'est pas épaissie

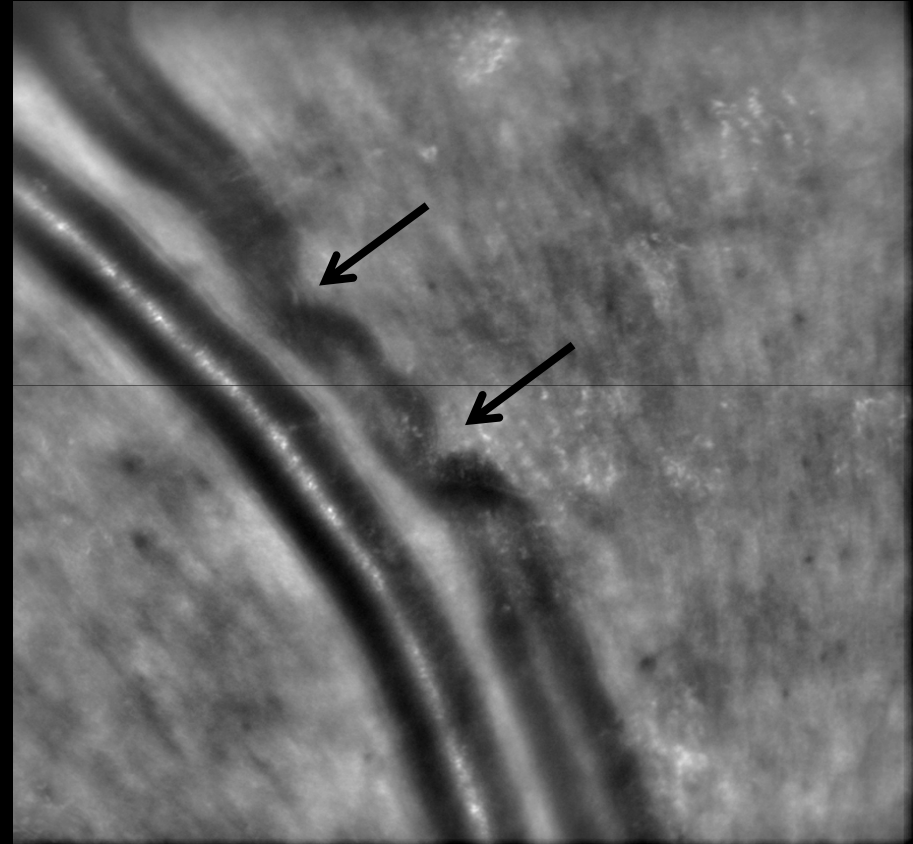
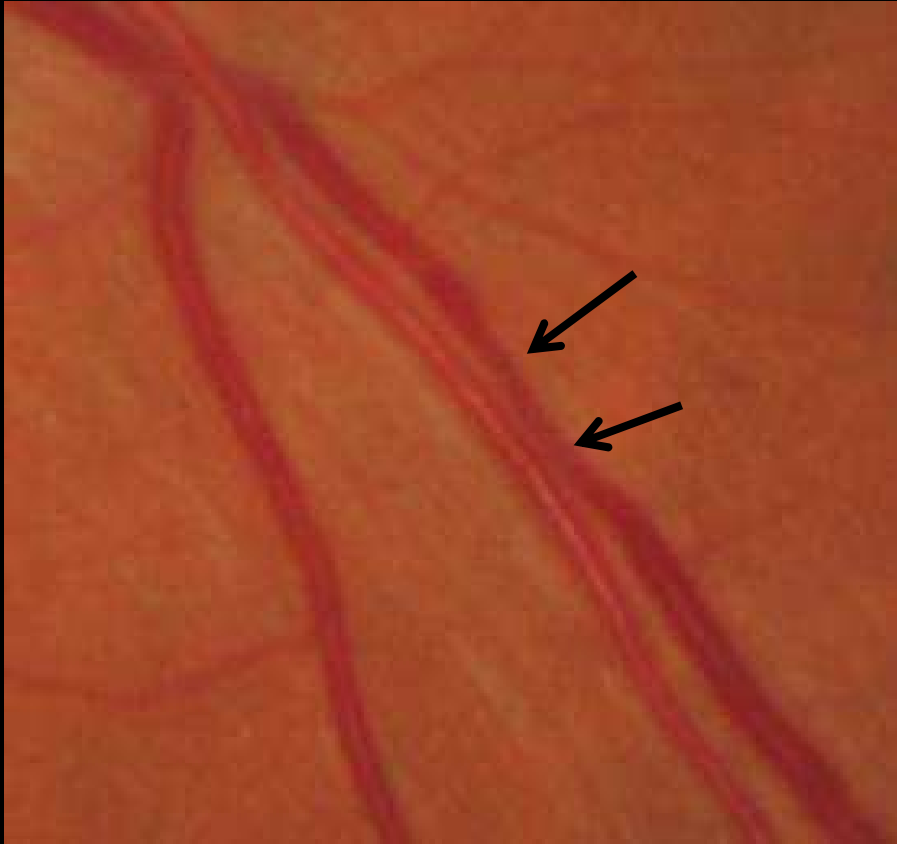


Exemples de signe du croisement... sans croisement

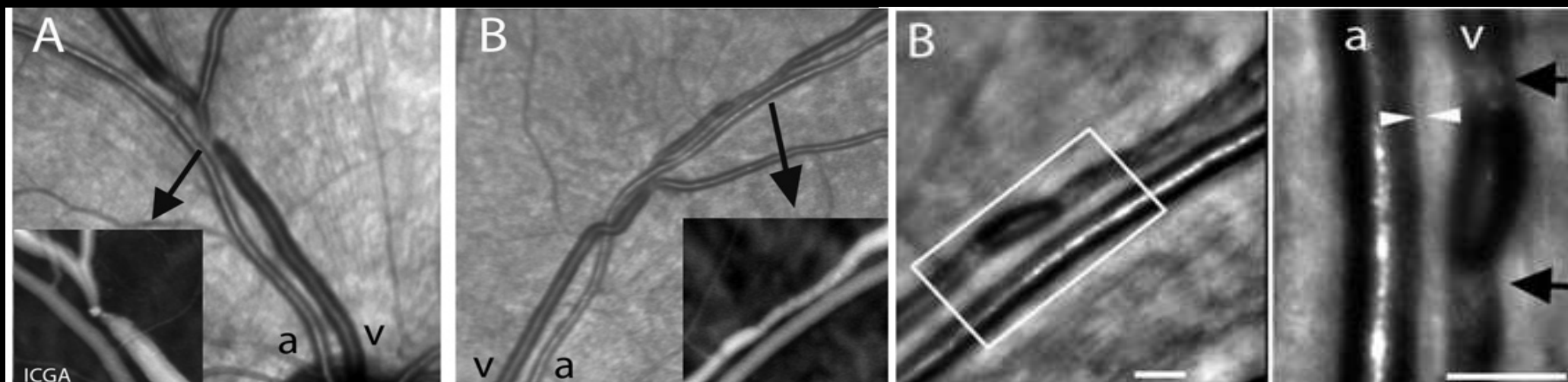
- La lumière veineuse peut être rétrécie au contact de l'artère, même sans croisement A-V



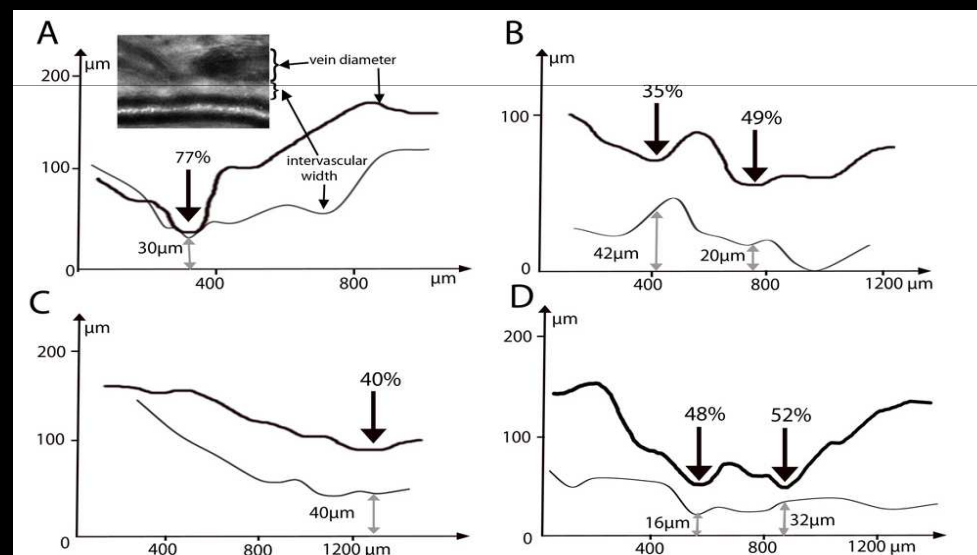
Au-delà des croisements artérioveineux: les interactions artérioveineuses



➔ *Une attraction veineuse peut s'observer à proximité d'une artère*



Le « signe du croisement » est en fait une pathologie de l'environnement artériel (et non une interaction mécanique)



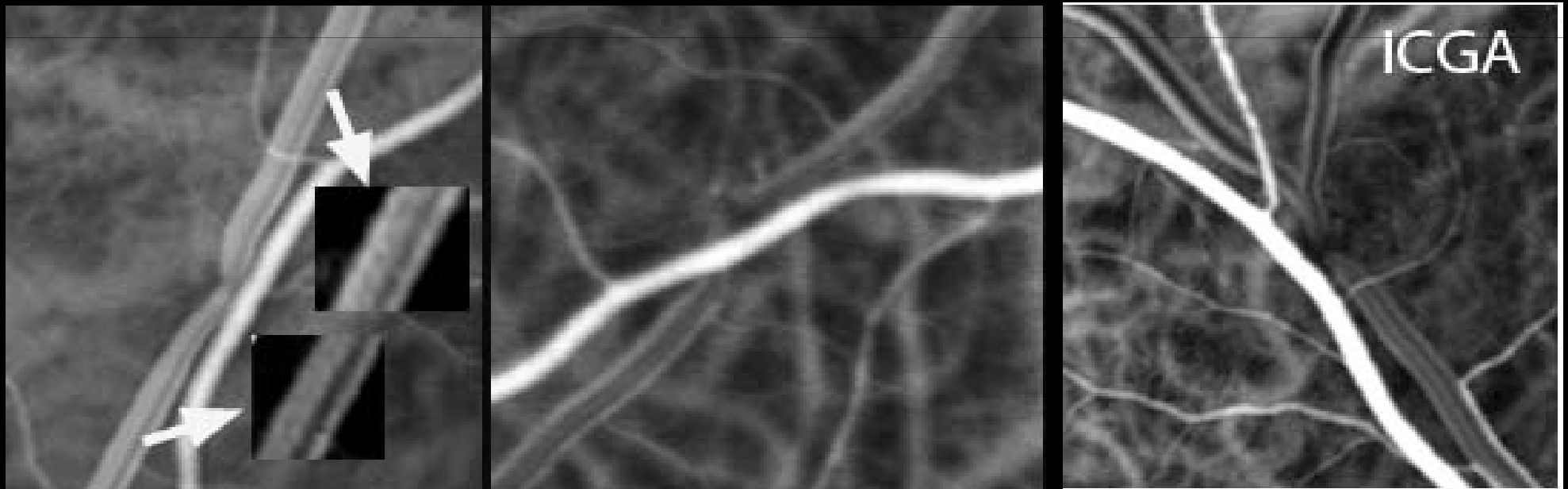
Brief Report

Venous Nicking Without Arteriovenous Contact The Role of the Arteriolar Microenvironment in Arteriovenous Nickings

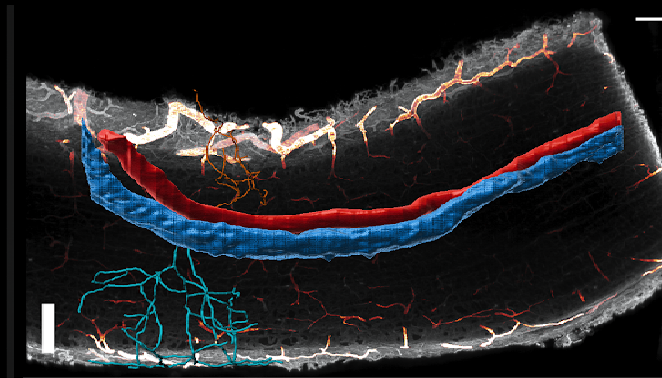
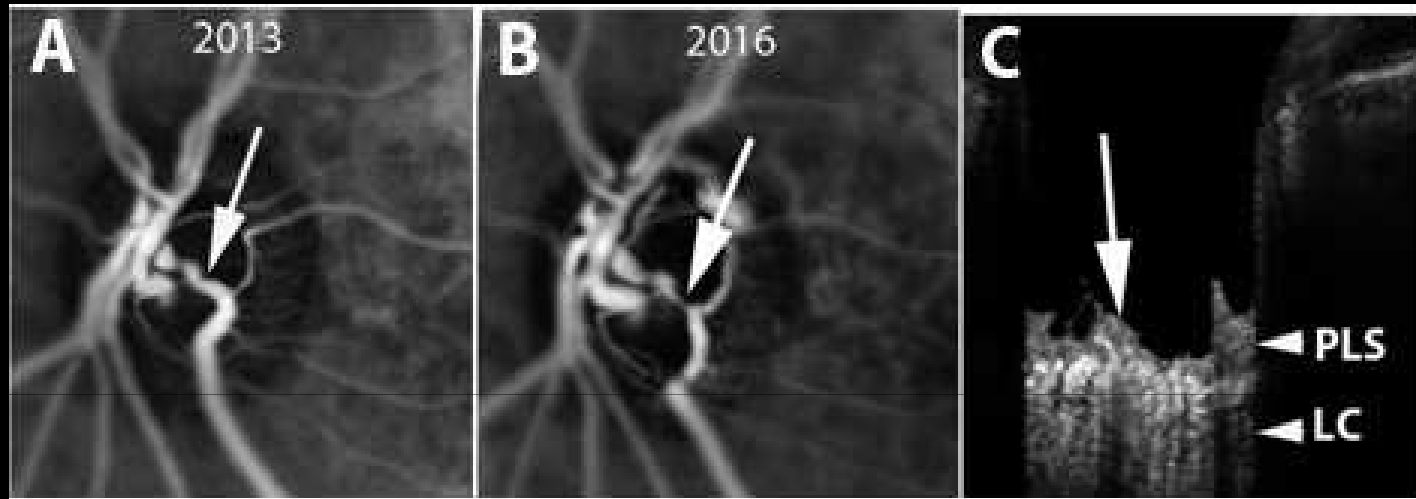
Michel Paques, MD, PhD; Aurélie Brolly, MD; Jonathan Benesty, MD, PhD; Nicolas Lermé, PhD; Edouard Koch, MD, PhD; Florence Rossant, PhD; Isabelle Bloch, PhD; Jean-François Girmens, MD

Courant laminaire et signe du croisement

- Le courant laminaire peut être préservé au niveau du croisement artérioveineux
- Pas de turbulence du flux même lors d'une sténose serrée



En dehors du cas des OBVR, le site d'une OVR reste le plus souvent incertain



Incidence OVR

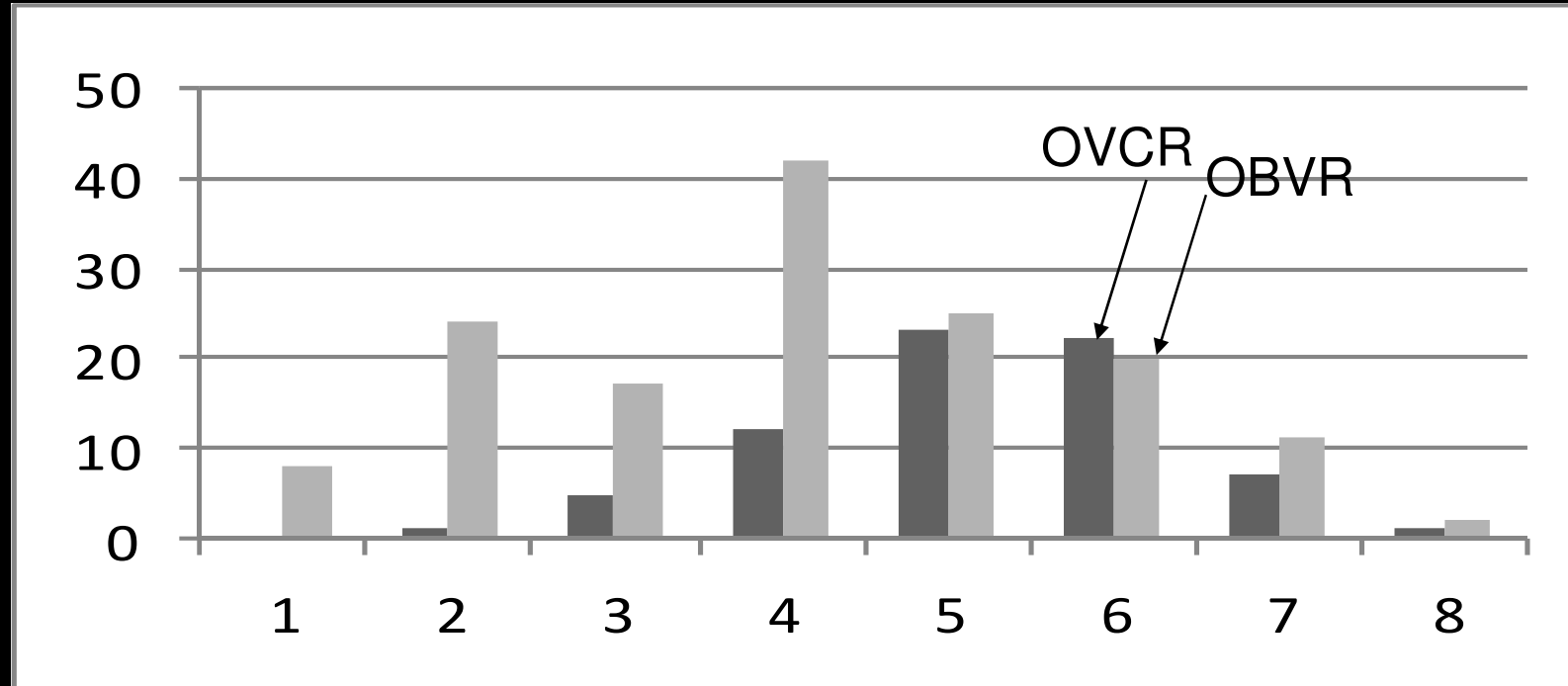
➤ Entre 1/1000 et 1/10 000 habitants /an

➤ 6000 à 60 000 par an en France (= DR)

➤ Age moyen

➤ OBVR: **65 ans**

➤ OVCR : **57 ans**

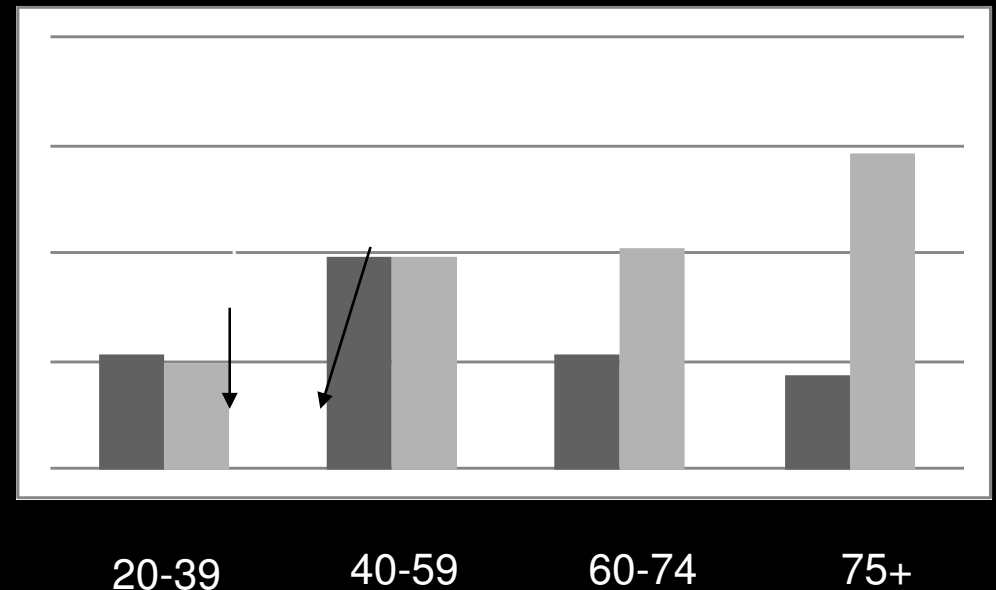


Le "bilan étiologique" des OVR

Voir le cours du Dr E Héron
Médecine interne
CHNO des 1520

Facteurs de risque des OVCR

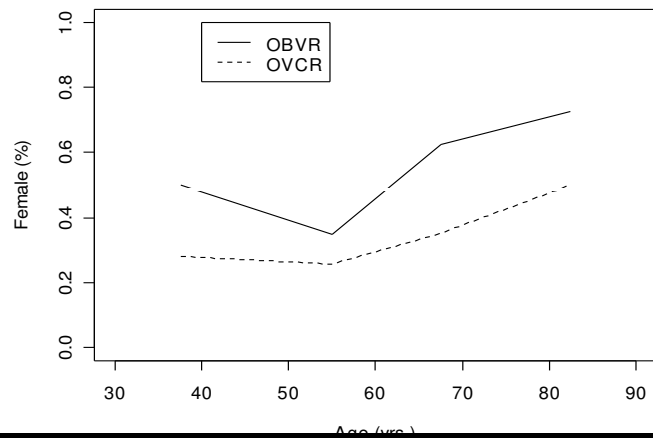
- Les plus de 75 ans représentent 7.3% de la population générale, mais 17% des OVCR
- Incidence des OVR augmente tout au long de la vie
- Age = premier facteur de risque



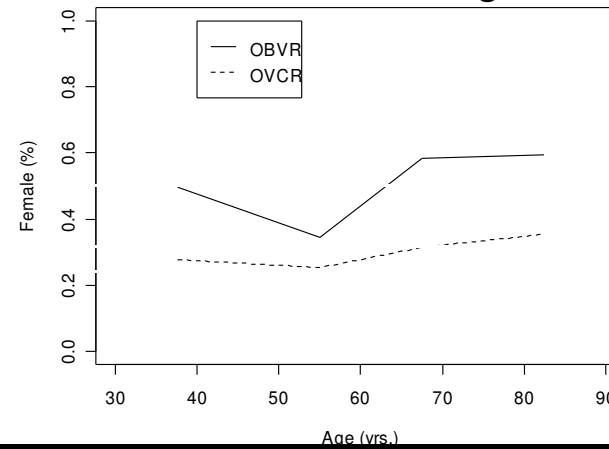
Les OVCR sont plus fréquentes chez les hommes

	n	OVCR	HémiOVR	OBVR
Mitchell		50		50
CRVO study	725	53		
Sperduto	357	53	53	53
Hayreh	1090	56	58	50
Paques	601	64	78	50
Weger				46
Koizumi	144	60		

Données brutes



Données corrigées



HTA

réf	Toutes OVR	OVCR	OBVR	OVHR
Rath 1992	3.9 (2.1-7.2)			
Mitchell 1996	2.0 (1.2-3.3)			
Sperduto 1998		2.1 (1.5-2.9)	3.3 (2.4-4.6)	2.9 (1.6-5.3)
Wong 2005	2.96 (1.4-6.1)			
Weger 2005			2.32 (1.6-3.)	

Glaucome

réf	Toutes OVR	OVCR	OBVR	OVHR
Rath 1992	2.9 (1.4-6)			
Mitchell 96	4.3 (2.1-9.1)			
Sperduto 98		5.4 (3.5-8.5)	2.5 (1.5-4.2)	4.6 (2.3-9.5)

Y a t-il des anomalies de la coagulation?

Dr E Héron
Médecine interne
CHNO des 1520

	Témoins (n = 1424)	Membres inf (n =971)	Membres sup (n = 176)	Cérébrale (n = 79)	Rétine (n =177)
FV Leiden	4.7	13.8	16	8.8	4.5
F II leiden	3.9	9.6	7.2	14.2	6
Déficit PC	2.5	1.8	0	0	0
Déficit PS	1	2	1	2.5	0
Déficit AT	0	1	0	0	0
Estrogènes	–	15.7	14.1	47.0	11.8
Sd myéloprolifératif	–	1.7	0	0	0

*Margaglione M, et coll. Haematologica 2001;86:634-9.
Bombeli T et coll. Am J Hematol, 2002;70:126-32.*

Les patients ayant une OVCR ne sont PAS à risque de faire un accident thromboembolique ultérieur

Liu et al. Retina 2018

OVR: un rôle de l'apnée du sommeil?

Glacet-Bernard A Obstructive sleep apnea among patients with retinal vein occlusion.

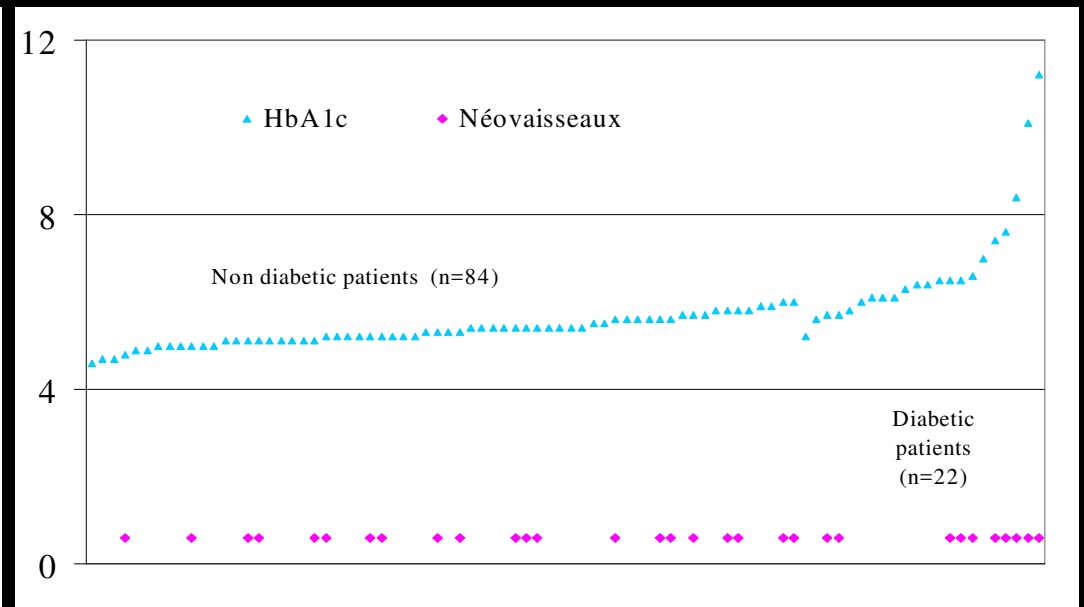
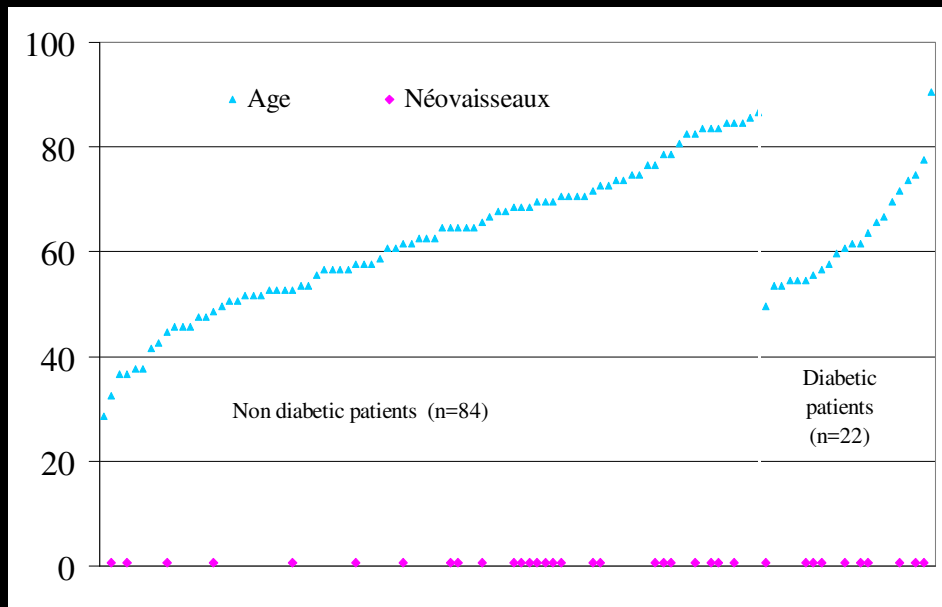
Arch Ophthalmol. 2010 Dec;128(12):1533-8.

Of the 30 selected patients, 23 (77%) had OSA. (...); OSA was mild (AHI <15) in 13 patients, moderate in 5 patients (AHI 15-30), and severe (AHI >30) in 5 patients. The AHI was correlated with body mass index (P = .02).

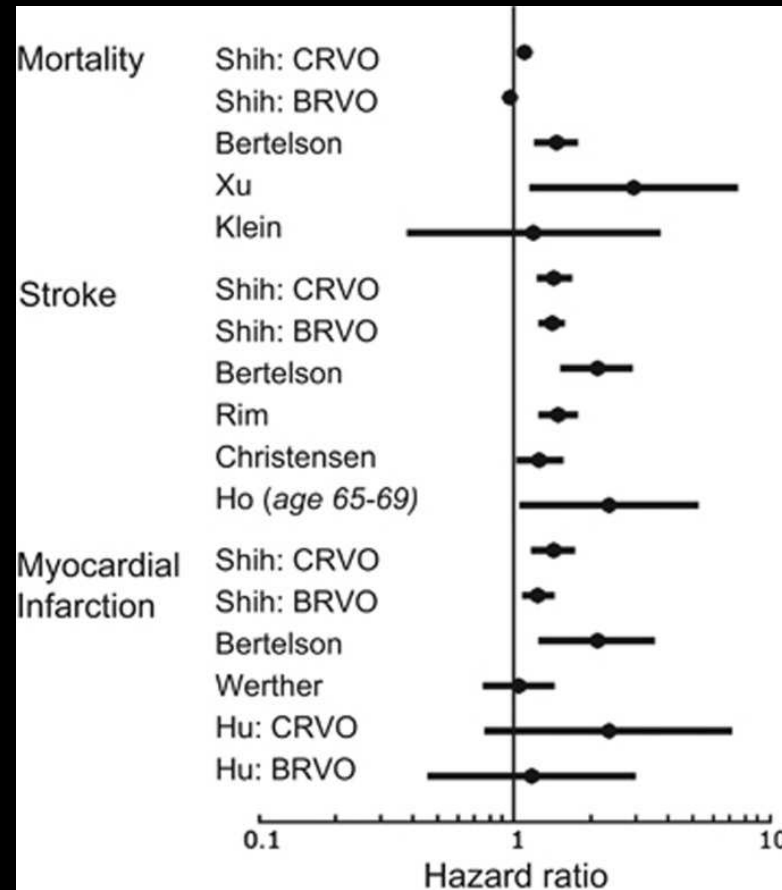
Chou KT¹, Sleep apnea and risk of retinal vein occlusion: a nationwide population-based study of Taiwanese. Am J Ophthalmol. 2012 Jul;154(1):200-205

Of the 35 634 sampled patients (5965 sleep apnea patients vs 29 669 controls), 52 (0.15%) experienced RVO during a mean follow-up period of 3.72 years, including 13 (0.22%, all branch RVO) from the sleep apnea cohort and 39 (0.13%, 39 branch RVO and 10 central RVO) from the control group. Kaplan-Meier analysis revealed the tendency of sleep apnea patients toward RVO development (P = .048, log-rank test). Patients with sleep apnea experienced a 1.94-fold increase (95% confidence interval, 1.03 to 3.65; P = .041) in incident RVO, which was independent of age, gender, and comorbidities

Le risque de rubéose irienne des OVCR est lié d'abord à l'HbA1c alors qu'il est lié à l'âge chez les sujets non diabétiques



L'OVR est-elle révélatrice d'un risque plus élevé de pathologie vasculaire?



Woo et al. Eye 2016

Facteurs de risque des OVR: ne pas oublier que l'OVR est dans la grande majorité des cas unilatérale...

- Facteurs favorisants:
 - L'âge
 - Sexe masculin?
 - HTA
 - HTO
- Facteurs d'aggravation
 - Age
 - Diabète déséquilibré
 - ATCD cardiovasculaires
- Facteurs sans incidence:
 - Tabagisme
 - Cholestérol
 - Hématocrite
- Facteurs incertains
 - Apnées du sommeil
 - Antécédants familiaux?
 - Mélanodermes? *Stem 2013*

•*Eye Disease Case Control Study ;258 patients et 1142 témoins dans 5 centres*

Table 1**Characteristics of the study subjects.**

Variable	RVO group n = 37921 n (%)	Comparison group n = 113763 n (%)	p-value
Variables to generate PS			
Age, year (mean±SD)	62.4±13.1	62.4±13.2	0.90
Age, categorical			>0.99
<50	6098 (16.1)	18302 (16.1)	
50–60	9038 (23.8)	27118 (23.8)	
60–70	10323 (27.2)	31003 (27.3)	
≥70	12462 (32.9)	37340 (32.8)	
Gender			>0.99
Male	19416 (51.2)	58249 (51.2)	
Female	18505 (48.8)	55514 (48.8)	
Antithrombotic drugs	9215 (24.3)	27752 (24.4)	0.71
Obesity	763 (2.0)	2396 (2.0)	0.78
Stroke	3992 (10.5)	11939 (10.5)	0.86
Hyperviscosity syndrome	429 (1.1)	1298 (1.1)	0.88
Glaucoma	3379 (8.9)	10077 (8.9)	0.75
PS (mean±SD)	0.124±0.102	0.124±0.102	>0.99
Variables for adjustment			
Diabetes	15020 (39.6)	35264 (31.0)	<0.0001
Hypertension	30194 (79.6)	69547 (61.1)	<0.0001
Hyperlipidemia	18852 (49.7)	45142 (39.7)	<0.0001
Congestive heart failure			<0.0001
Yes, with CHD	1499 (3.9)	3555 (3.1)	
Yes, without CHD	3743 (9.9)	9900 (8.7)	
No	32679 (86.2)	100308 (88.2)	
Chronic renal failure	4945 (13.0)	8208 (7.2)	<0.0001
Follow-up period, year (mean±SD)	5.52±3.56	5.55±3.66	0.16
Incident AMI	1240 (3.27)	2616 (2.30)	<0.0001

!!

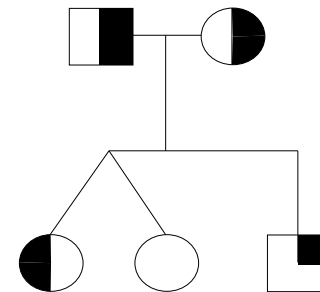
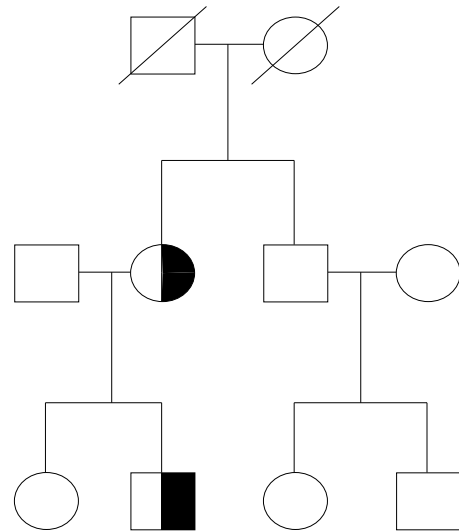
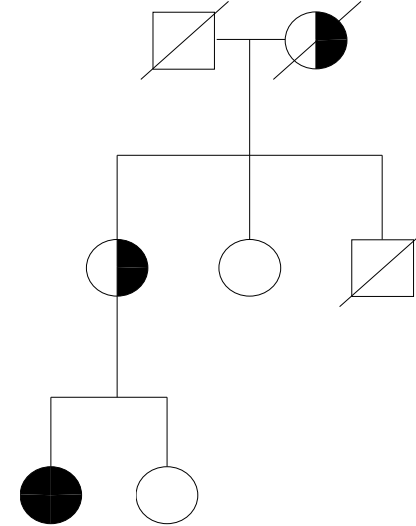
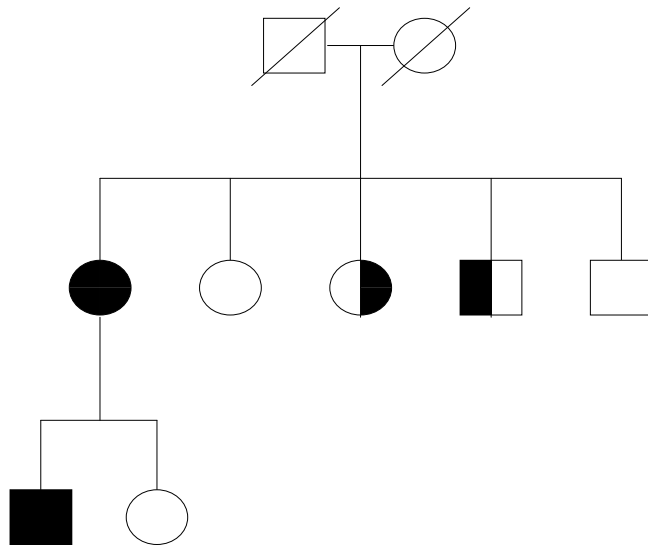
Characteristics of the CRVO and BRVO subjects.

Variable	CRVO group n = 11855 n (%)	BRVO group n = 26066 n (%)	p-value
Variables to generate PS			
Age, year (mean±SD)	61.3±14.5	62.8±12.4	<0.0001
Age, categorical			<0.0001
<50	2396 (20.2)	3702 (14.2)	
50–60	2656 (22.4)	6382 (24.5)	
60–70	2911 (24.6)	7412 (28.4)	
≥70	3892 (32.8)	8570 (32.9)	
Gender			<0.0001
Male	6367 (53.7)	13049 (50.1)	
Female	5488 (46.3)	13017 (49.9)	
Antithrombotic drugs	2782 (23.5)	6385 (24.5)	0.03
Obesity	218 (1.8)	443 (1.7)	0.35
Stroke	1223 (10.3)	2769 (10.6)	0.38
Hyperviscosity syndrome	176 (1.5)	253 (1.0)	<0.0001
Glaucoma	1600 (13.5)	1779 (6.8)	<0.0001
PS (mean±SD)	0.121±0.10	0.131±0.12	0.015
Variables for adjustment			
Diabetes	4964 (41.9)	10056 (38.6)	<0.0001
Hypertension	8934 (75.4)	21260 (81.6)	<0.0001
Hyperlipidemia	5713 (48.2)	13139 (50.4)	<0.0001
Congestive heart failure			0.67
Yes, with CHD	483 (4.1)	1016 (3.9)	
Yes, without CHD	1179 (10.0)	2564 (9.8)	
No	10193 (86.0)	22486 (86.3)	
Chronic renal failure	1854 (15.6)	3091 (11.9)	<0.0001
Follow-up period, year (mean±SD)	5.51±3.50	5.53±3.58	0.59
Incident AMI	427 (3.60)	813 (3.12)	<0.0001

Ne demandez plus de
« grand bilan »!

Sauf cas exceptionnel...
ou recherche

Formes familiales d' OVR



Athérosclérose vs artériolosclérose

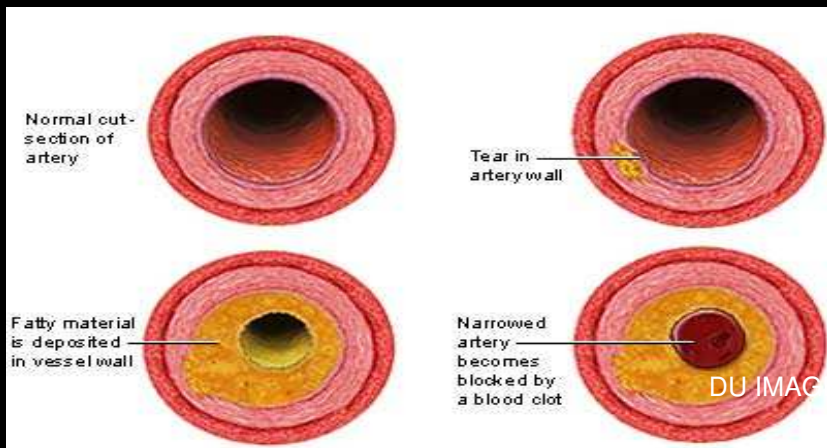
- Athérome

- Dépôts de cholestérol intracellulaire sous l'intima des grosses artères
- Thrombose artérielle
- FDR: HCT, HTA, Hérité, diabète

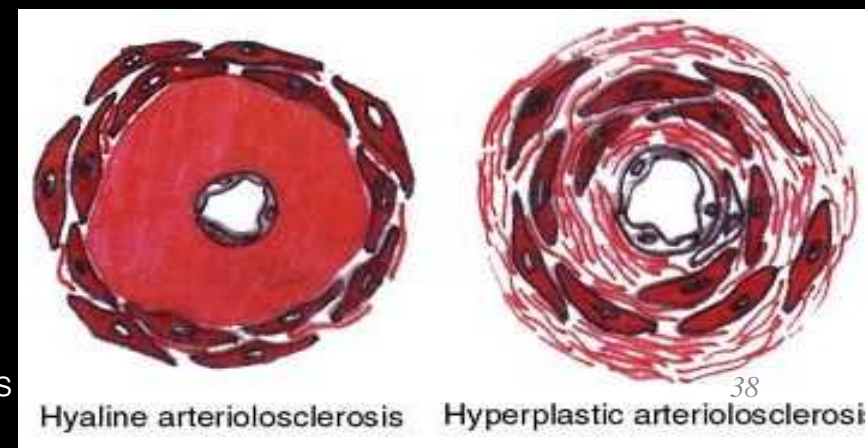


Artériolosclérose

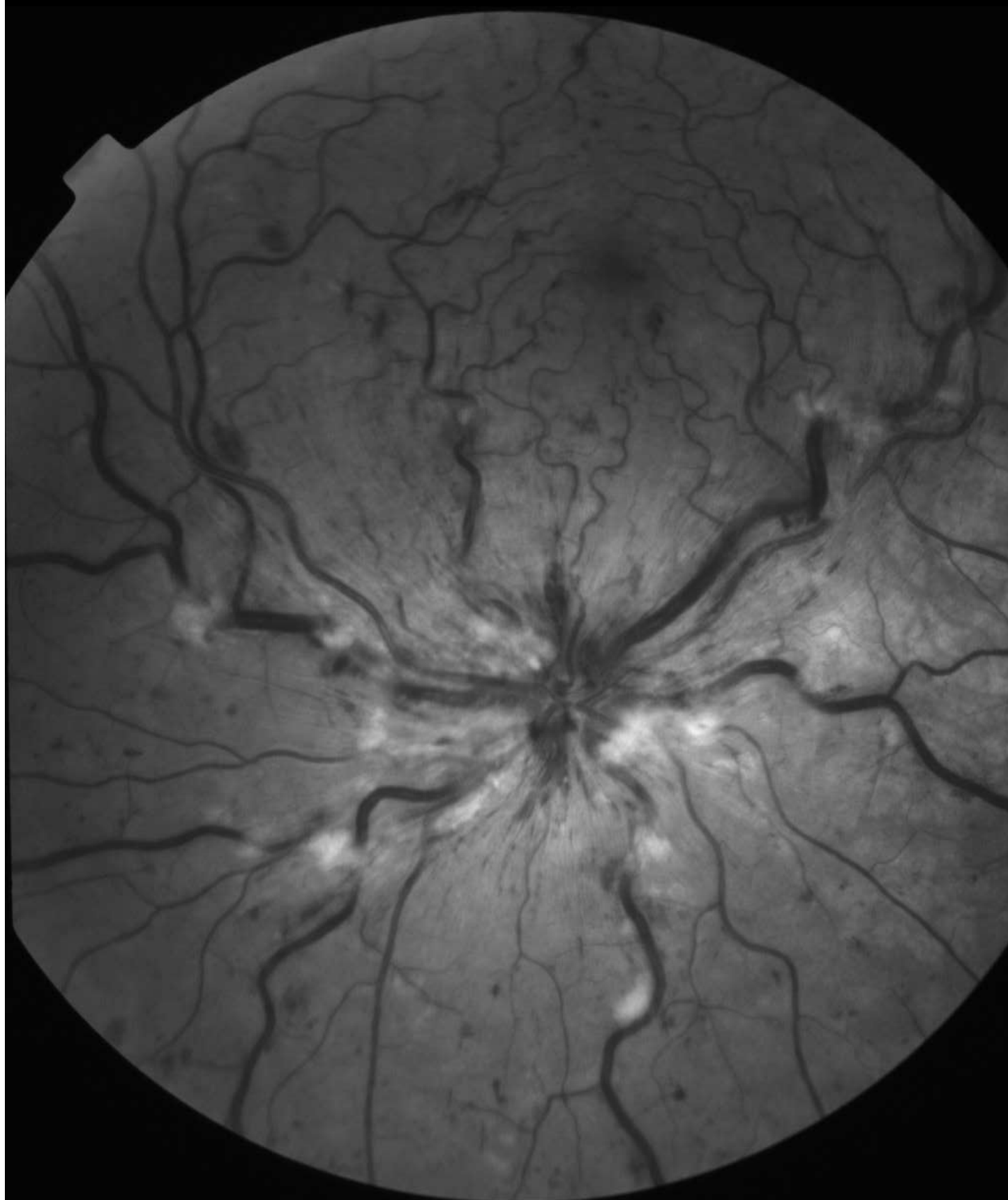
Hyperplasie et fibrose de l'adventice des artérioles
FDR: HTA, âge



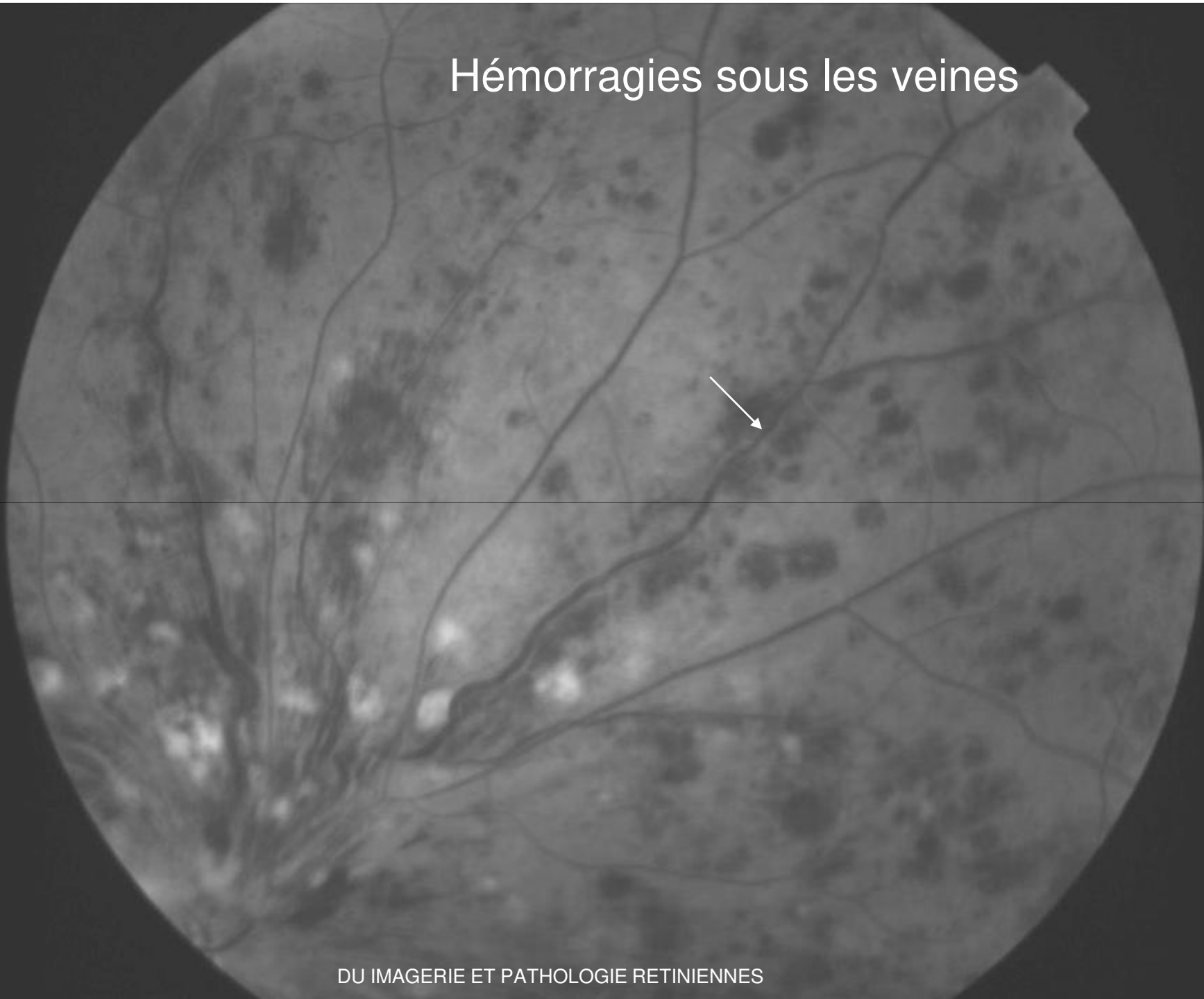
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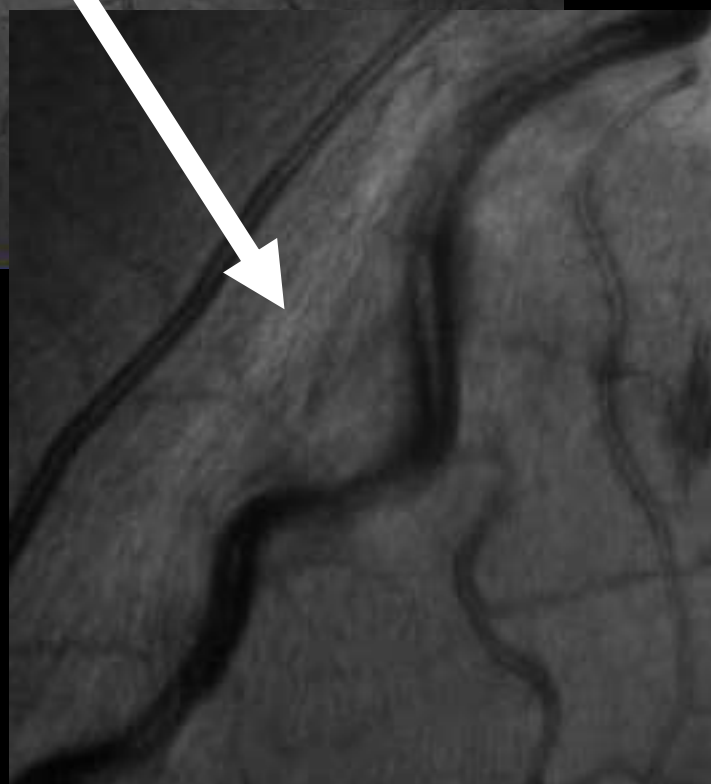


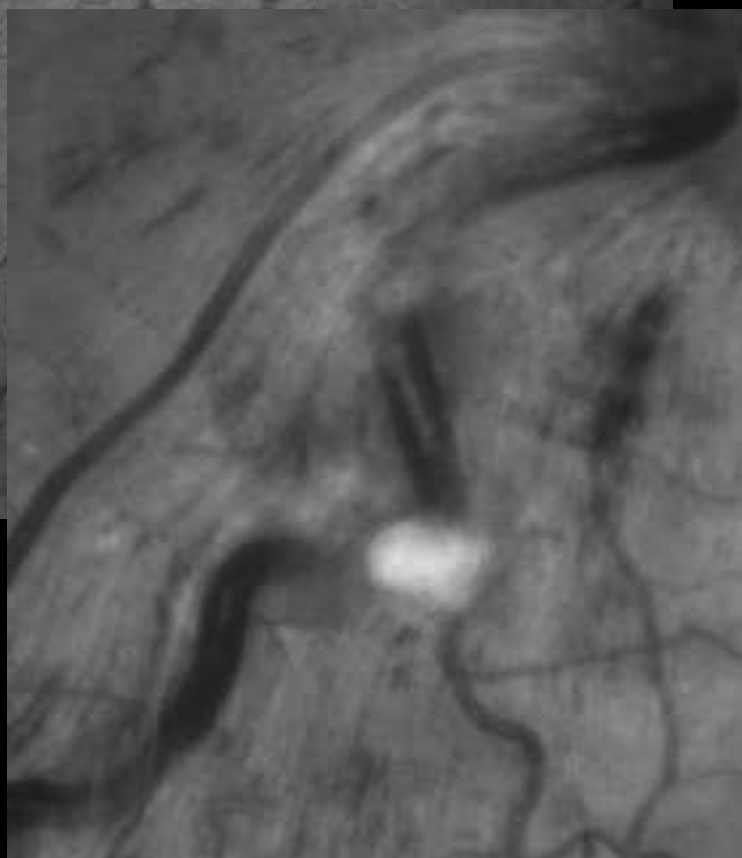
OVCN: Aspects cliniques



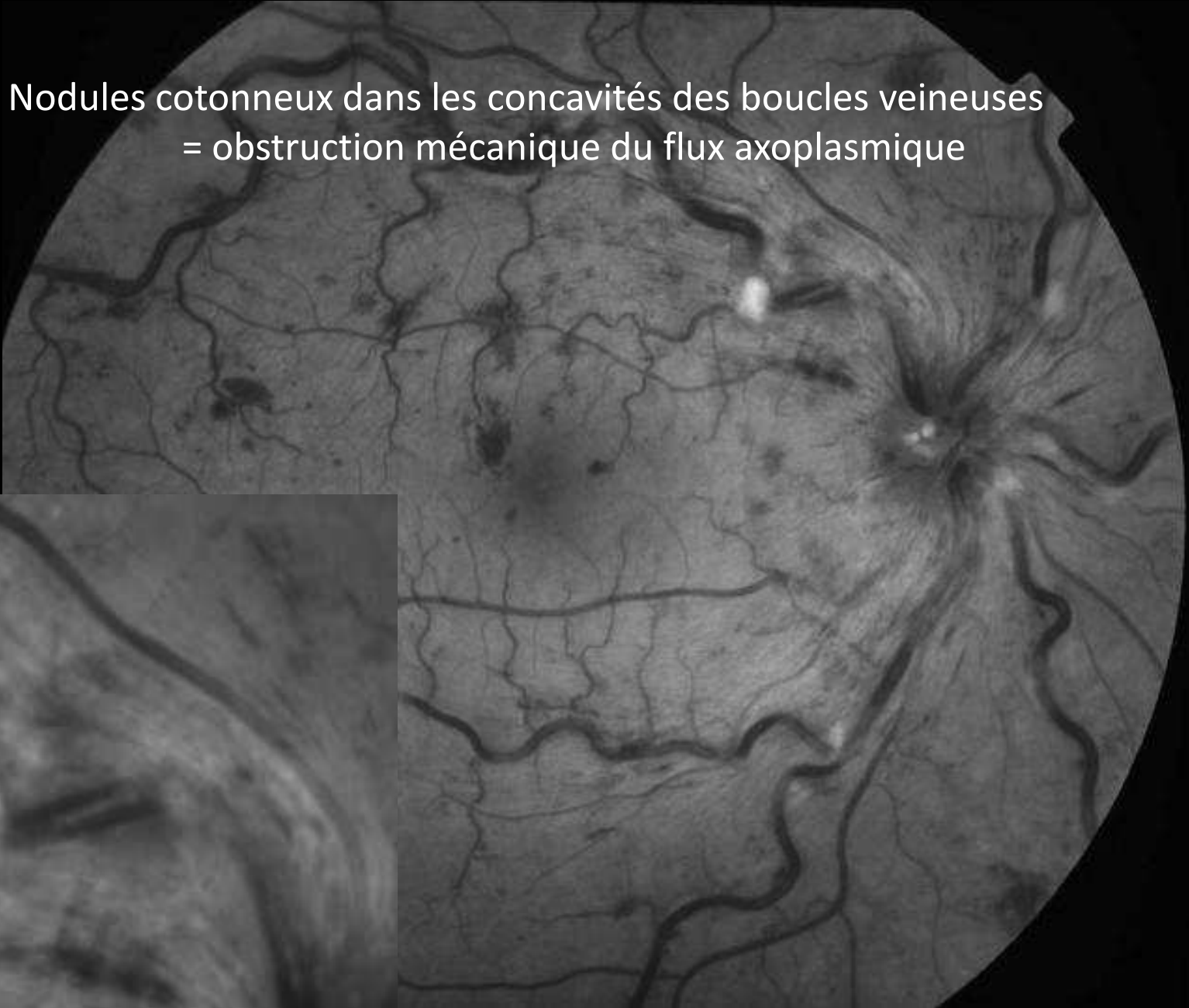
Hémorragies sous les veines



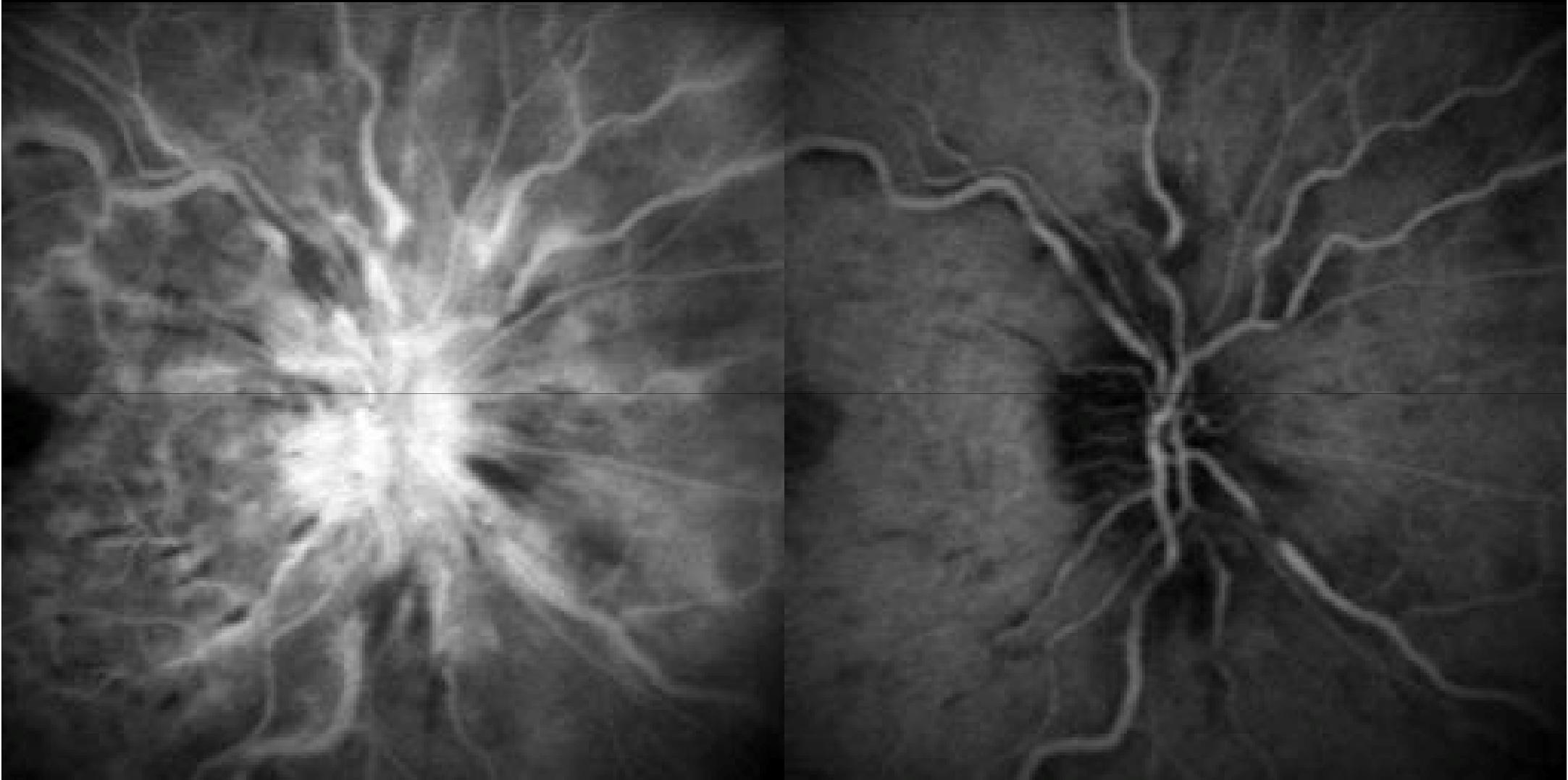




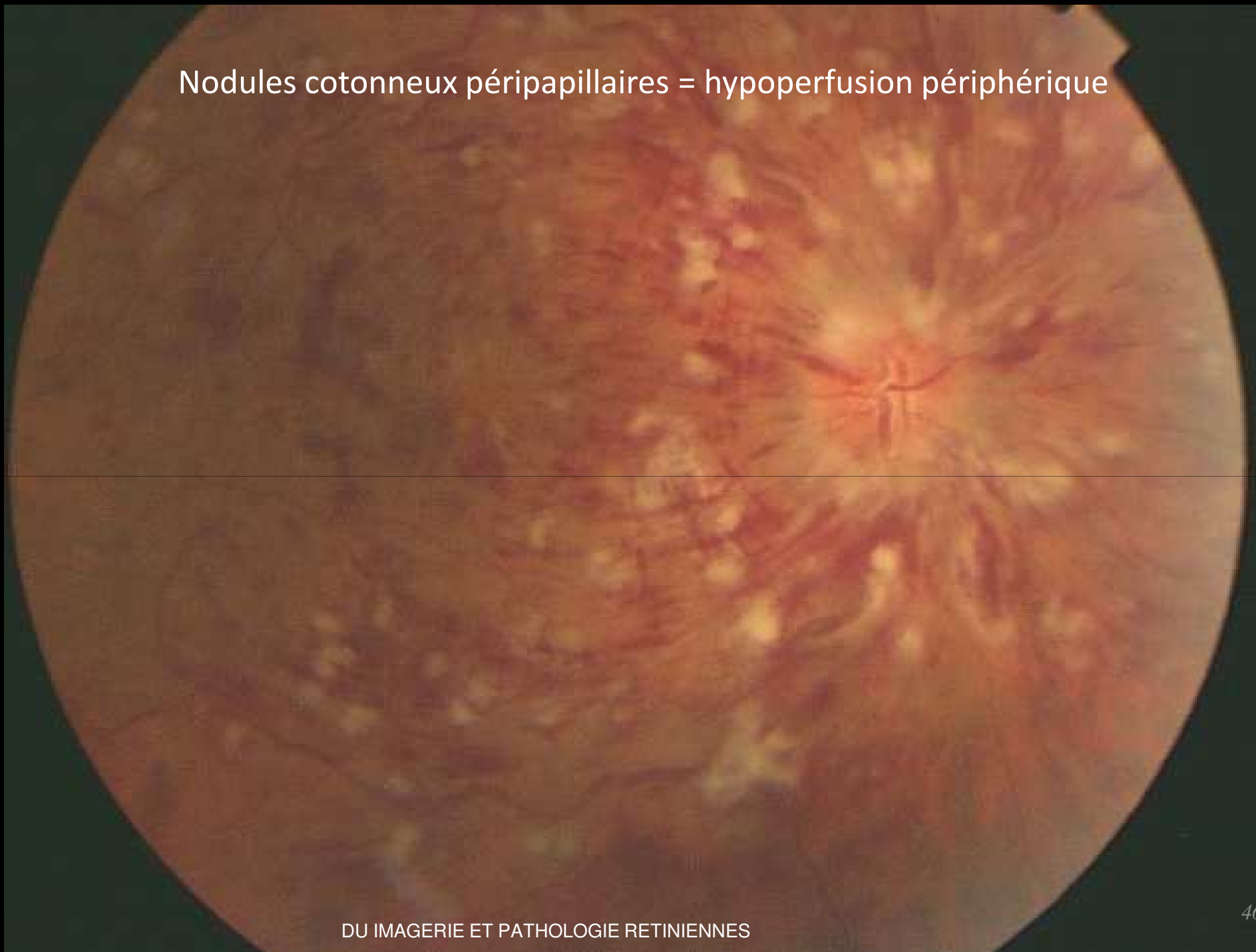
Nodules cotonneux dans les concavités des boucles veineuses
= obstruction mécanique du flux axoplasmique



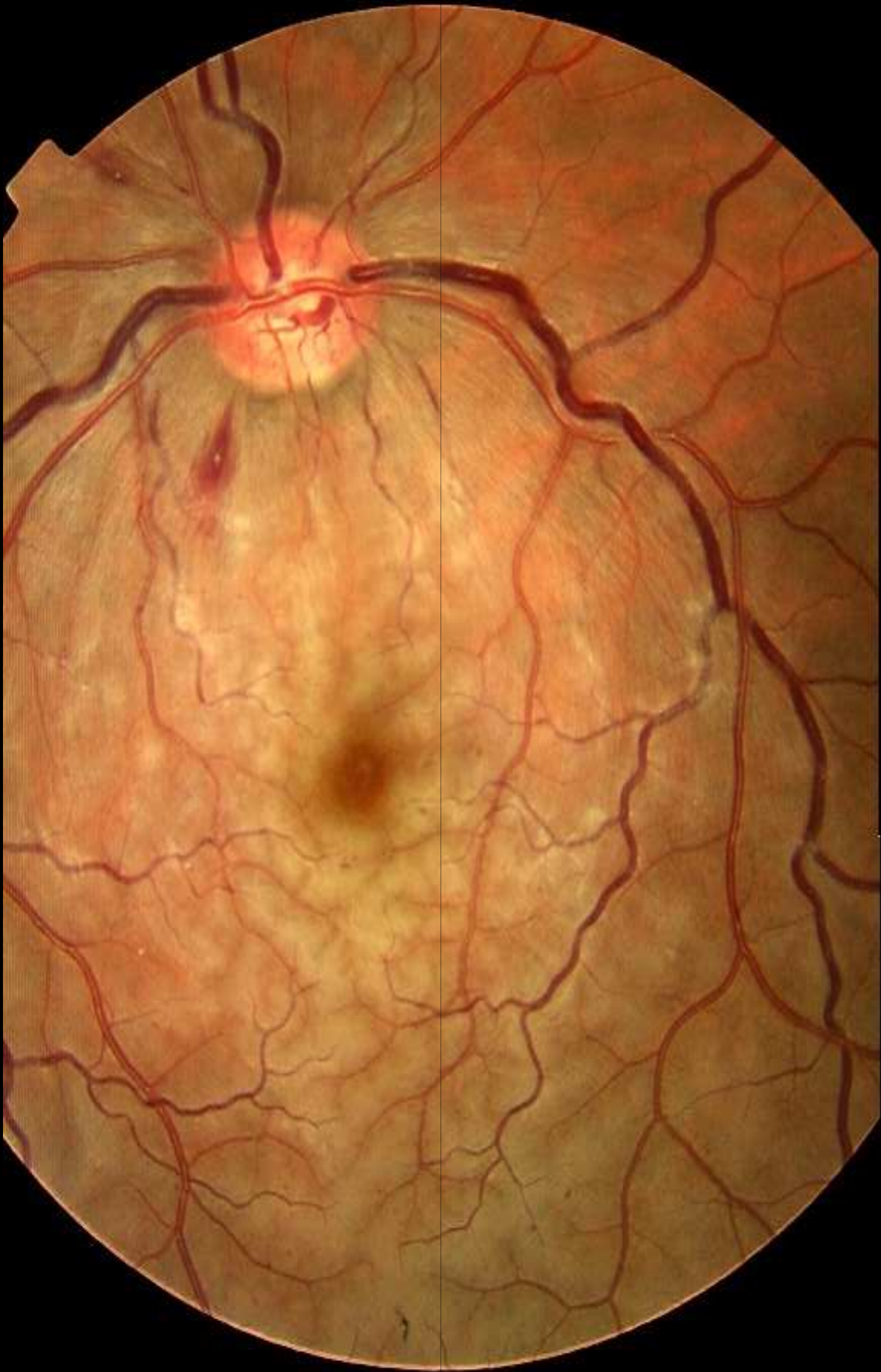
Œdème papillaire



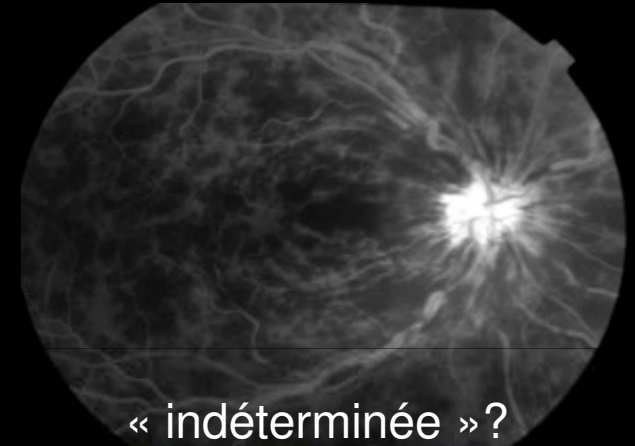
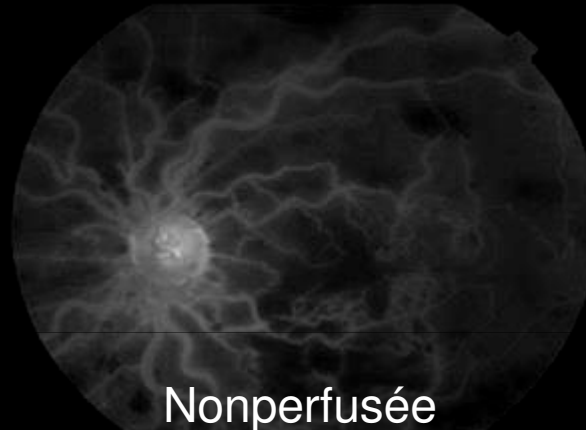
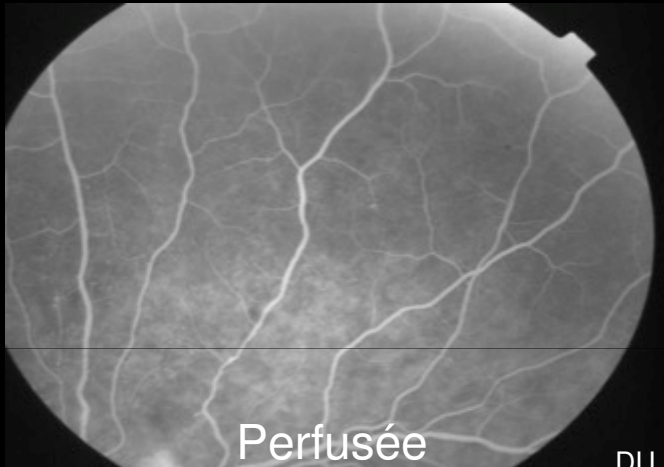
Nodules cotonneux péripapillaires = hypoperfusion périphérique







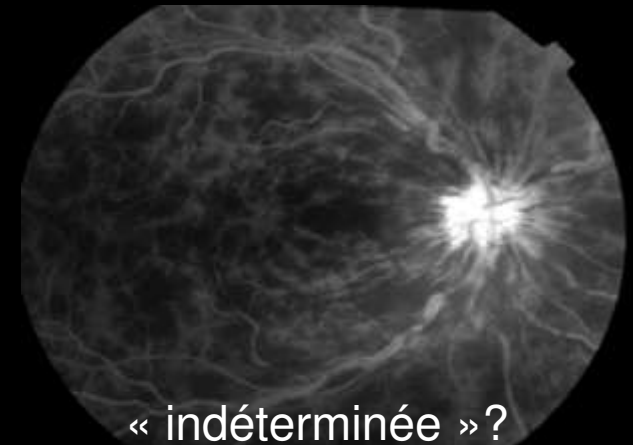
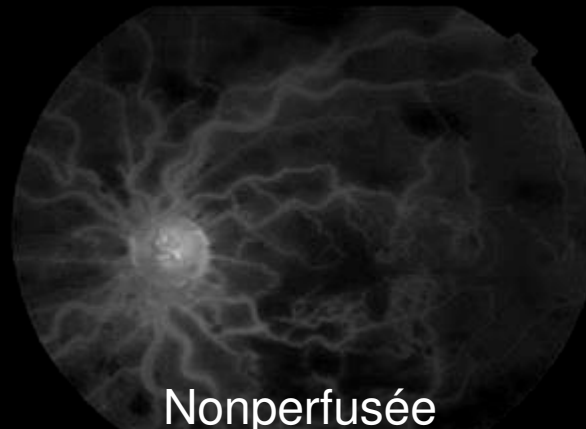
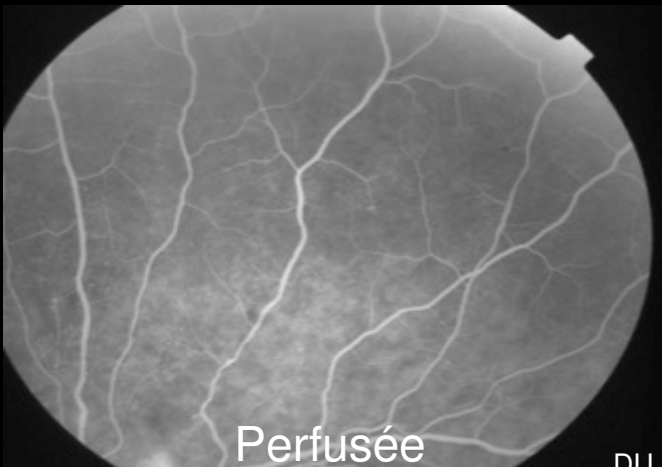
Classification des OVR



DU IMAGERIE ET PATHOLOGIE RETINIENNES

Limites des classifications actuelles

- Essentiellement basée sur des critères angiographiques
 - « Perfusée / non-perfusée »
 - « Ischemique / non-ischémique »
- Définition peut varier (champ visuel, ERG, pupille...)
- Beaucoup de cas « inclassables » (/ex hémorragies nombreuses)
- Peu de valeur pronostique dans les cas bien perfusés
- L'acuité visuelle est un meilleur élément pronostic



Que se passe t-il au début d'une OVR?

June 2003

March 2004



DU IMAGERIE ET PATHOLOGIE RETINIENNES

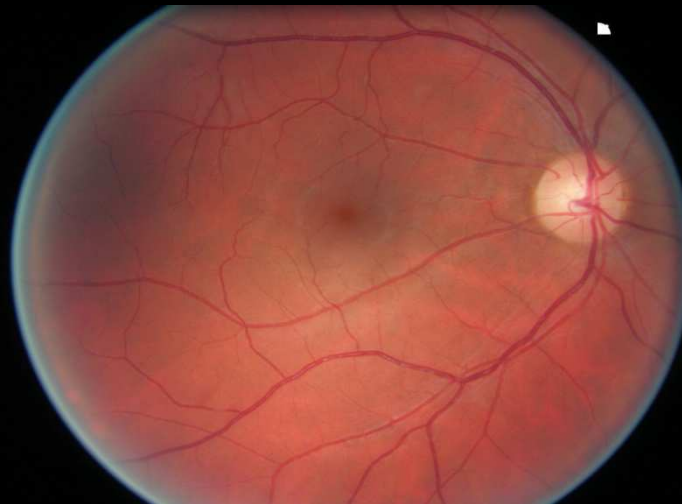
June 2004



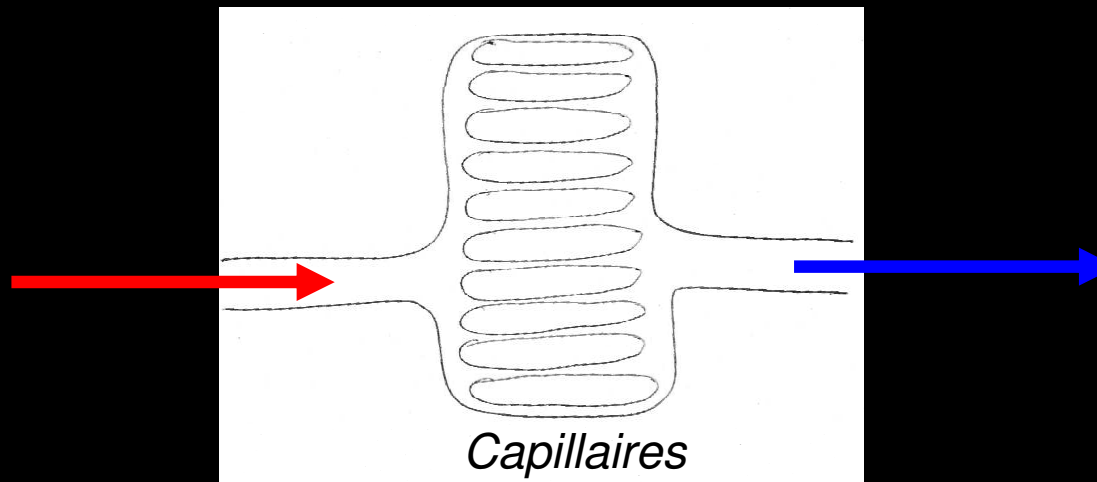
- Notre expérience nous amène à postuler que l' évolution clinique des OVR dépend du mode de début
 - Mode aigu: type A
 - Mode chronique: type B

- Cliniquement, ces deux types ne sont pas forcément distingués par les symptômes visuels, mais surtout par:
 - le fond d' œil
 - l' OCT

- Indépendamment de l' acuité visuelle, de la localisation, de l' angiographie
 - OBVR est toujours du type B
 - Les hémioclusions peuvent être de type A

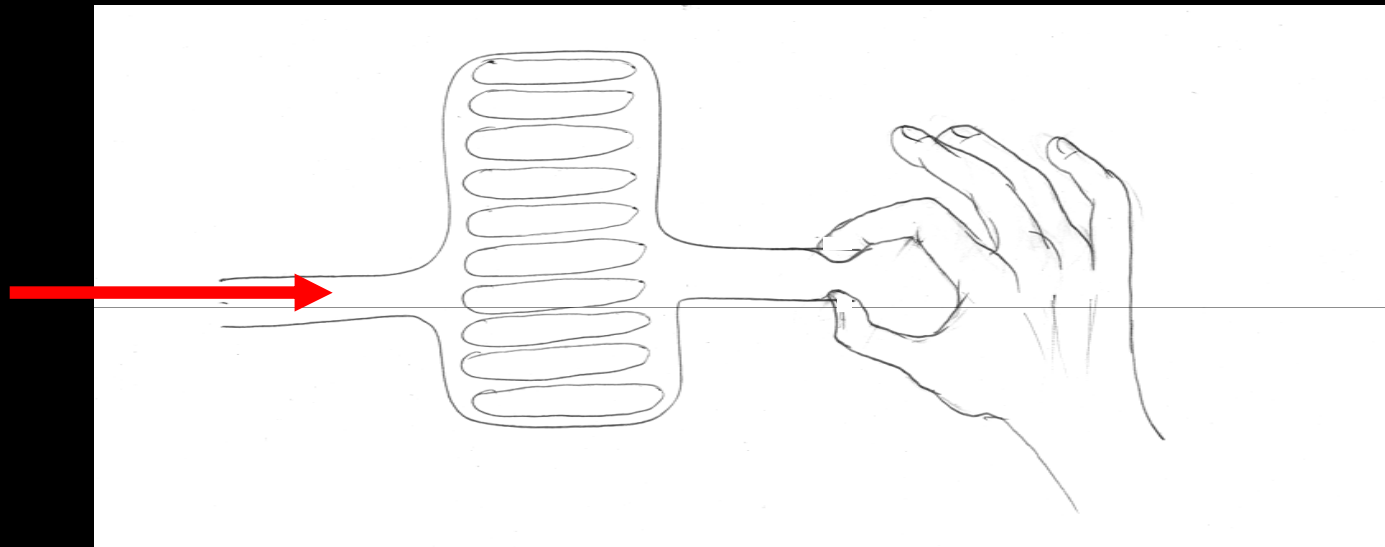


*Artère
centrale de
la rétine*

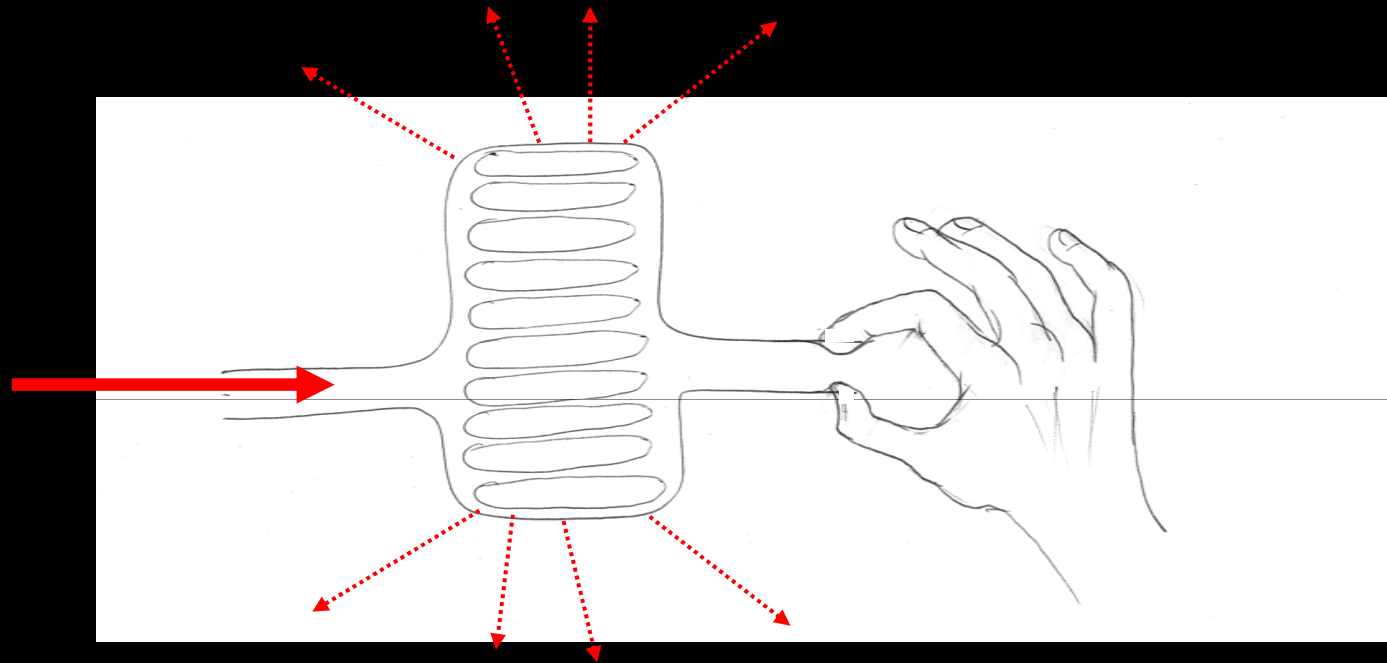


*Veine centrale
de la rétine*

Survenue d'une OVR



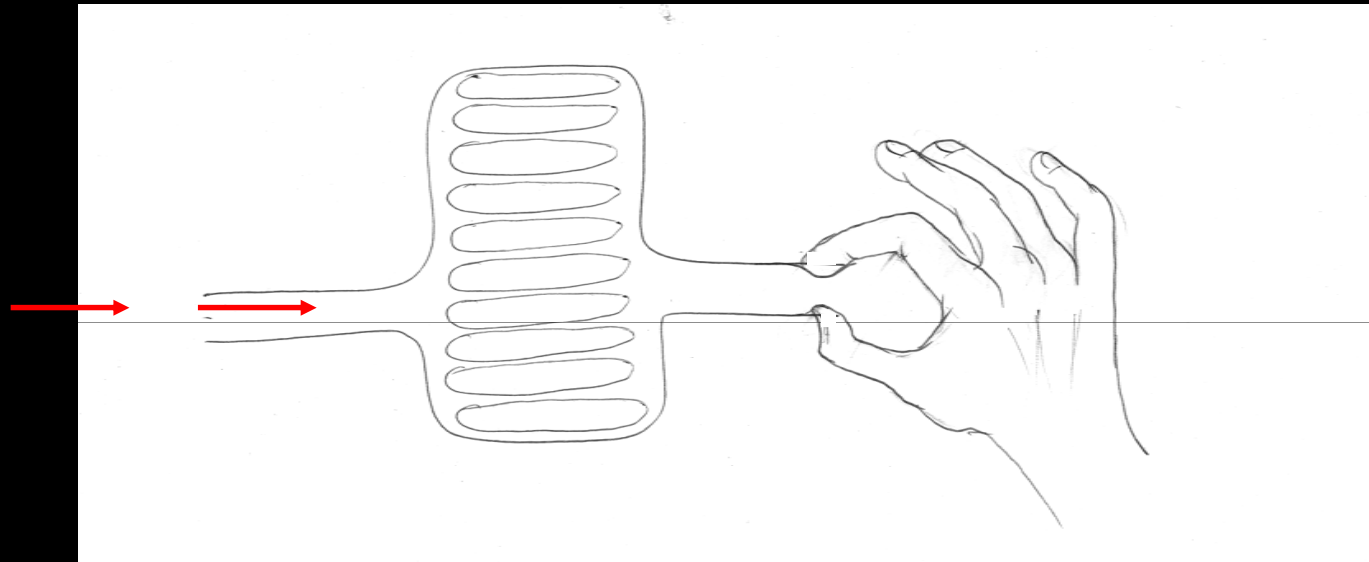
Si les capillaires cèdent



Rupture de la barrière hématorétinienne
→ hémorragies et œdème maculaire



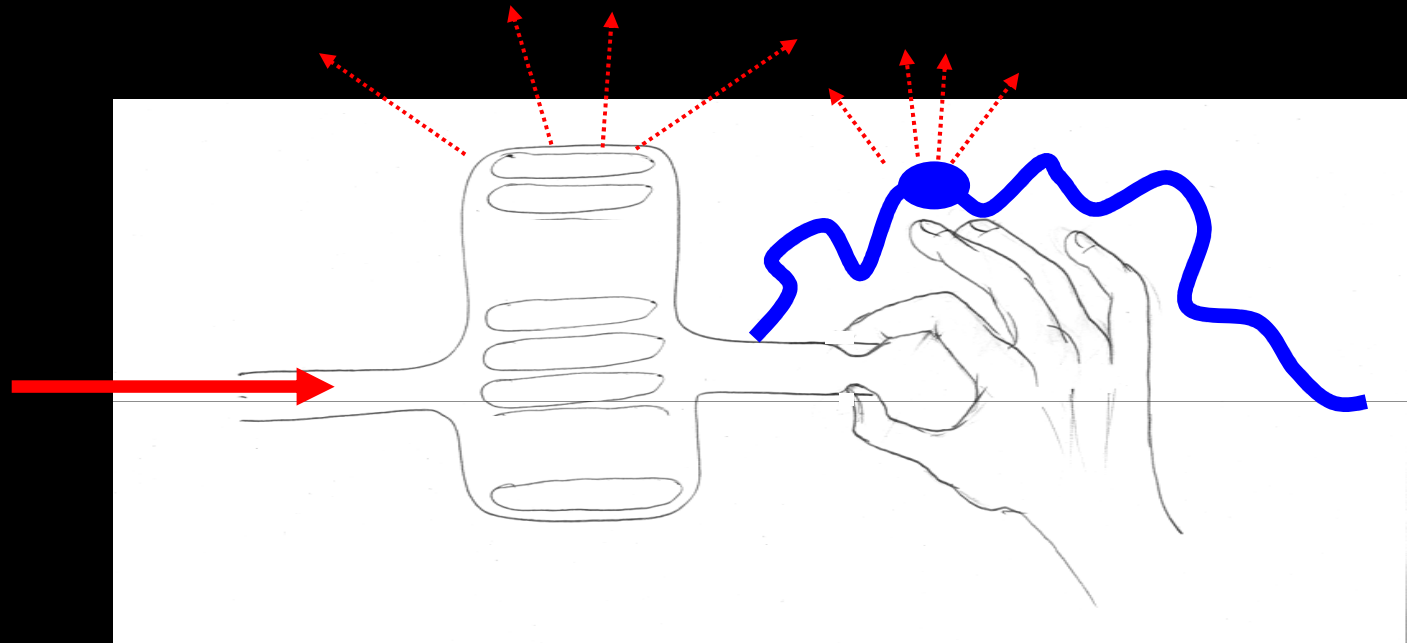
Si les capillaires résistent à la pression



→ Bas débit global

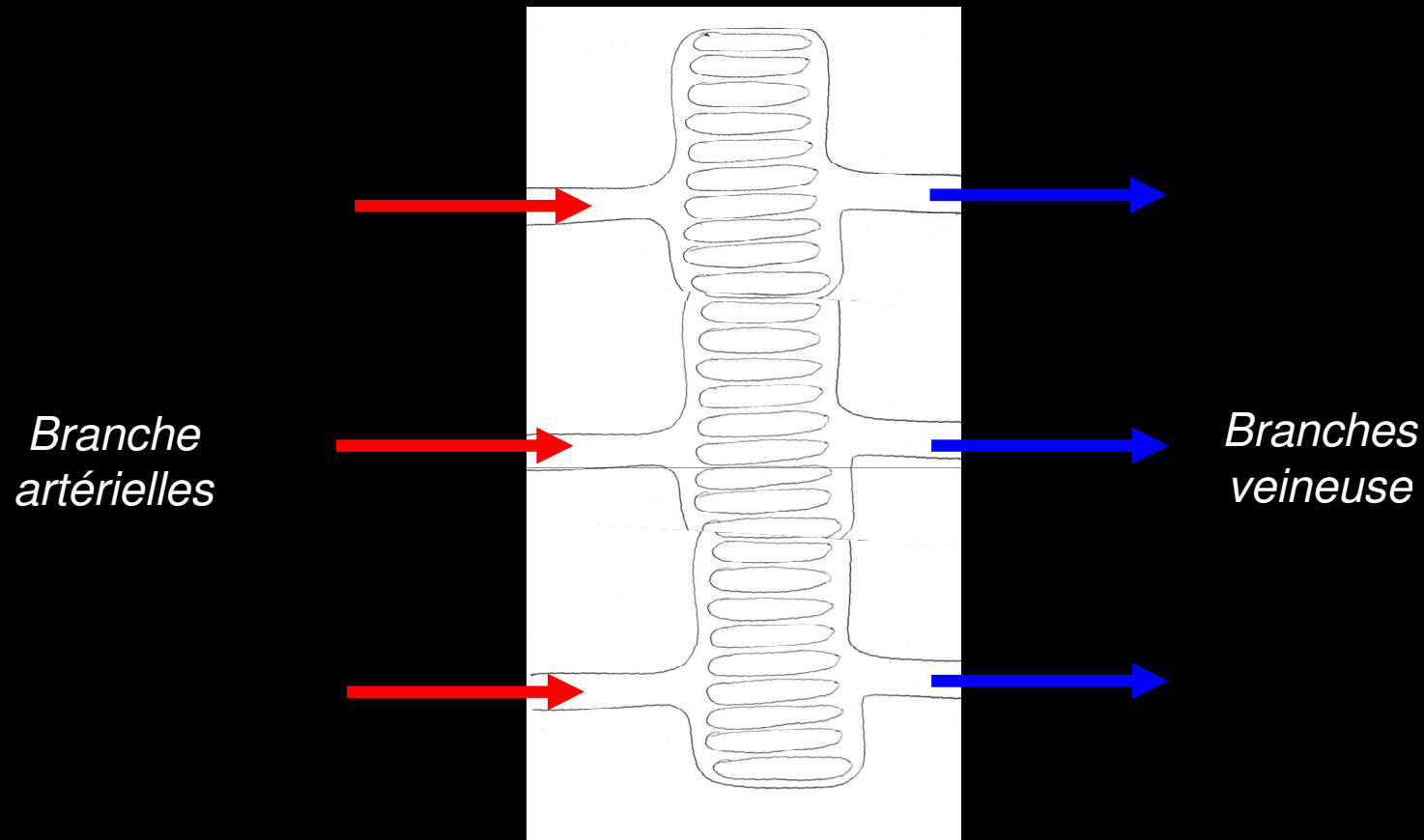


*Un remodelage microvasculaire peut compliquer une OVR,
quel que soit son type initial*



→ nonperfusion, collatérales, macroanévrysmes

Qu'en est-il des OBVR?



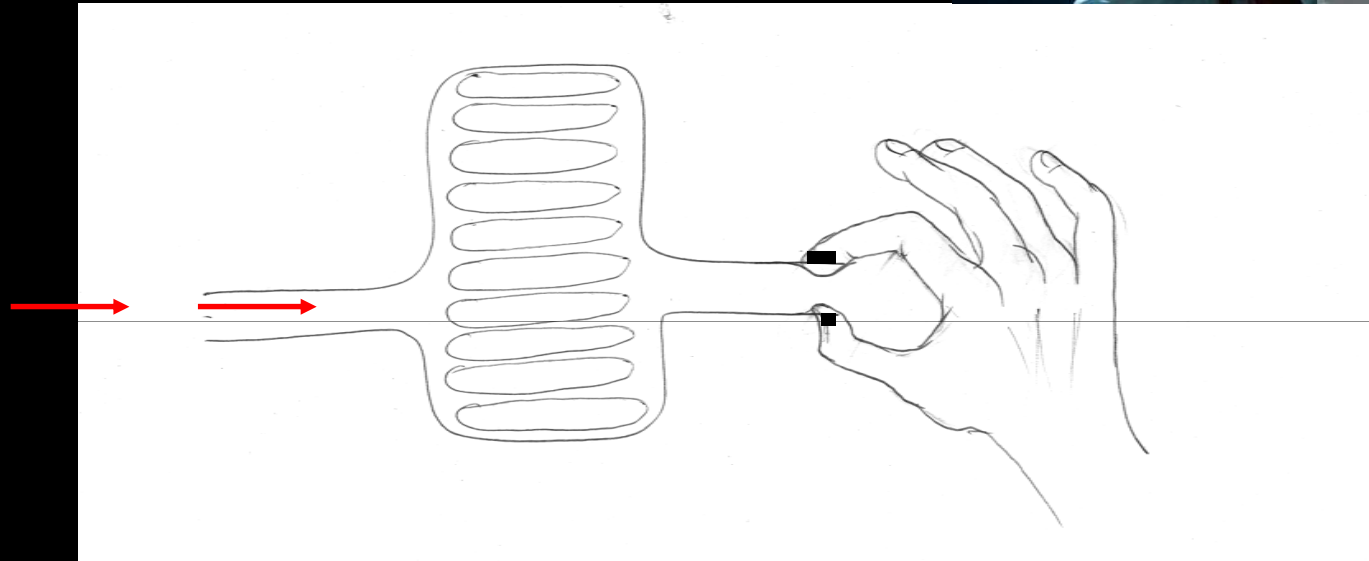
Par rapport aux OVCR, la surface anastomotique autour d'un territoire d'OBVR est beaucoup plus importante ce qui pourrait expliquer la raison de l'absence de complications aiguës des OBVR (le flux veineux trouve toujours le moyen de sortir)

Notions préalables

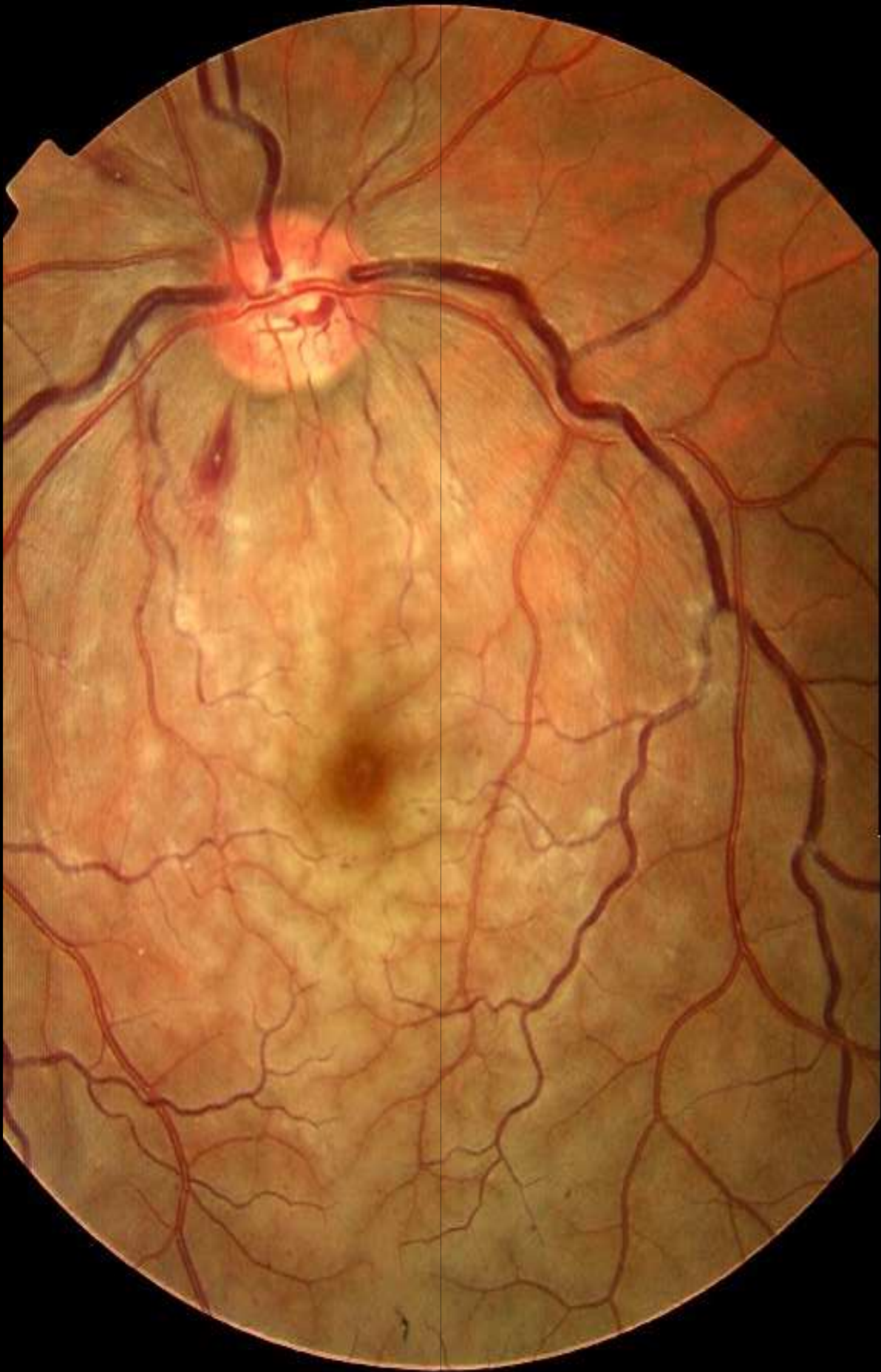
- Lors d'une baisse du flux sanguin, sans arrêt circulatoire, une *ischémie* rétinienne peut survenir: c'est la manifestation cellulaire résultant de l'hypoxie (donc pas seulement présente en cas d'occlusion capillaire)
- Cette ischémie entraîne une vasodilatation artérielle et veineuse
- Une cellule nerveuse en état d'ischémie aiguë devient oedémateuse, *et donc s'opacifie*
- Un vaisseau soumis à un stress *chronique* va modifier son architecture et sa fonction (concept de remodelage microvasculaire)

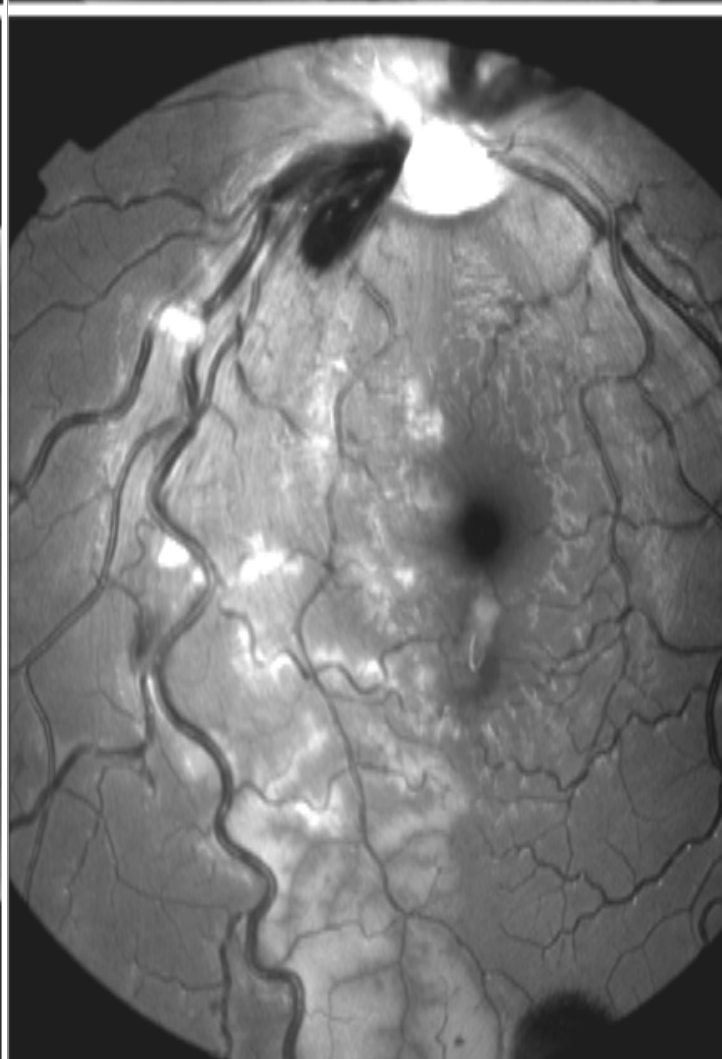
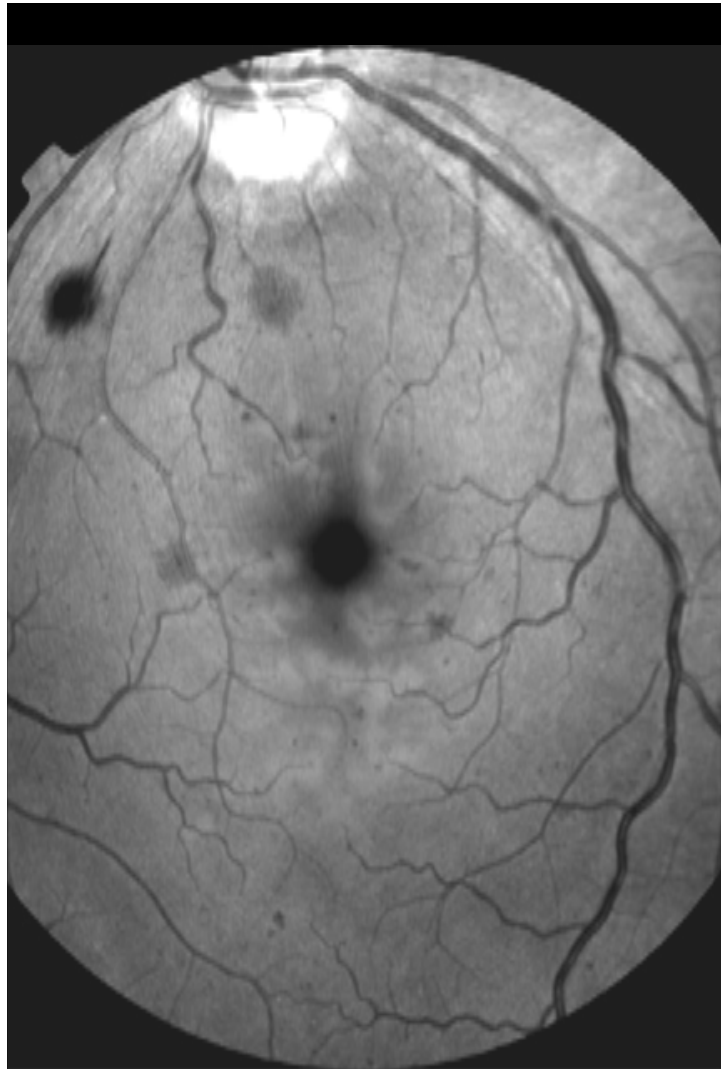
OVCR type A:

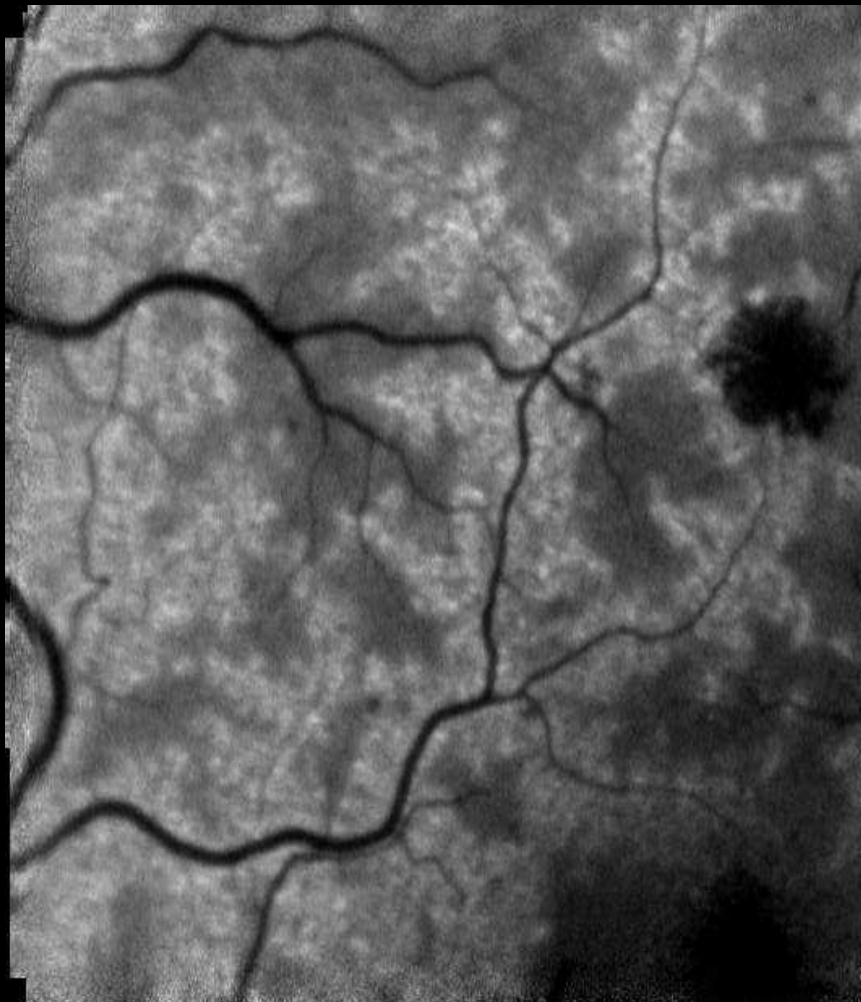
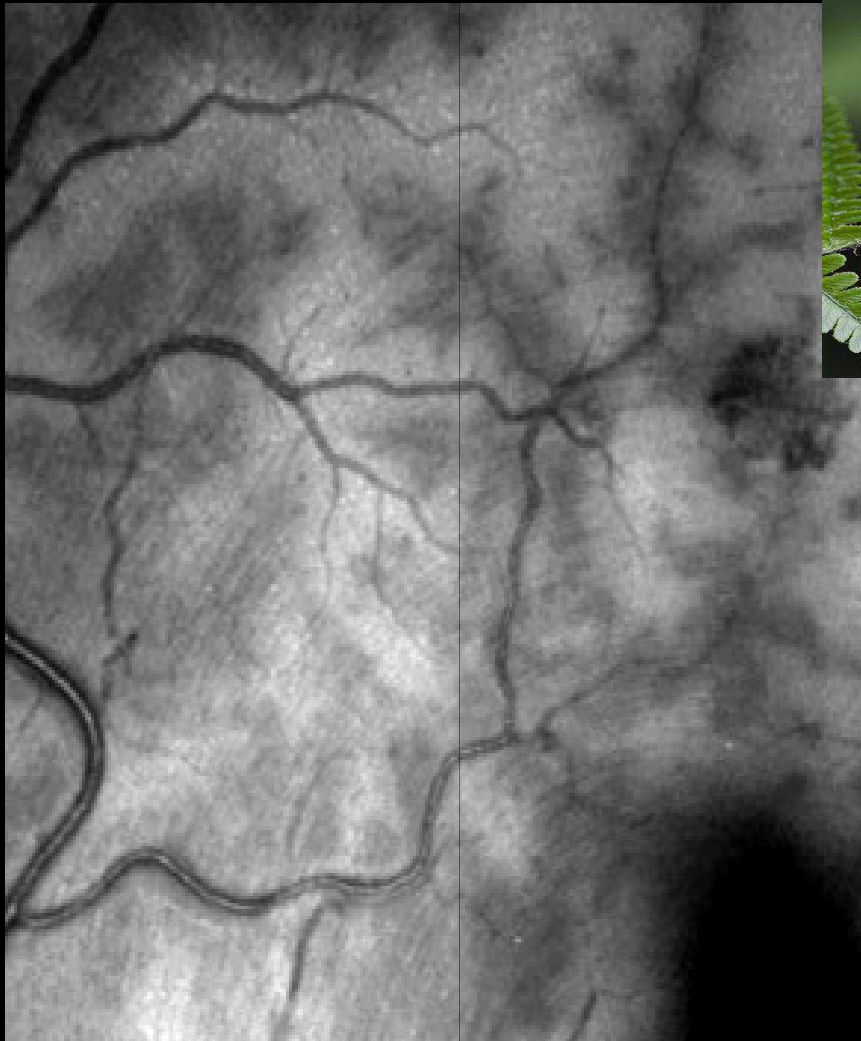
*Survenue brutale d'une OVCR
sur un réseau vasculaire sain*



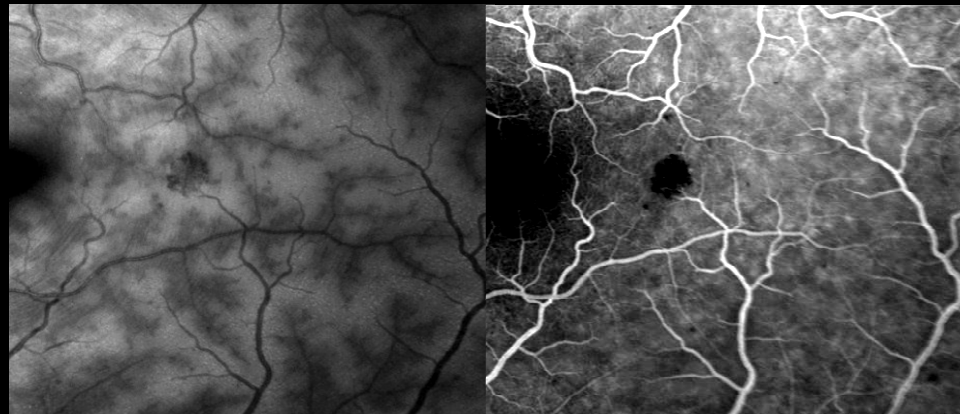
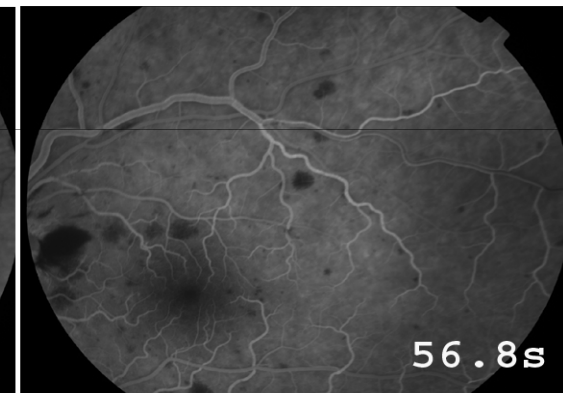
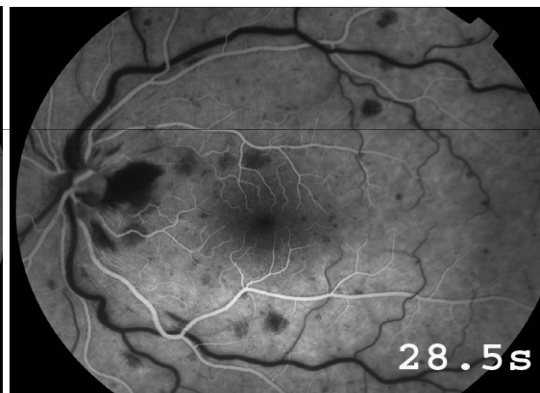
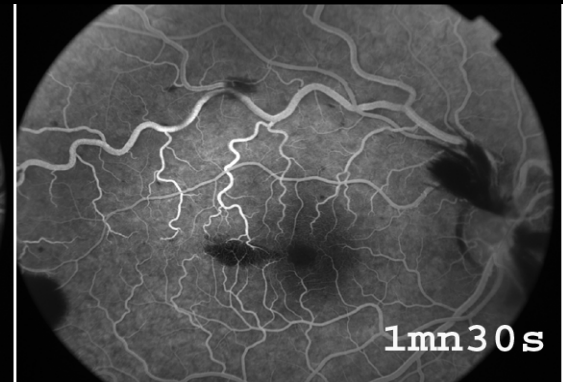
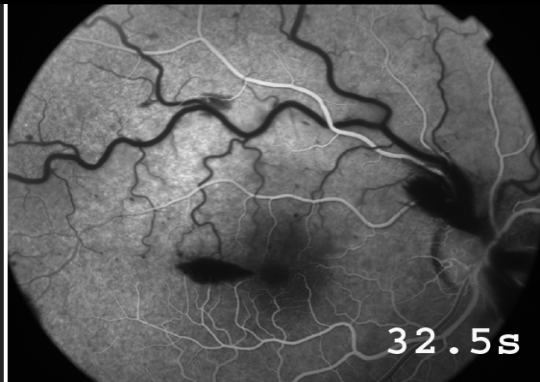
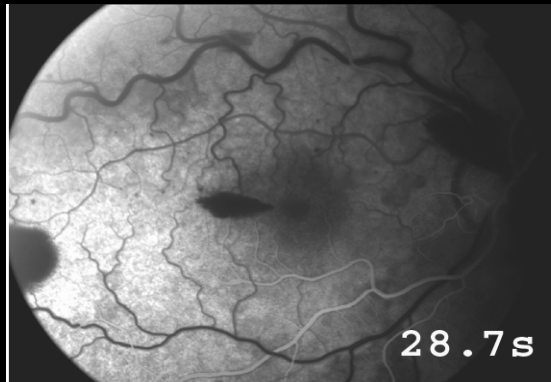
- ➔ Bas débit généralisé
- ➔ Apparition du blanc périveinulaire







Panretinal hypoperfusion

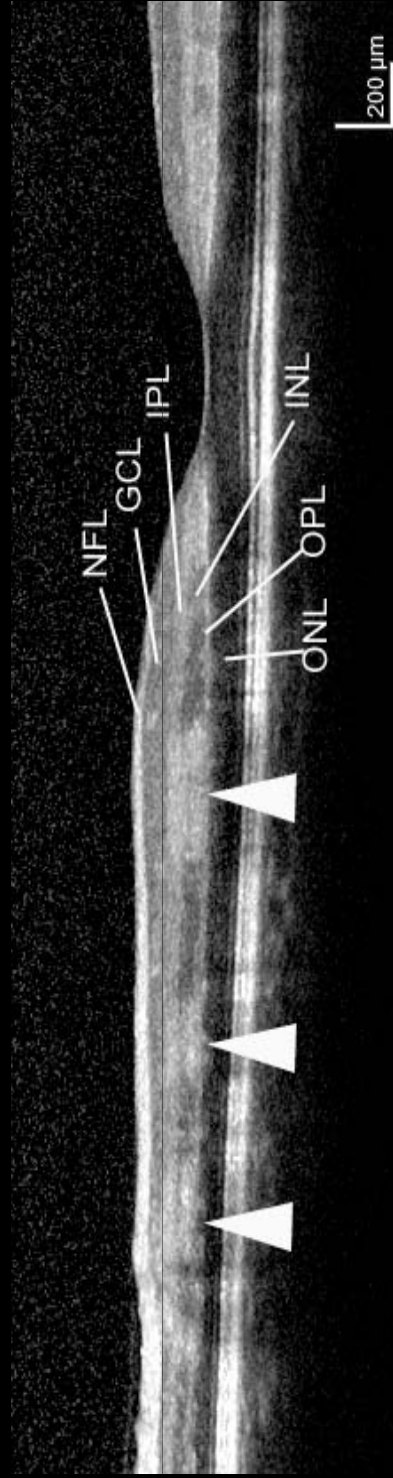
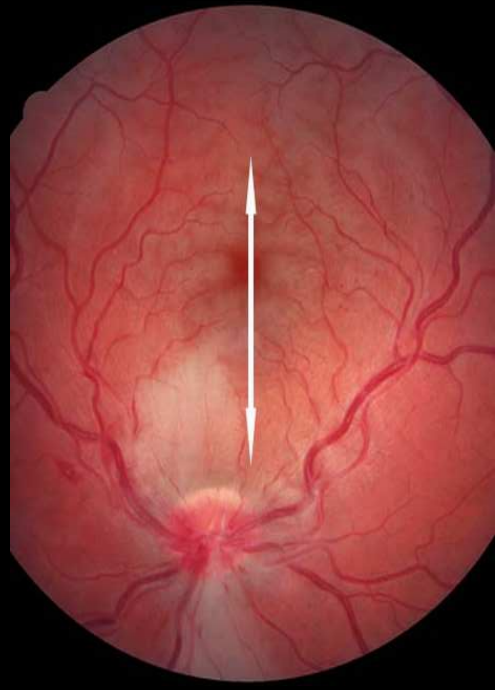




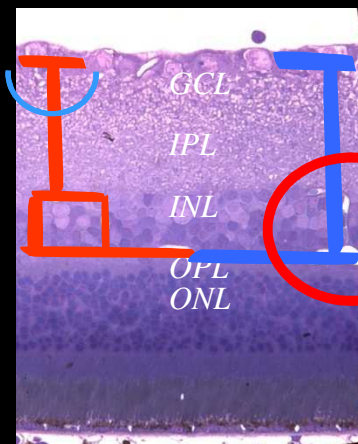
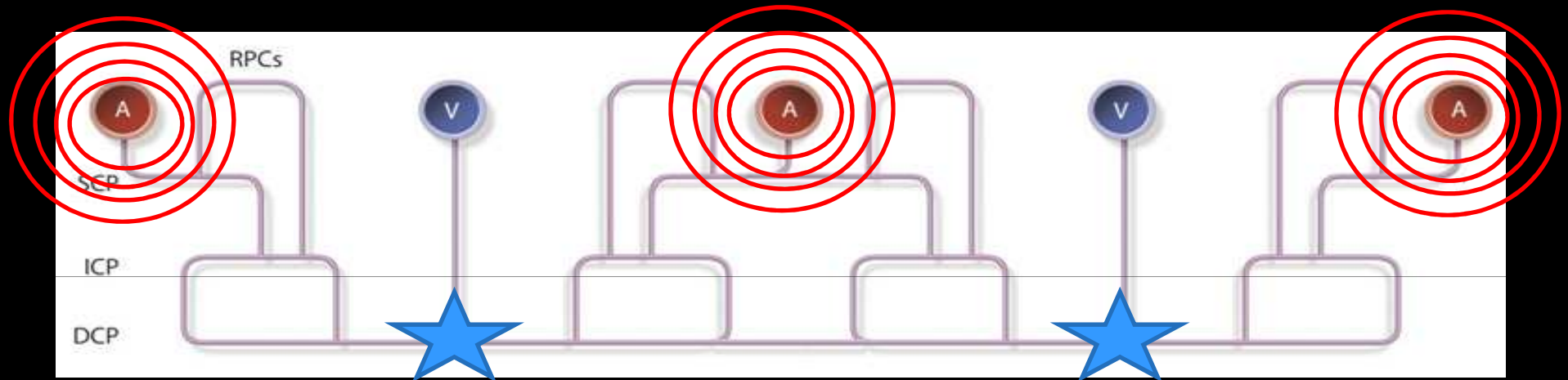


Le BPV représente une opacification ischémique aiguë des neurones, secondaire à une hypoperfusion généralisée

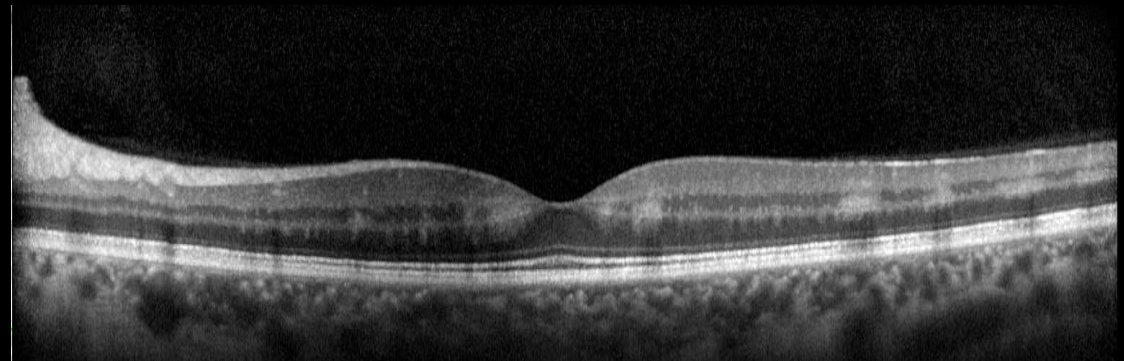
MacLeod Acta Ophthalmologica 2010

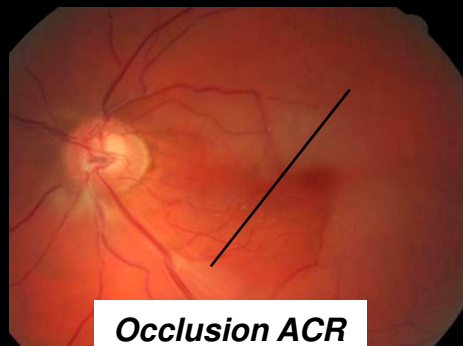


Effect of decreased perfusion pressure on retinal metabolism: The microvascular anatomy explains the localisation of preinflammatory whitening (« PAMM »)

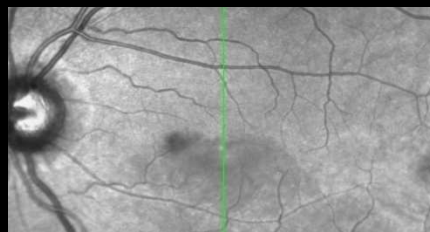
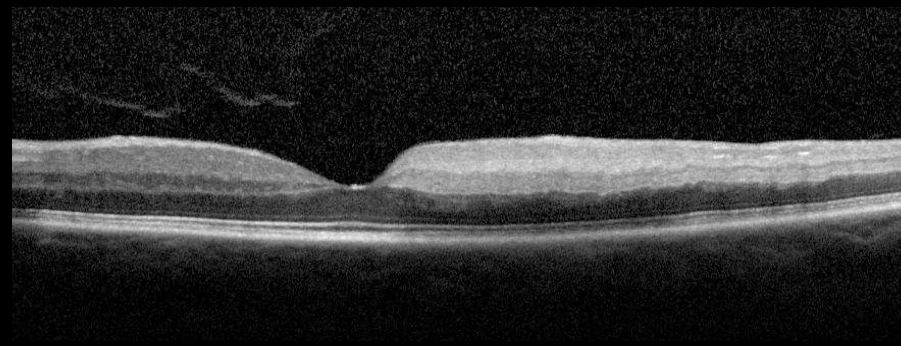


Area of retinal thinning

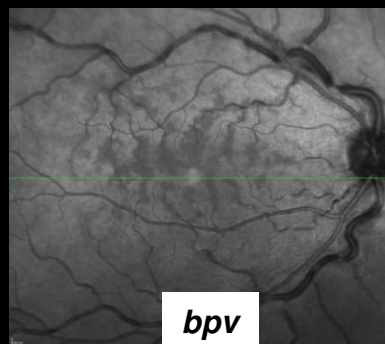
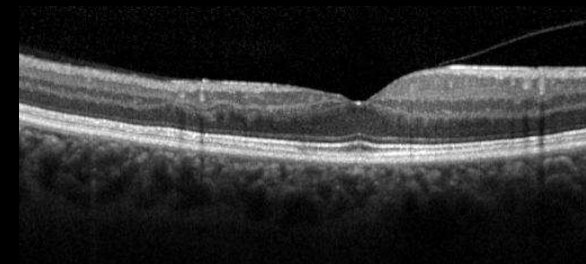
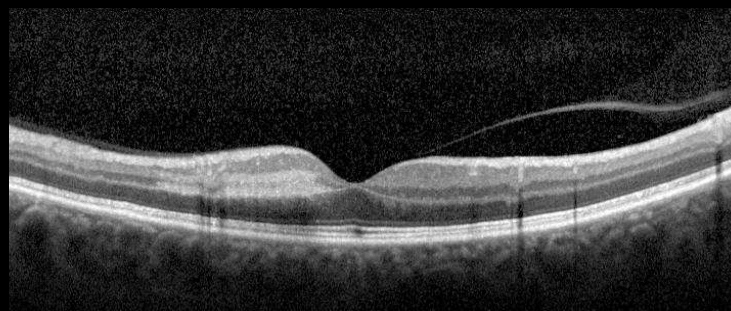




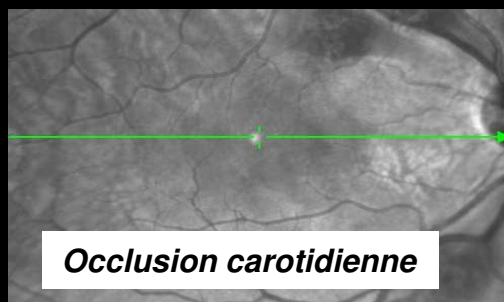
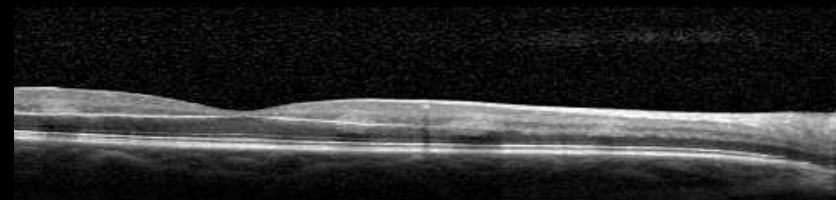
Occlusion ACR



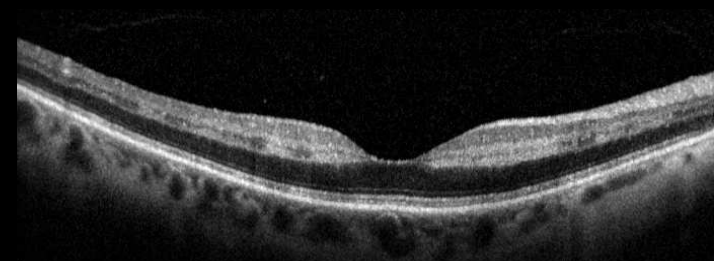
Occlusion ciliorétinienne



bpv



Occlusion carotidienne



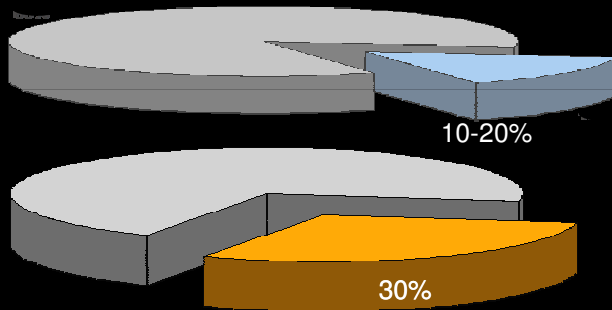
Facteurs de risque selon le type d'OVR

- La pression de perfusion oculaire est

$$PP = (0,67 \text{ PAM}) - \text{PIO}$$

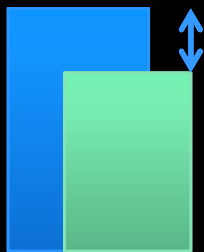
- Donc le débit sanguin rétinien
 - Augmente avec la pression artérielle
 - Diminue avec la pression intraoculaire

Type A



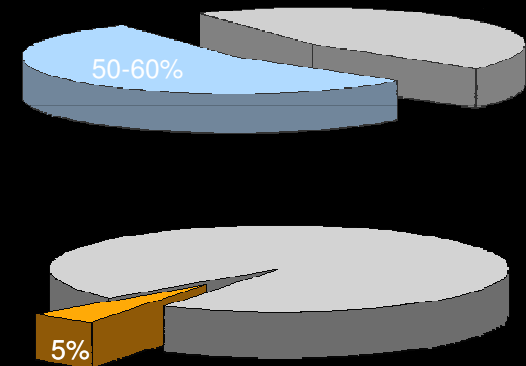
HTA

Glaucome



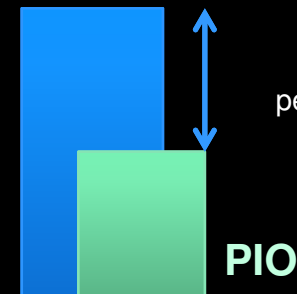
Glaucome
+
Hypotension
artérielle

Type B



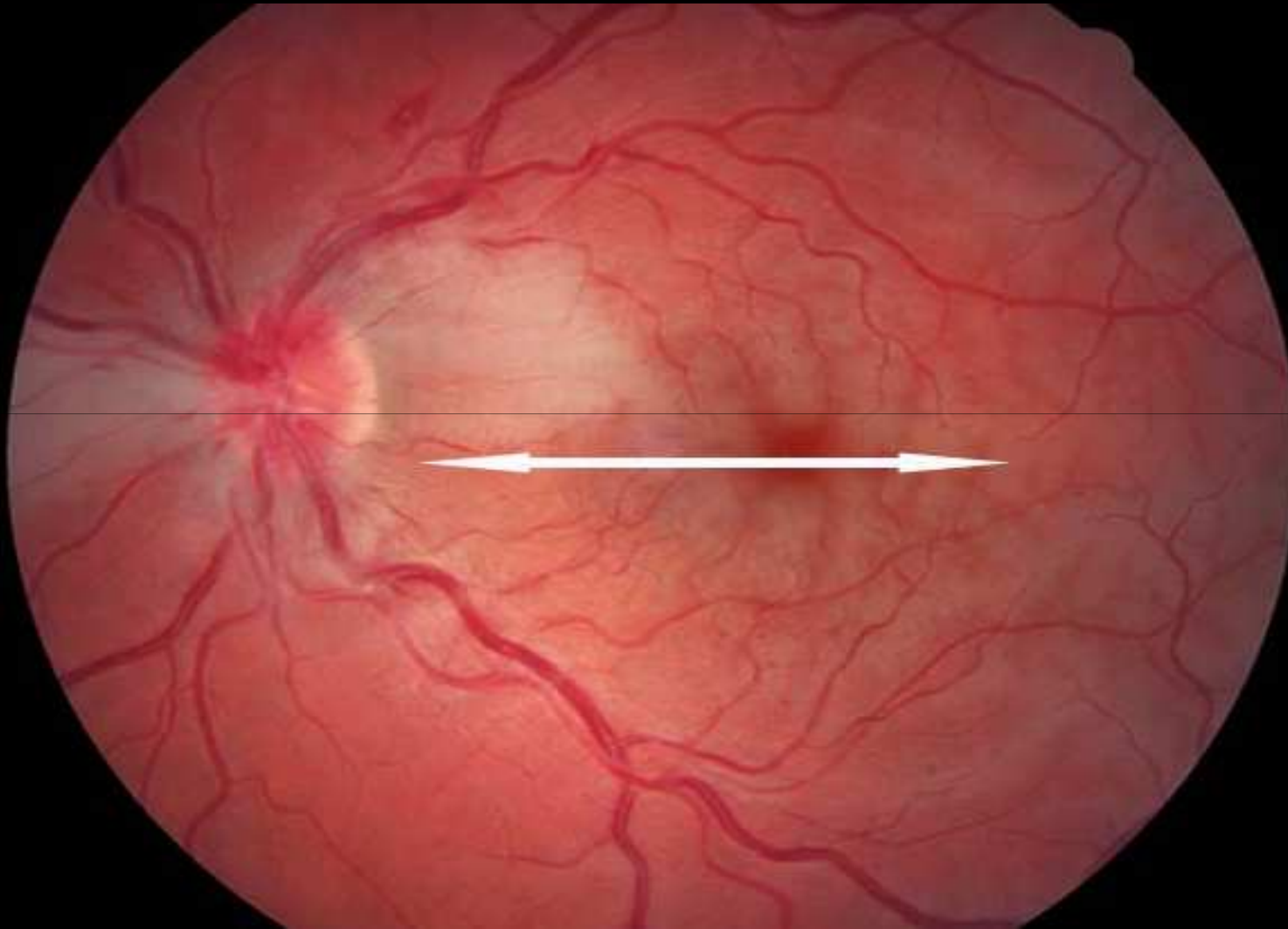
PA

Pression de
perfusion oculaire



PIO

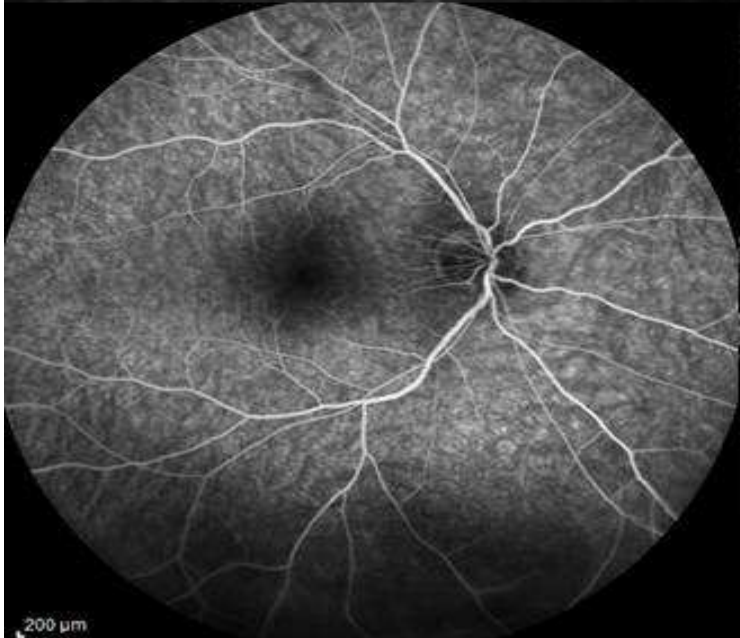
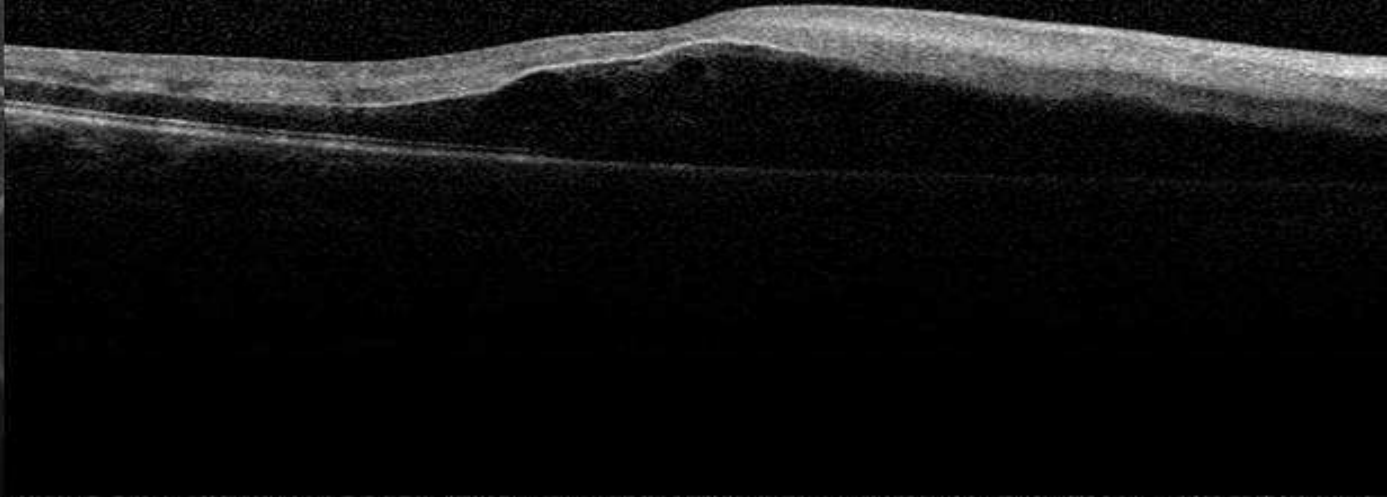
Patient 38 ans. BAV = CLD. PIO = 43 mmHg



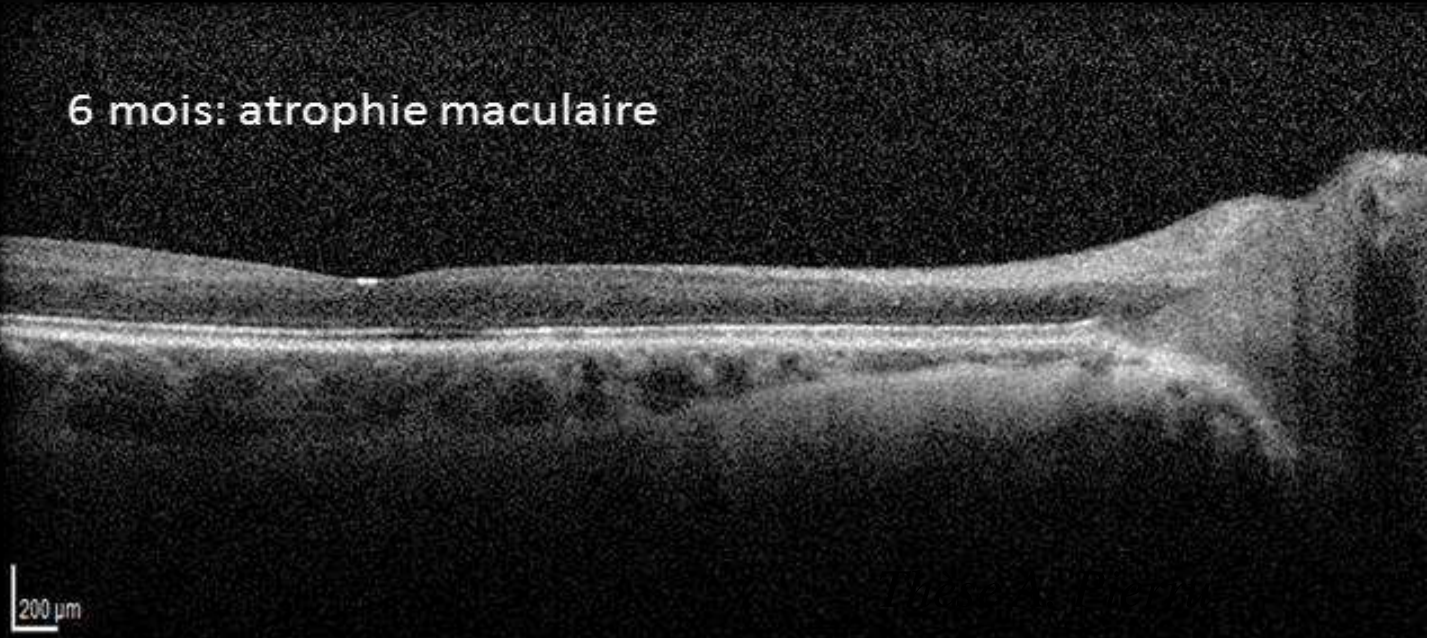
Le type A est souvent suivi d'un œdème papillaire

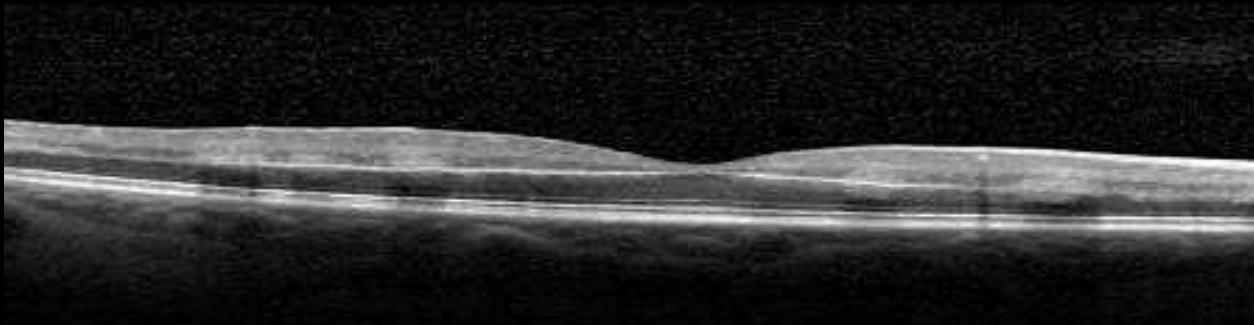
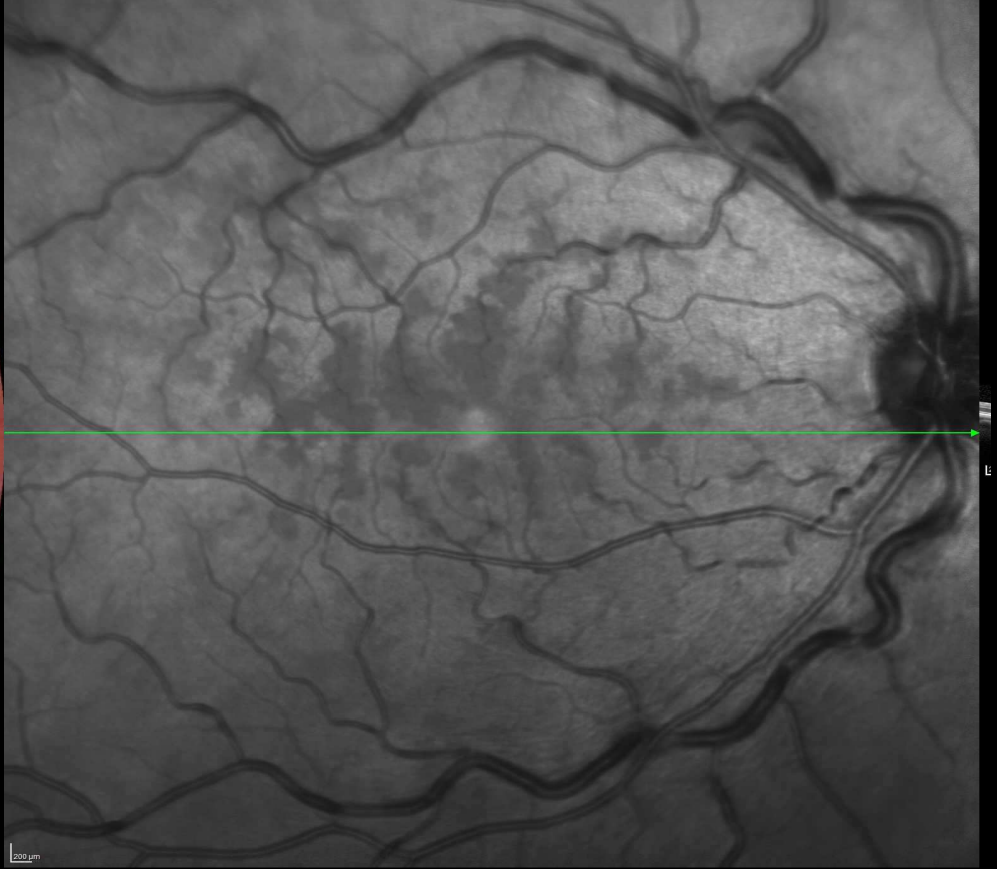


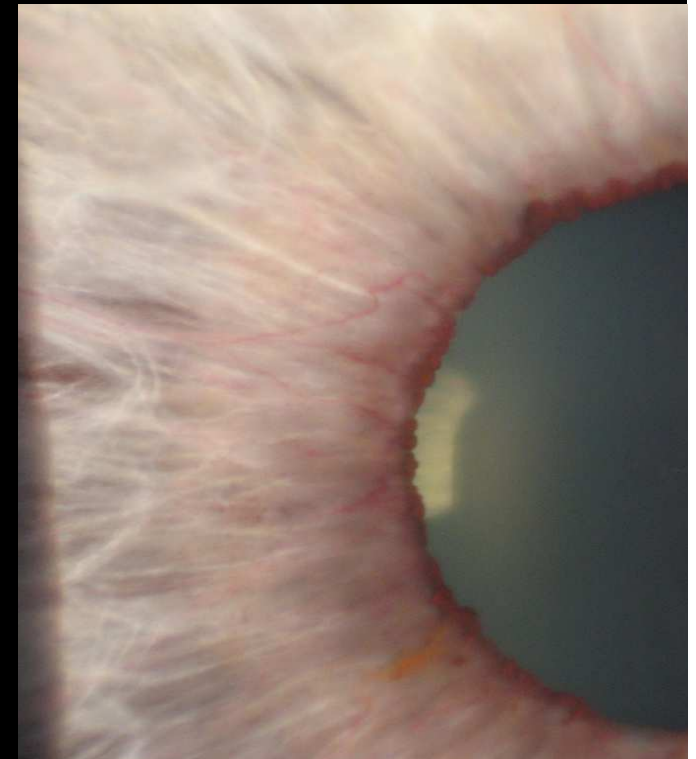
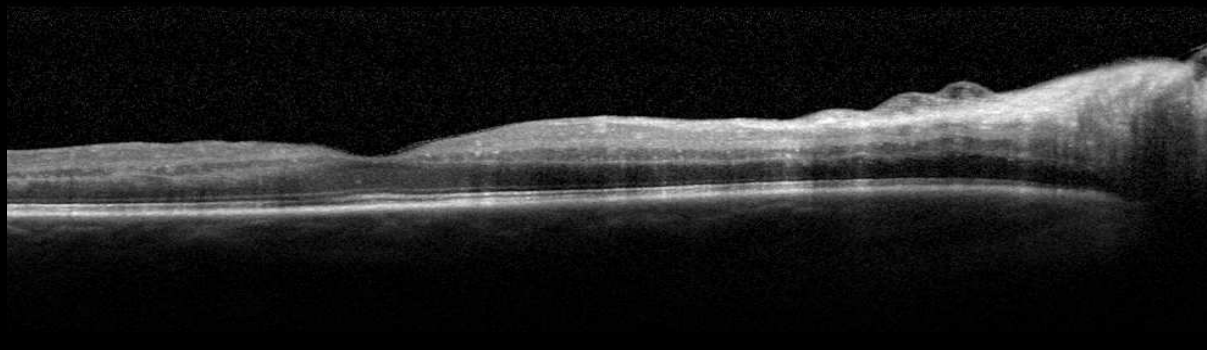
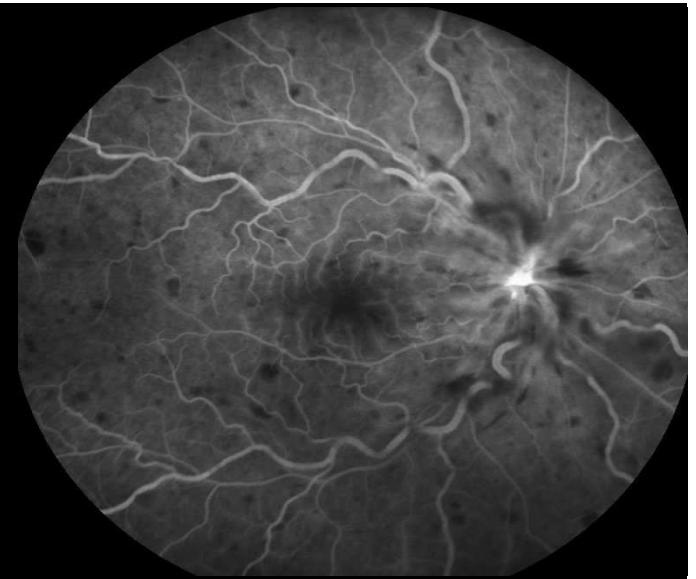
Examen initial: BPV et diffusion papillaire

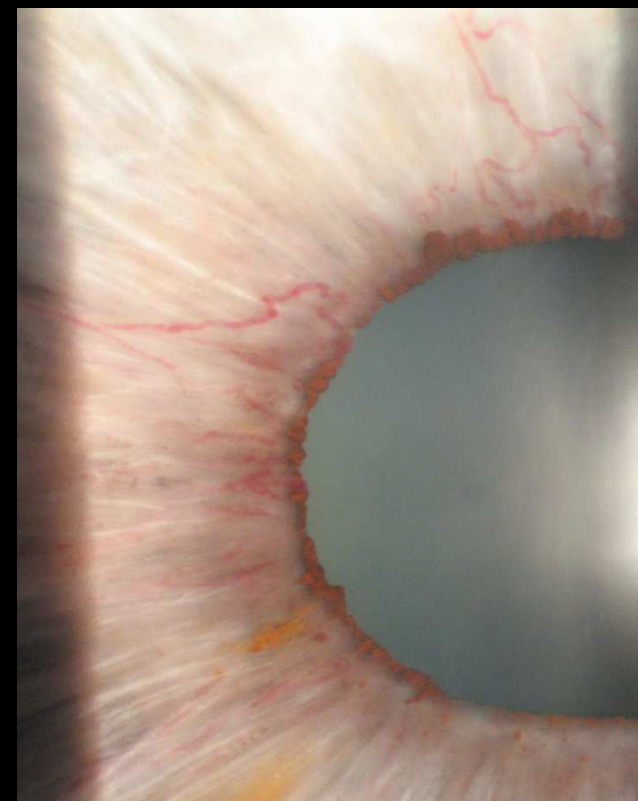
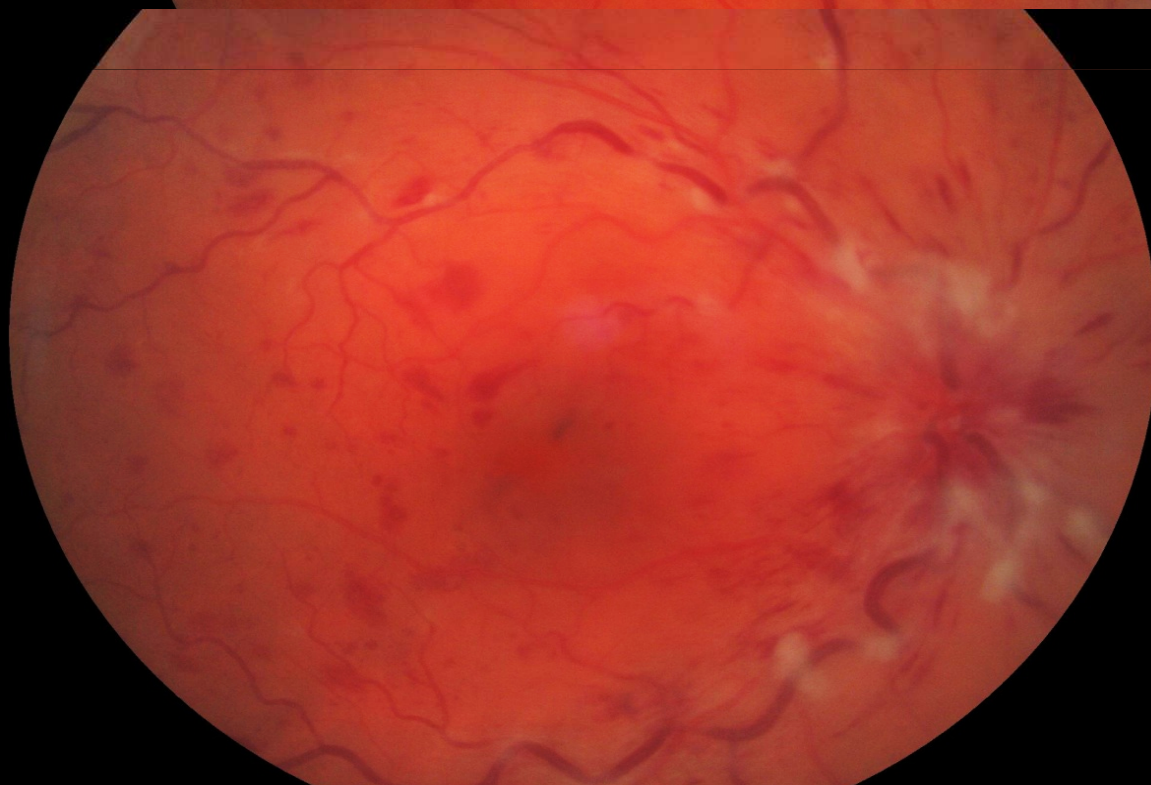


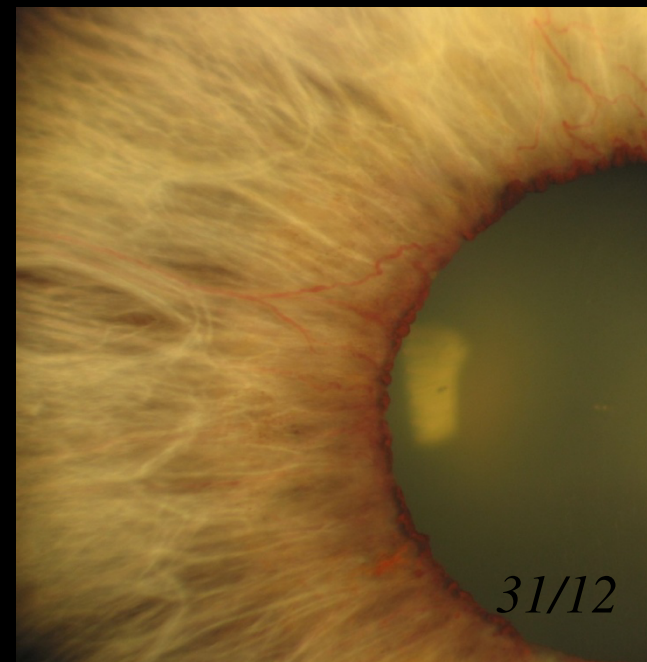
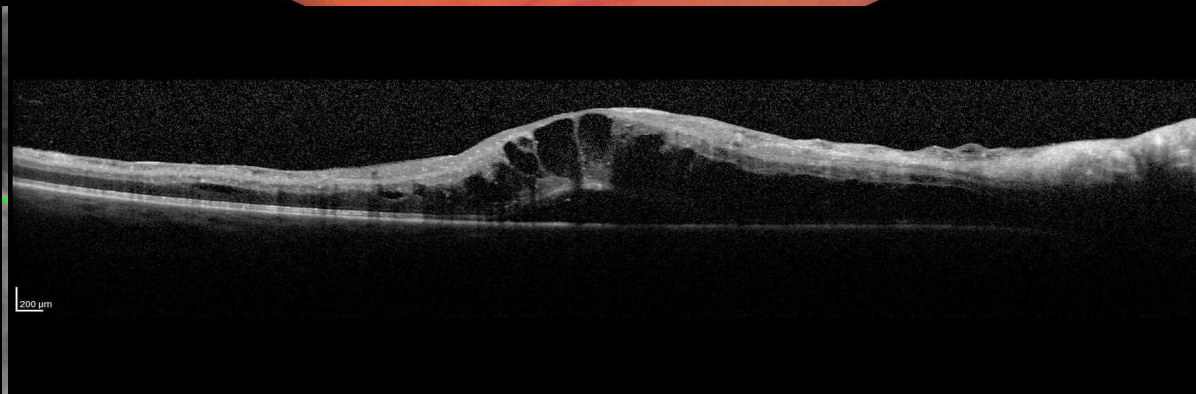
6 mois: atrophie maculaire

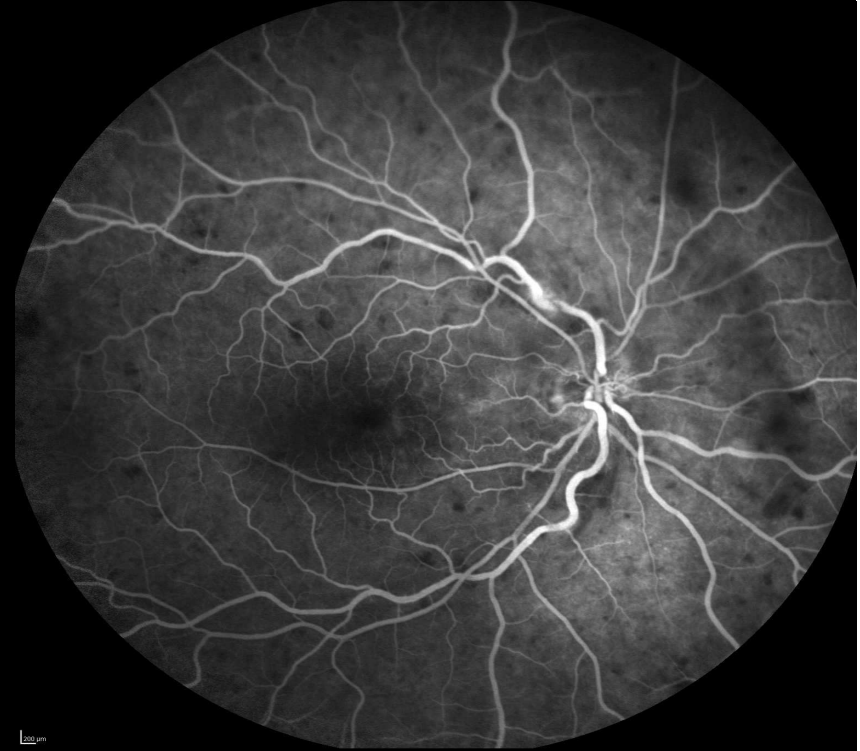
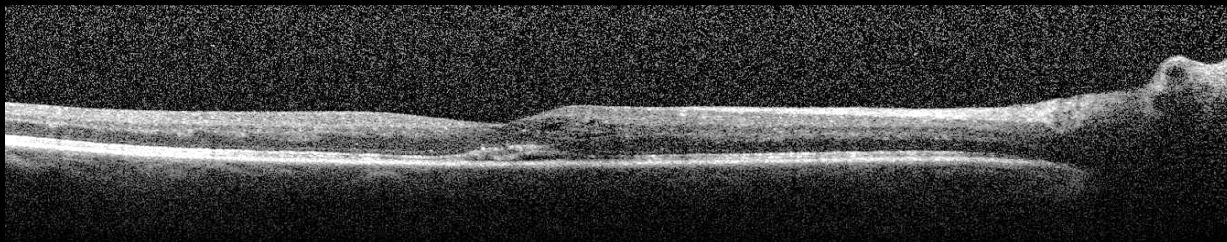
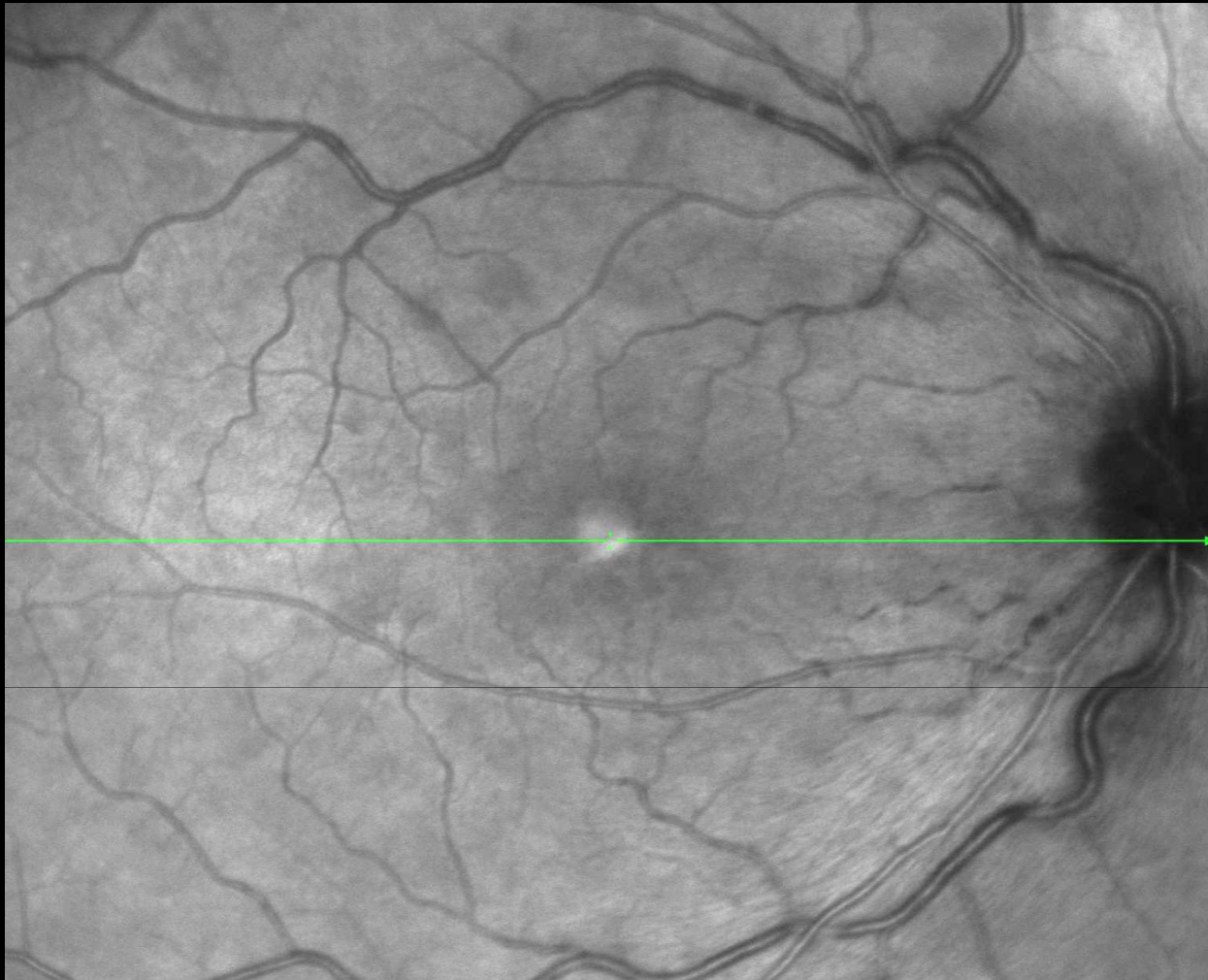






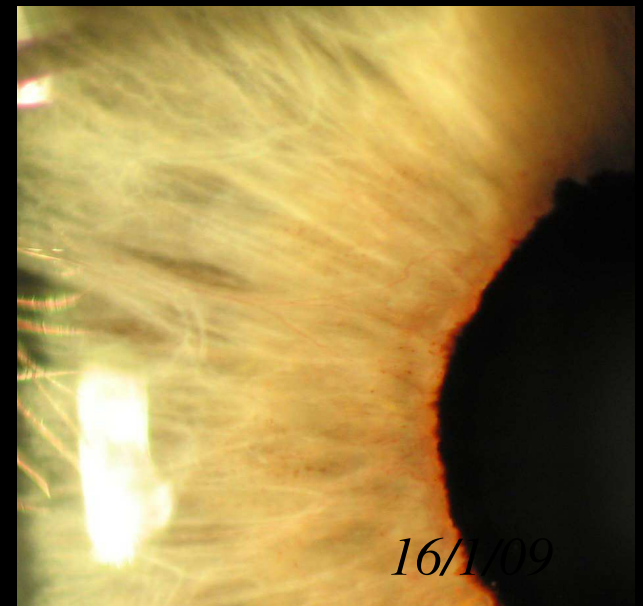




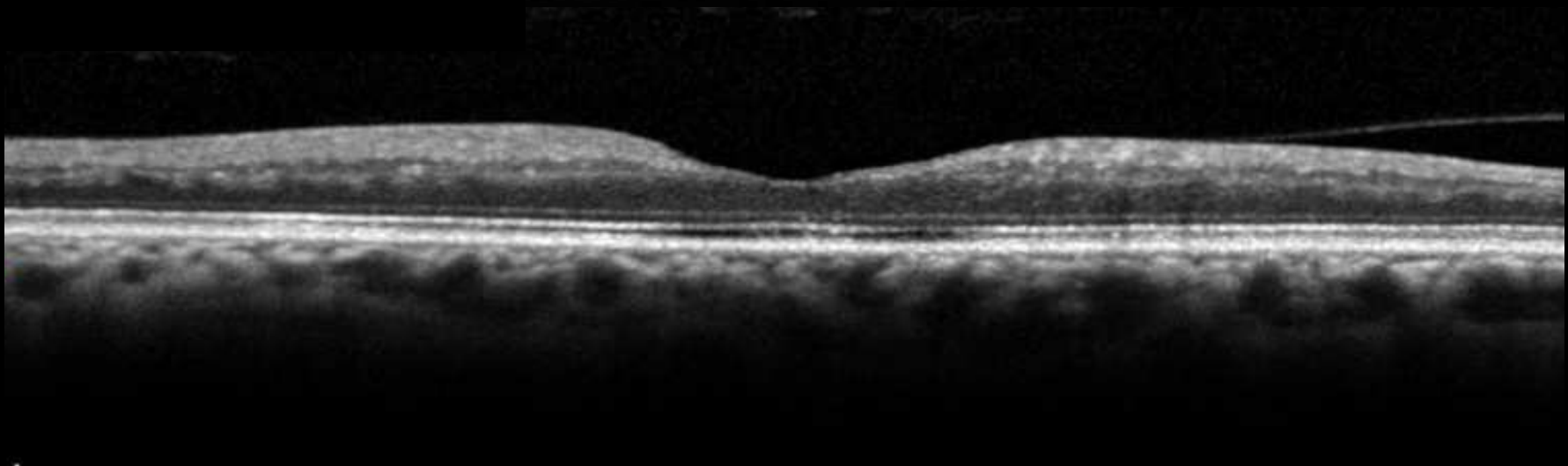
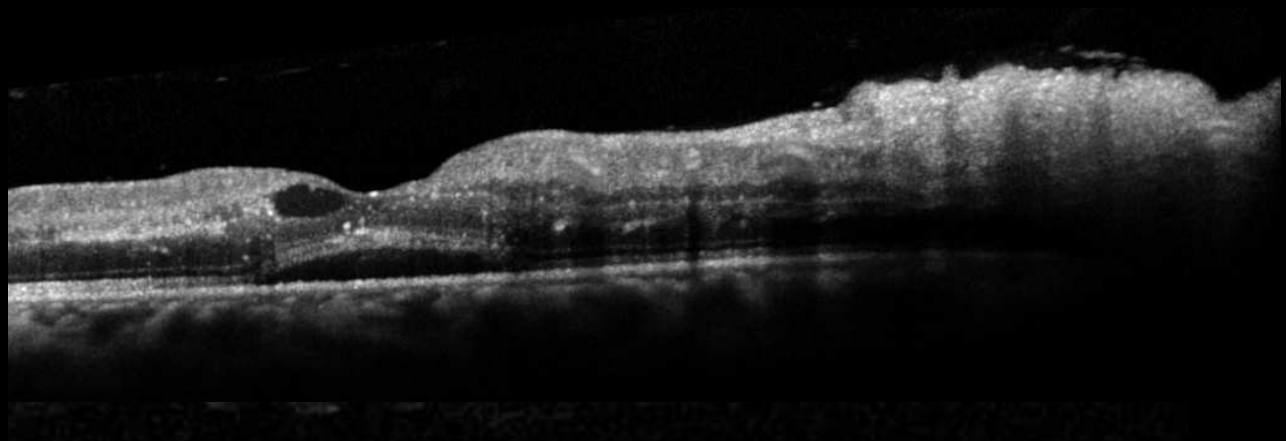


100 μ m
09/02/1992
16/01/2009, OD
FA 3.08.61 30° ART

HEIDELBERG
RETINA



PVW may be subtle, and disc/macular edema more prominent

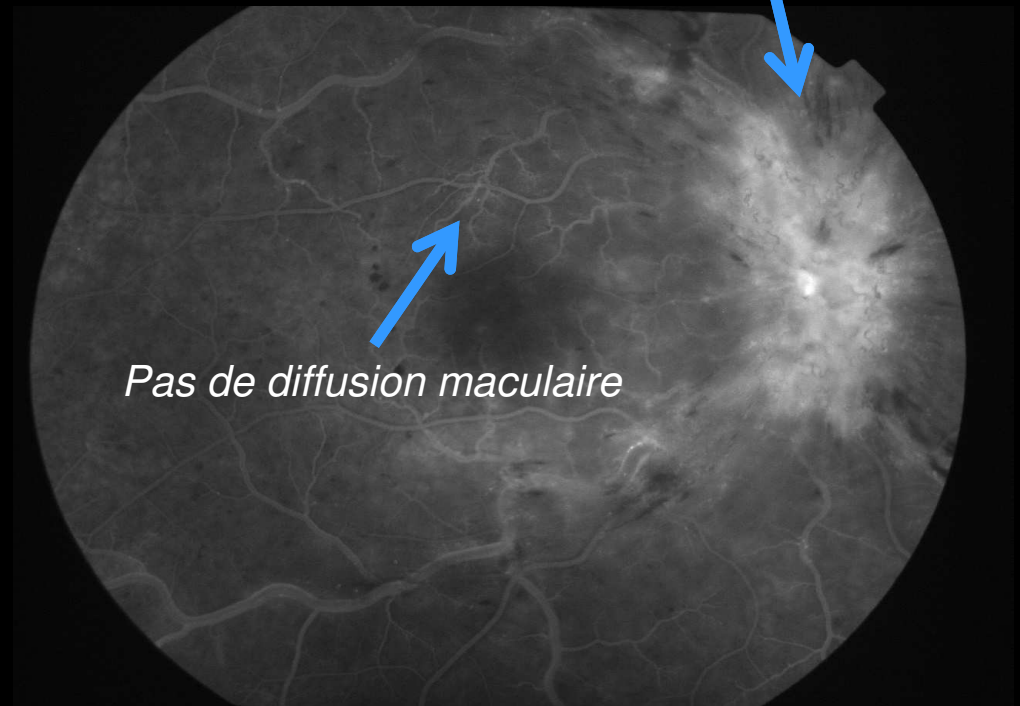


Evolution du type A

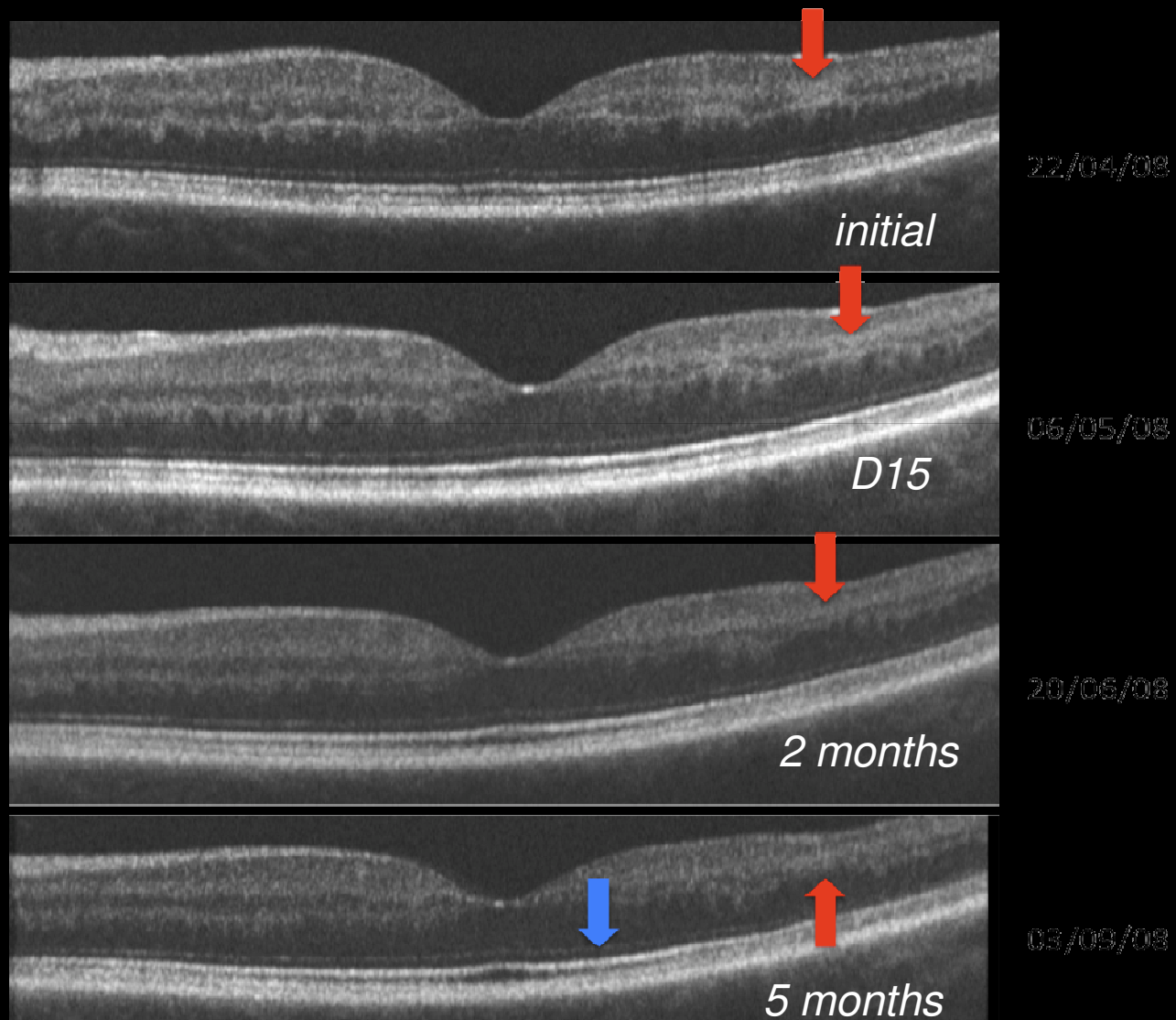
- Debut: Veinoconstriction aigue
 - Ralentissement circulatoire généralisé
 - Occlusion ciliorétinienne en raison des dilatation capillaires de la tête du nerf optique qui impose un double obstacle à la ciliorétinienne
- Puis rupture de la barrière hématorétinienne des capillaires de la tête du nerf optique
 - Oedème papillaire
 - Hémorragies papillaires
 - Possible oedème maculaire par propagation
- Et evolution à long terme selon le degré de reperfusion de la veine centrale

*OVCR avec diffusion de fluorescéine par la papille, sans diffusion maculaire:
possiblement OVCR de type A*

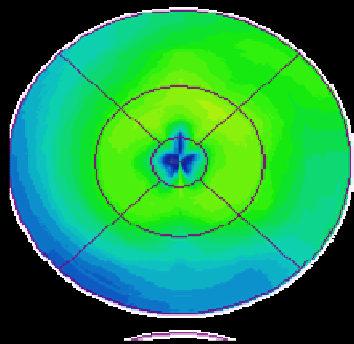
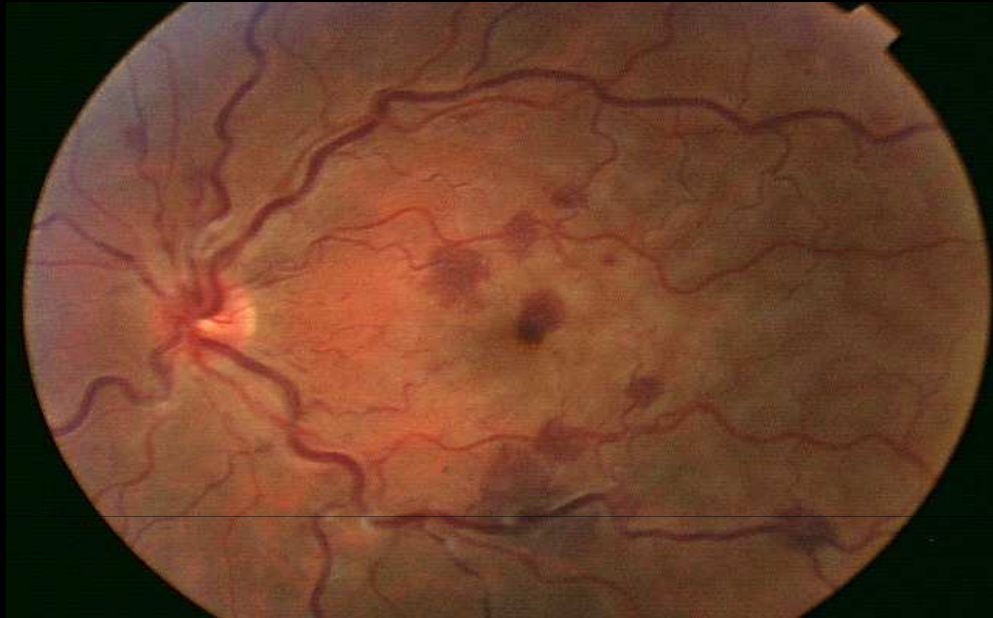
→ un OM qui prédomine du côté nasal de la macula est sans doute une diffusion latérale d'un œdème papillaire



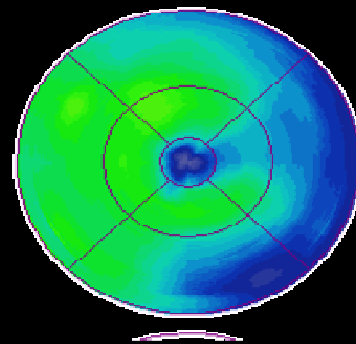
Evolution des lésions rétinienne



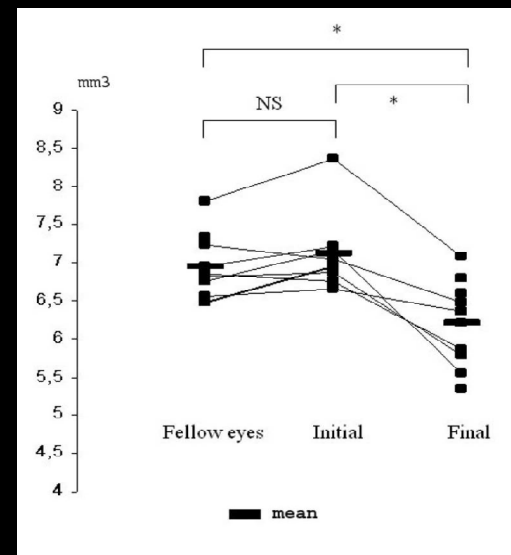
Anatomical results



Fellow



Affected eye



*Mean loss
of macular volume
10.3%*

Functional results

At last visit:

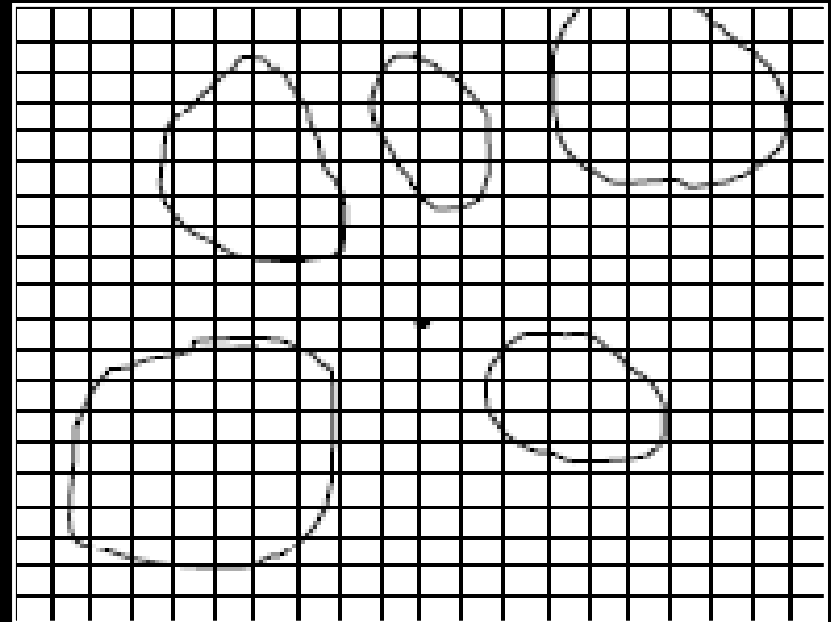
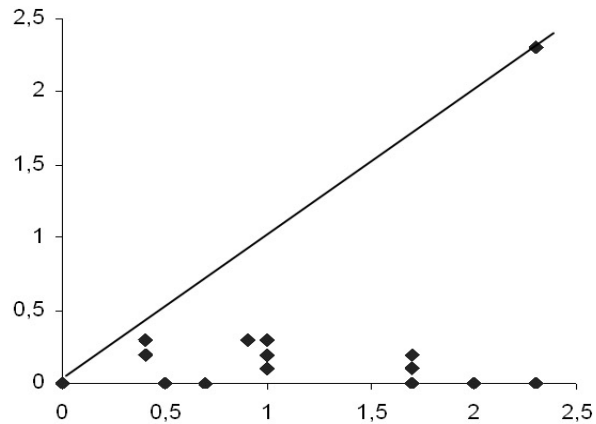
median VA 20/200 → 20/25

13 out of 20 complained of pericentral scotomas

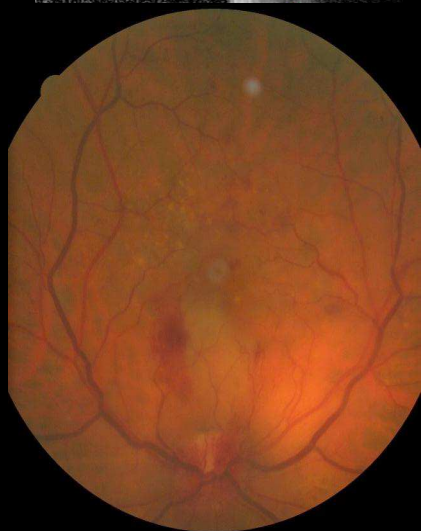
1 had developed preretinal newvessels

No chronic macular edema

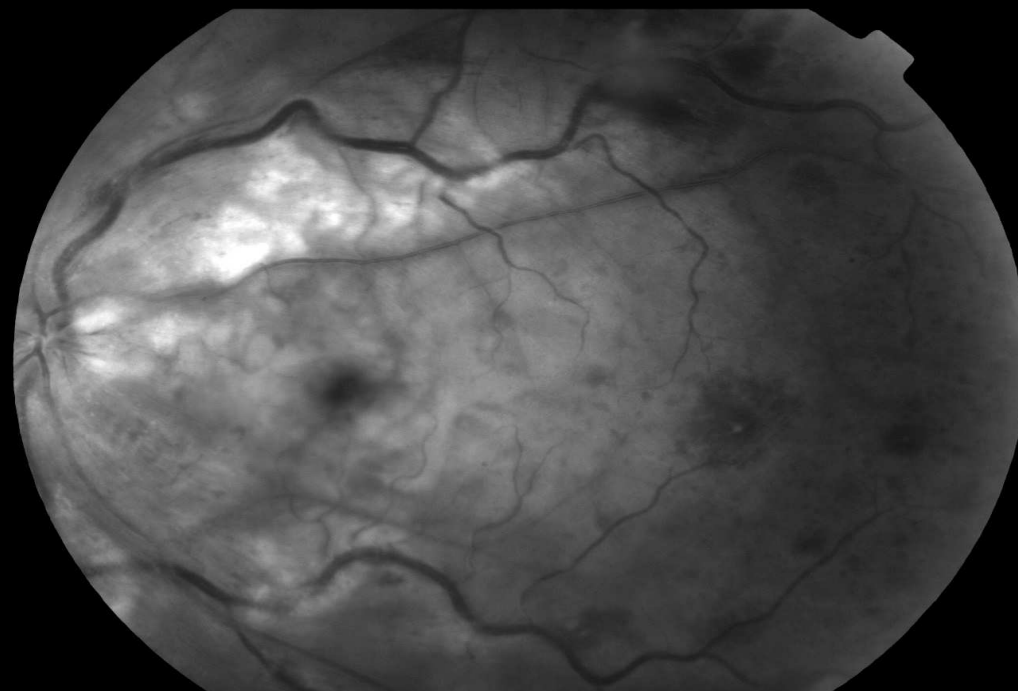
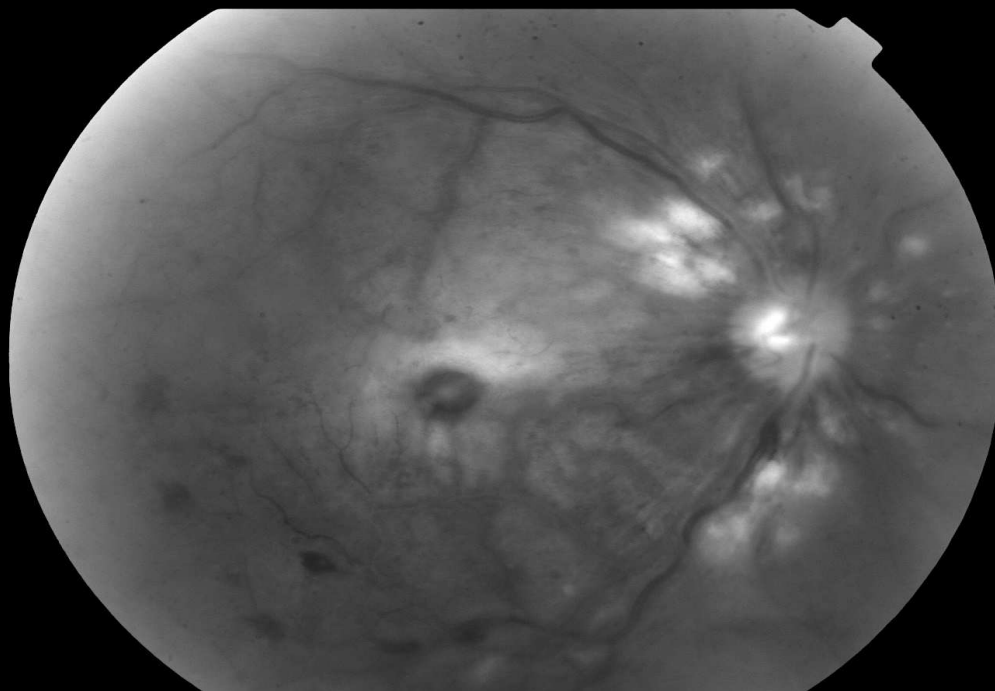
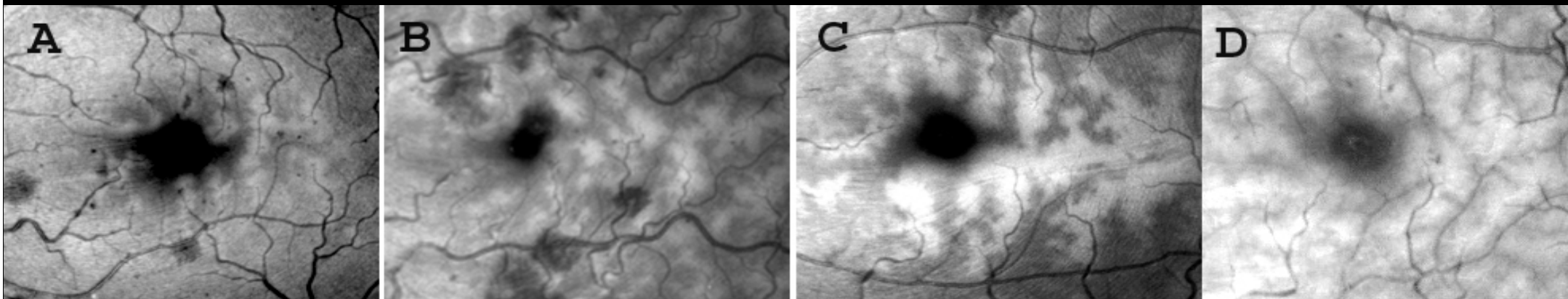
LogMAR

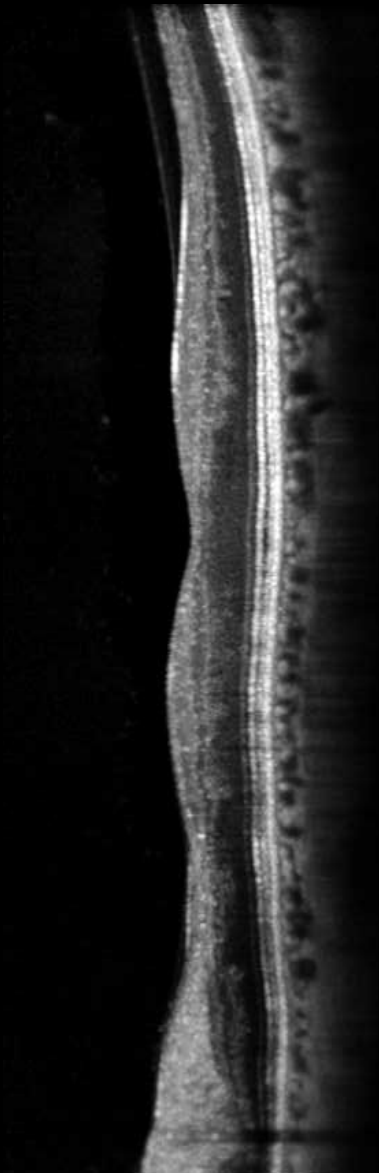


Quelques exemples de type A



continuum de PVW à occlusion dite « combinée »





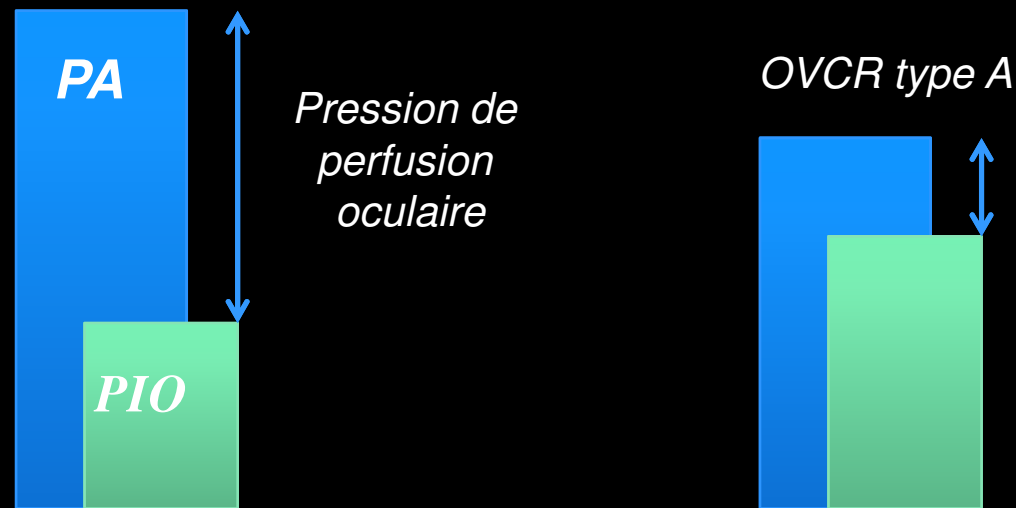
Traitement du type A

En principe, en cas d'OVCR de type A vue au stade précoce:

-Augmenter la pression artérielle

-Baisser la pression intraoculaire

Afin d'augmenter la pression de perfusion oculaire



Type A ou B?



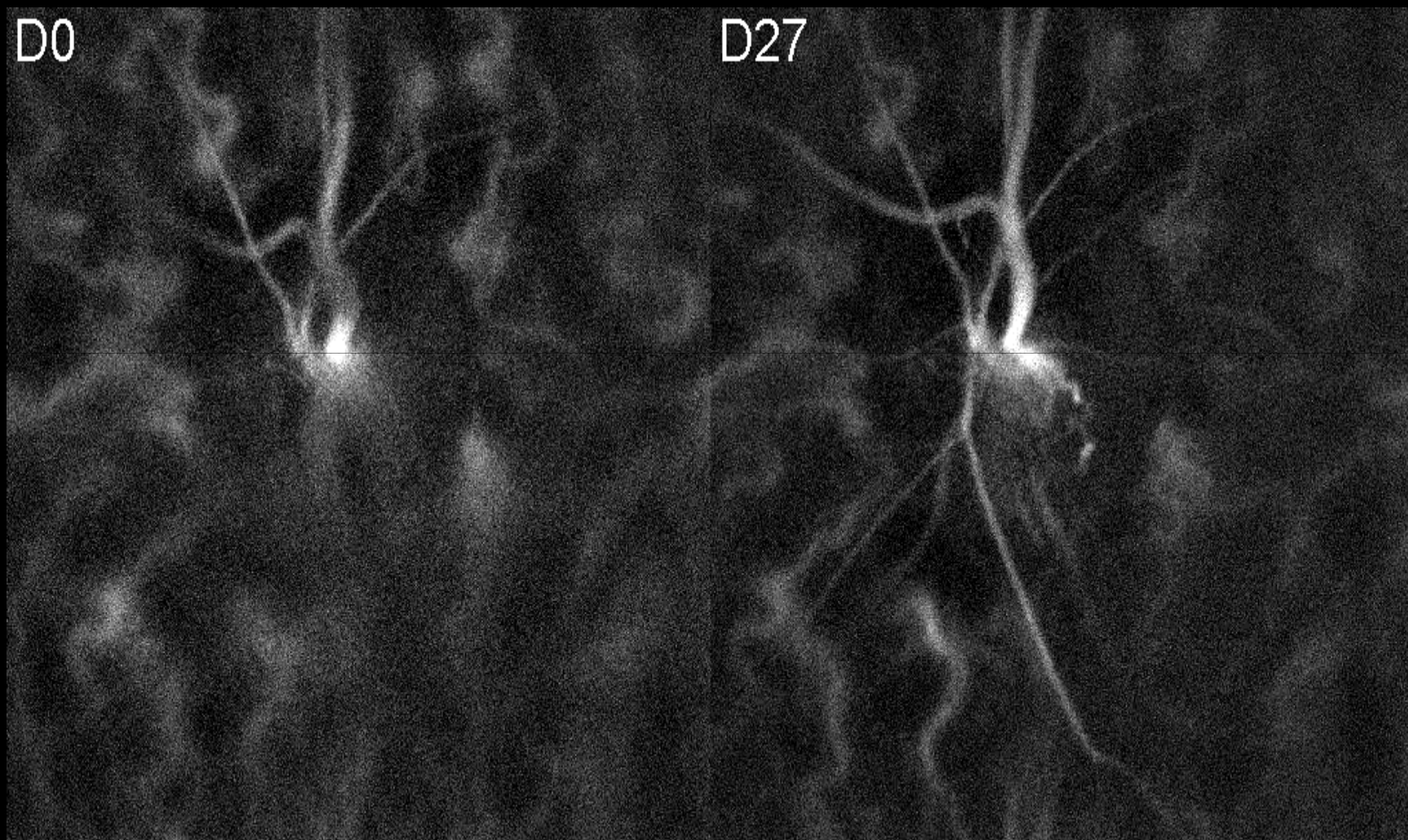


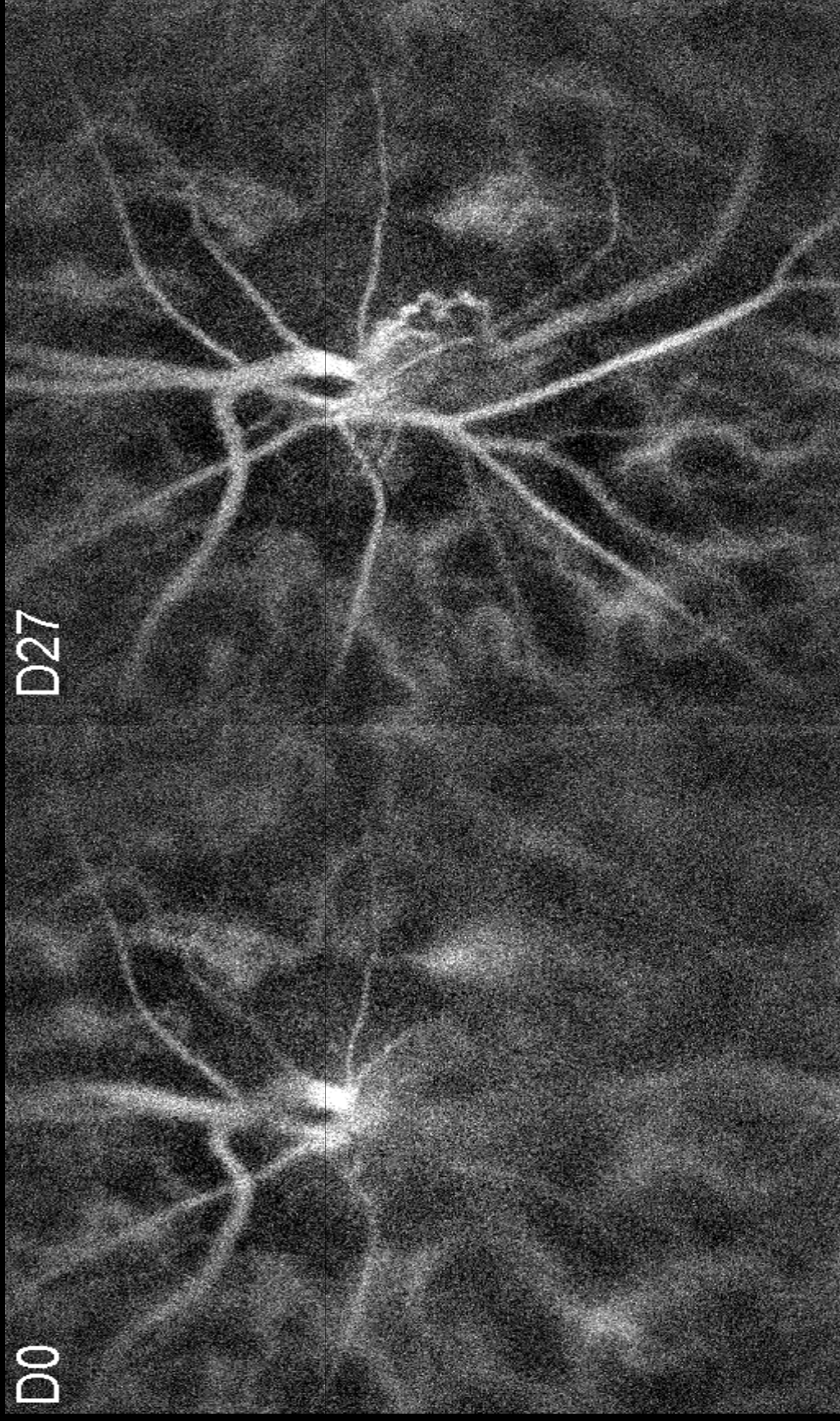


200 μm

200 μm

hemioclusion

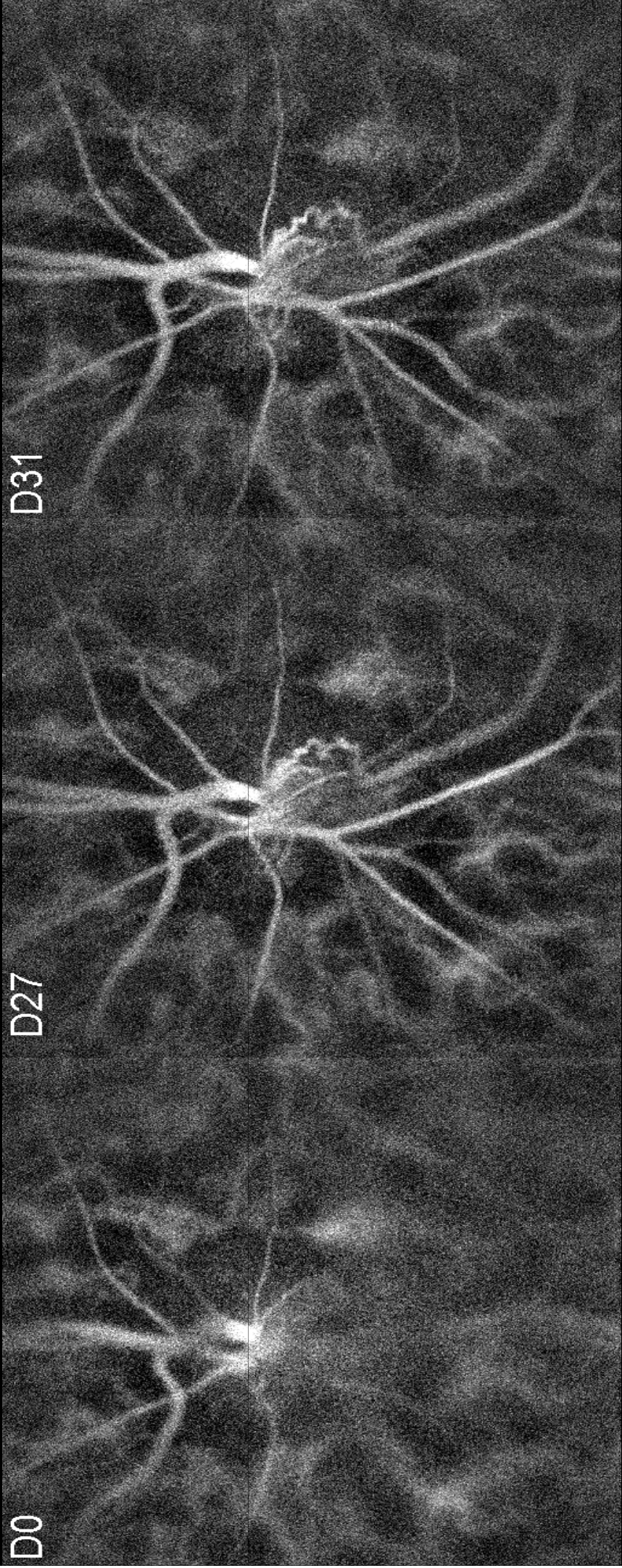




D0

D27

D31

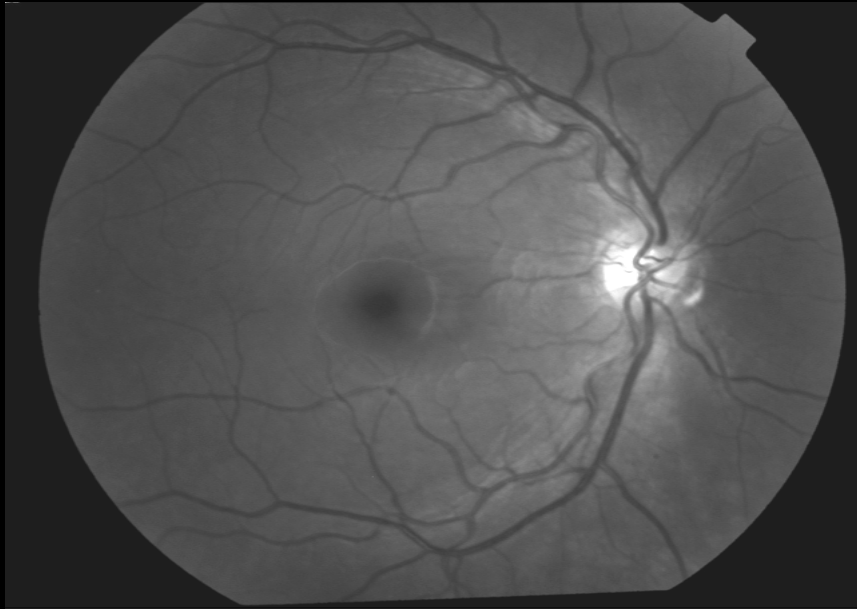


Que se passe-t-il au cours des OVCR de
type B?

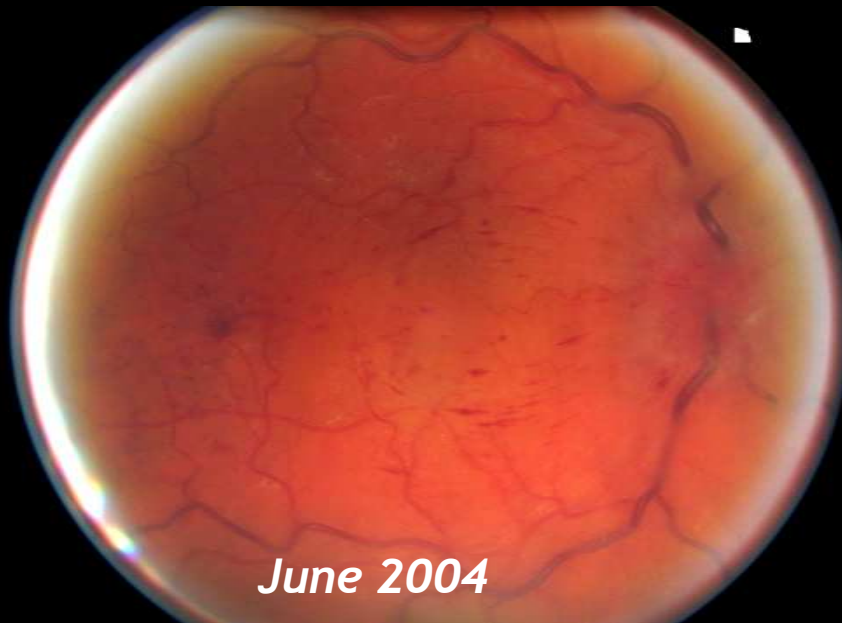
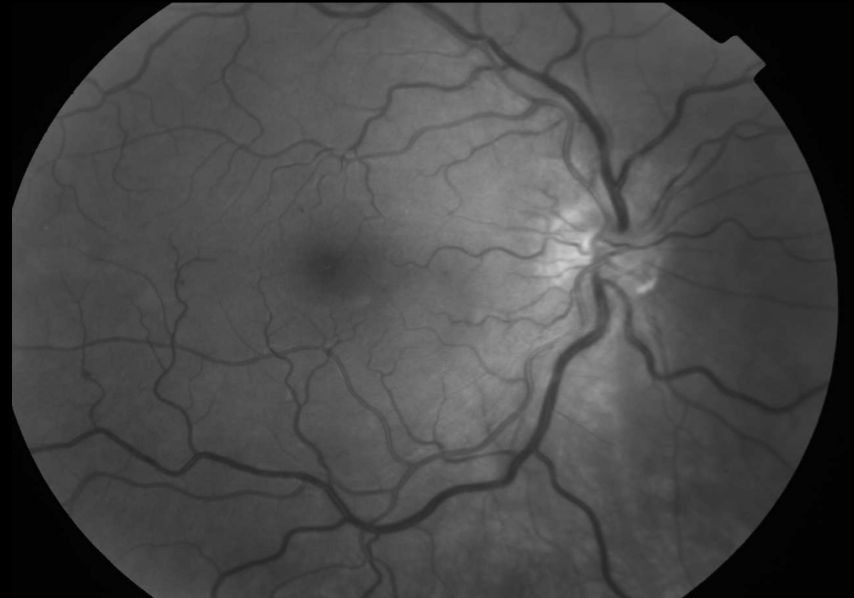




June 2003

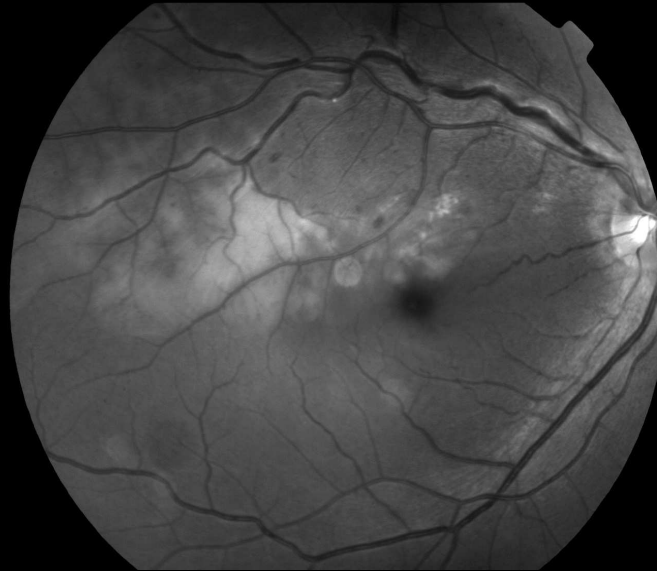


March 2004



June 2004

➤ Les h mi-occlusions peuvent  tre de type A ou B



➤ Les OBVR sont (virtuellement) toutes des types B



DU IMAGERIE ET PATHOLOGIE RETINIENNES

Evolution

➤ Quel que soit le type d' OVR, un remodelage micro vasculaire peut apparaître:

➤ Occlusion capillaire

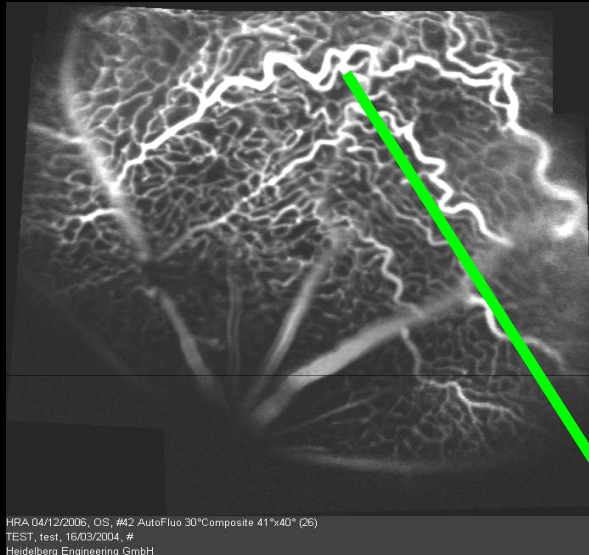
➤ Collatérales

➤ Macroanévrismes

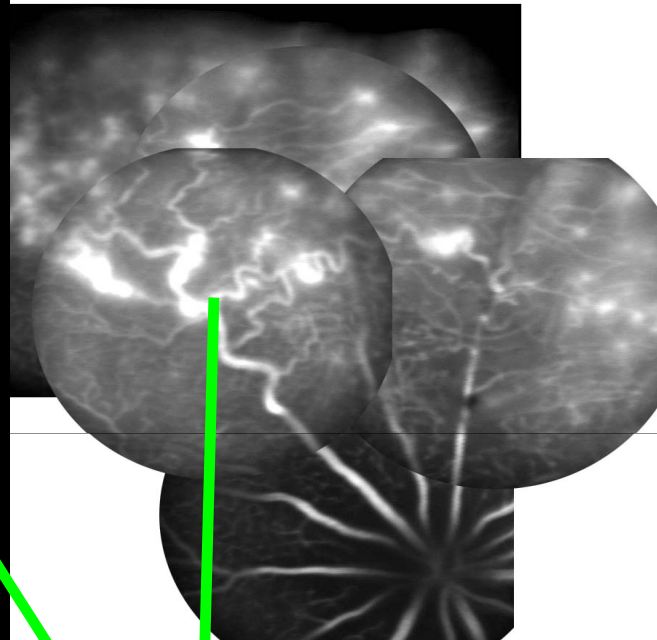
- Remodelage micro vasculaire
 - Rupture de la BHR
 - Collatérale
 - Nonperfusion

L'occlusion veineuse chez la souris peut reproduire certains aspects cliniques du RMV

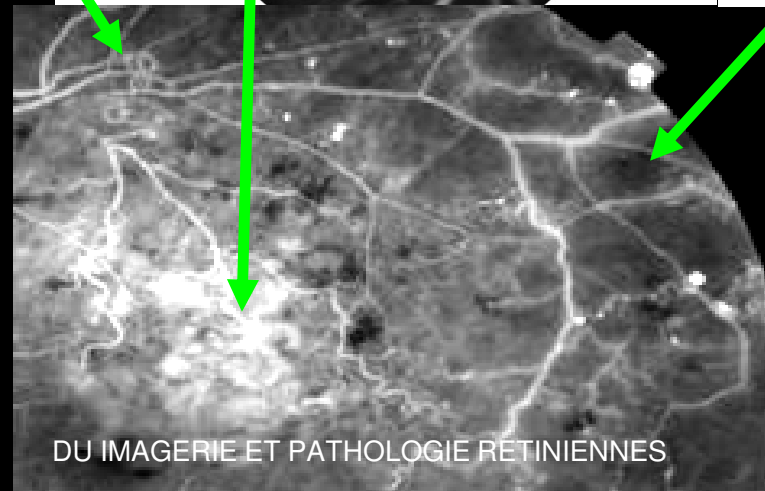
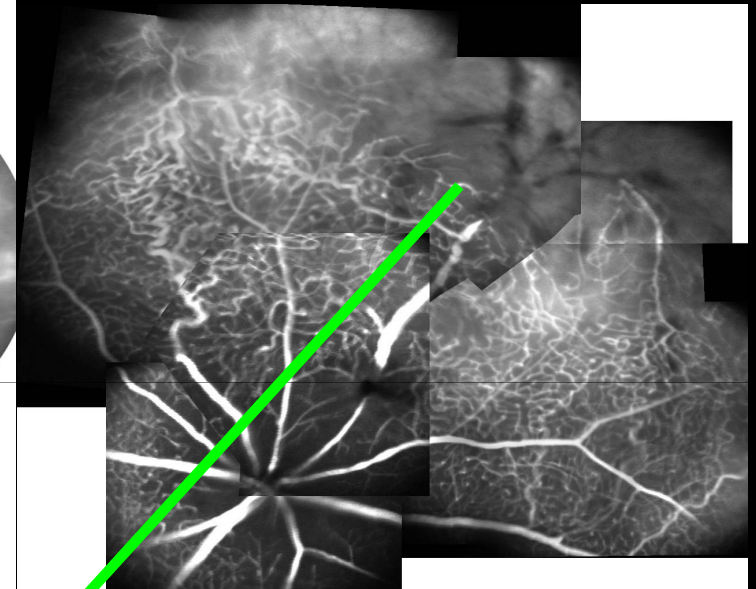
Circulation collatérale



Rupture de la BHR



Occlusion capillaire



Occlusion capillaire

➤ Définit la forme dite « non perfusée »

- En fait, la présence de capillaires occlus est très fréquente comme l' a montré l' imagerie grand champ
- Il n' y a pas de seuil défini entre « forme non perfusée » et « formes perfusées »

➤ Cause de l' occlusion capillaire?

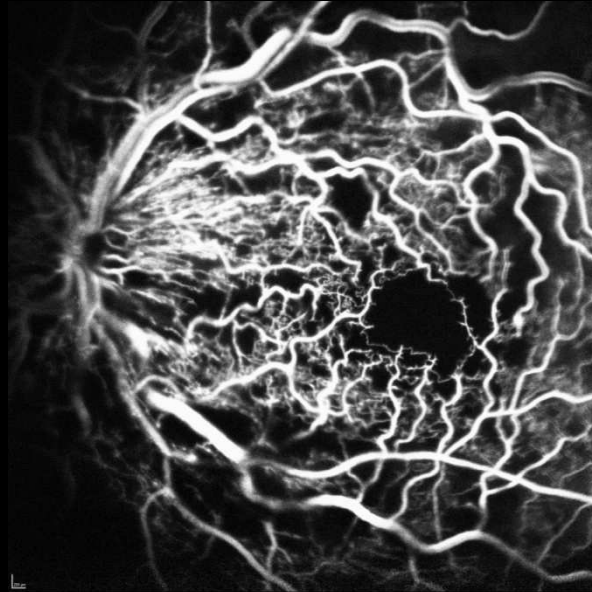
➤ Seuls facteurs de risque connus:

- Age
- Diabète

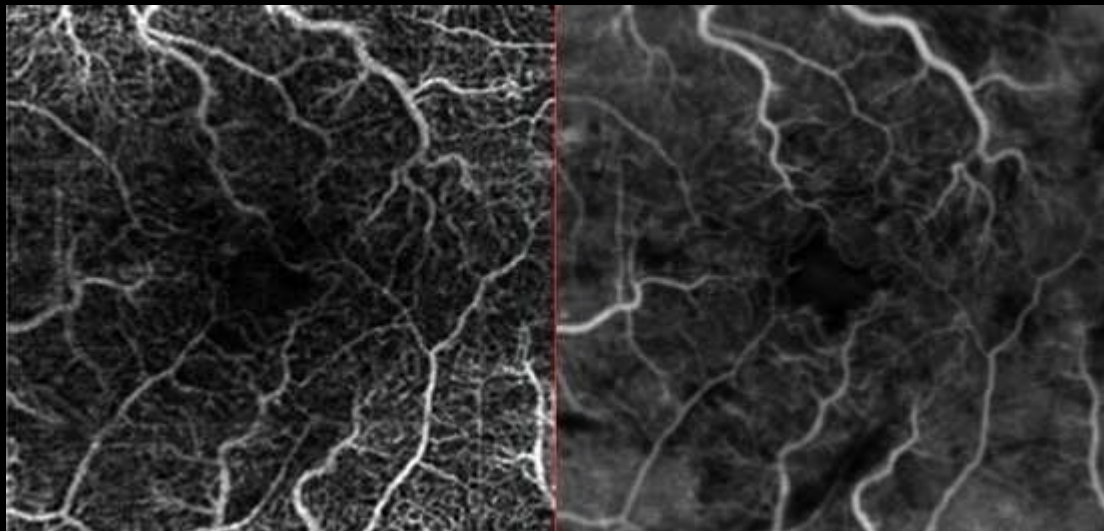
➤ Conséquences:

- Baisse acuité visuelle
- Synthèse de facteurs de croissance néo-vasculaires

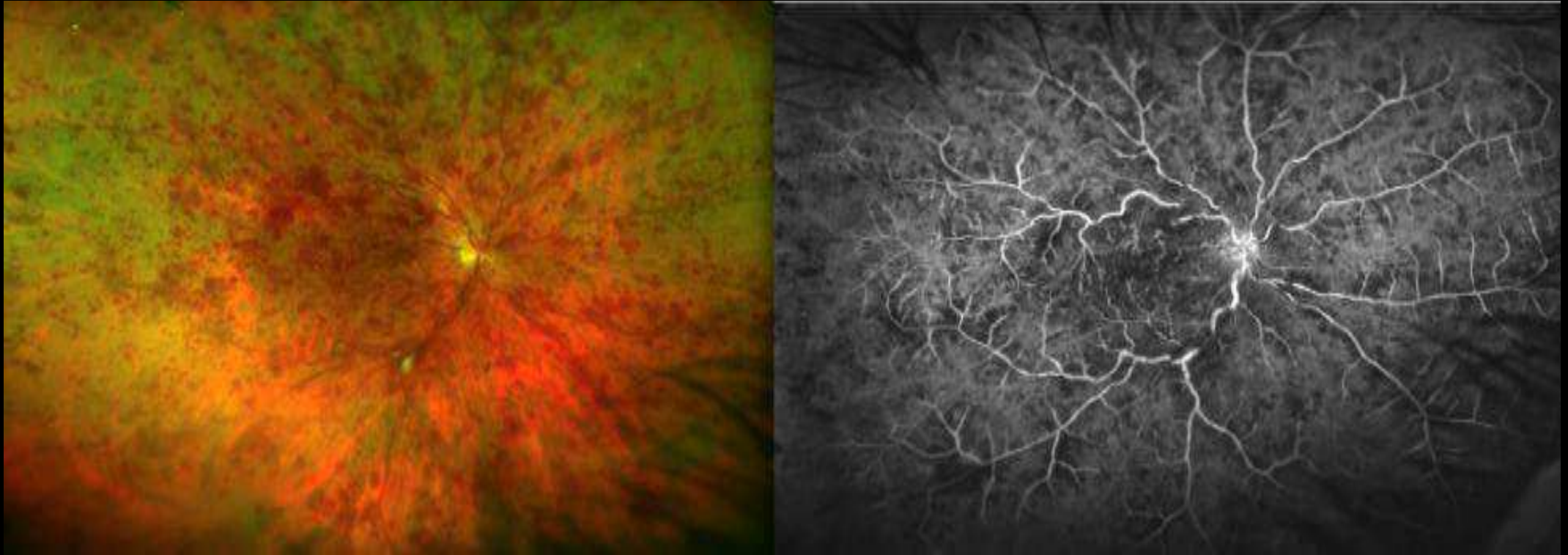
La non perfusion peut affecter la macula



16/12/2015, 08
FA 0.25 71 307 ANTIO (H)



... Mais le plus souvent commence par la périphérie



The Central Vein Occlusion Study

Evolution vers une « non perfusion étendue »

➤ 1/3 en 3 ans- Médiane 4 mois

Evolution vers une rubéose

➤ 16% (dont la moitié avaient été catégorisés initialement comme « perfusés »)

➤ 10 yeux (1,5%) ont perdu la vision par GNV non contrôlé

➔ le risque de rubéose est maximal au début de l'évolution puis décroît sans s'annuler

Les antiVEGF ne font que décaler le risque de GNV, sans le modifier à long terme

L'OCTA du pôle postérieur est corrélé à l'index d'ischémie périphérique au cours des OV

► [Ophthalmologica](#). 2022;245(3):275-284. doi: 10.1159/000522503. Epub 2022 Feb 10.

Optical Coherence Tomography Angiography in Central Retinal Vein Occlusion: Macular Changes and Their Correlation with Peripheral Nonperfusion at Ultra-Widefield Fluorescein Angiography

Michele Cavalleri ¹ ², Riccardo Sacconi ³, Mariacristina Parravano ⁴, Eliana Costanzo ⁴, Mario Pezzella ⁵, Francesco Bandello ³ ⁵, Giuseppe Querques ³ ⁵

Affiliations + expand

PMID: 35144261 DOI: 10.1159/000522503

Abstract

Introduction: The aim of this study was to investigate the correlation between ischemic index (ISI) measured on ultra-widefield (UWF) fluorescein angiography (FA) images and macular parameters obtained by optical coherence tomography angiography (OCT-A) in eyes affected by central retinal vein occlusion (CRVO).

Methods: Retrospective study of data from 12 eyes affected by treatment-naïve CRVO. All patients underwent a comprehensive ocular examination including structural OCT, OCT-A, and UWF FA.

Variables analyzed included best corrected visual acuity (BCVA) measured with the ETDRS chart; foveal avascular zone (FAZ) area at full-thickness OCT-A angiogram; perfusion density (PD) in the superficial (SCP) and deep capillary plexus (DCP); ISI; and central macular thickness (CMT).

Results: ISI showed a significant positive correlation with FAZ area ($r = 0.63$, $p = 0.019$) and a significant negative correlation with PD in the SCP ($r = -0.62$, $p = 0.022$), PD in the DCP ($r = -0.66$, $p = 0.011$), and BCVA ($r = -0.75$, $p = 0.002$). FAZ area also negatively correlated to PD in the SCP ($r = -0.75$, $p = 0.002$) and DCP ($r = -0.64$, $p = 0.016$). BCVA positively correlated to PD in the SCP ($r = 0.67$, $p = 0.009$) and DCP ($r = 0.68$, $p = 0.008$), while a negative correlation was found with FAZ area ($r = -0.65$, $p = 0.013$) and CMT ($r = -0.70$, $p = 0.006$).

Discussion/conclusion: OCT-A macular parameters (namely, FAZ area and PD of SCP and DCP) significantly correlated with ISI, a quantitative way to assess peripheral retinal nonperfusion on UWF FA. Macular OCT-A analysis may help in assessing the need for additional UWF FA testing in eyes affected by CRVO.

► [Am J Ophthalmol](#). 2021 May;225:57-68. doi: 10.1016/j.ajo.2020.12.026. Epub 2021 Jan 4.

Macular Microvascular Changes and Their Correlation With Peripheral Nonperfusion in Branch Retinal Vein Occlusion

Gahyung Ryu ¹, Donggeun Park ¹, Jinam Lim ¹, Jano van Hemert ², Min Sagong ³

Affiliations + expand

PMID: 33412121 DOI: 10.1016/j.ajo.2020.12.026

Abstract

Purpose: To investigate the correlation between macular microvascular alterations on optical coherence tomography angiography (OCTA) and retinal ischemia on ultra-widefield fluorescein angiography (UWF FA) in eyes with branch retinal vein occlusion (BRVO).

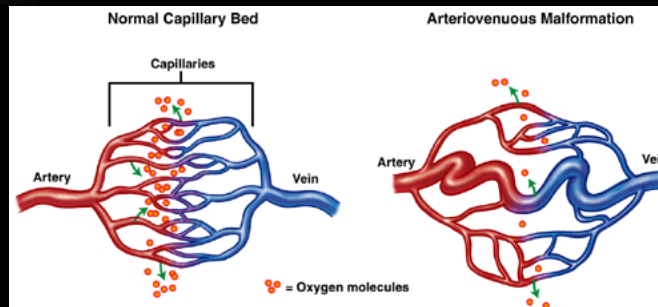
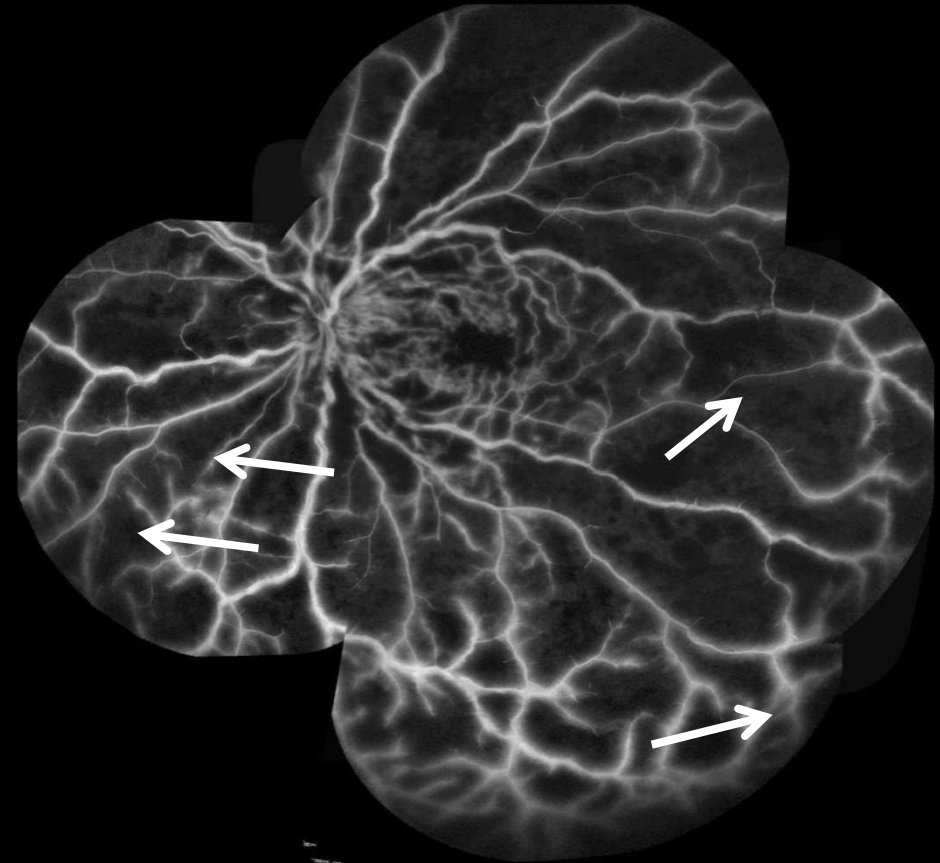
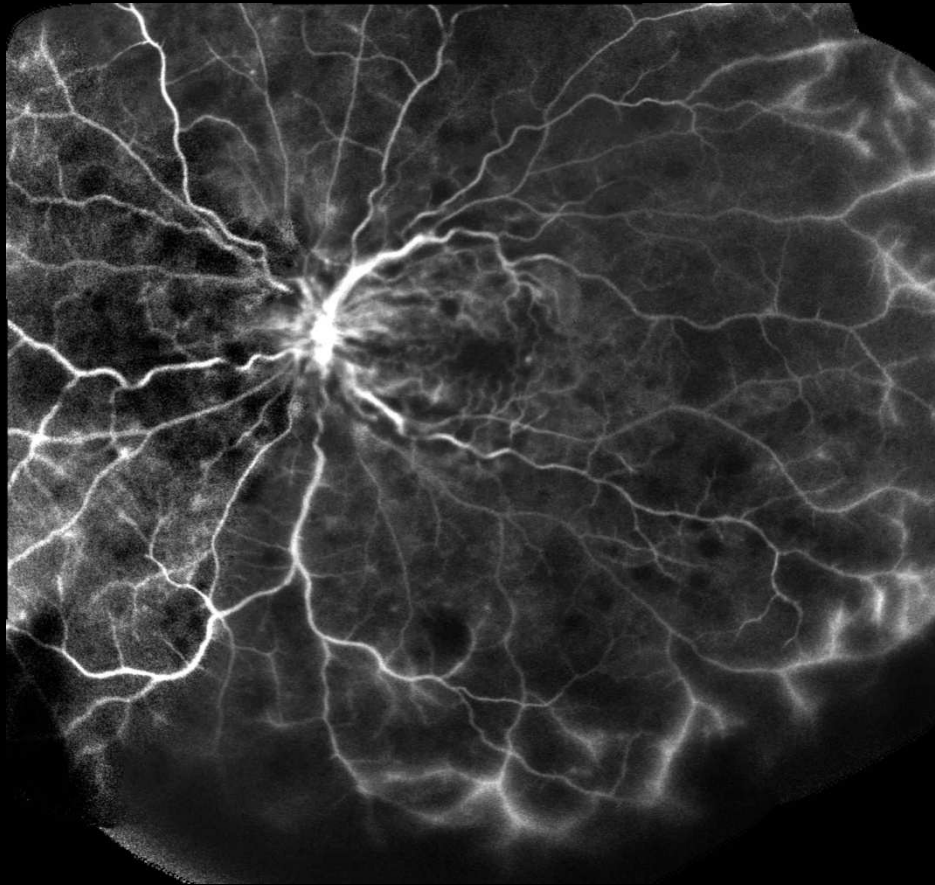
Design: Cross-sectional study.

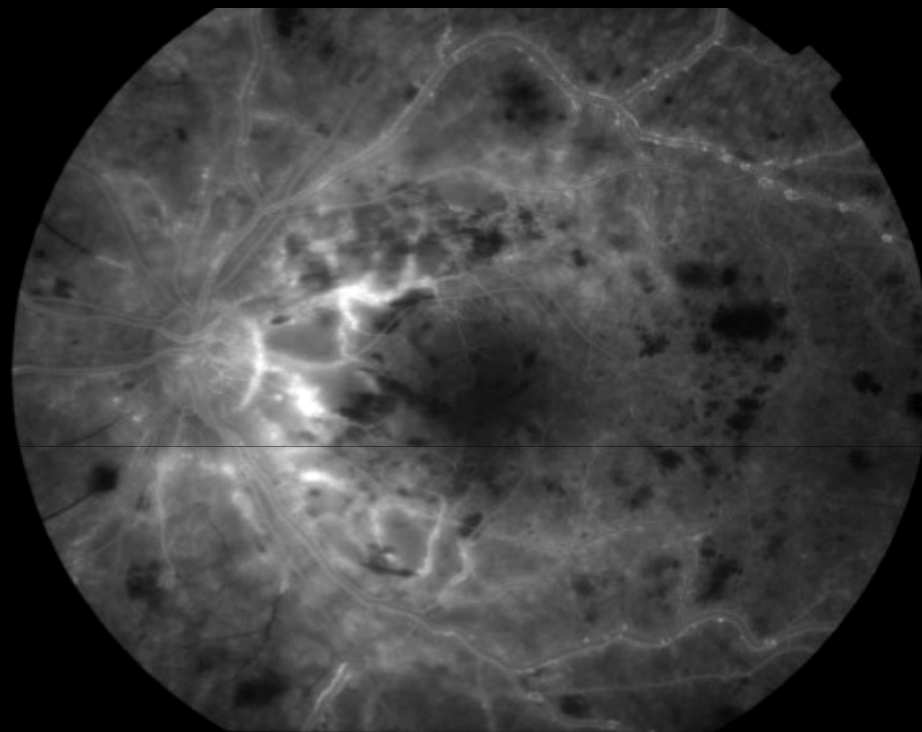
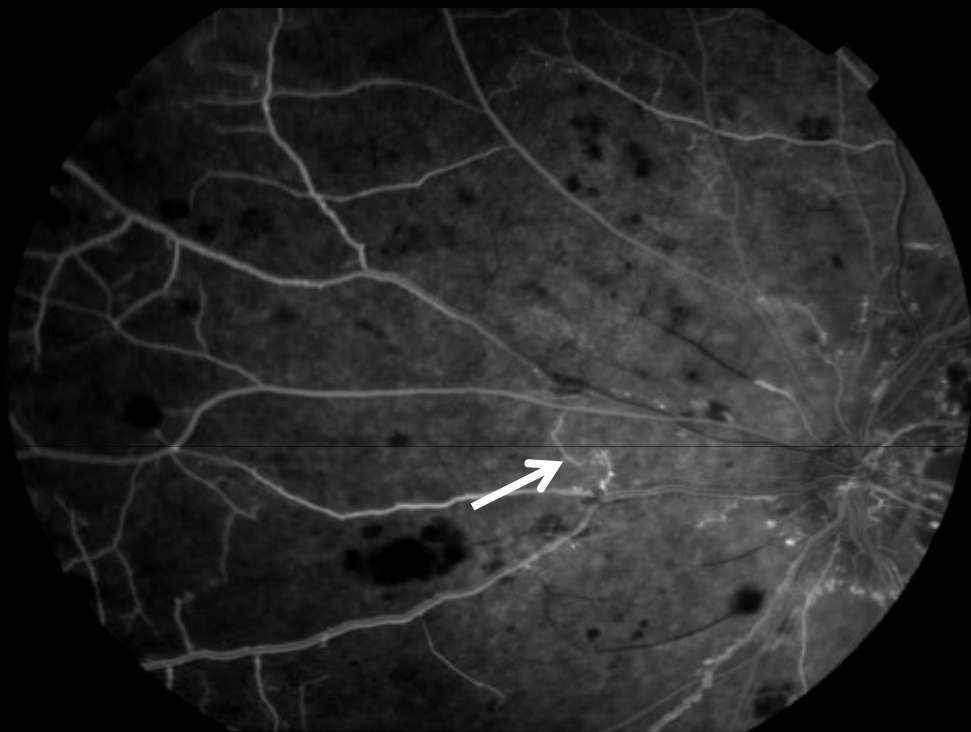
Methods: This prospective study was performed from September 2019 to June 2020 at Yeungnam University Medical Center. We included 60 patients with treatment-naïve BRVO. Two independent, masked graders analyzed OCTA parameters, including vessel density, skeletal density, and fractal dimension (FD), and UWF FA parameters, including retinal nonperfusion area (NPA) and ischemic index (ISI), from various concentric regions (perimacular region, 0.5-3 mm radius; near-peripheral region, 3-10 mm; midperipheral region, 10-15 mm; far-peripheral region, >15 mm). A repeated-measures analysis of variance test and a paired t test were performed for inter-visit and inter-regional comparisons, and Pearson correlation coefficient and multivariate regression analyses were performed to examine the correlation between UWF FA and OCTA parameters.

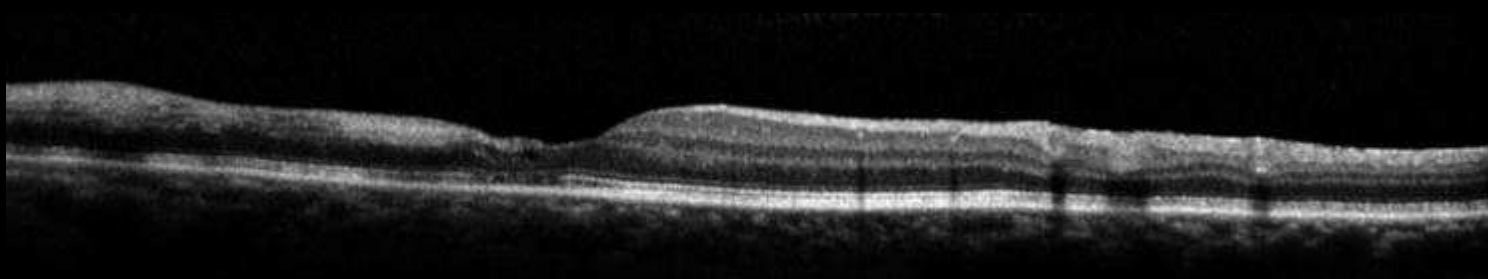
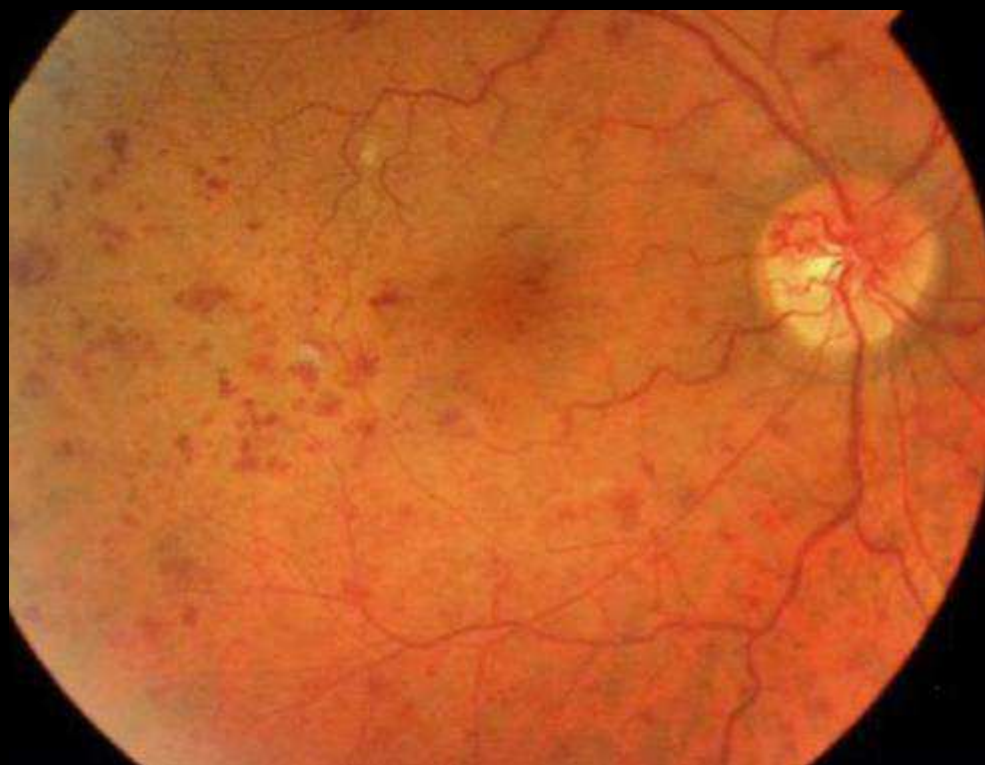
Results: The OCTA parameters from both the superficial and deep capillary plexuses (DCP) were significantly correlated with NPA and ISI in all concentric regions. Even after adjusting for several covariates, all OCTA parameters revealed a significant association with ISI on UWF FA. Moreover, OCTA parameters from DCP were significantly correlated with concentrations of placental growth factor and vascular endothelial growth factor. Although all OCTA parameters achieved excellent results of area under the curve (AUC) > 0.9 for detecting severe retinal ischemia, defined as ISI >10%, FD reduction in DCP was the most reliable parameter (AUC = 0.948, $P < .001$), and 5.39% was the best cut-off point for predicting ISI > 10%.

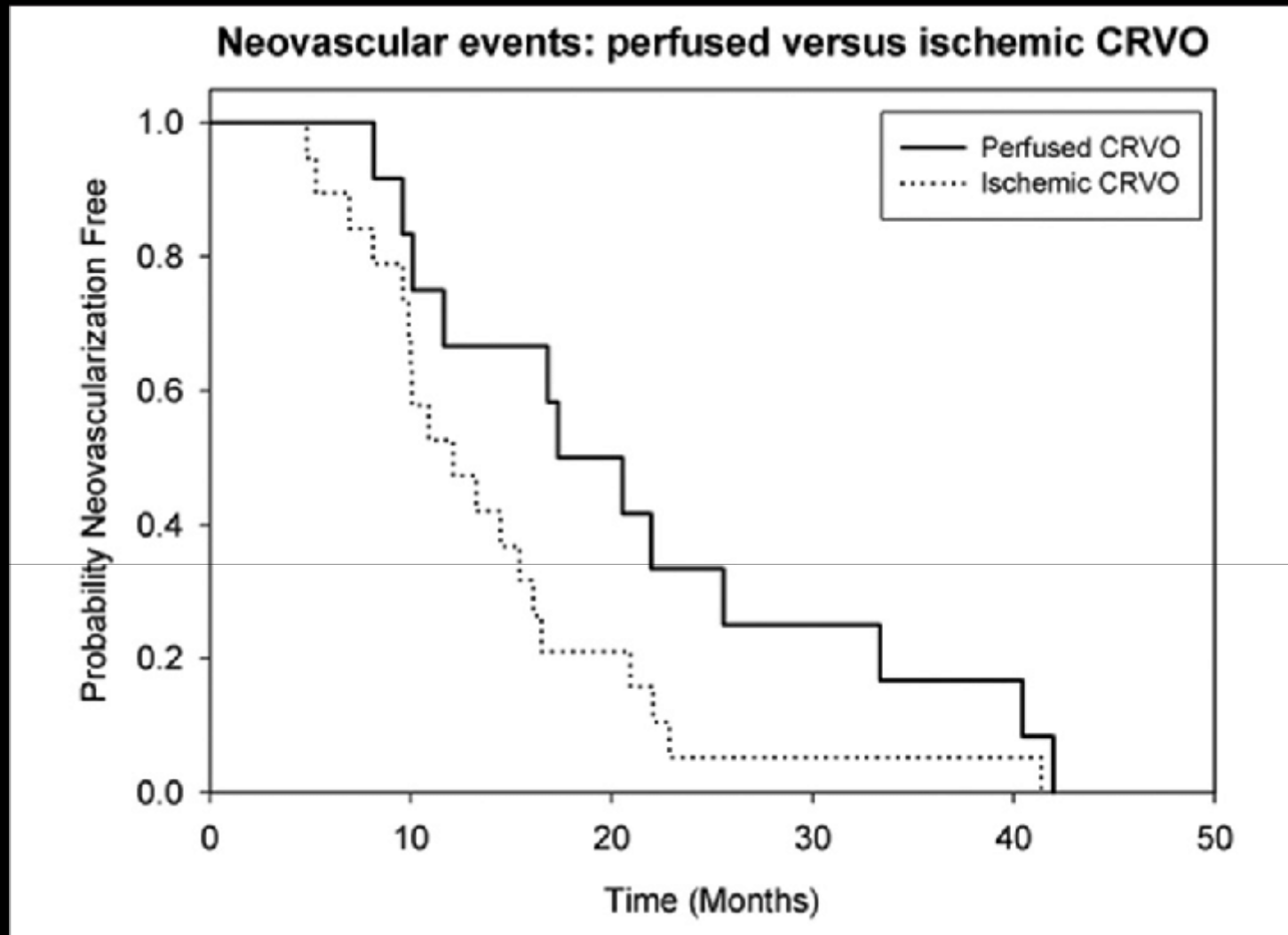
Conclusions: OCTA is a useful noninvasive tool not only for evaluation of macular microvasculature but for supposition of peripheral nonperfusion in eyes with BRVO.

Développement de nonperfusion capillaire: un effet shunt?









DeCroos FC, et al. Neovascular events in eyes with central retinal vein occlusion undergoing serial bevacizumab or ranibizumab intravitreal injections: a retrospective review. J Ophthalmic Vis Res. 2014 Oct-Dec;9(4):461-8.

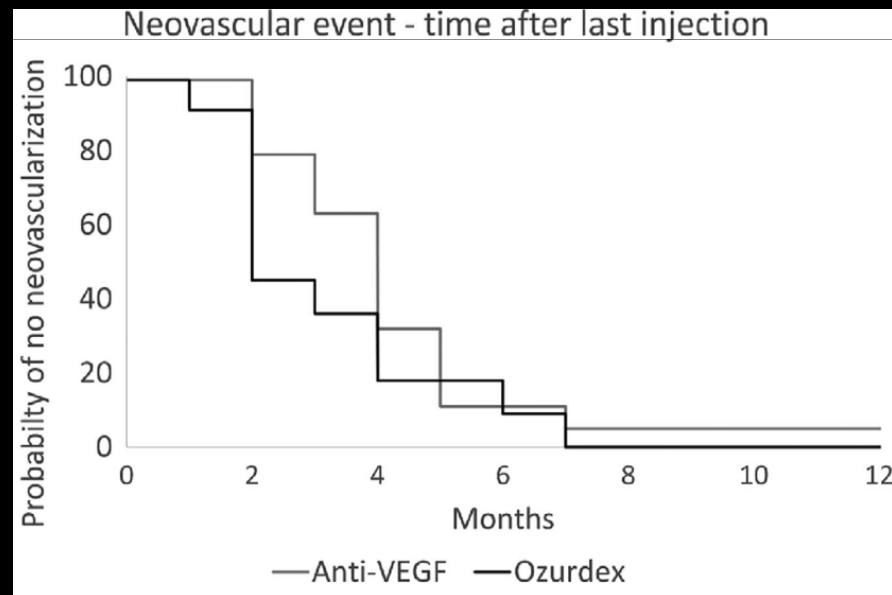
Casselhom de Salles Acta Ophthalmologica 2014

Retrospective real-life study

72 patients. Twenty-eight eyes (39%) developed NE after discontinuation of intravitreal therapy for macular oedema (ME).

NE occurred 15.6 ± 13.8 months after the baseline visit and 4.1 ± 2.6 months after the last injection.

Final best-corrected visual acuity was 8.6



L'acuité visuelle et l'OCT sont des excellents indicateurs de la présence d'une non perfusion étendue

- Sans doute parce qu'une non perfusion étendue affecte la macula au moins à un certain degré
- Une acuité visuelle $> 2/10$ est peu souvent compliquée d'une rubéose
- Au contraire, une acuité à « voit bouger la main » est beaucoup plus à risque de développer une rubéose
- Et une non perfusion s'accompagne d'une atrophie

Néovascularisation des OVR

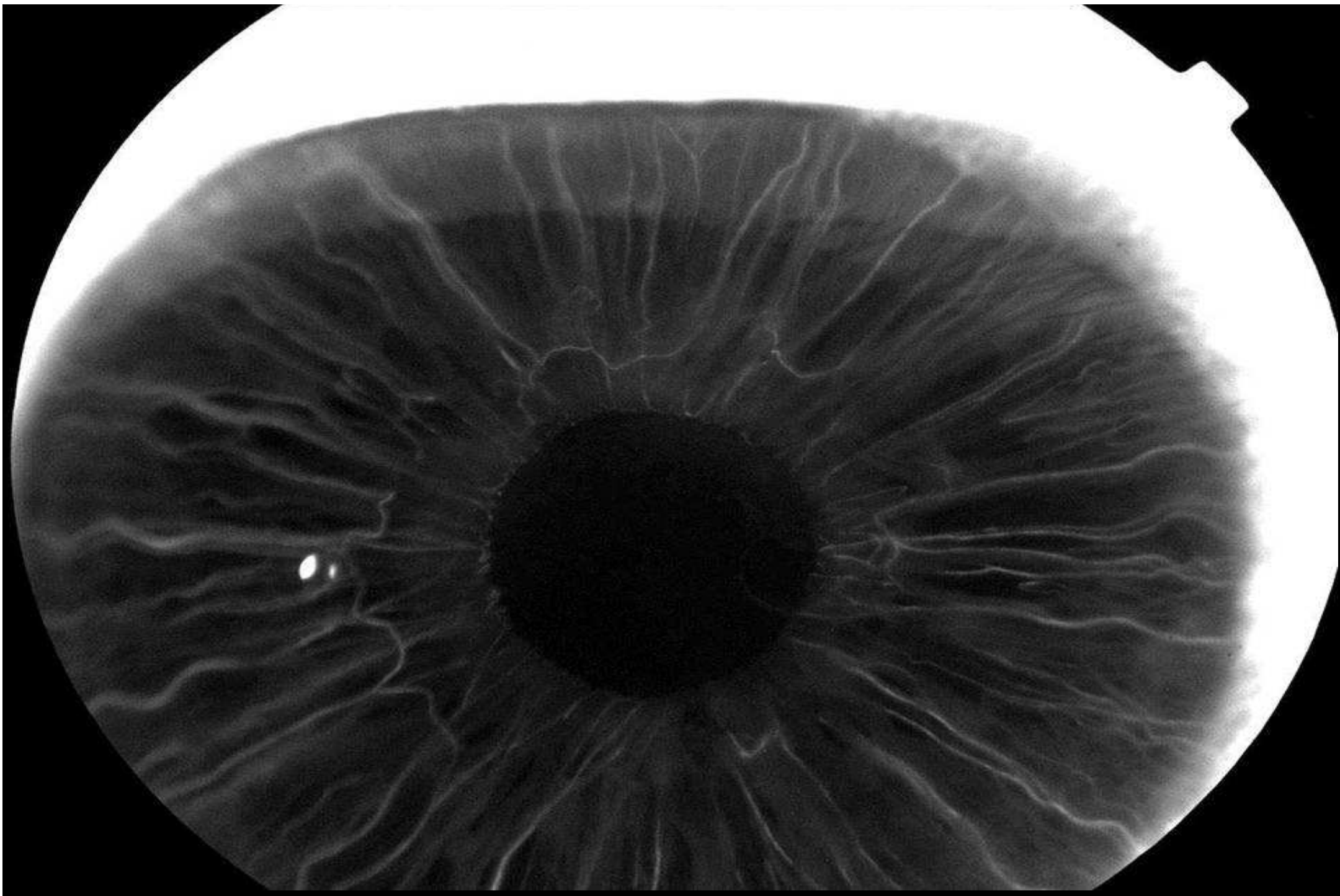
Néovaisseaux:

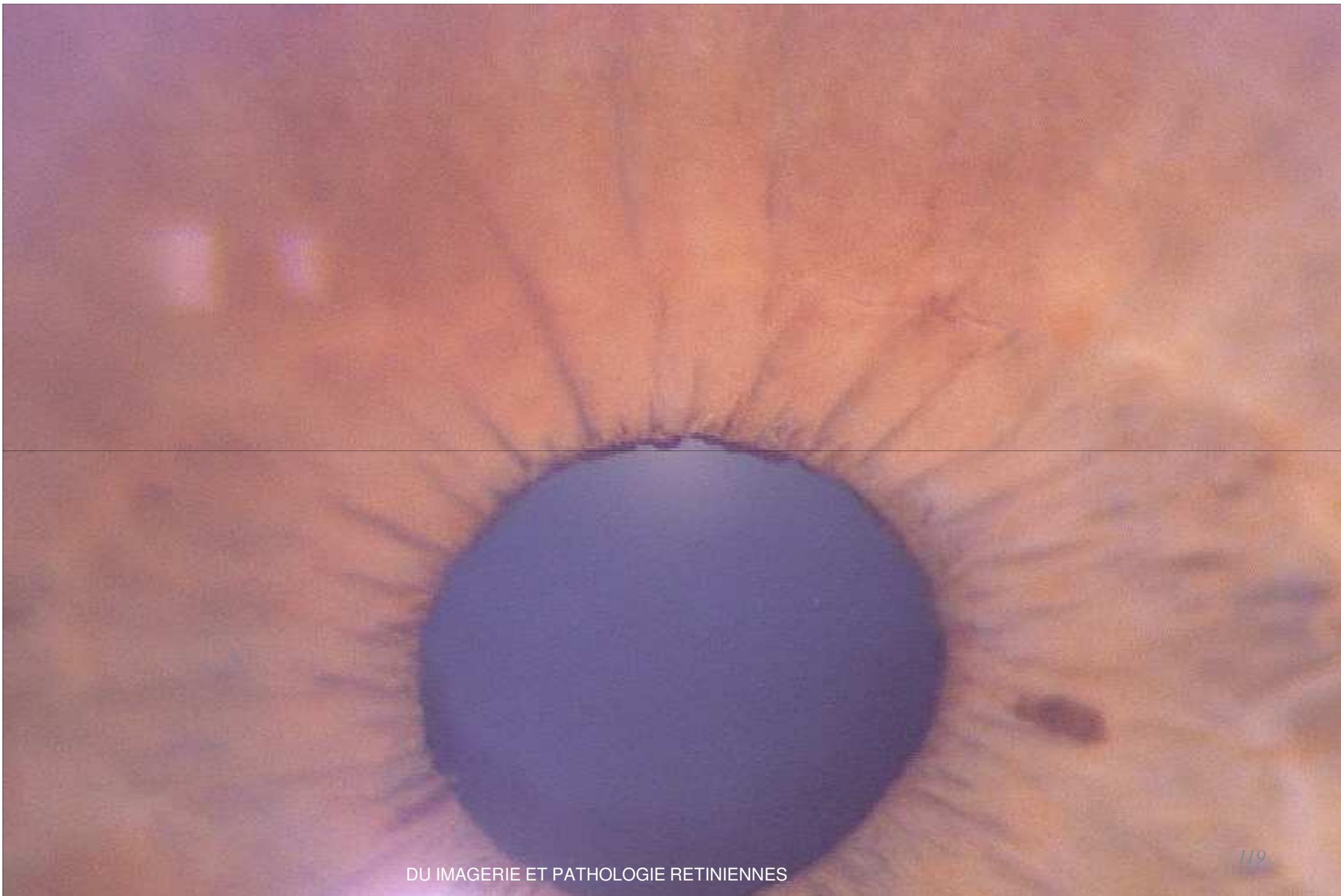
- Prépapillaires

- Prérétiniens

- Iriens ++ (risque de glaucome néovasculaire)

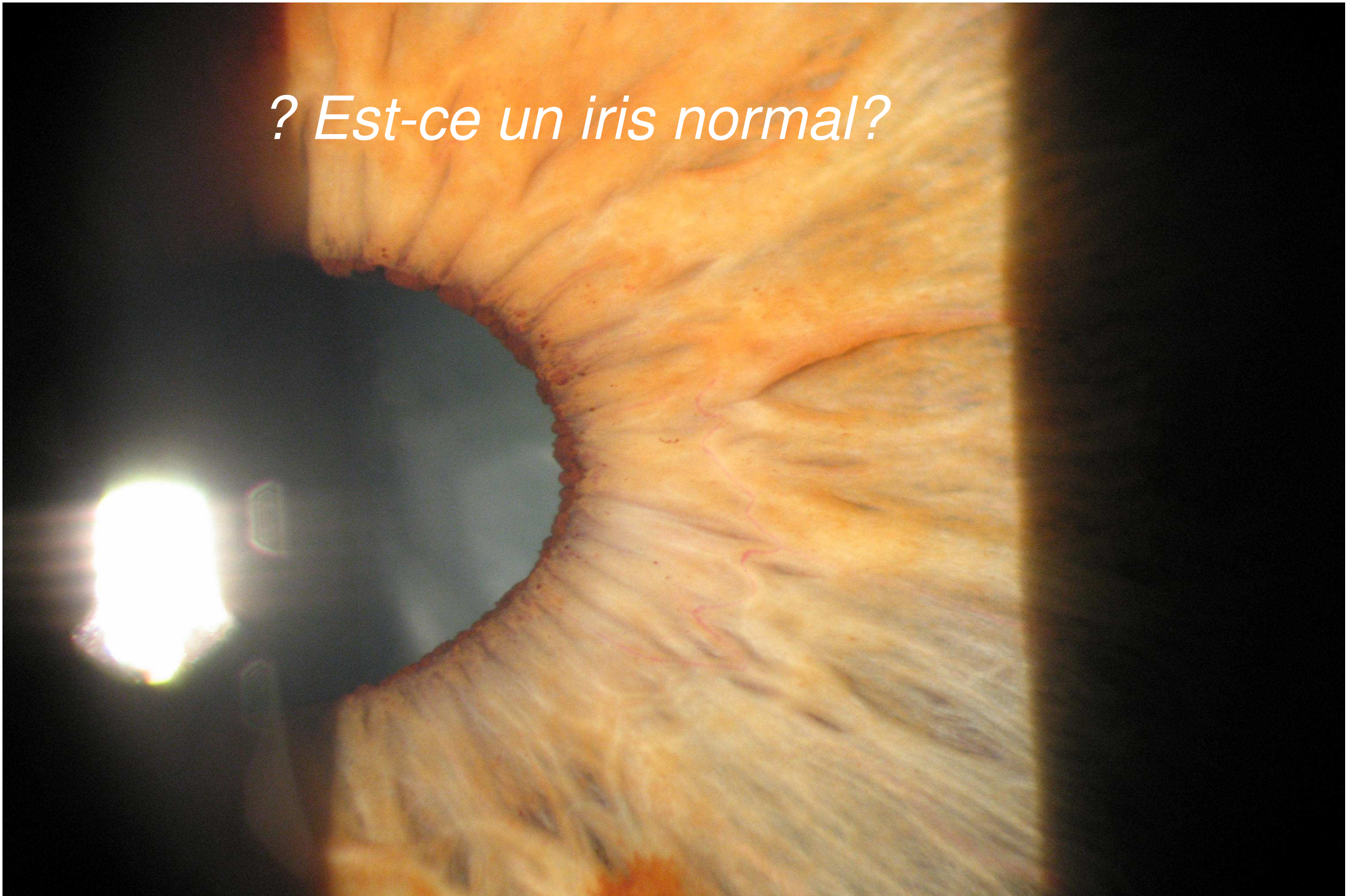
 - Seulement si OVCR



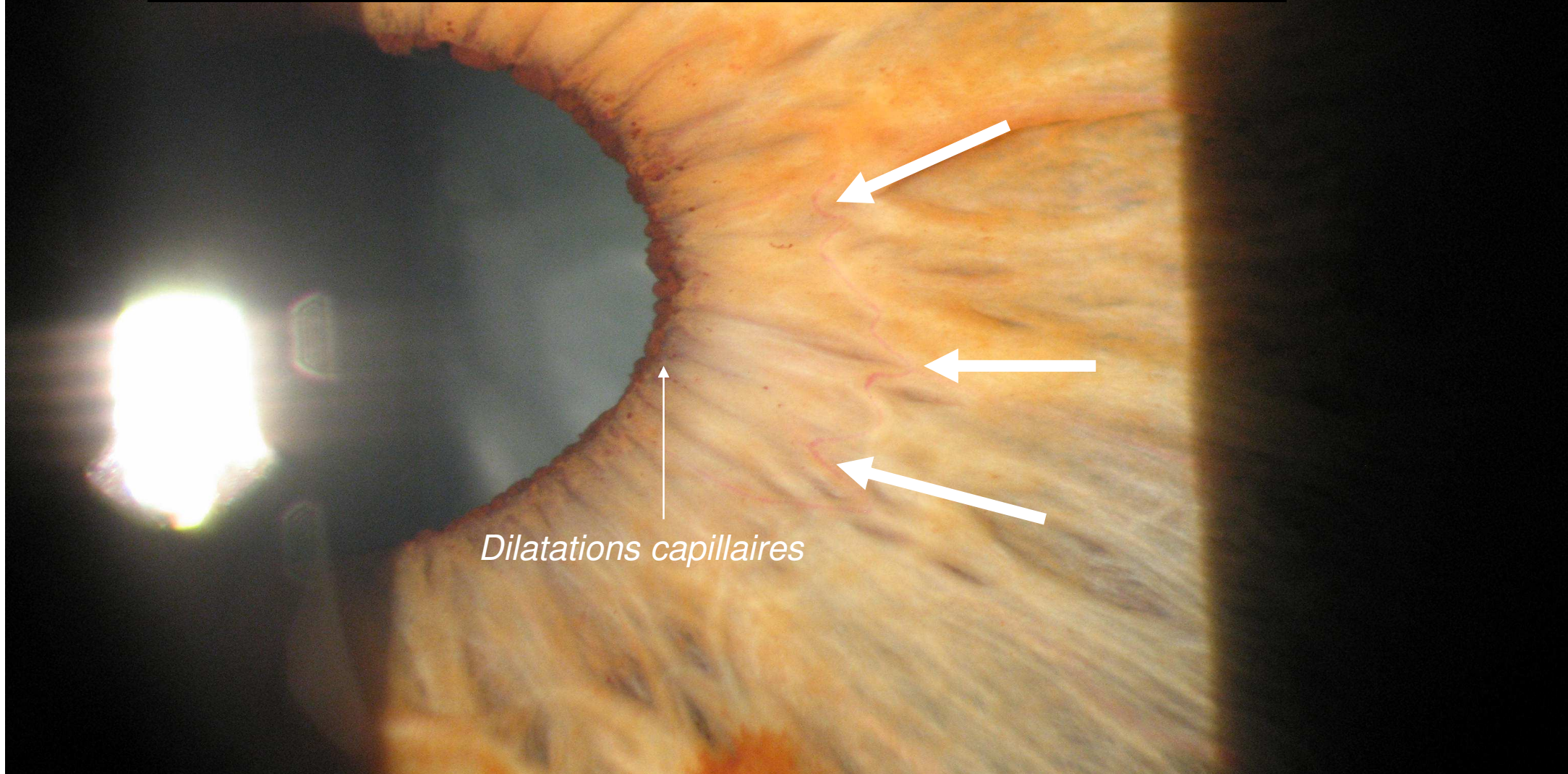




? *Est-ce un iris normal?*



*? Est-ce un iris normal?
Non, car reperfusion de la collerette*





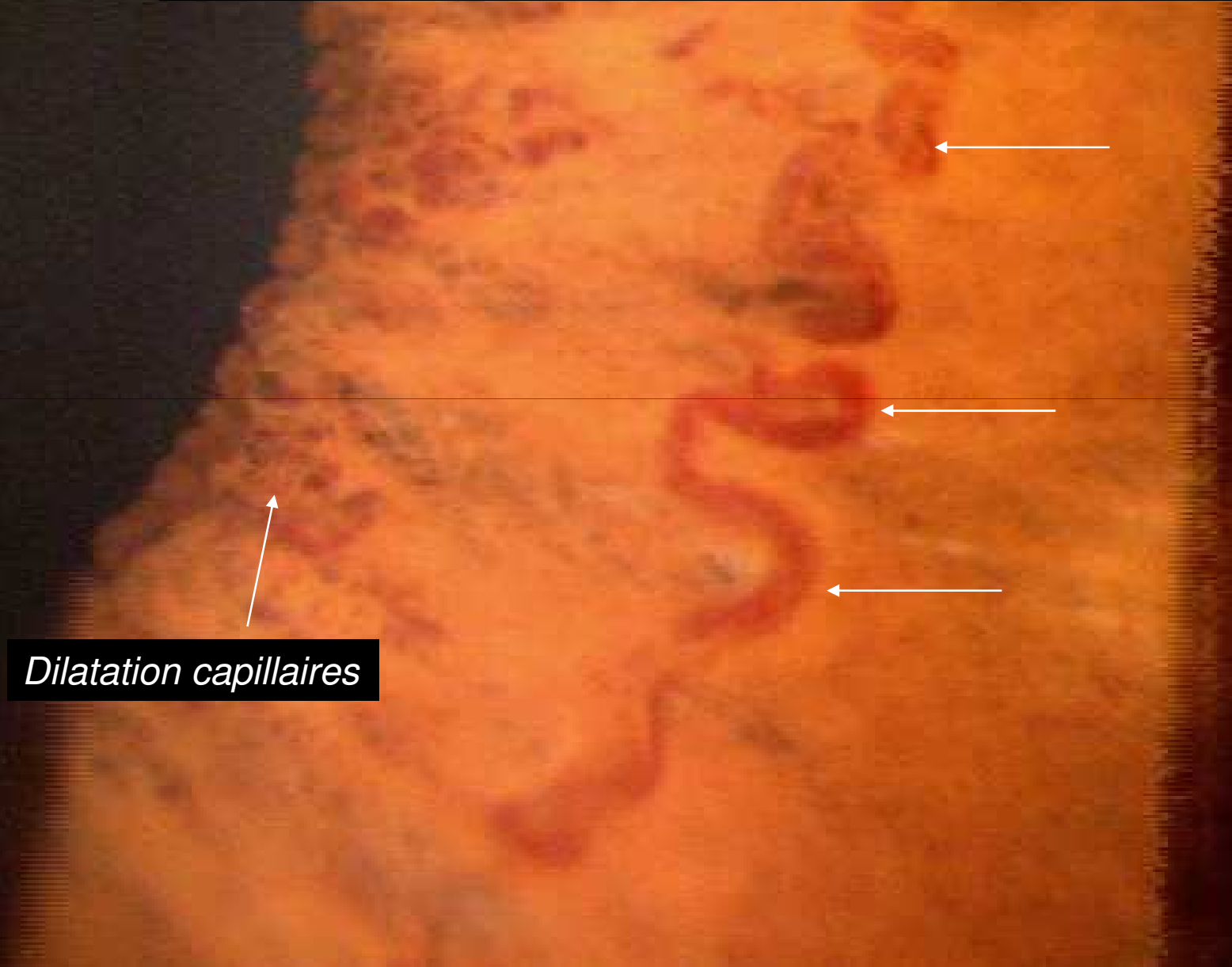
Dilatation vasculaire irienne



Pour rechercher une rubéose irienne,
il faut un iris NON dilaté
et un FORT grandissement

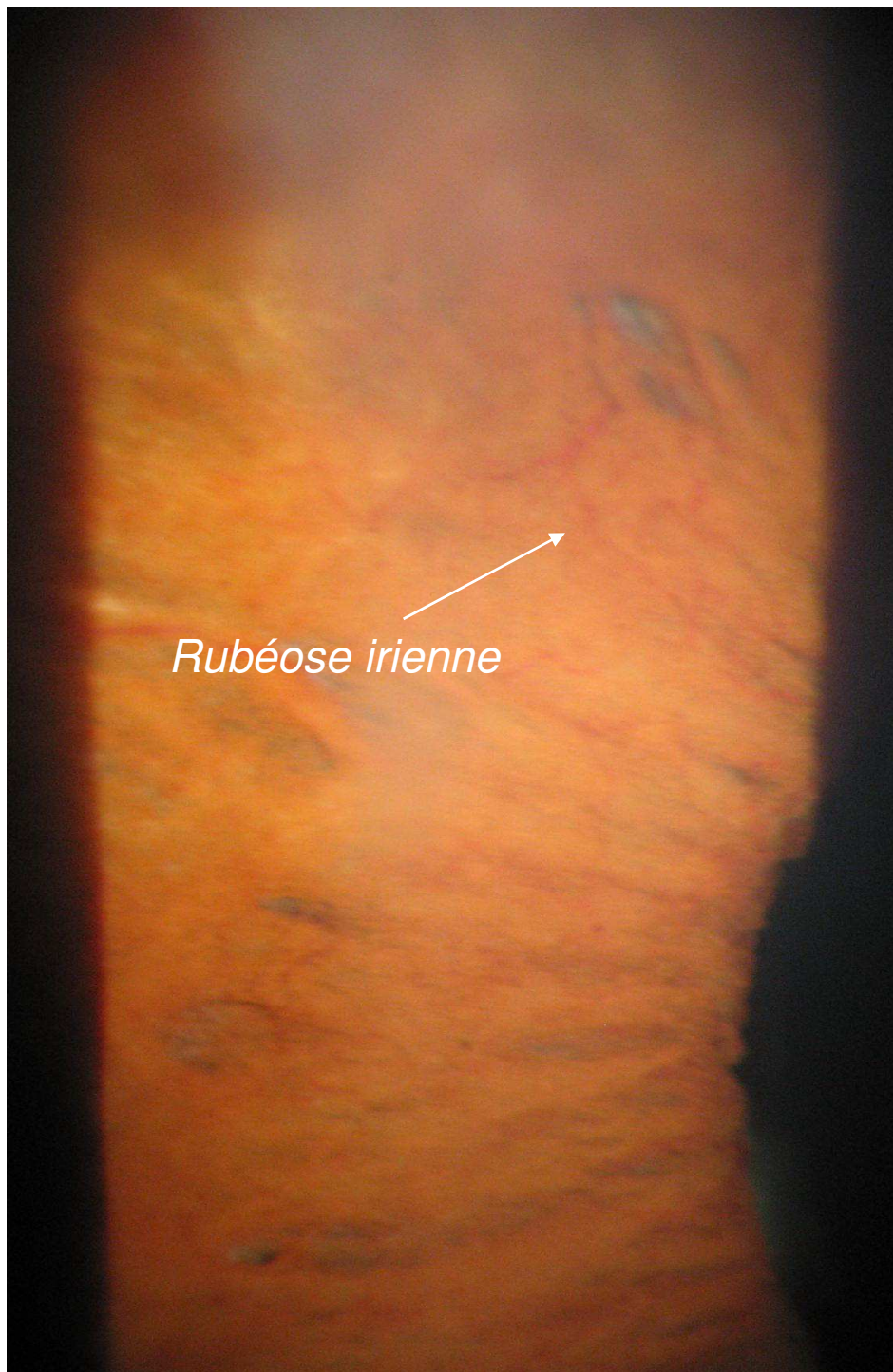
*Reperfusion très avancée de la collerette
(ce n'est pas une néovascularisation!)*

Dilatation capillaires



iridodilatation irienne sur iris pigmenté





Rubéose irienne



Néovaisseaux dans l'angle

Attention aux pièges
la rubéose peut commencer dans l'angle

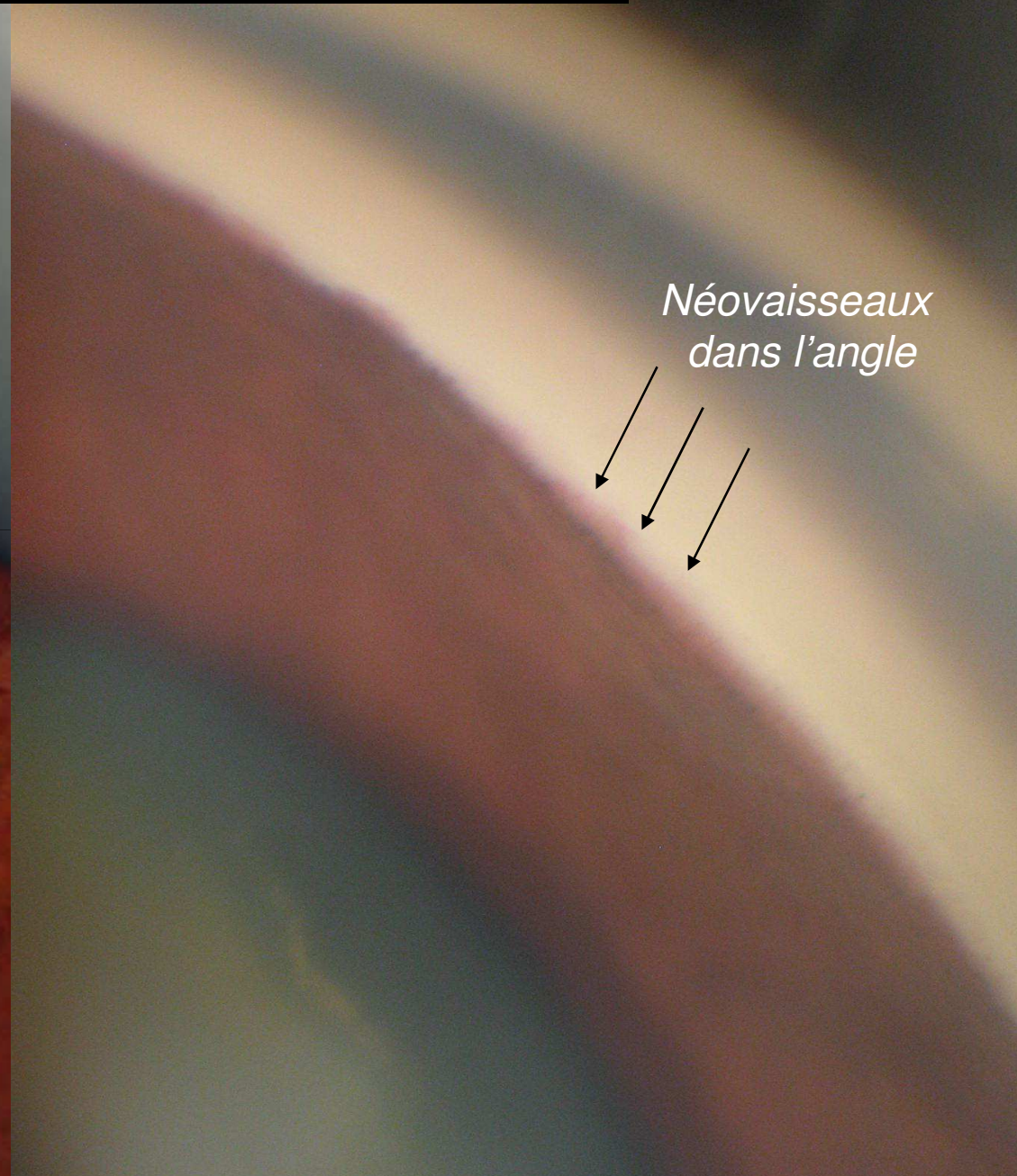
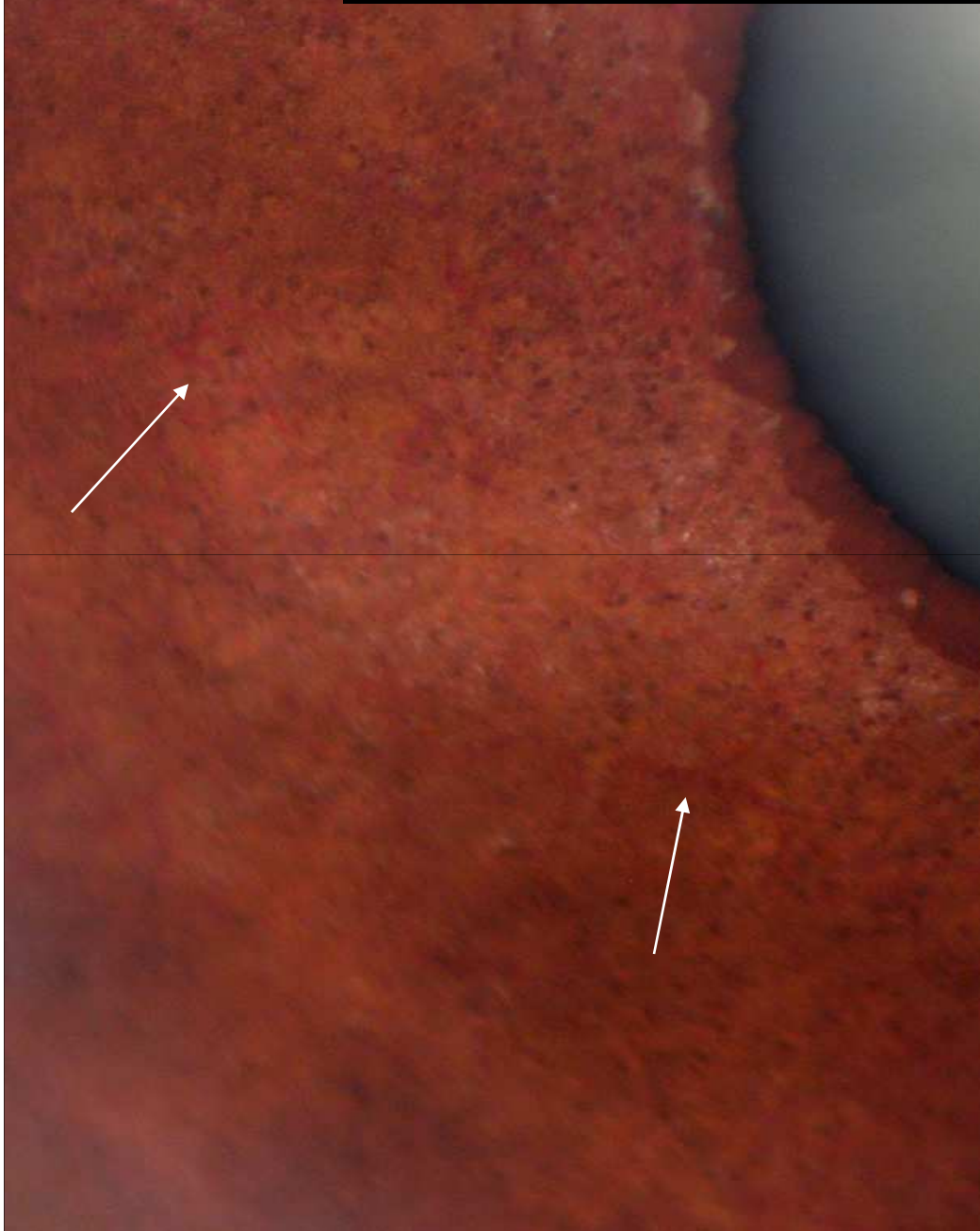


Vasodilatation irienne



Néovaisseaux dans l'angle

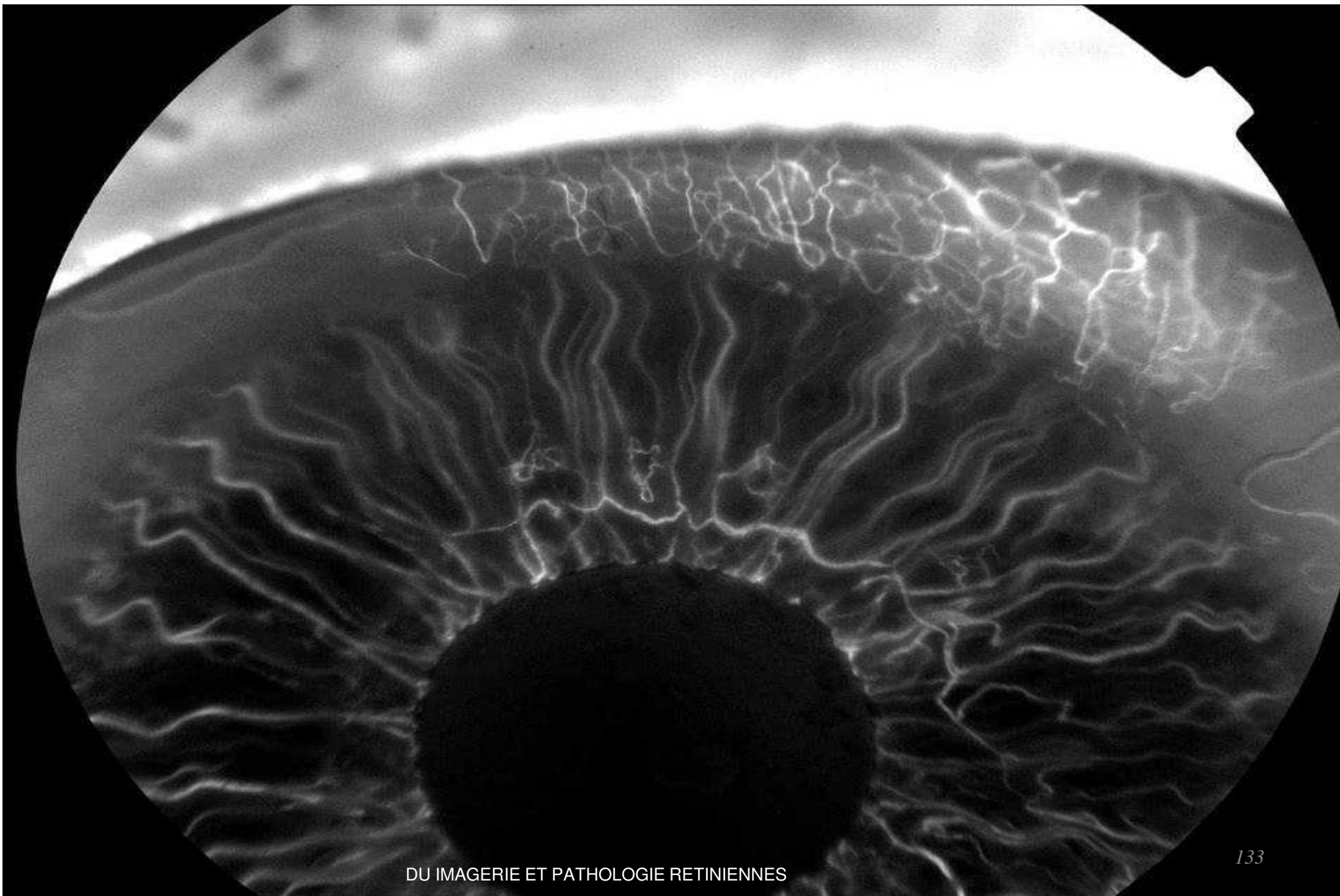
Examen d'un iris très pigmenté

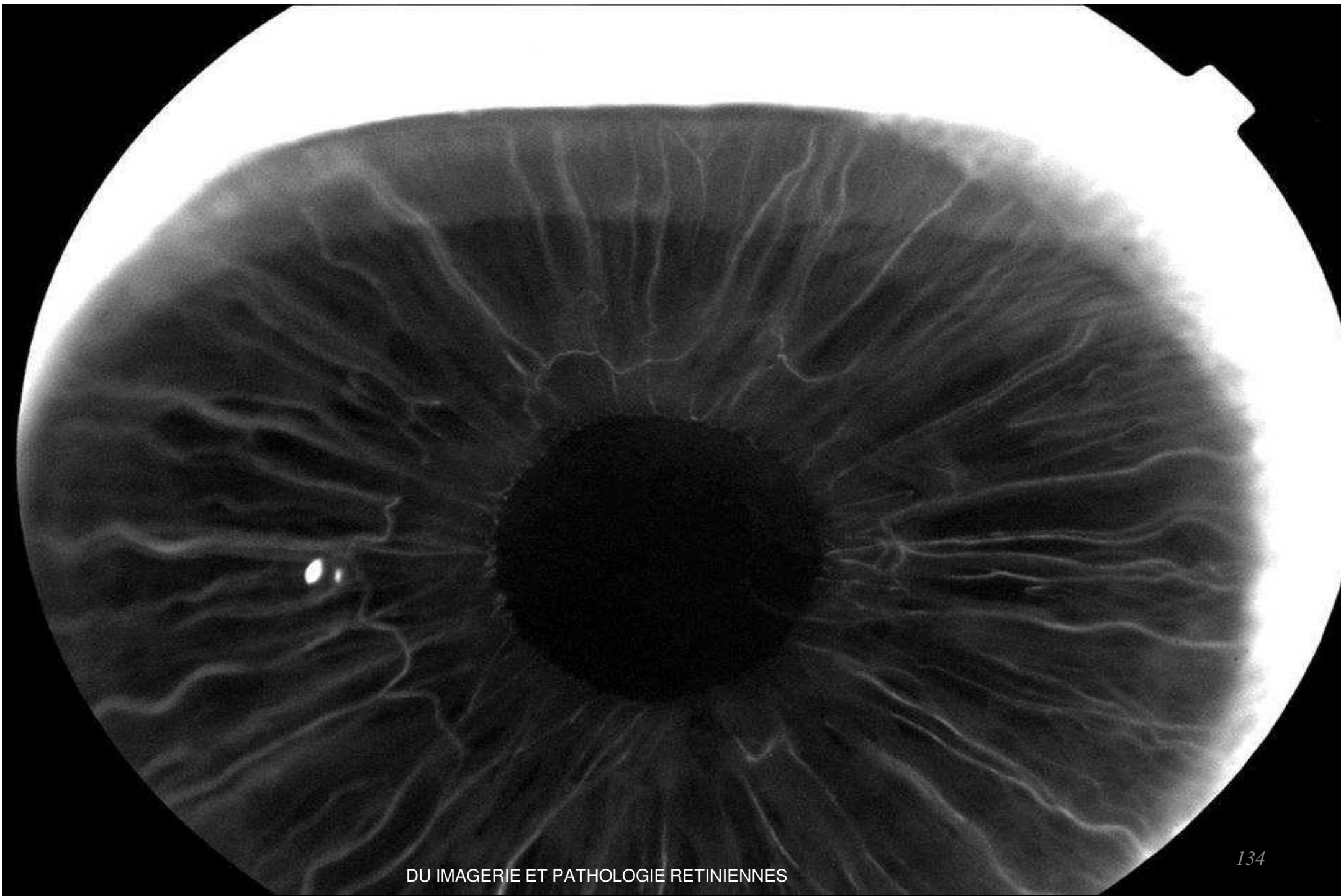


*Néovaisseaux
dans l'angle*









Reperfusion de la collerette irienne

Le cercle artériel mineur de l'iris

- Participe à la vascularisation de la membrane pupillaire fœtale
- Régresse en périnatal
- Reliquat : collerette irienne

Cependant, persistance d'une perfusion segmentaire par la fluorescéine

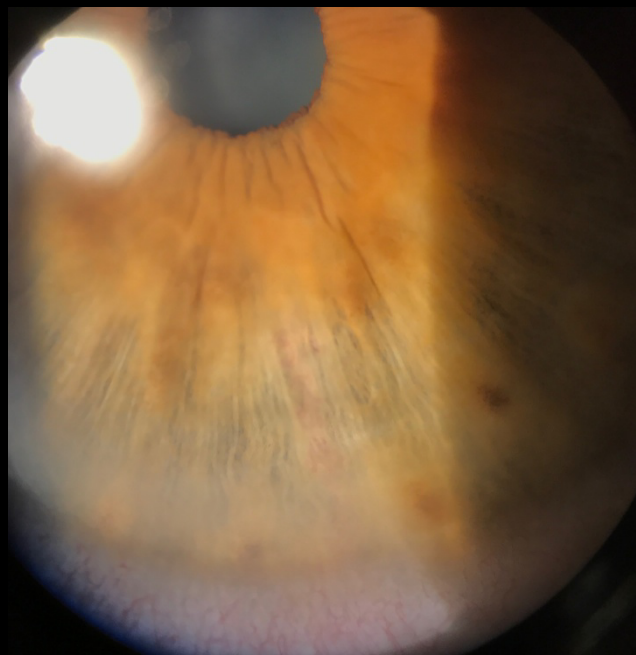
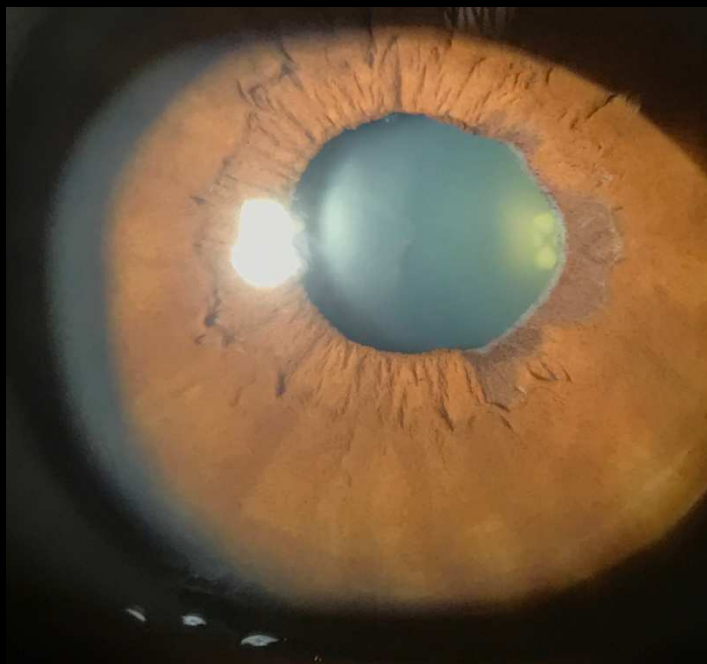
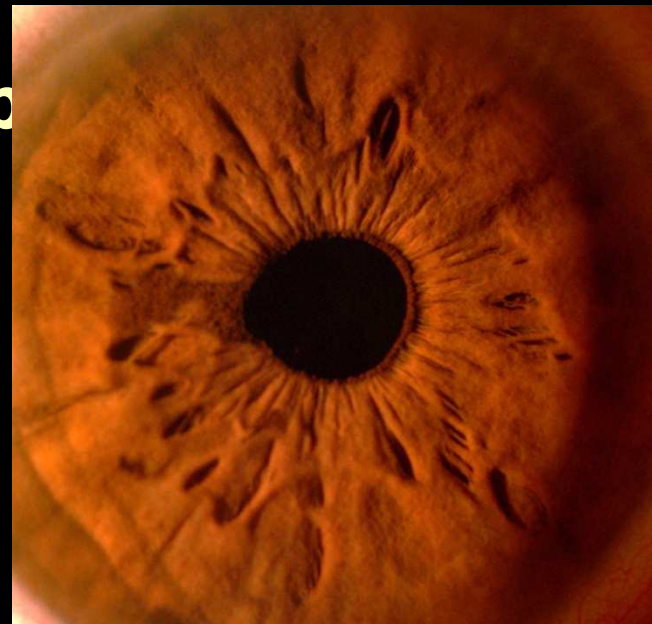
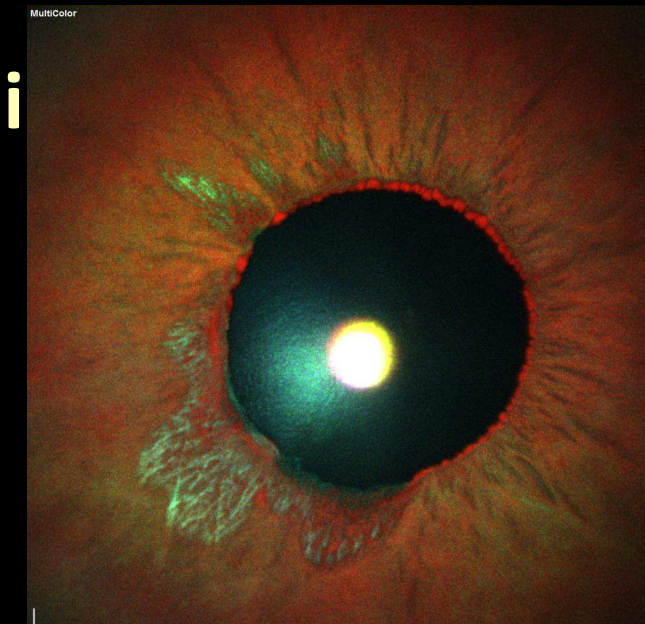
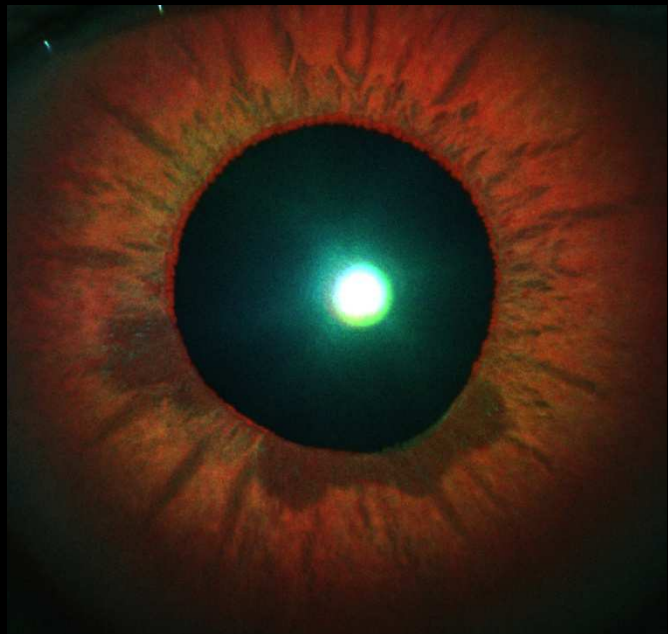
- Perfusion par plasma seul

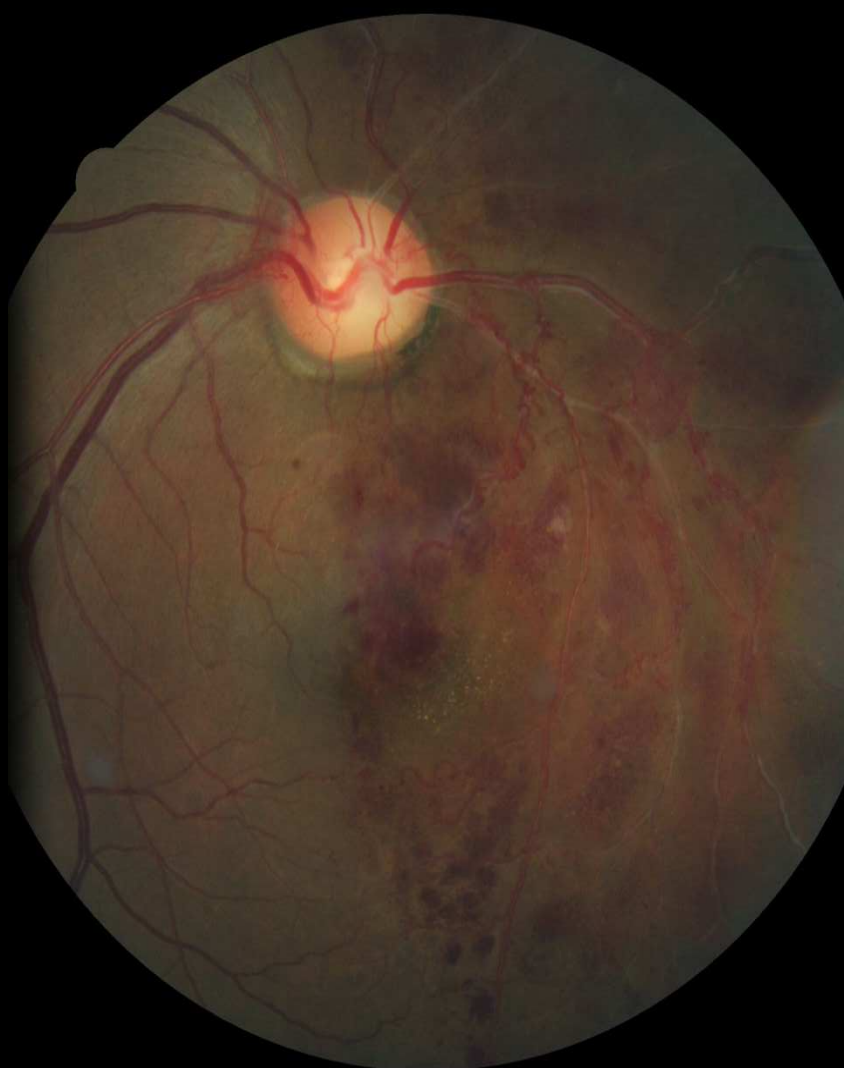
Hayreh 1978

L'iris est un biosenseur de la présence de facteurs angiogéniques

L'ischémie rétinienne quelle qu'en soit l'origine, peut entraîner

- Diffusion antérieure de facteurs vasodilatateurs et angiogéniques
 - VEGF
 - Boyd 2002
- Dilatation des vaisseaux stromaux
- Augmentation de la pression de perfusion de segments de la collerette déjà perfusés
- Signe précoce de vasodilatation des vaisseaux iriens, pouvant précéder la rubéose
- (≠ néovaisseaux)
- Également rencontrée au cours rétinopathie diabétique, inflammation intraoculaire...

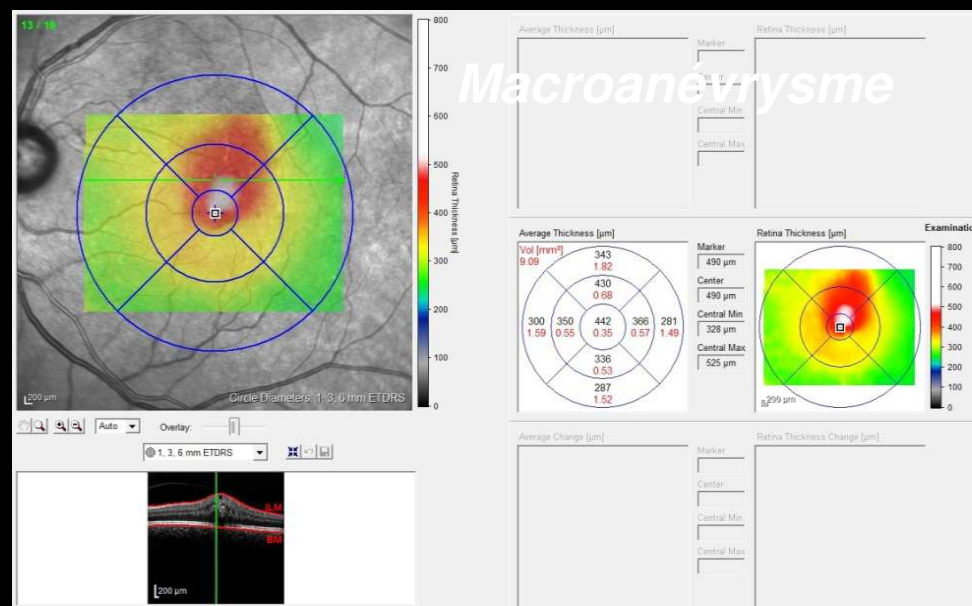
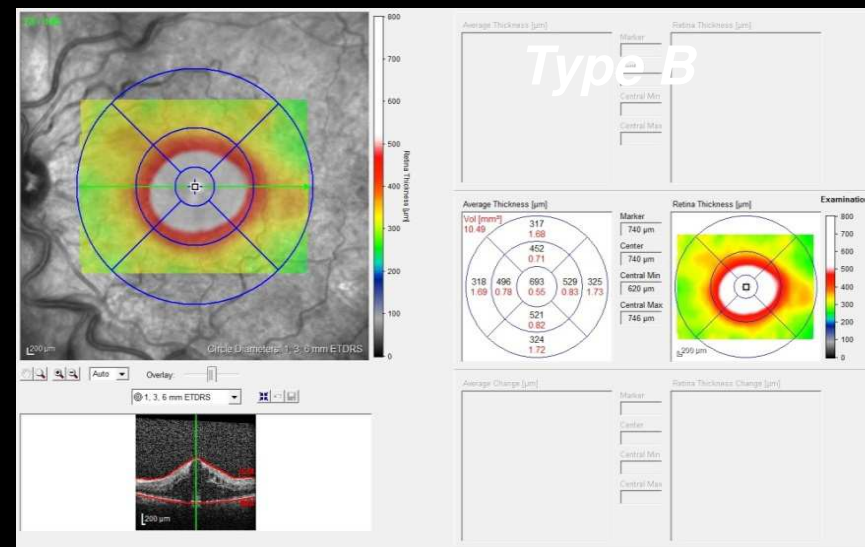
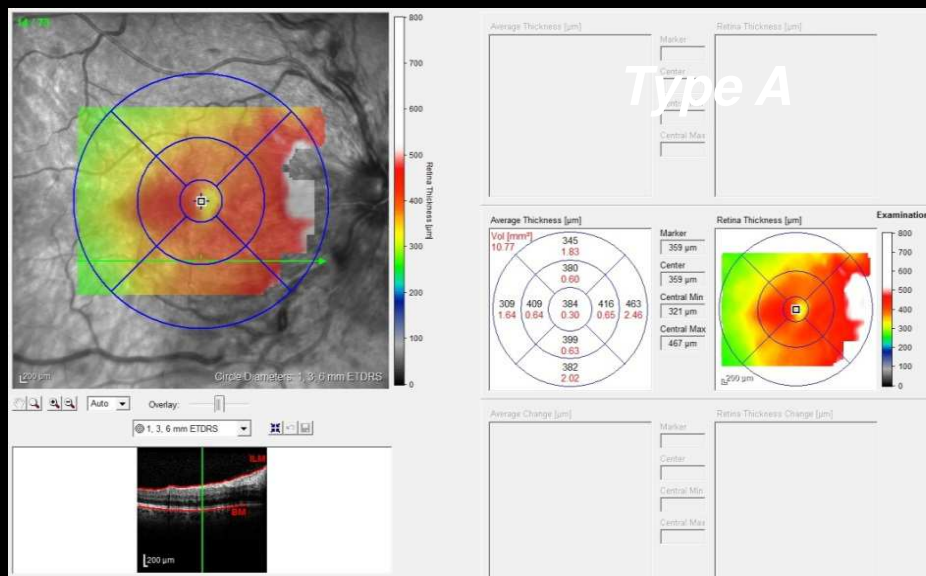


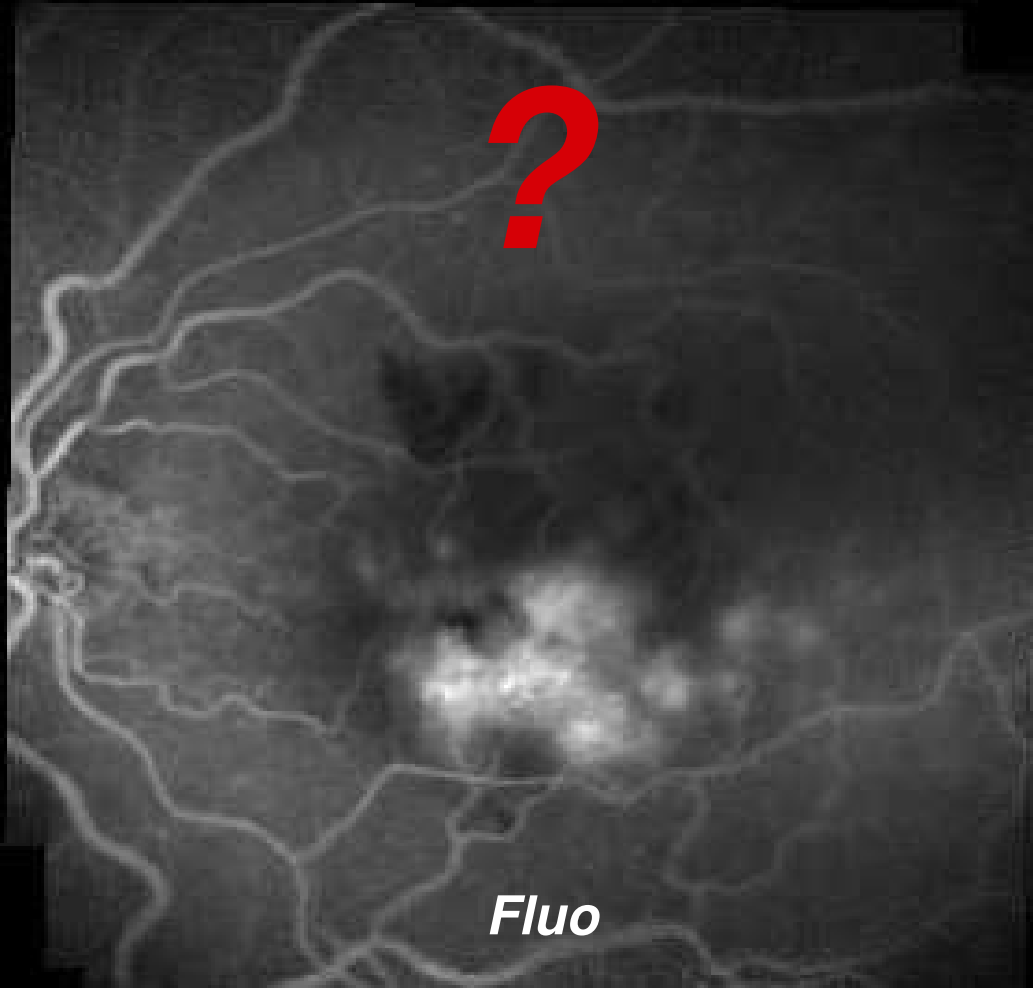


Que faire devant une rubéose? (ou une atteinte sévère chez un sujet âgé, diabétique...)

- *PPR en une séance et IVT antiVEGF*
- Si PPR difficile, envisager d'emblée vitrectomie endolaser
 - Tenter de répéter une PPR difficile est épuisant pour tout le monde...et pendant ce temps la néovascularisation évolue
 - La vitrectomie endolaser est possible même en présence d'une cataracte
- Si le pôle postérieur est déjà atrophique, envisager un grid maculaire

L'œdème maculaire



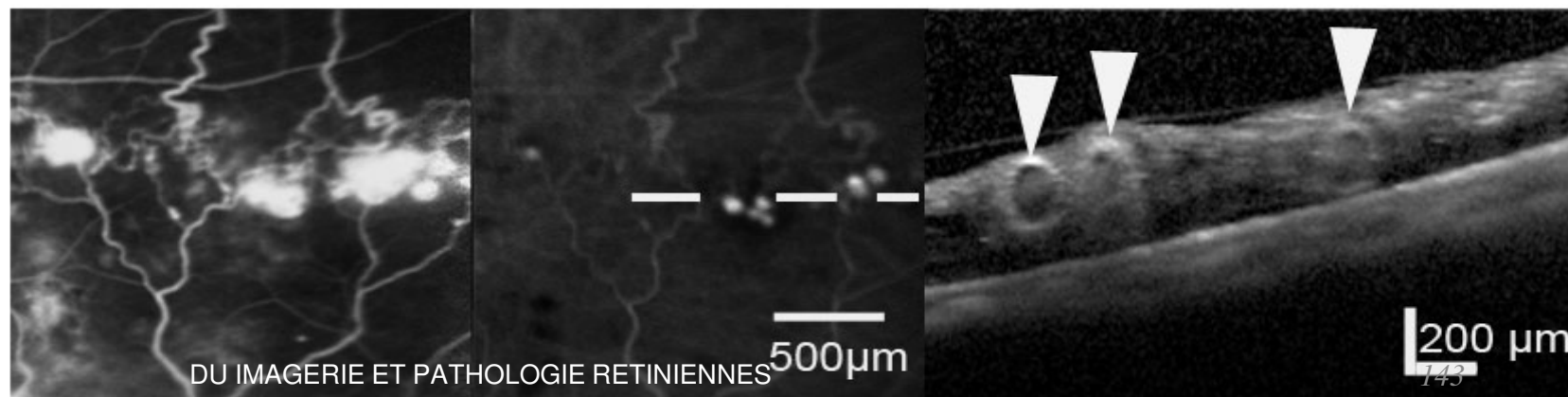
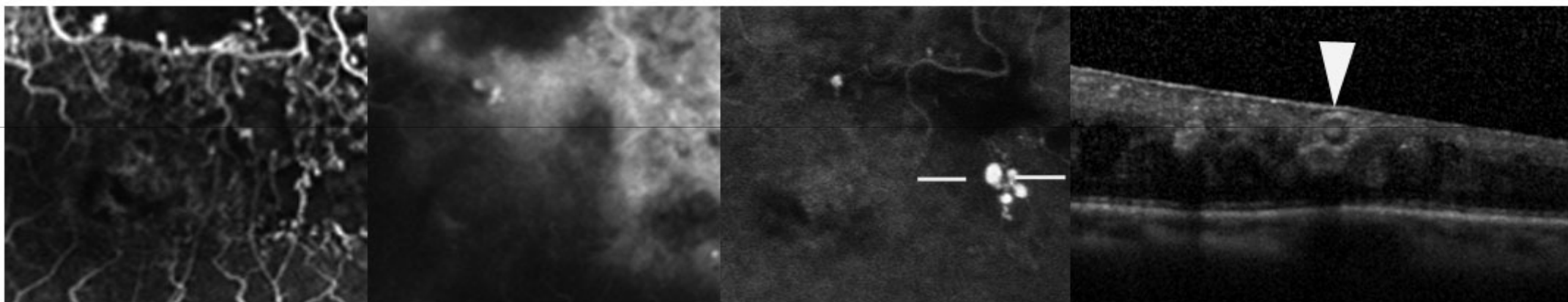
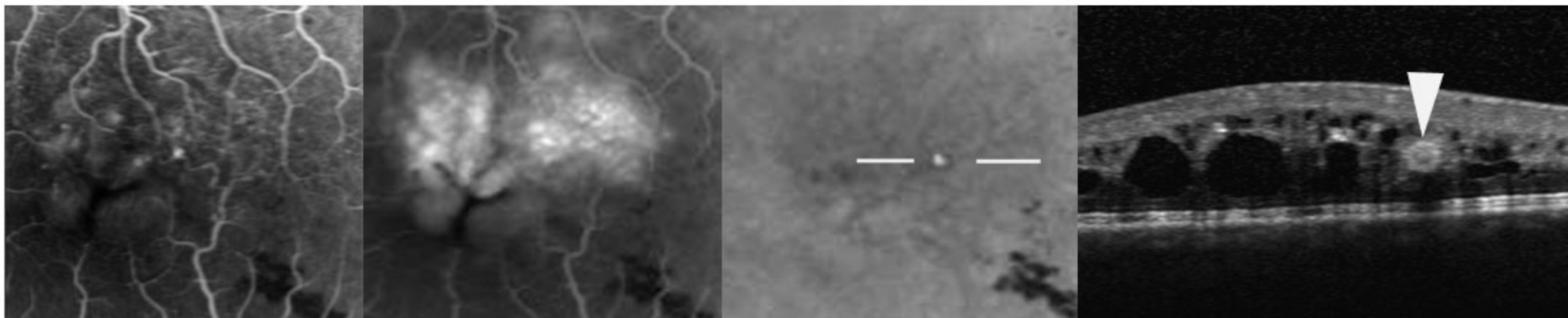


FA early

FA late

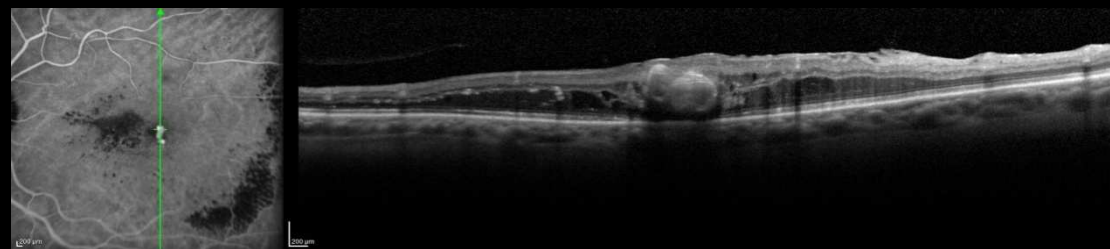
ICGA late

OCT

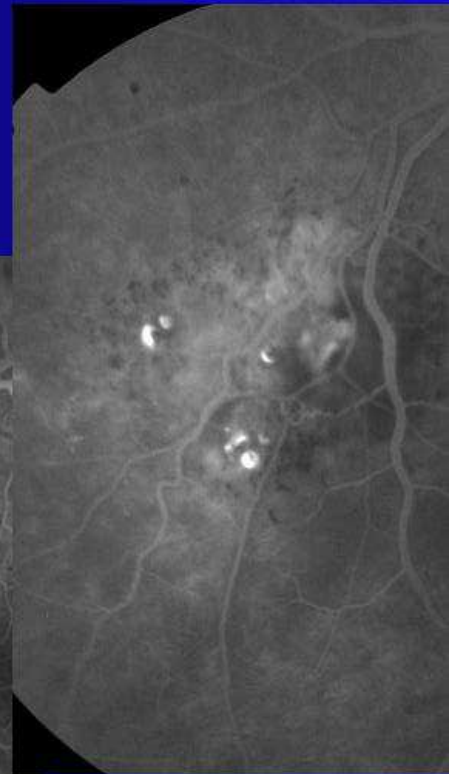


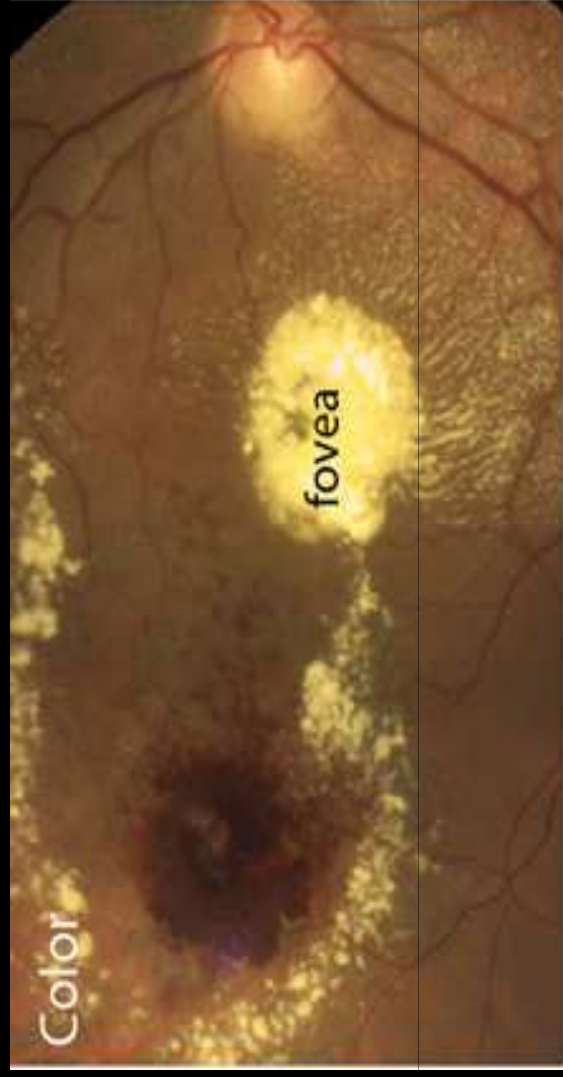


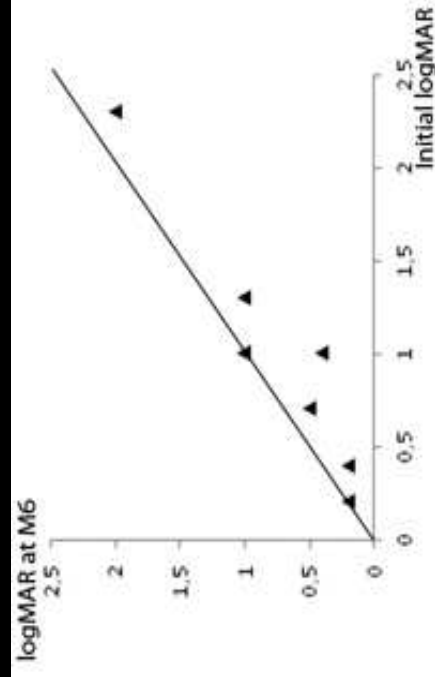
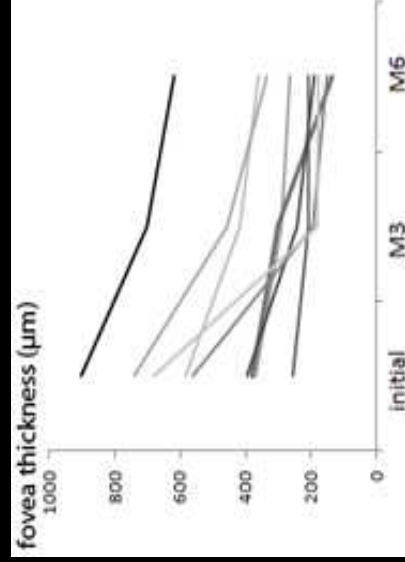
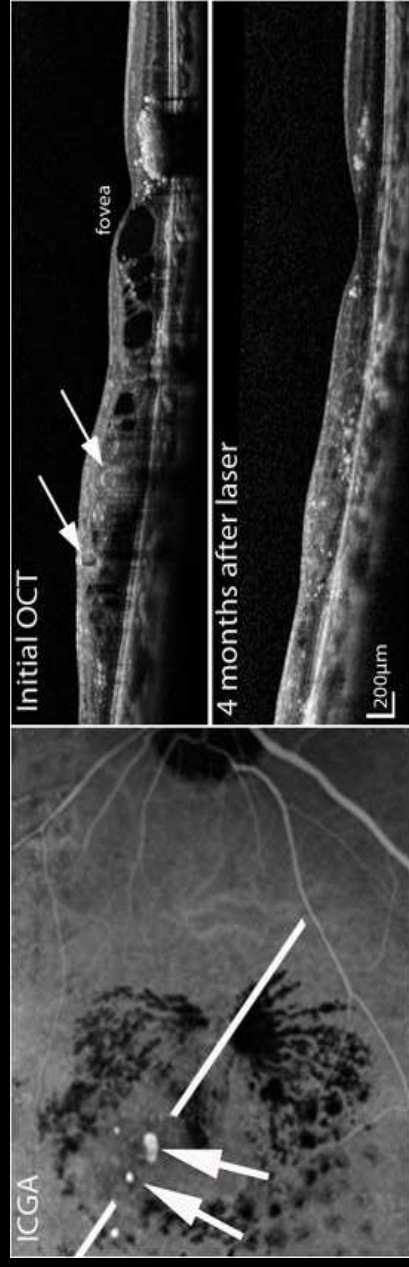
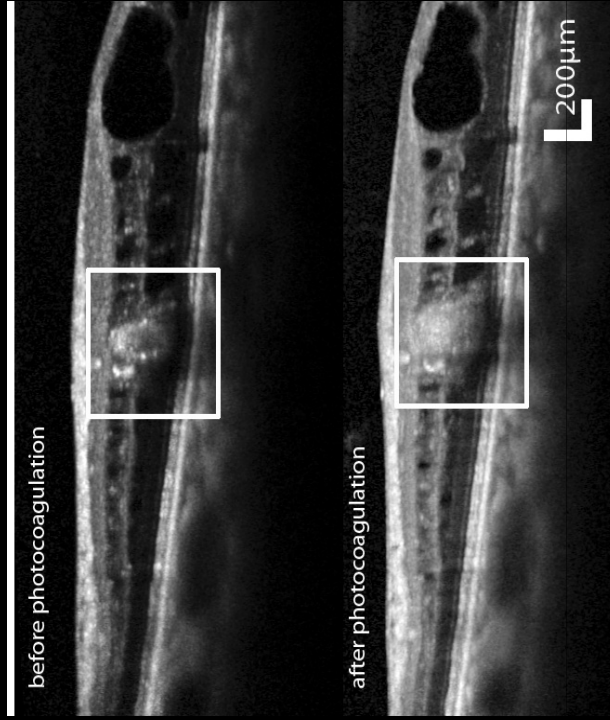
Bourhis et al, Graefes Arch 2010

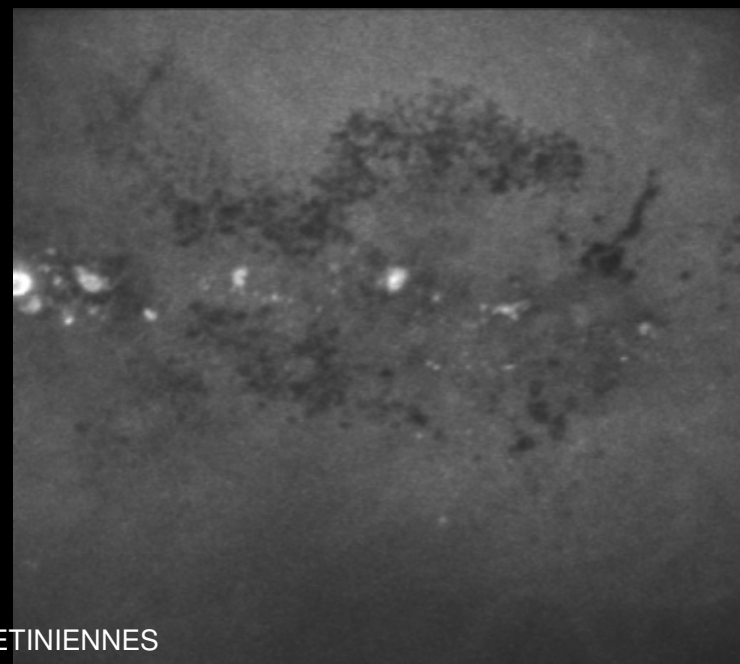
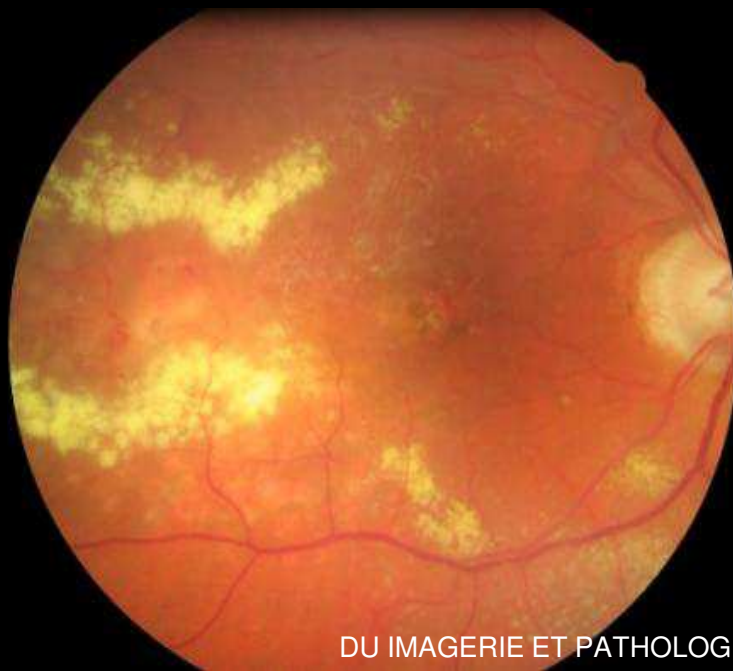
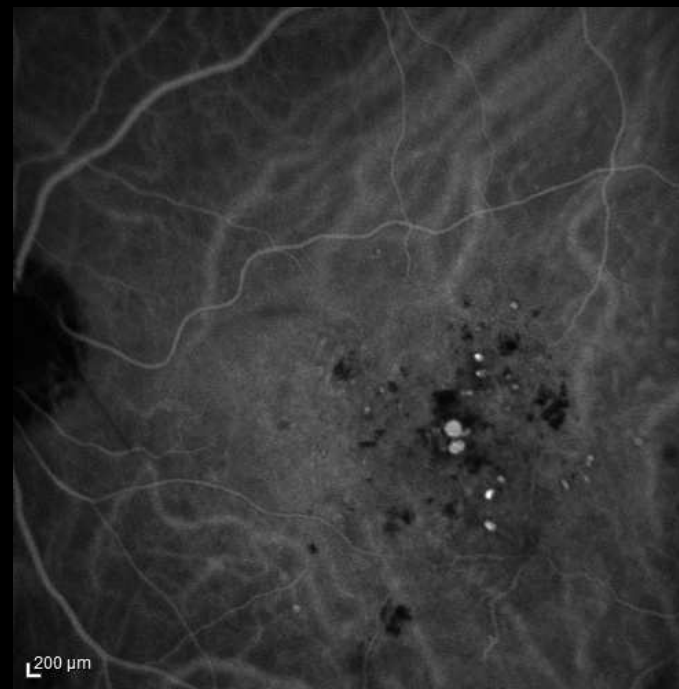


*Courtoisie
Dr Favard*



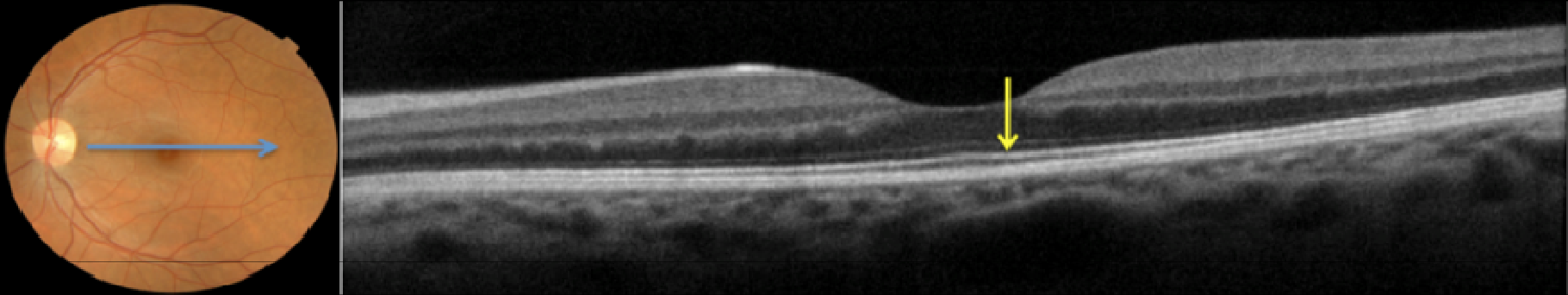




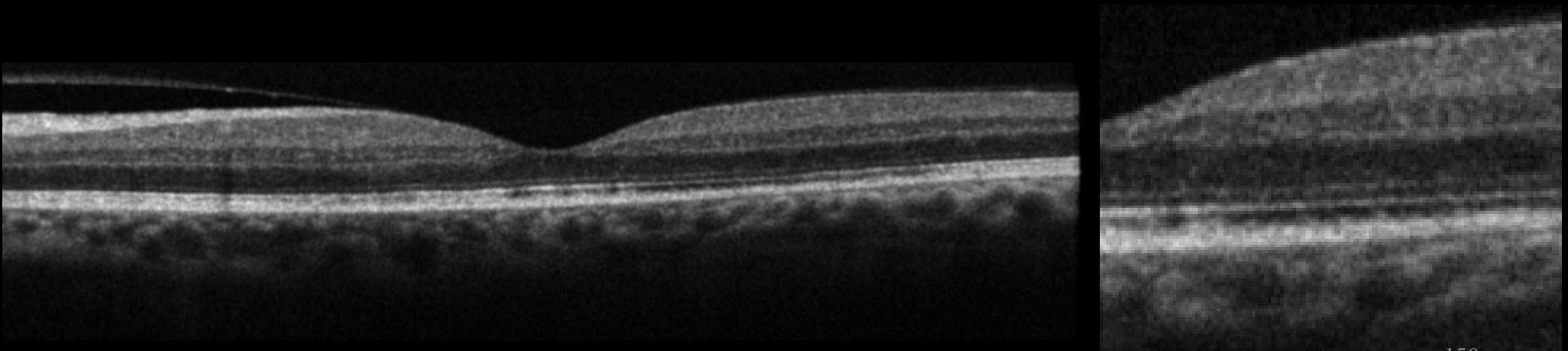


Résultats morphologiques (2): analyse qualitative

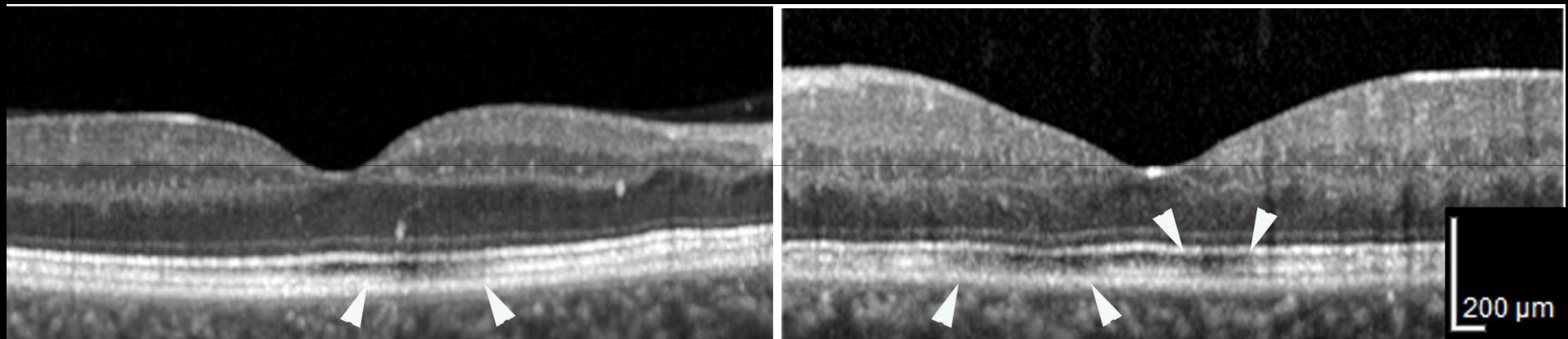
- Aspect normal: 27 % (tous AV $\geq 10/10$)



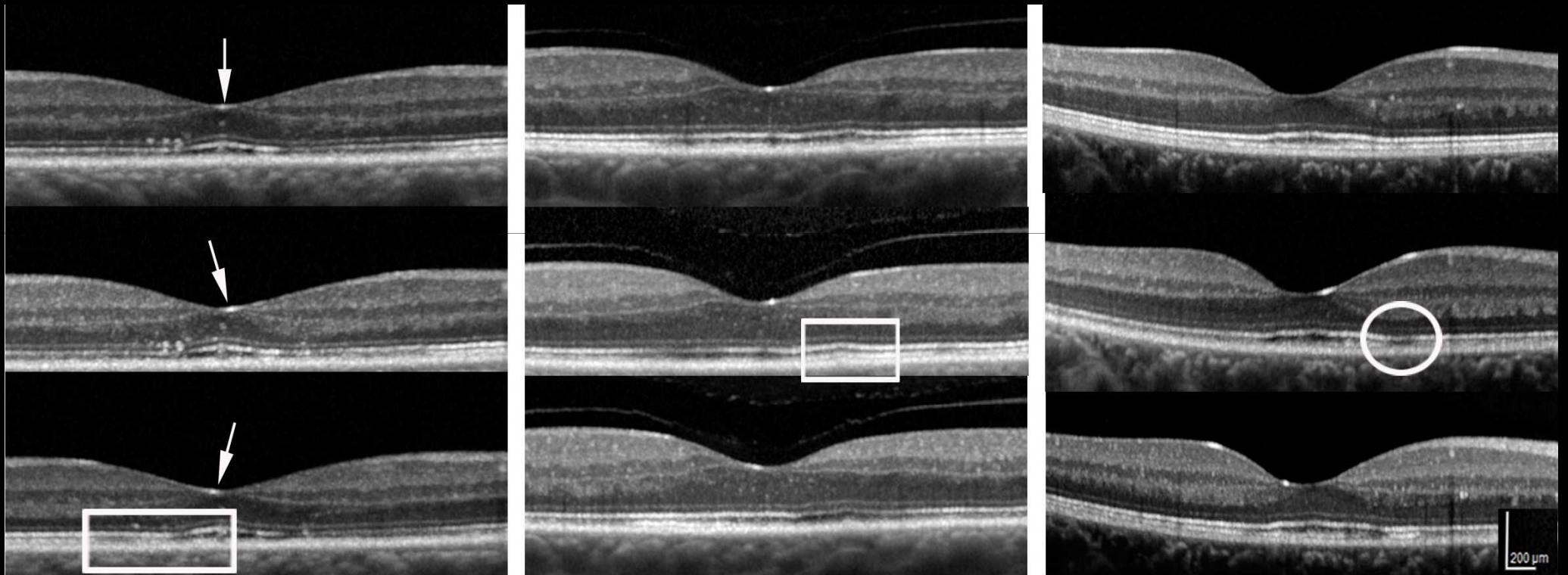
- Anomalie la plus fréquente: altération de la « ligne centrale » des photorécepteurs

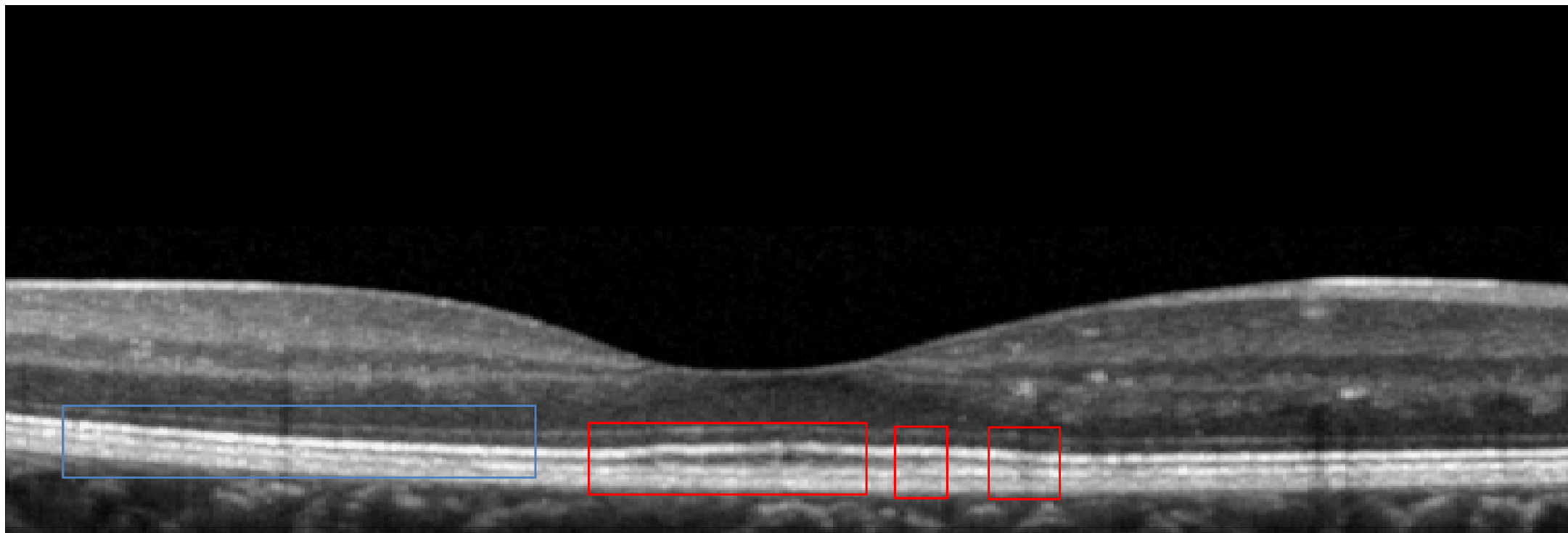


Séquelle d'OM: les segments externes sont très souvent désalignés



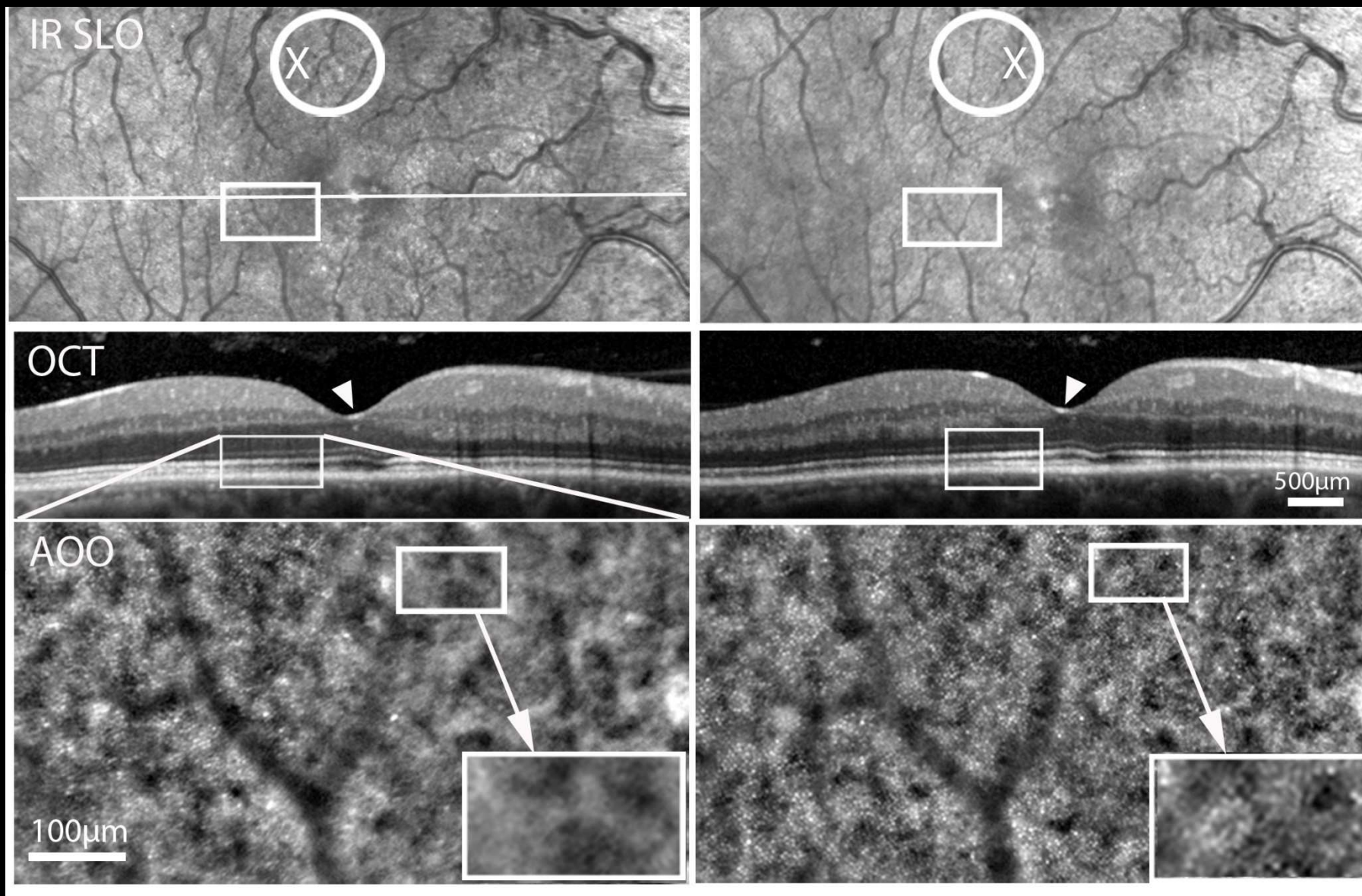
OCT multiangle après œdème maculaire

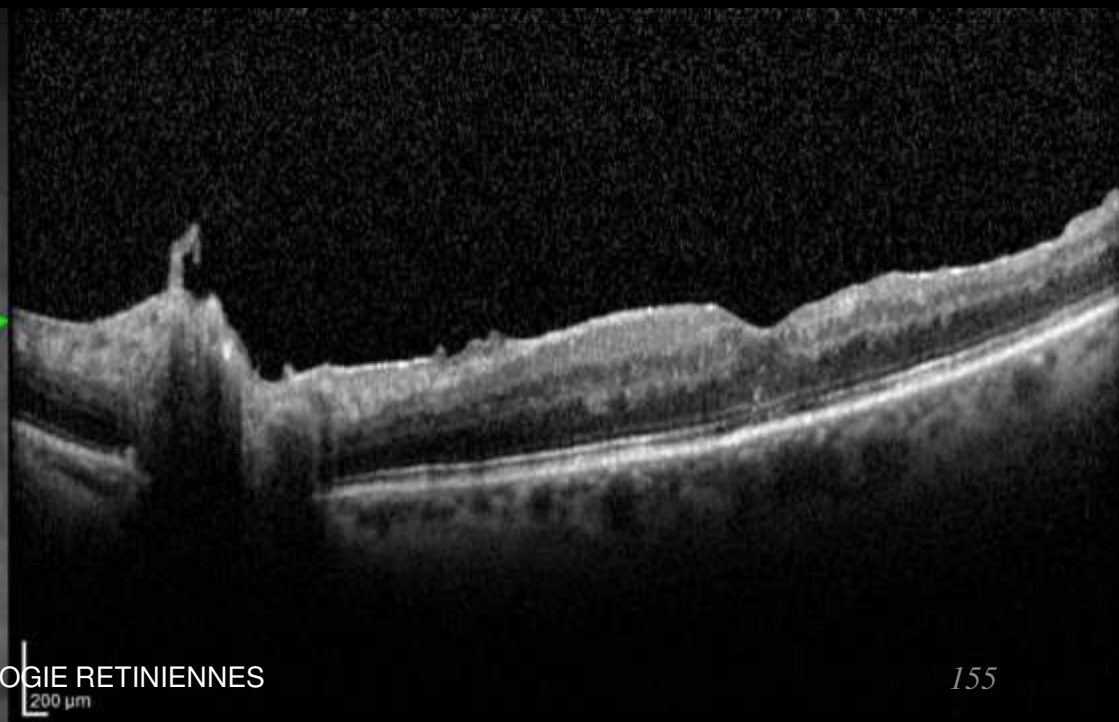
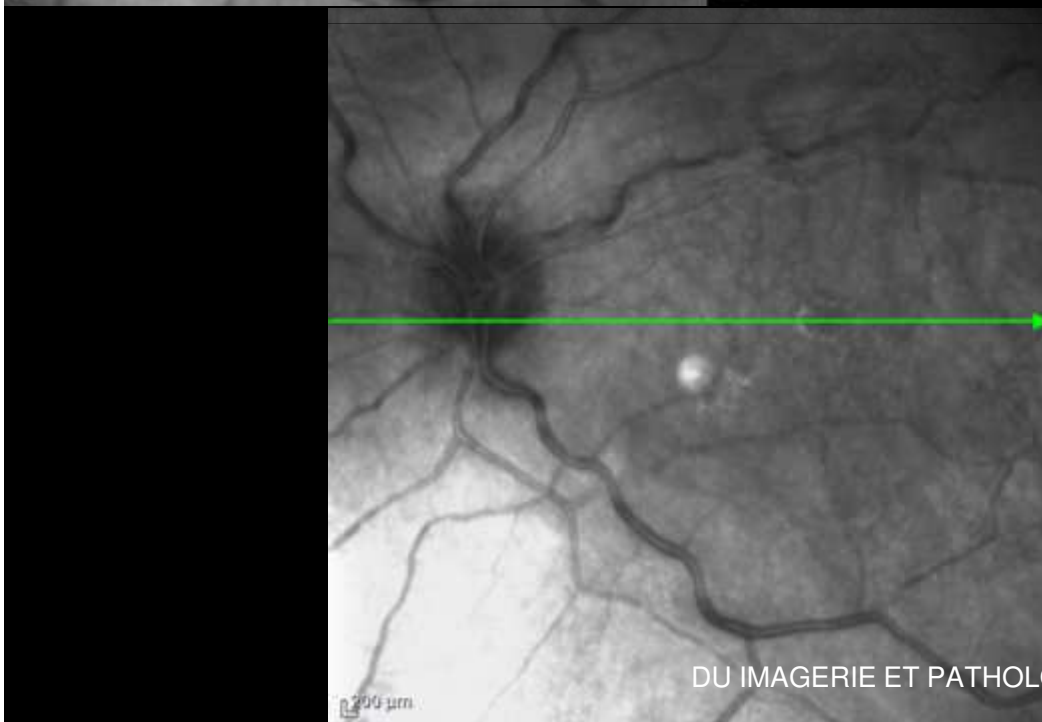
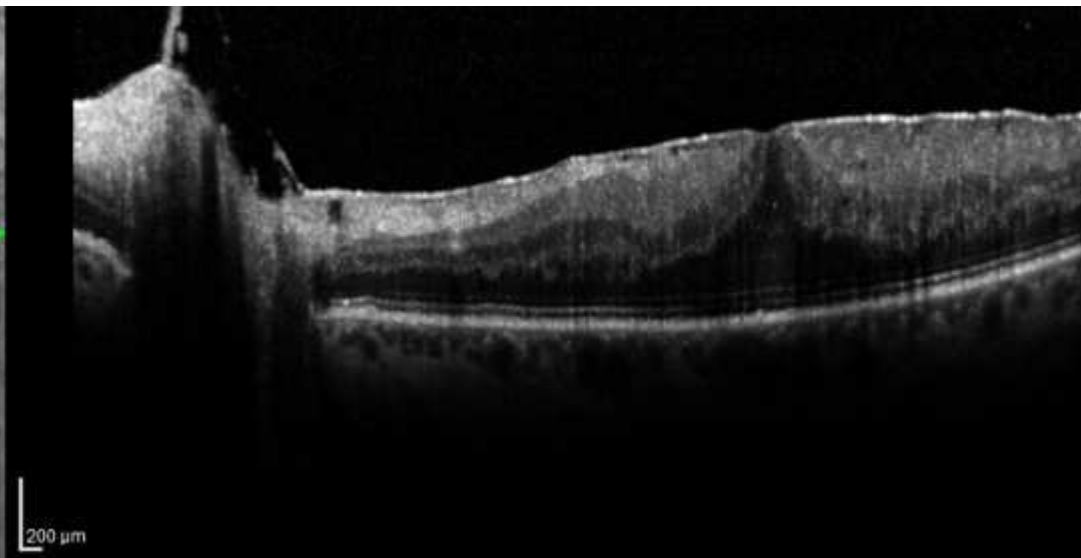
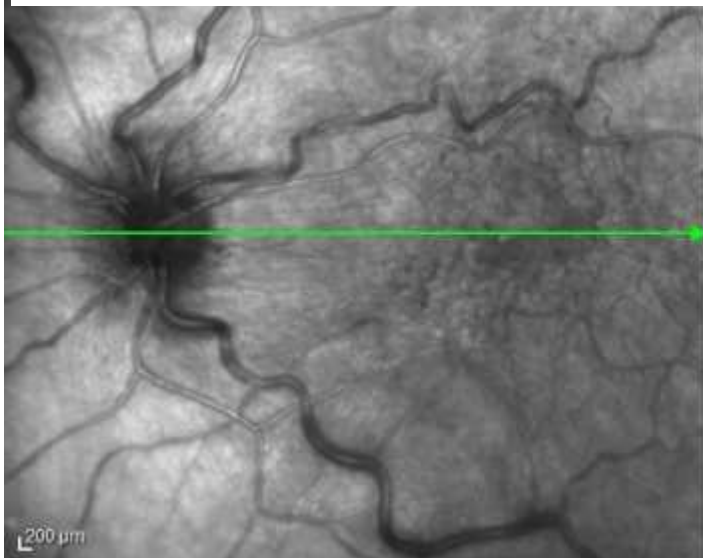




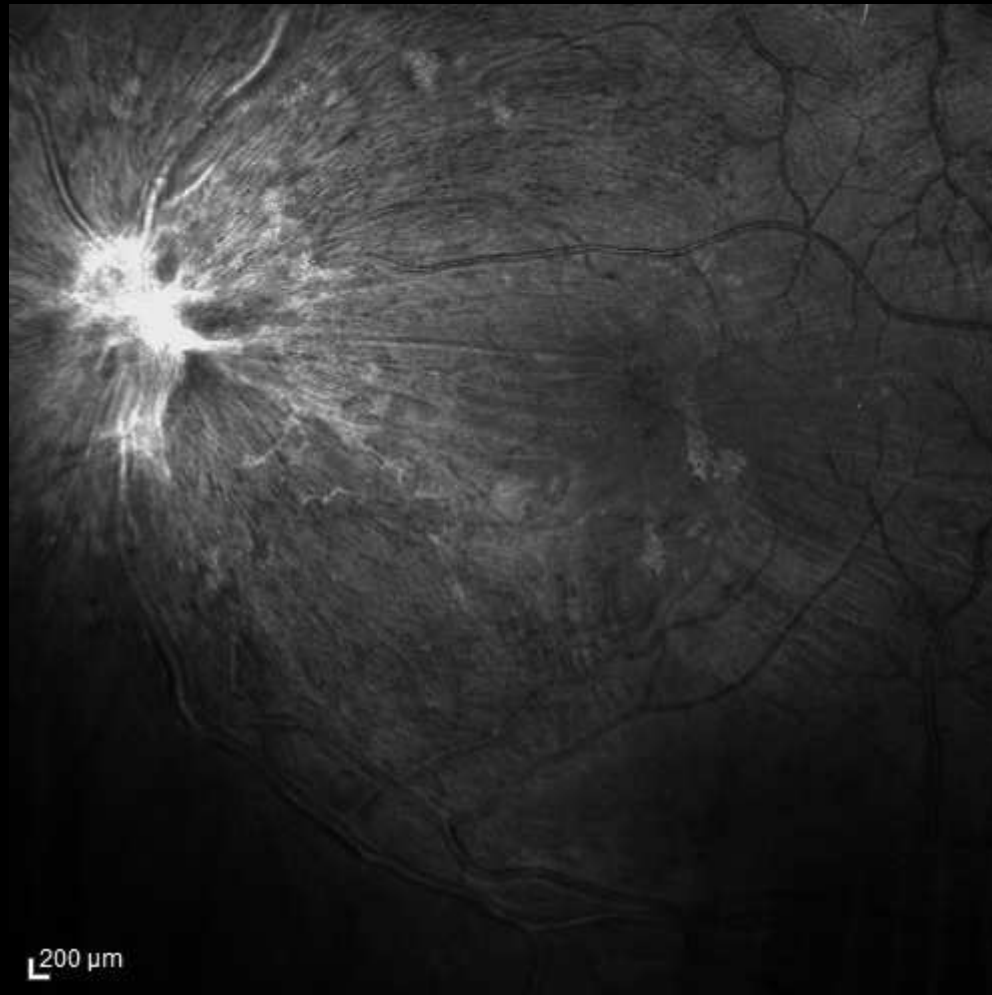
STD 2-3





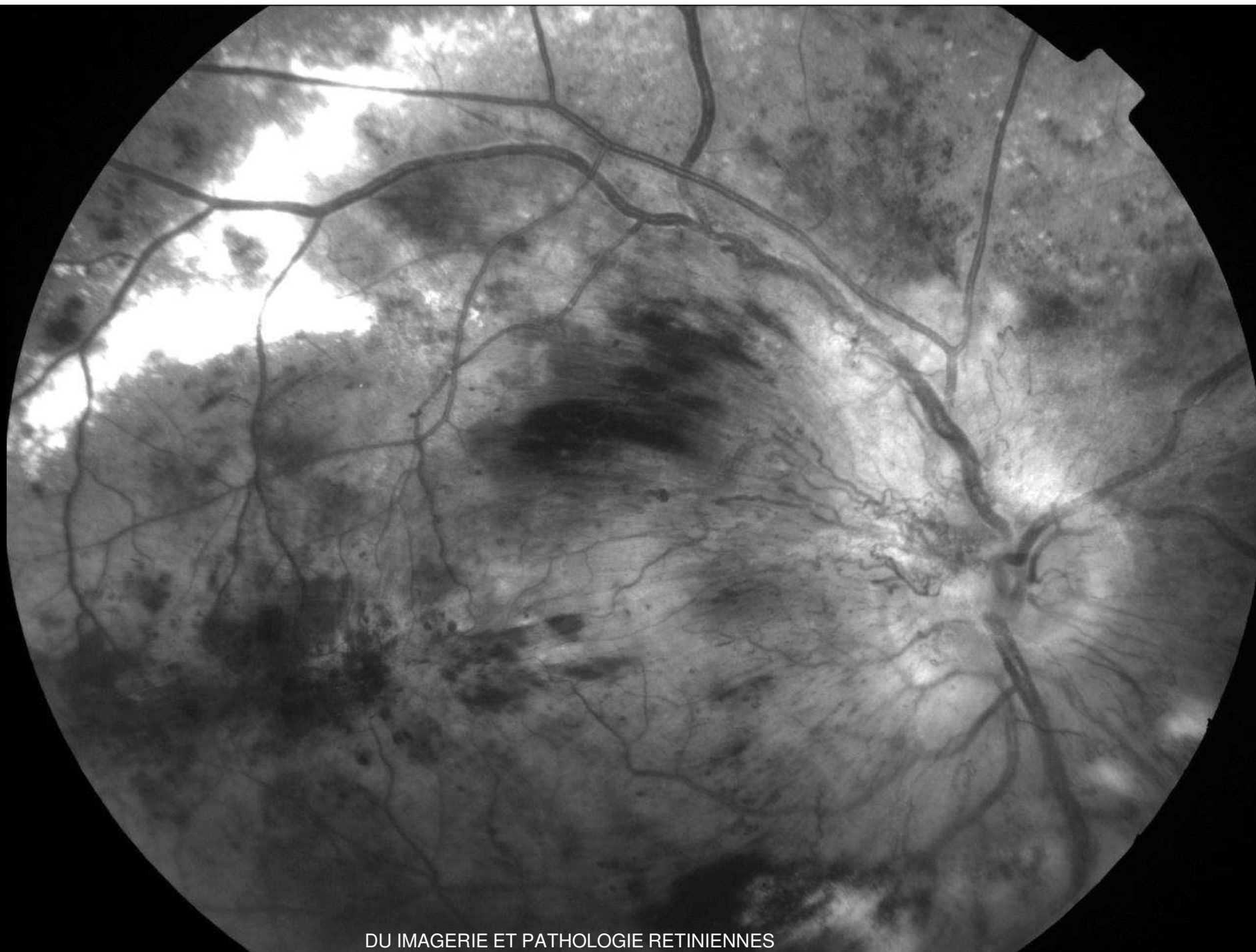


Les membranes épirétiniennes: plus fréquentes après anti-VEGF?



Cas particulier: l'OVCR du diabétique

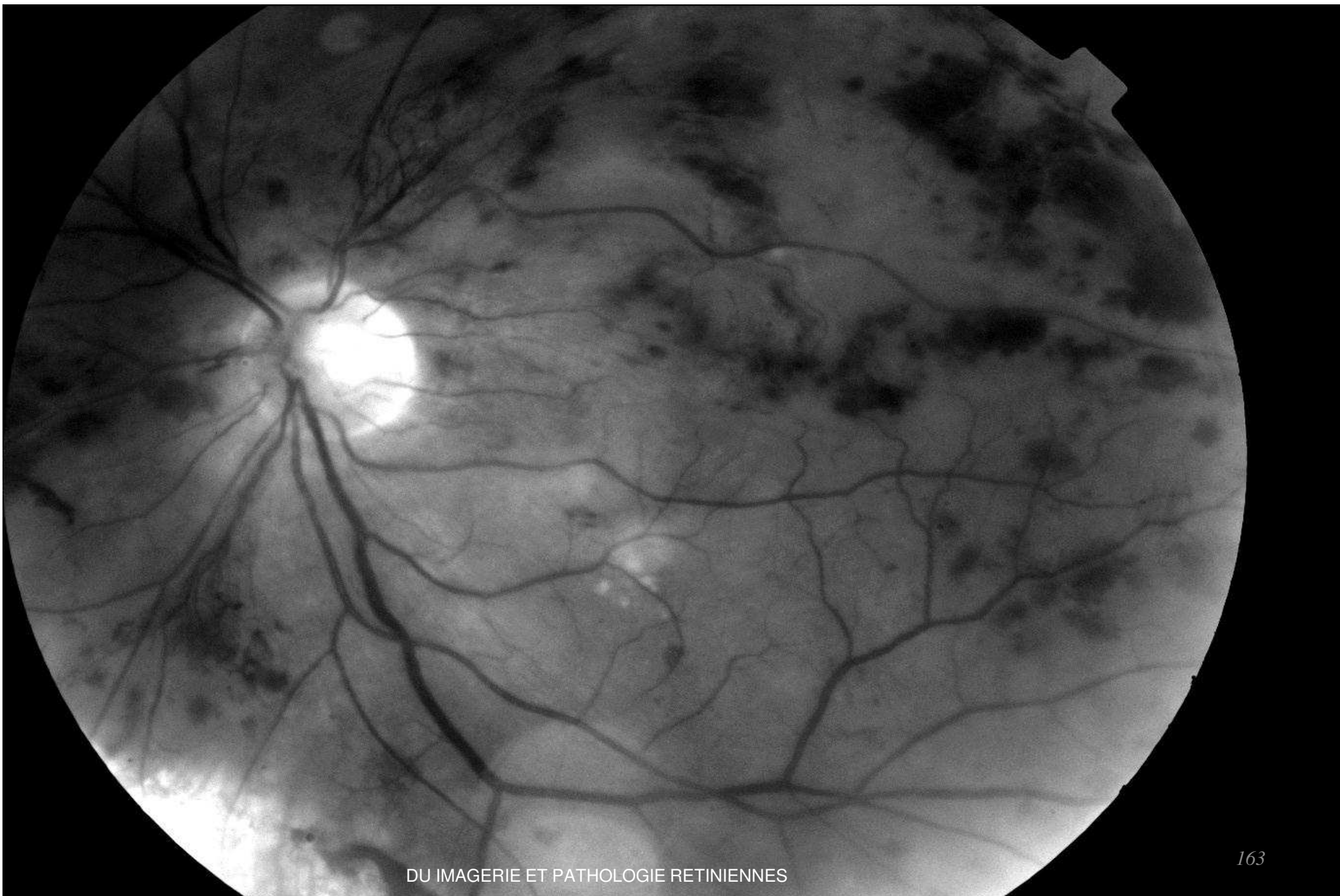


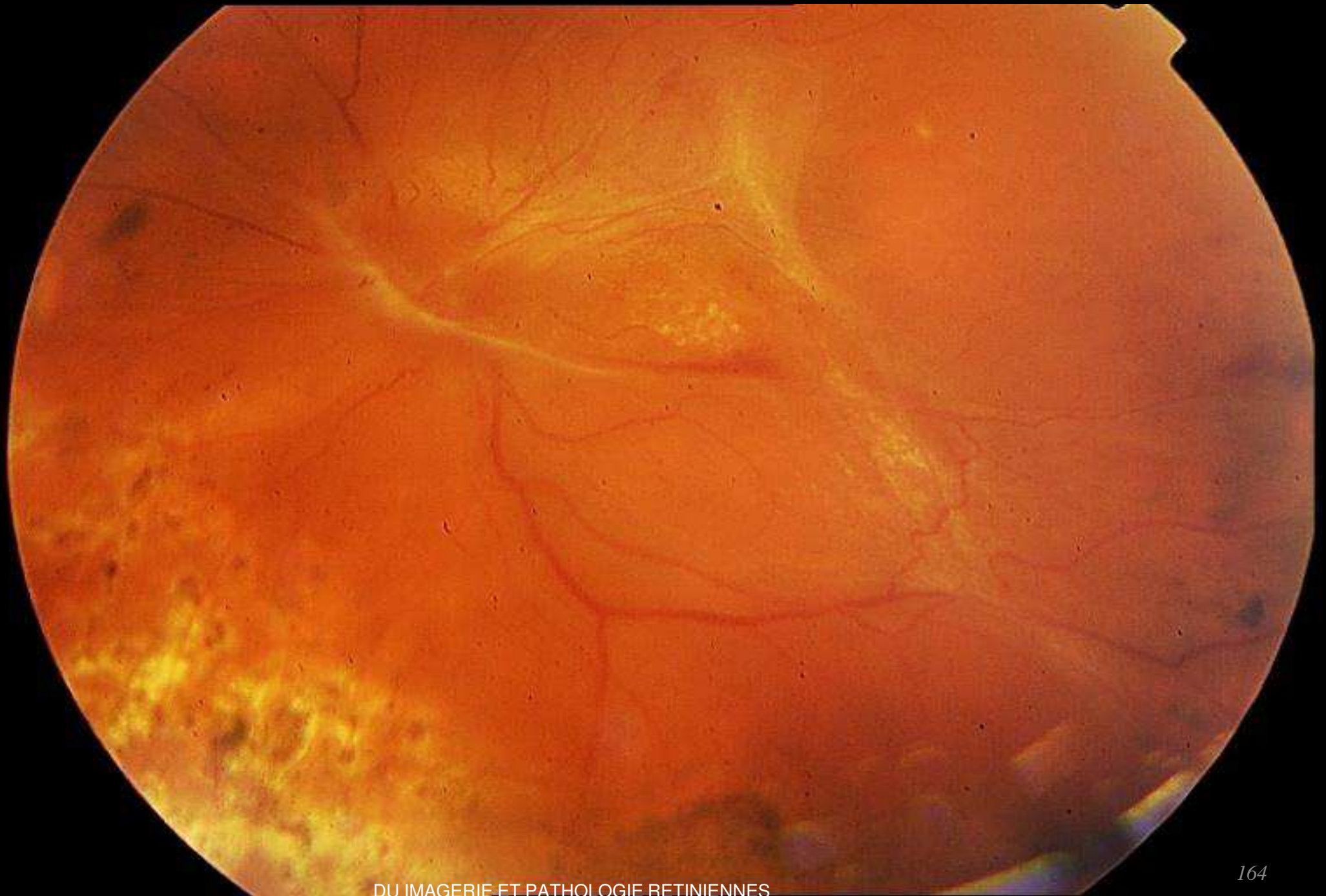














Results

- **106 patients**
- **Median age : 62.5 yrs [29-91]**
- **Median follow-up : 18 months [6-120]**
- **New vessels : 34%**

Non diabetic patients (n=84)

	No new vessels (n=58)	New vessels (n=26)	p
Age $\pm$ SD	60.7 $\pm$ 13.8	66.8 $\pm$ 14.0	0.041
Age $\geq$ 65 yrs (%)	38	73	0.003
Men, % patients	48	46	0.858
Duration of the disease (months) $\pm$SD	25 $\pm$ 24	23 $\pm$ 19	0.753
History of ocular hypertension (%)	34	46	0.311
Hypotensive therapy (%)	40	42	0.820
Lipid lowering medications (%)	33	35	0.868
Current smokers (%)	7	4	0.587
Familial history of diabetes (%)	34	15	0.075
Body mass index (kg/m ²) $\pm$ SD	24.8 $\pm$ 4.5	26.8 $\pm$ 3.2	0.002
Waist circumference (cm) $\pm$ SD	91 $\pm$ 13	97 $\pm$ 12	0.042
Systolic blood pressure (mmHg) $\pm$SD	143 $\pm$ 22	146 $\pm$ 16	0.268
Diastolic blood pressure (mmHg) $\pm$SD	84 $\pm$ 10	81 $\pm$ 8	0.280
HbA1c (%) $\pm$SD	5.7 $\pm$ 0.3	5.8 $\pm$ 0.3	0.169
HbA1c $\geq$ 6% (%)	21	38	0.148

Diabetic patients (n=22)

	No new vessels (n=12)	New vessels (n=10)*	p
Age $\pm$SD	63.8$\pm$10.9	63.0$\pm$9.3	0.869
Men (%)	67	90	0.204
Duration of the disease (months) $\pm$SD	35$\pm$26	53$\pm$27	0.114
History of ocular hypertension (%)	50	20	0.155
Hypotensive therapy (%)	67	80	0.495
Lipid lowering medications (%)	75	20	0.012
Current smokers (%)	8	20	0.438
Familial history of diabetes (%)	42	80	0.075
Diabetes duration (yrs)	4$\pm$3	11$\pm$13	0.137
Body mass index (kg/m²) $\pm$SD	27.3$\pm$5	28.5$\pm$2.7	0.166
Waist circumference (cm) $\pm$SD	100$\pm$8	107$\pm$9.2	0.060
Systolic blood pressure (mmHg) $\pm$SD	144$\pm$20	146$\pm$22	0.895
Diastolic blood pressure (mmHg) $\pm$SD	84$\pm$13	79$\pm$13	0.338
HbA1c (%) $\pm$SD	6.5$\pm$0.5	8.0$\pm$1.8	0.017
HbA1c $\geq$ 6% (%)	92	100	0.361

* Diabetic retinopathy: 2 patients ; diabetic macular edema: 1 patient

Predictors for new vessels

- In multivariate regression analysis model, only HbA1c level (0.01) and age ≥ 65 yrs (0.04) were significant predictors for new vessels.
- In multivariate regression analysis model adjusting for known or unknown diabetes, only HbA1c level (0.003) and age ≥ 65 yrs (0.024) were significant predictors of new vessels.

Multivariate logistic regression

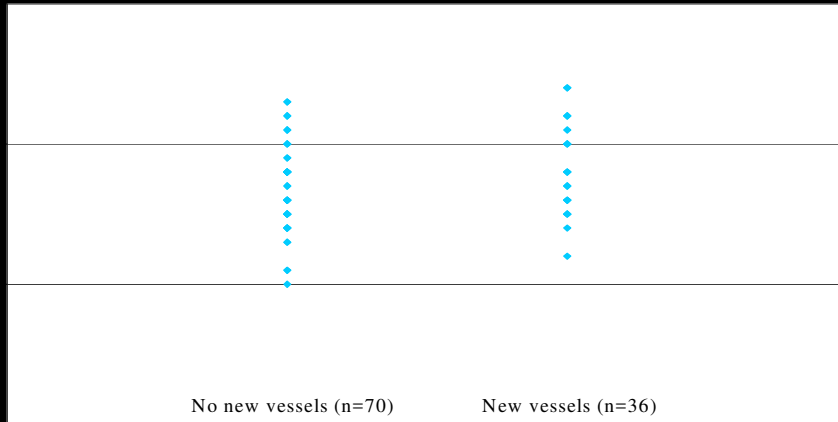
	OR	IC (95%)	p
Age ≥ 65 yrs	2.96	[1.12-7.76]	0.028
HbA1c*	2.40	[1.21-4.76]	0.012

* Adjusted for known or unknown diabetes

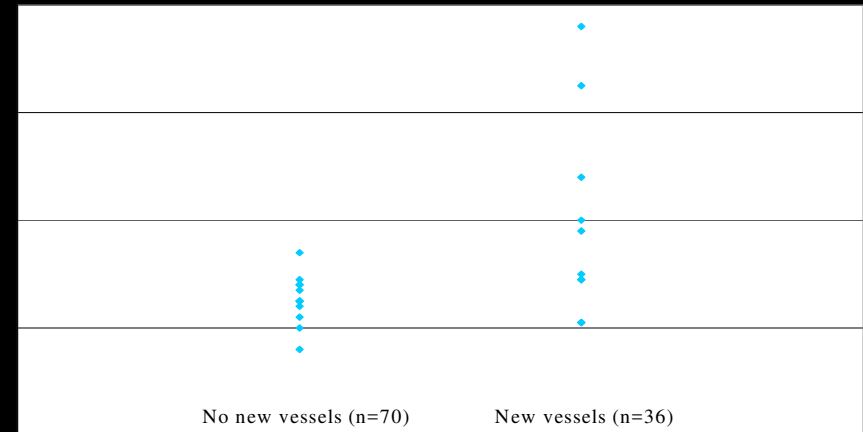
	OR°	IC (95%)	p
Age ≥ 65 yrs	2.80	[1.06-7.43]	0.037
HbA1c*	3.98	[1.26-12.55]	0.018

* For every 1% more of HbA1c

HbA1c and new vessels



Nondiabetic patients



Diabetic patients

Risk factors for severe CRVOs differ between diabetic and nondiabetic subjects

- Diabetics are at high risk for post-CRVO neovascularisation
 - even without preexisting RD
 - The risk linearly increases with HbA1c level
 - Males are possibly at higher risk
 - Interaction between sex and diabetes for ocular complications?
 - More patients needed
- In nondiabetics, age and BMI are strongly correlated to the outcome of CRVO

Predictors of Neovascular Glaucoma in Central Retinal Vein Occlusion

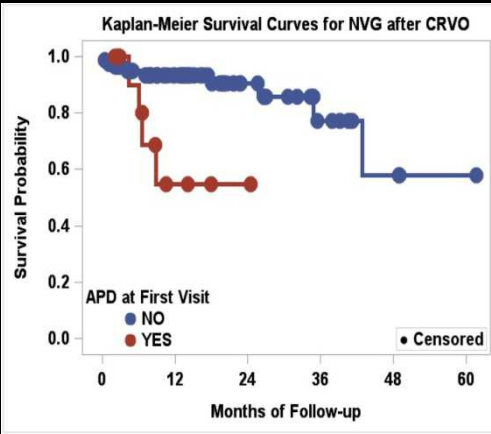
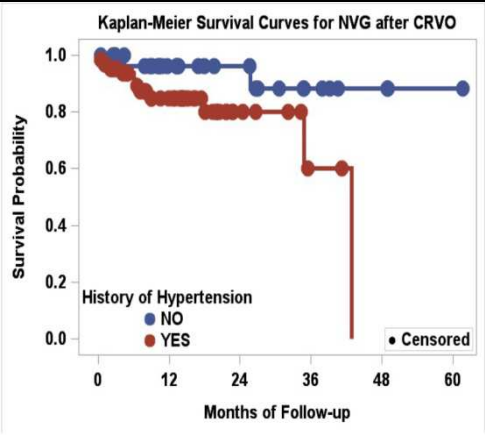
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¹Department of Ophthalmology, Bascom Palmer Eye Institute, University of Miami Miller School of Medicine, Miami, FL

²Department of Ophthalmology, Duke Eye Center, Duke University, Durham, NC

meeting inclusion criteria

Categorical Variables	Value	All Patients (n = 98)	No Neovascular (n = 85, 86.7%)	Neovascular (n = 13, 13.3%)
Gender	Female	47 (48%)	40 (47%)	7 (53.8%)
	Male	51 (52%)	45 (53%)	6 (46.2%)
Age less than or equal to 50 years	No	78 (79.6%)	65 (76.4%)	13 (100%)
	Yes	20 (20.4%)	20 (23.6%)	0 (0%)
Afferent Pupillary Defect	No	84 (85.7%)	75 (88.3%)	9 (69.2%)
	Yes	14 (14.3%)	10 (11.7%)	4 (30.8%)
Anti-VEGF at first visit?	No	36 (36.7%)	30 (35.2%)	6 (46.2%)
	Yes	62 (63.3%)	55 (64.8%)	7 (53.8%)
History of Primary Open Angle Glaucoma	No	74 (75.5%)	65 (76.4%)	9 (69.2%)
	Yes	24 (24.4%)	20 (23.5%)	4 (30.8%)
History Hypertension	No	32 (32.7%)	30 (35.2%)	2 (15.4%)
	Yes	66 (67.4%)	55 (64.8%)	11 (84.6%)
History Diabetes	No	65 (66.3%)	58 (68.2%)	7 (53.8%)
	Yes	33 (33.7%)	27 (31.8%)	6 (46.2%)
Diabetic Retinopathy (DR)	No	21 (63.6%)	17 (63%)	4 (66.6%)



Comment expliquer ce risque plus élevé de complications?

Le diabète est associé à une réduction de la densité vasculaire dans la DVP (Couturier et al. Retina 2015)

Cette DVP est le lieu où se développent les collatérales (Freund JAMA ophth 2018)

Il est donc logique que en présence d'une OVR, les diabétiques soient moins bien armés pour en affronter les conséquences

Conclusions

Les OVCR et la rétinopathie diabétique proliférante ont de nombreux points communs, et de plus peuvent se potentialiser

- Validation de l' étude expérimentale des OBVR pour mieux comprendre la RD

La mauvaise tolérance de la rétine du diabétique à un stress vasculaire (HTA, OVCR) souligne l' existence de lésions microvasculaires infracliniques

- Notion de dépopulation capillaire précoce dans la DVP, justement là où se forment les collatérales (*Dupas et coll, soumis*)
- Un suivi plus rapproché des patients diabétiques, surtout s' ils sont déséquilibrés, paraît raisonnable

Guérison

➤ Reperfusion de la VCR

Et/ou

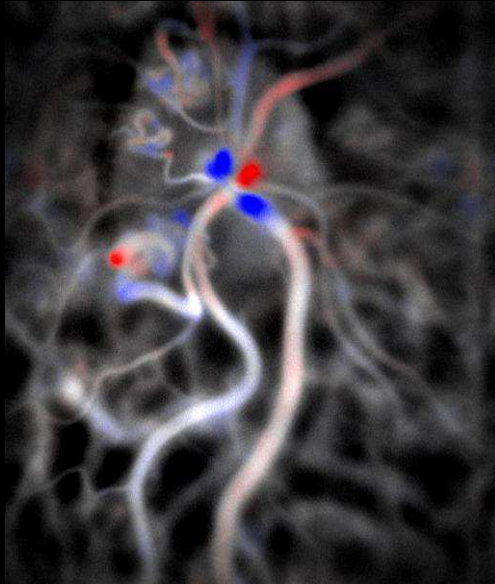
➤ Circulation collatérale



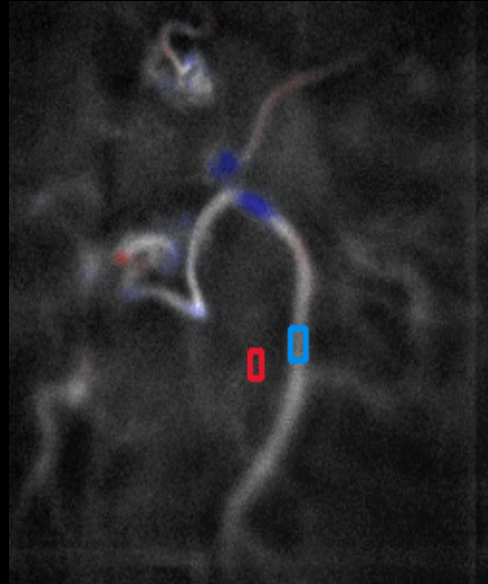
- 191011
- AHM0117
- OD

RI 0.96
 = 0.07 s
 t_{SRT} 0.21 s
 = 0.52 s
 $t_{\text{mean}, A}$ 0.31 s
 =
 $t_{\text{mean}, V} = 75 \text{ kHz}$
 $\Delta f_{\text{flow}} = 1-6 \text{ kHz}$
 $t_{\text{mean}, A-V} = 8-37 \text{ kHz}$
 Δf_{high}

- Blood volume

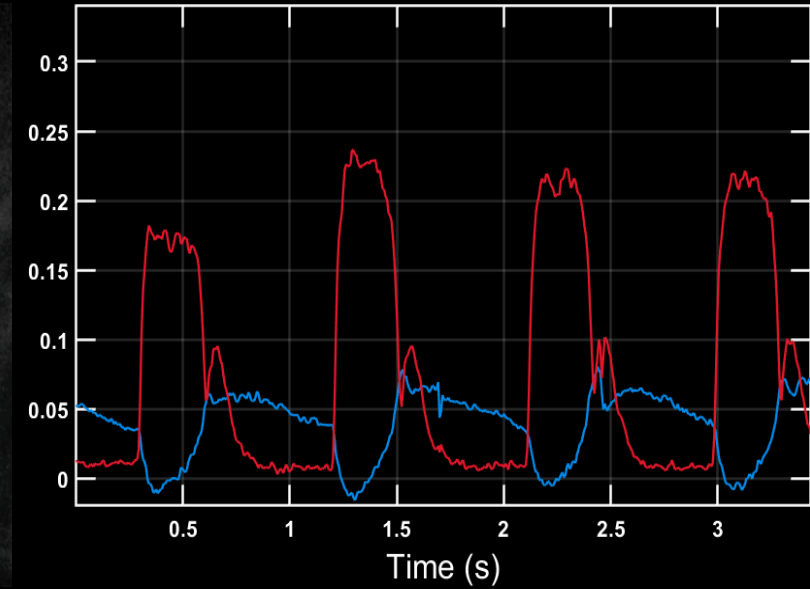


- Blood flow movie



- Blood flow variations

V6



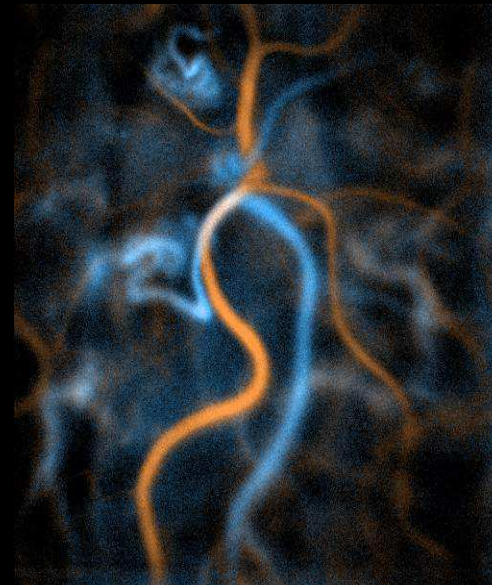
- Blood velocity



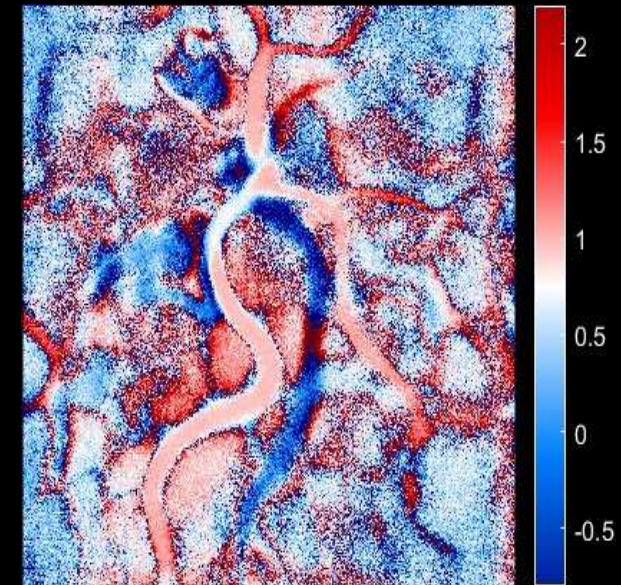
Low flow / high flow

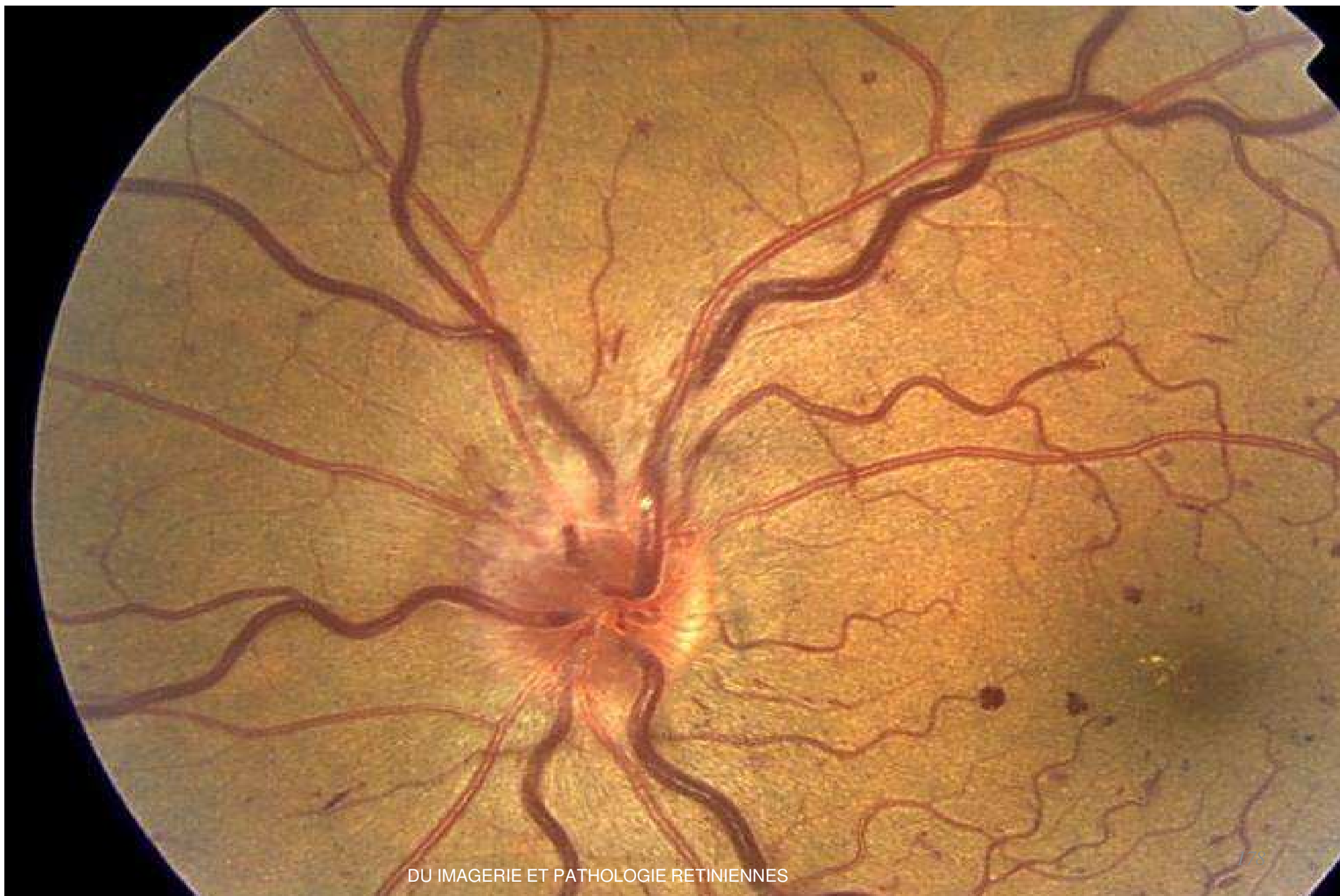


Systole / Diastole



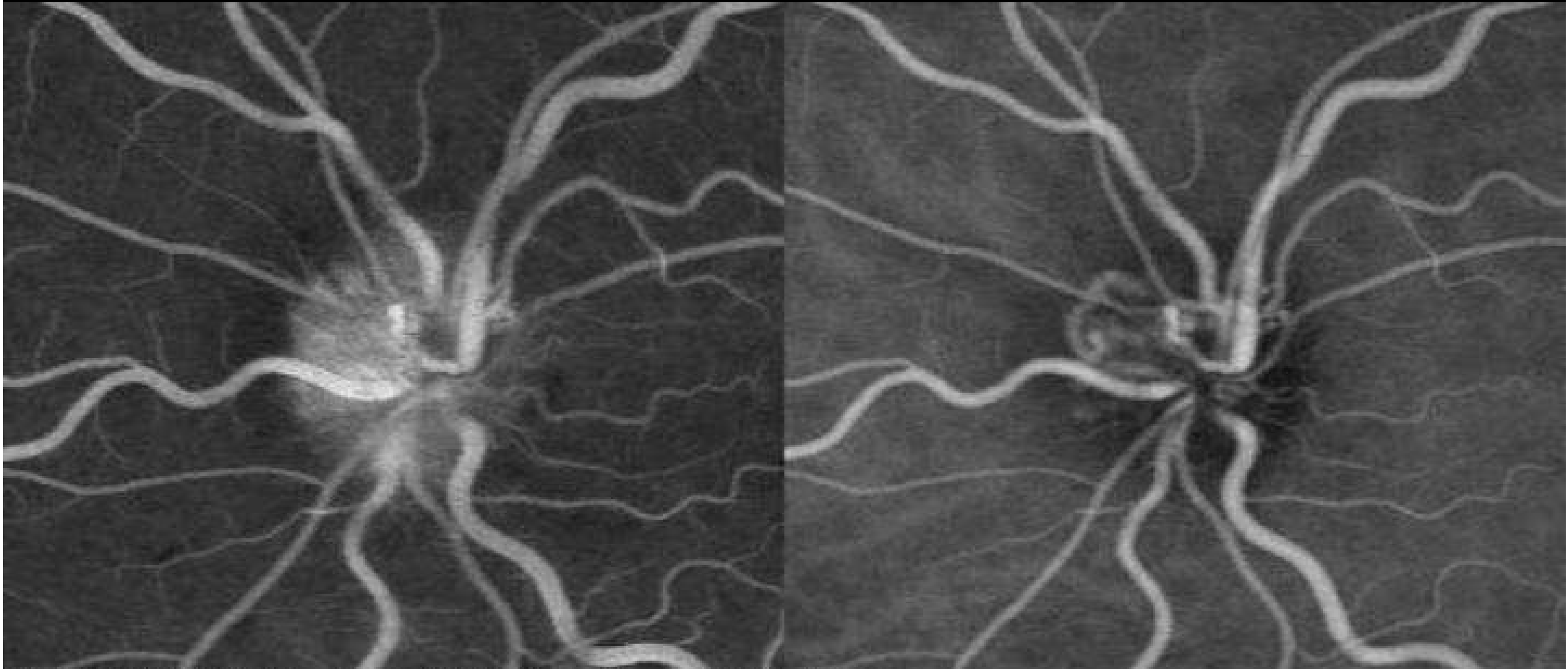
- Resistivity





Fluo

ICG





200 μ m



OD

IRAF 55° ART(10) 91732.1 [HR]

HEIDELBERG
ENGINEERING





Rechute

1) Sur le même oeil

- Effet protecteur d' un réseau collatéral développé?

2) Sur l' autre oeil

- Environ 1% par an
- Traitement de l' HTA et du glaucome?

Classification des OVCR

Il semble exister deux groupes d'OVR, avec des formes intermédiaires...

Type A : bas débit aigu suivi d'un œdème papillaire

Type B: début insidieux suivi d'un œdème maculaire

Pouvant toutes deux pouvant se compliquer de remodelage microvasculaire

Classification des OVR

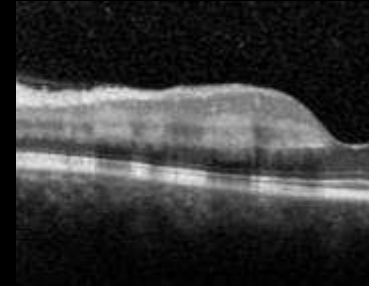
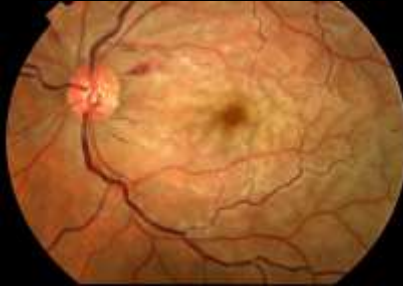
- Type A: Occlusion brutale révélée par bas débit aigu, suivi d' un oedème papillaire
 - Résolution habituelle en quelques semaines
 - Risque essentiel: atrophie de la rétine intermédiaire
 - Possibilité de formes graves si bas débit sévère

- Type B: Occlusion progressive révélée par un œdème maculaire
 - Peut durer plusieurs mois, voire se chroniciser
 - Risque essentiel: lésion des photorécepteurs
 - Possibilités de formes graves si OM chronique et/ou non perfusion

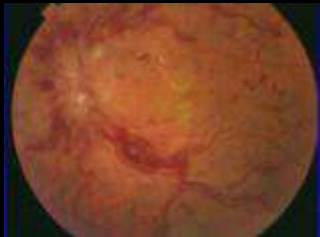
- Remodelage micro vasculaire proportionnel à la durée de l' OVR
 - Collatéralisation
 - Non perfusion ($\pm$ Angiogenèse)
 - Macro anévrysmes

Vers une nouvelle classification des OVR?

➤ Type A précoce: ischémie aiguë



➤ Type A tardif: œdème papillaire



➤ Type B précoce: dilatation veineuse



➤ Type B tardif: œdème maculaire



