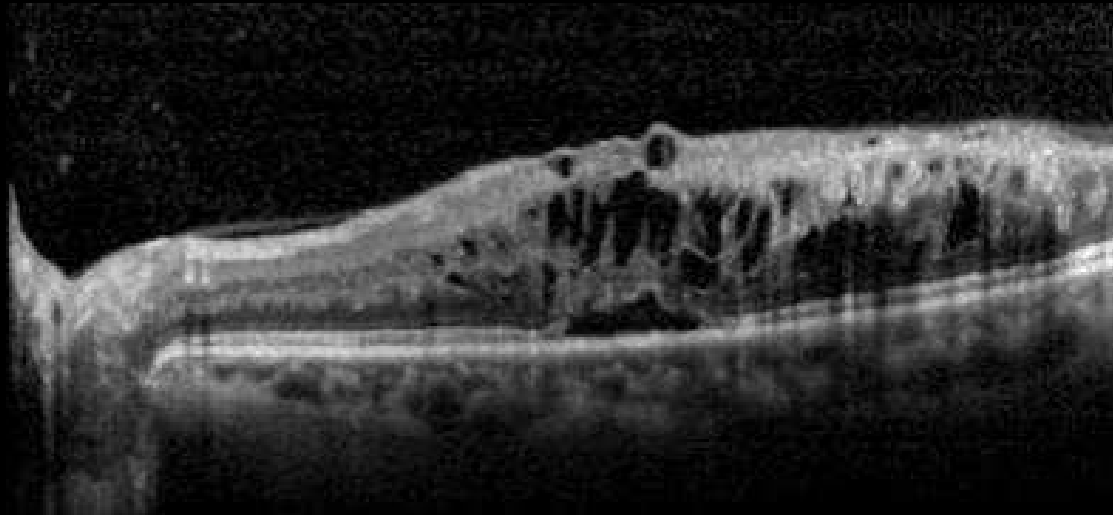


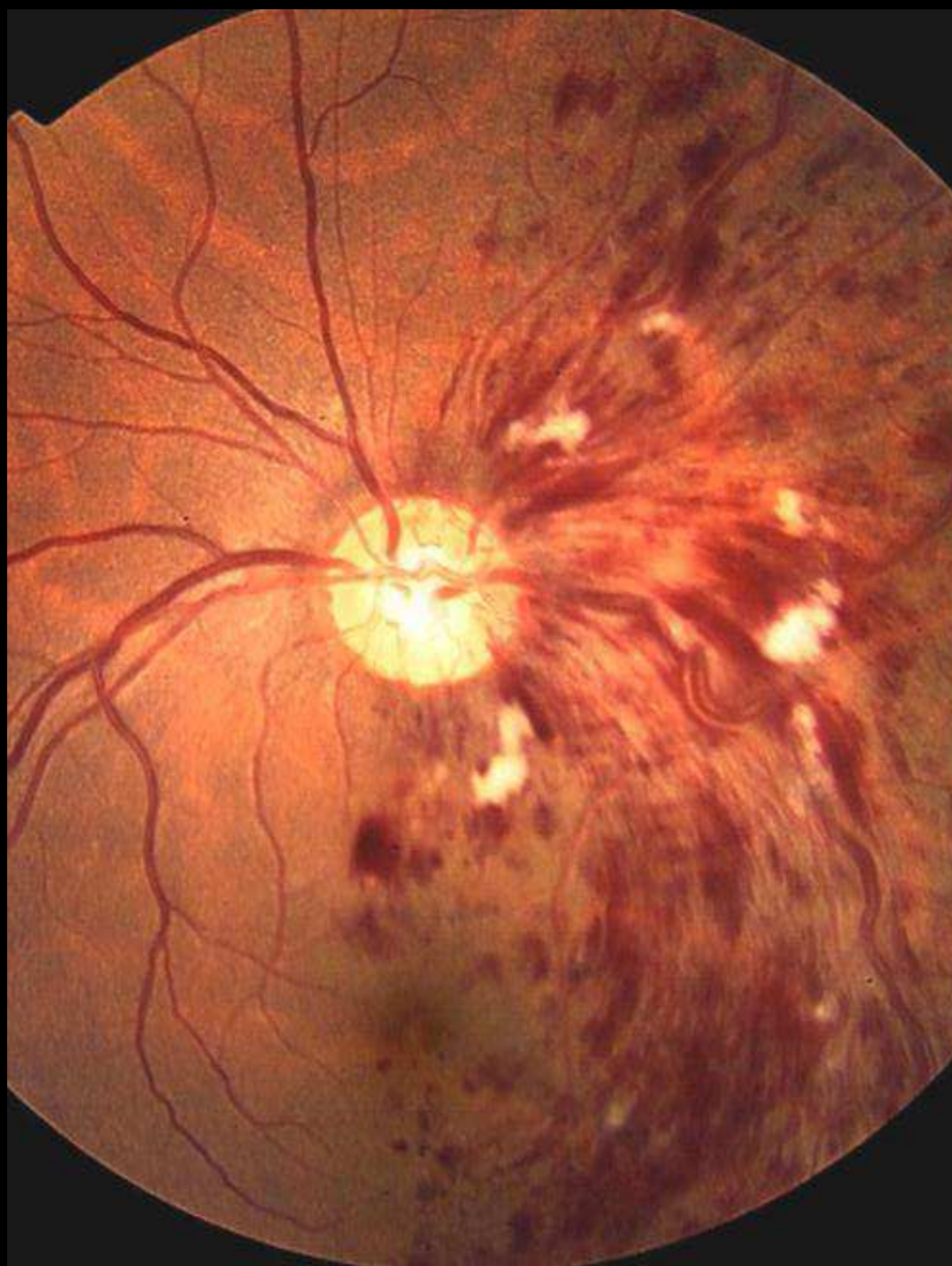
OCCLUSIONS VEINEUSES RETINIENNES

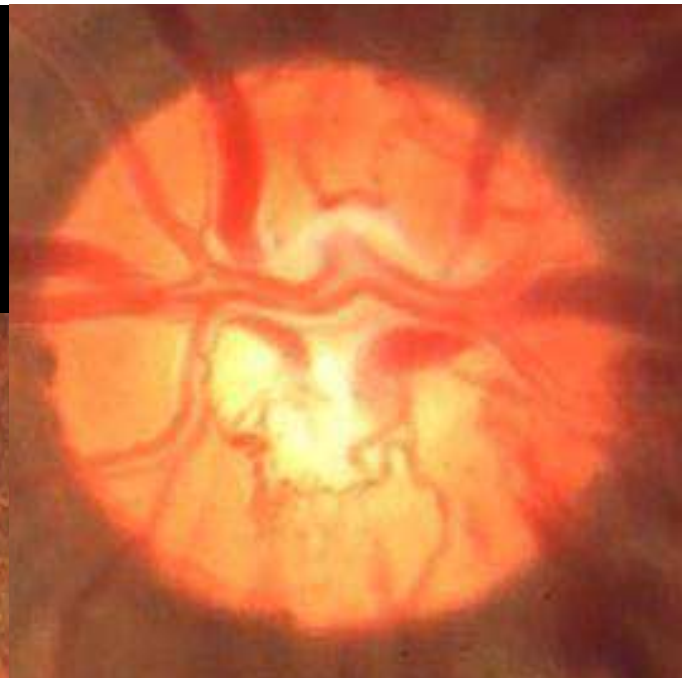


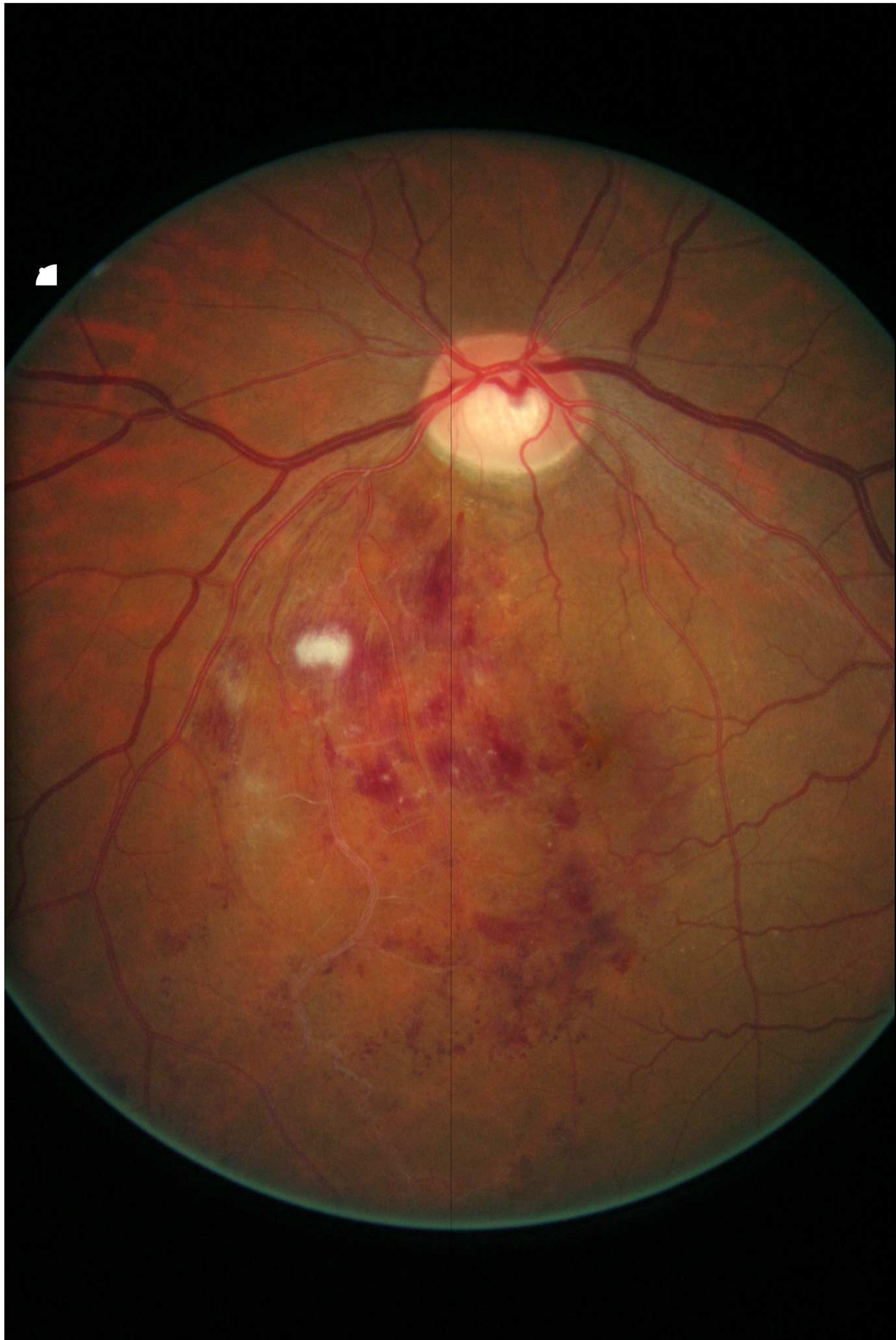
M. PAQUES, JF GIRMENS

AUCUN CONFLITS D'INTÉRÊTS













OCCLUSIONS VEINEUSES RÉTINIENNES

4 territoires:

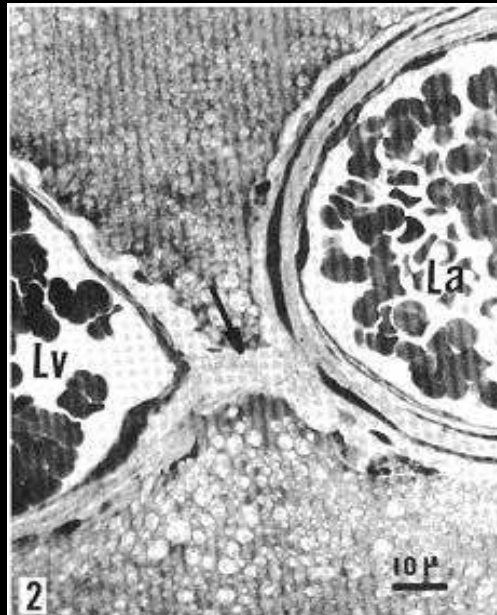
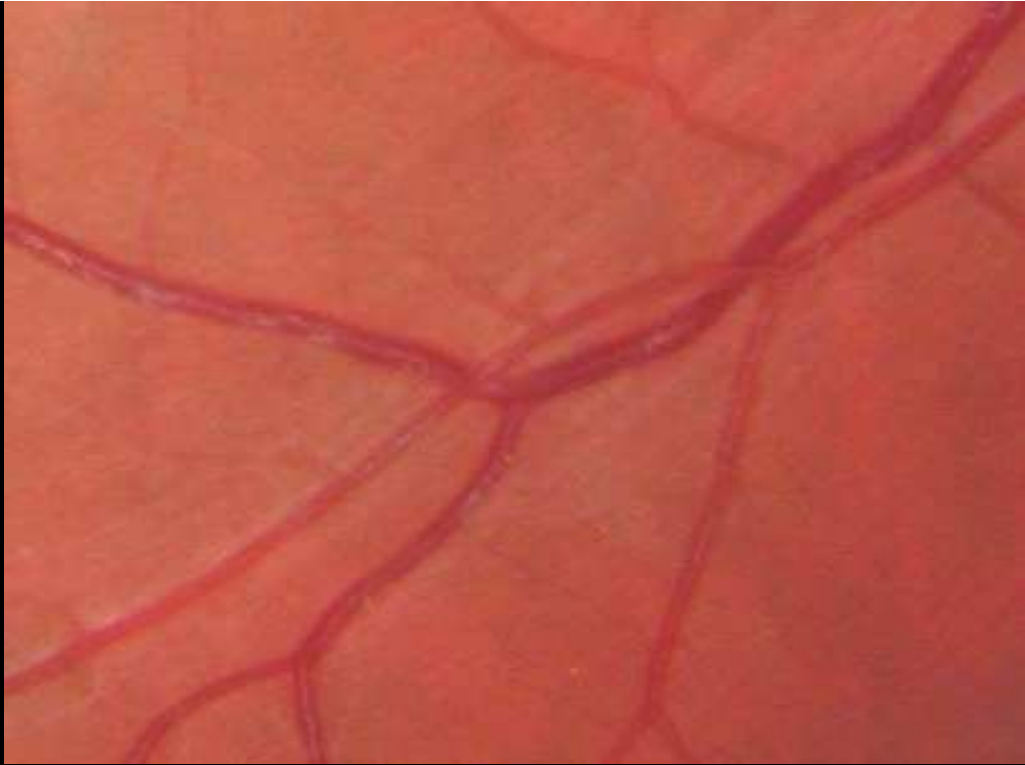
- Veine centrale de la rétine
- Héli-veine centrale
- Branche veineuse
- Excavation papillaire



OCCLUSIONS VEINEUSES RÉTINIENNES

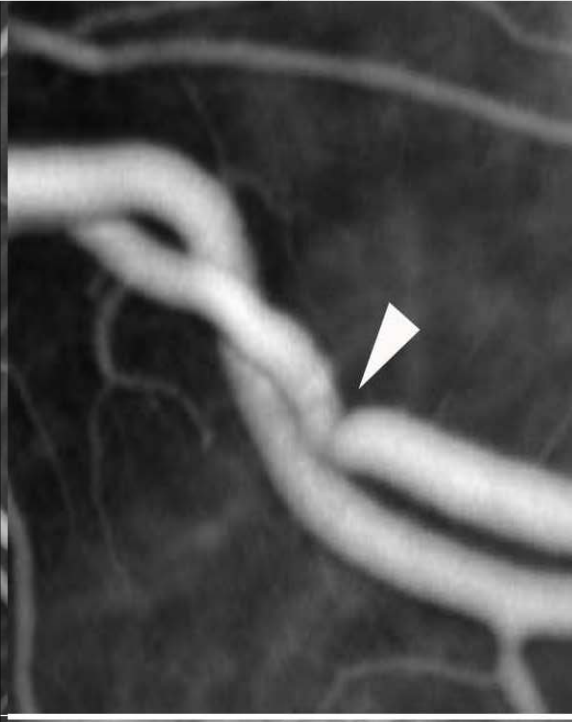
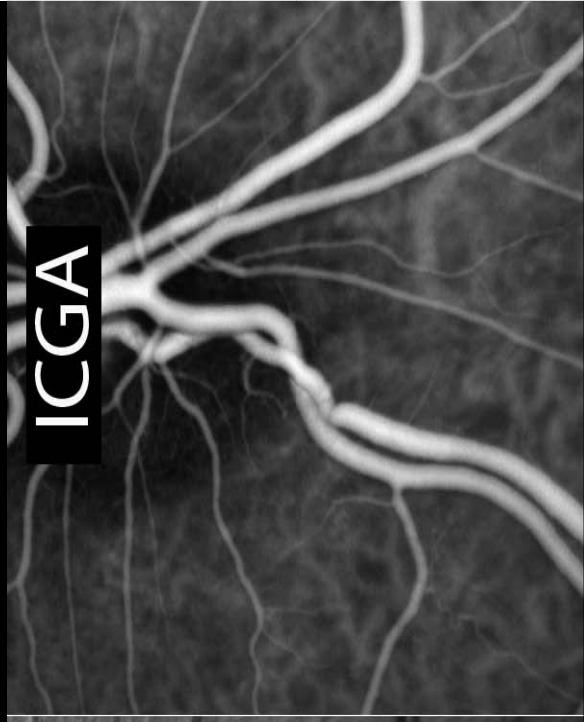
- Pathologie spécifique de l'œil
- Thrombose incertaine
- ≠ Occlusion complète
 - ➔ Maladie obstructive veineuse rétinienne

Quelle est la cause des OVR?

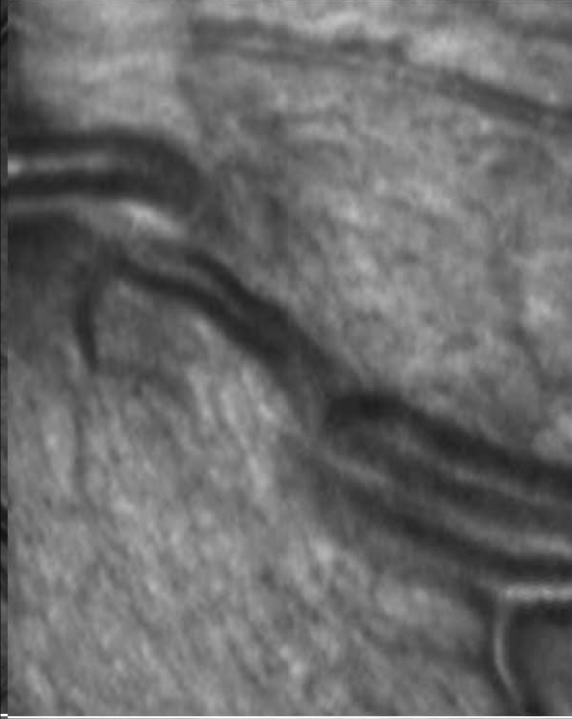
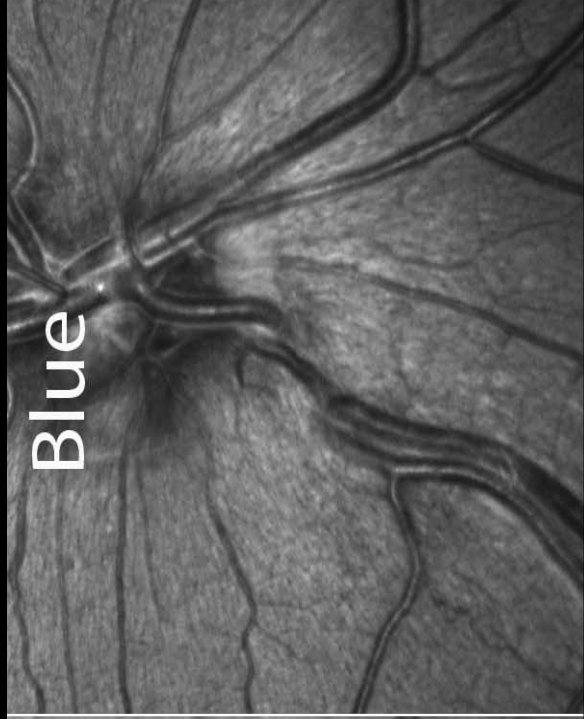


Adventice commune:
Fibres solidarissant l'artère et la veine
aux croisements AV

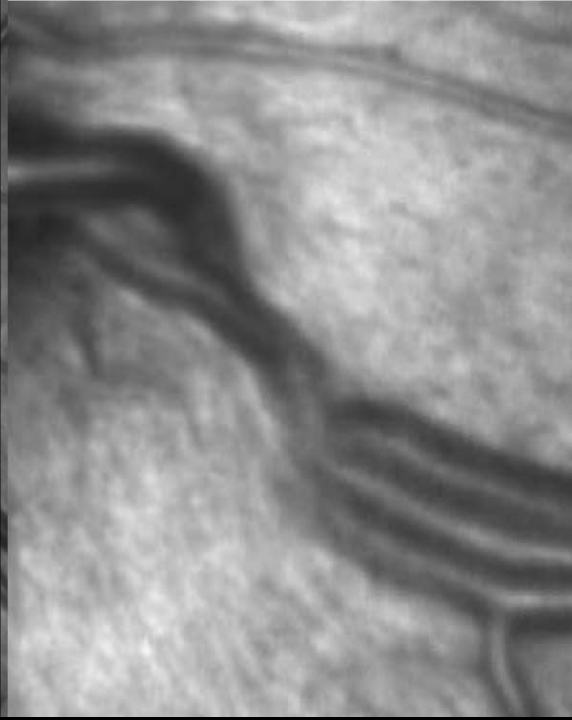
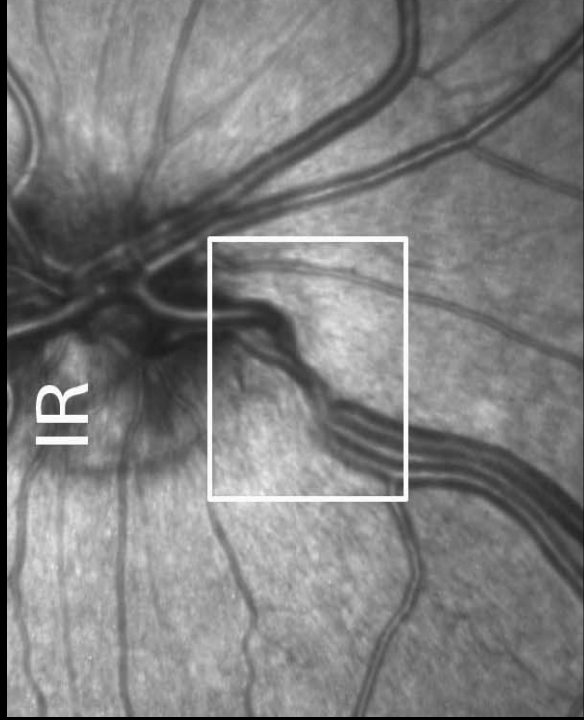
ICGA



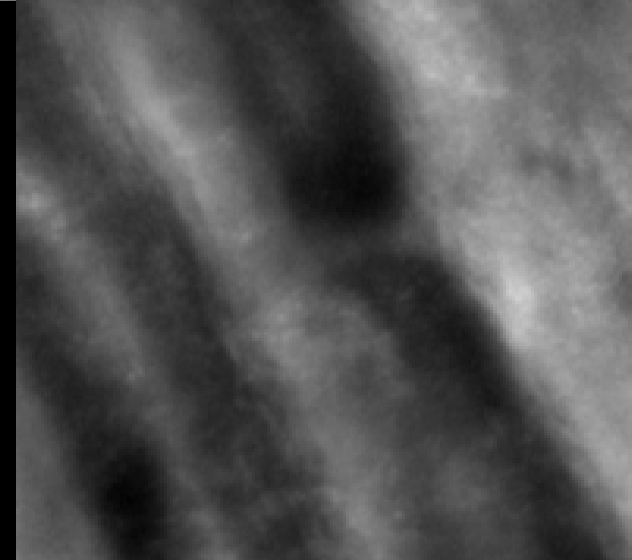
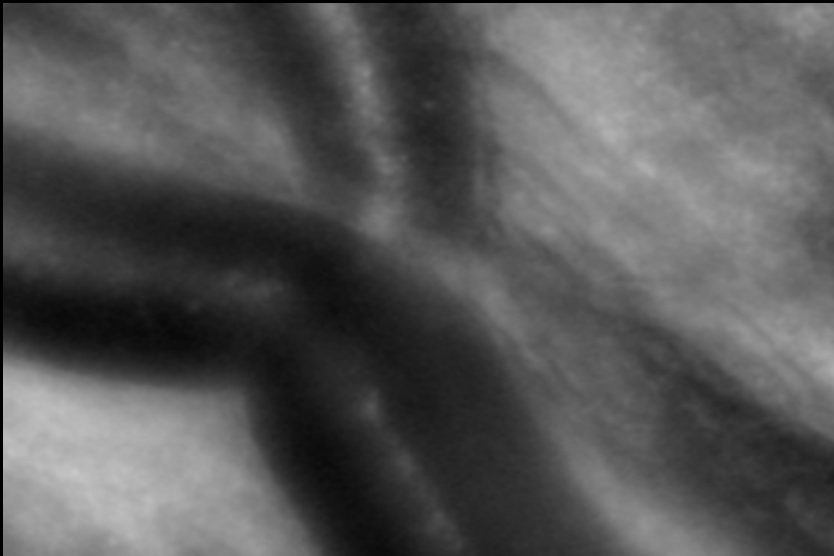
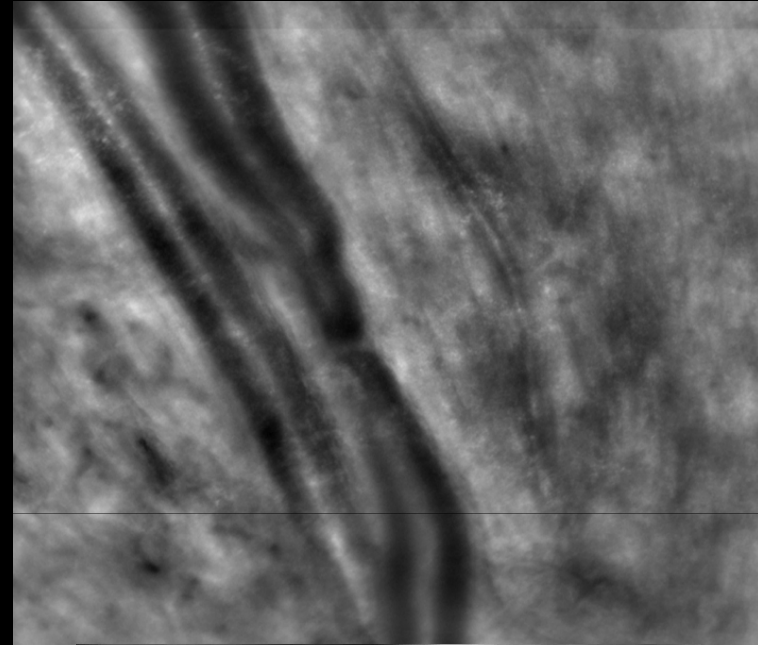
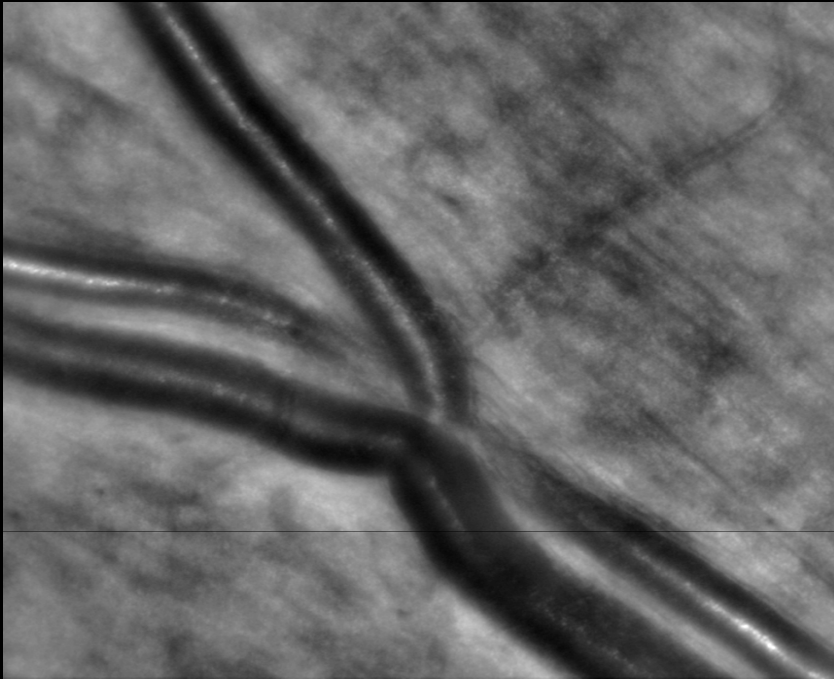
Blue



IR



Le « signe du croisement »; l'artère écrase-t-elle vraiment la veine?



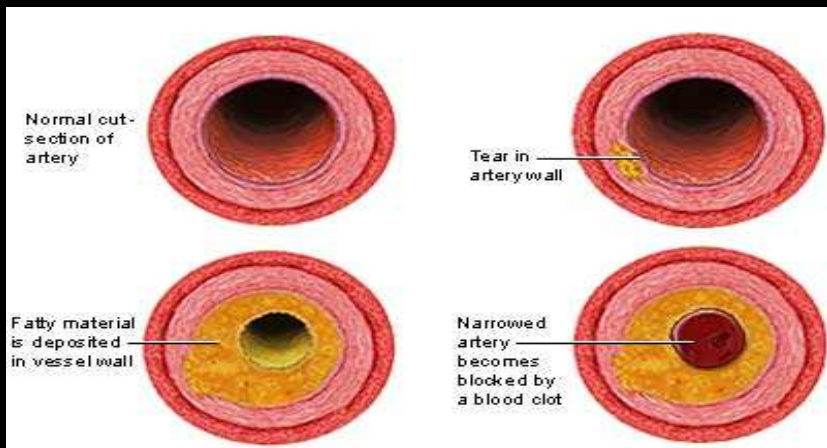
ATHÉROSCLÉROSE VS ARTÉRIOLOSCLÉROSE

Athérome

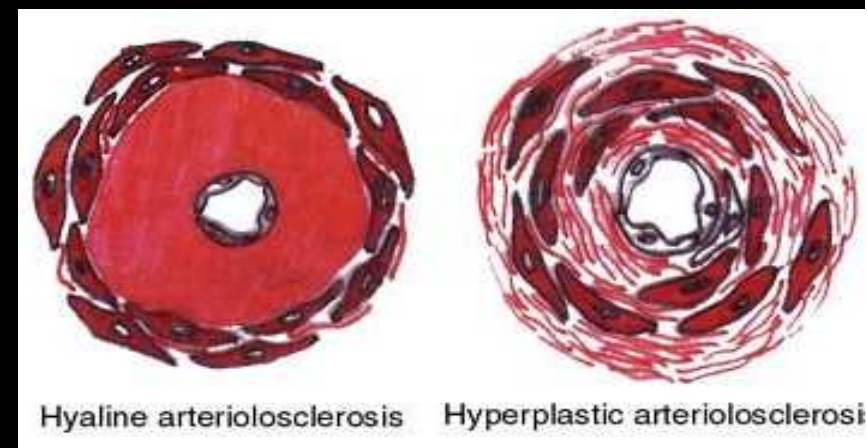
- Dépôts de cholestérol intracellulaire sous l'intima des grosses artères
- Thrombose artérielle
- FDR: HCT, HTA, Hérité, diabète

Artériolosclérose

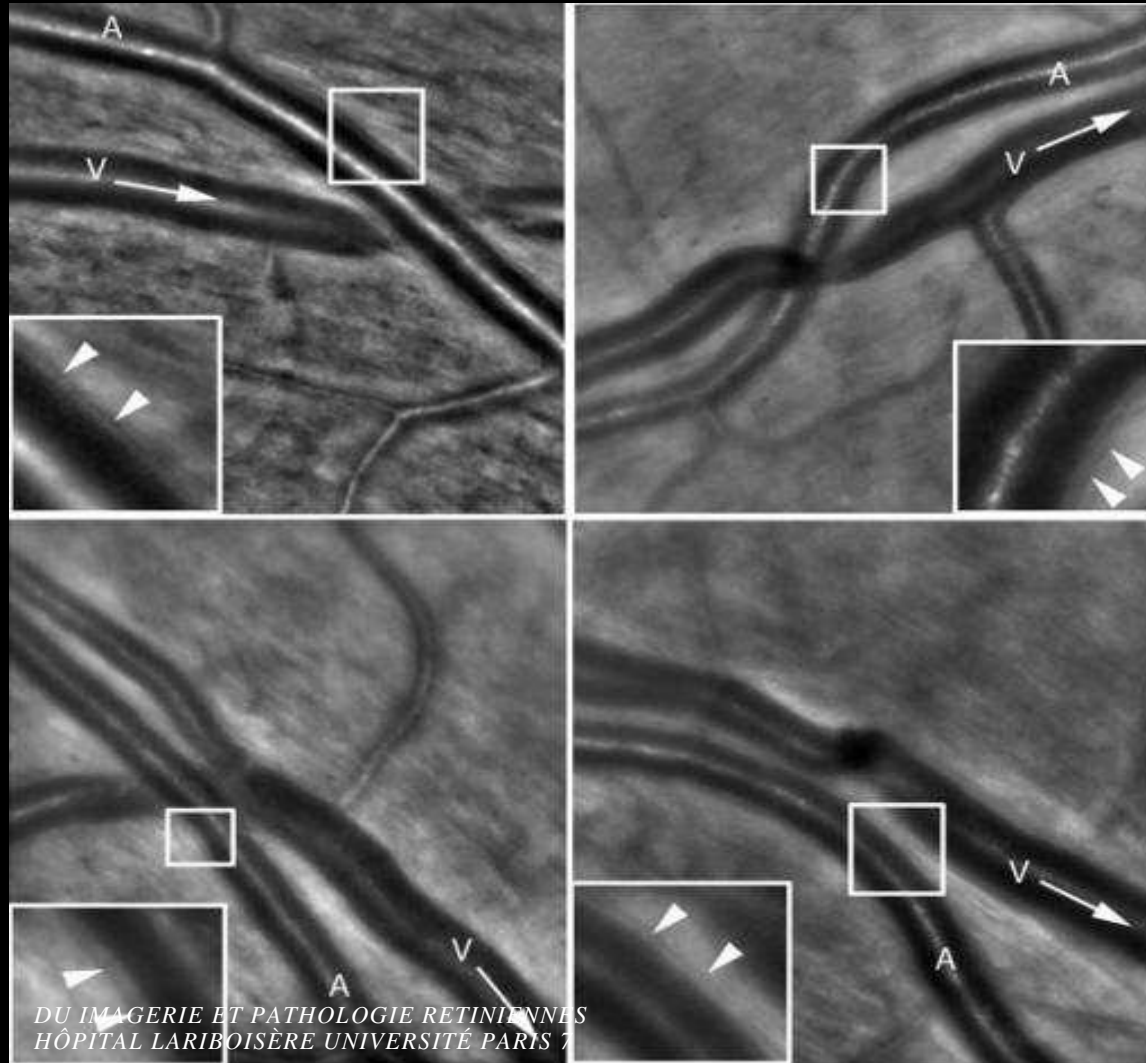
Hyperplasie et fibrose de l'adventice des artérioles
FDR: HTA, âge



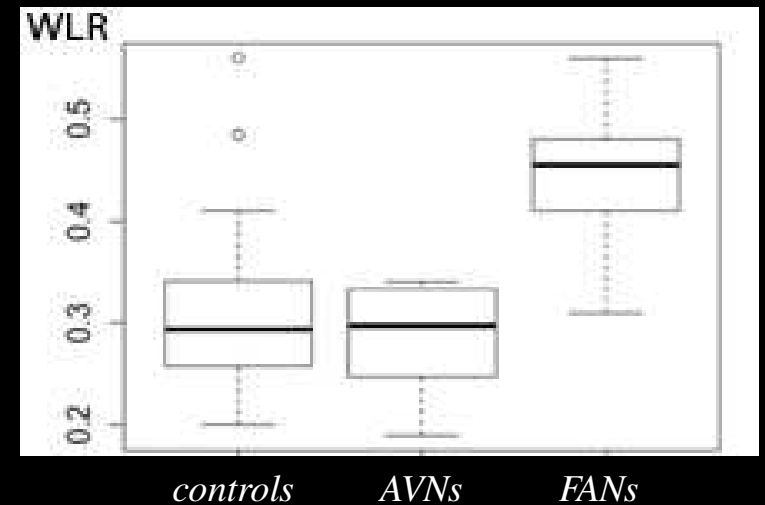
PATHOLOGIE RETINIENNES



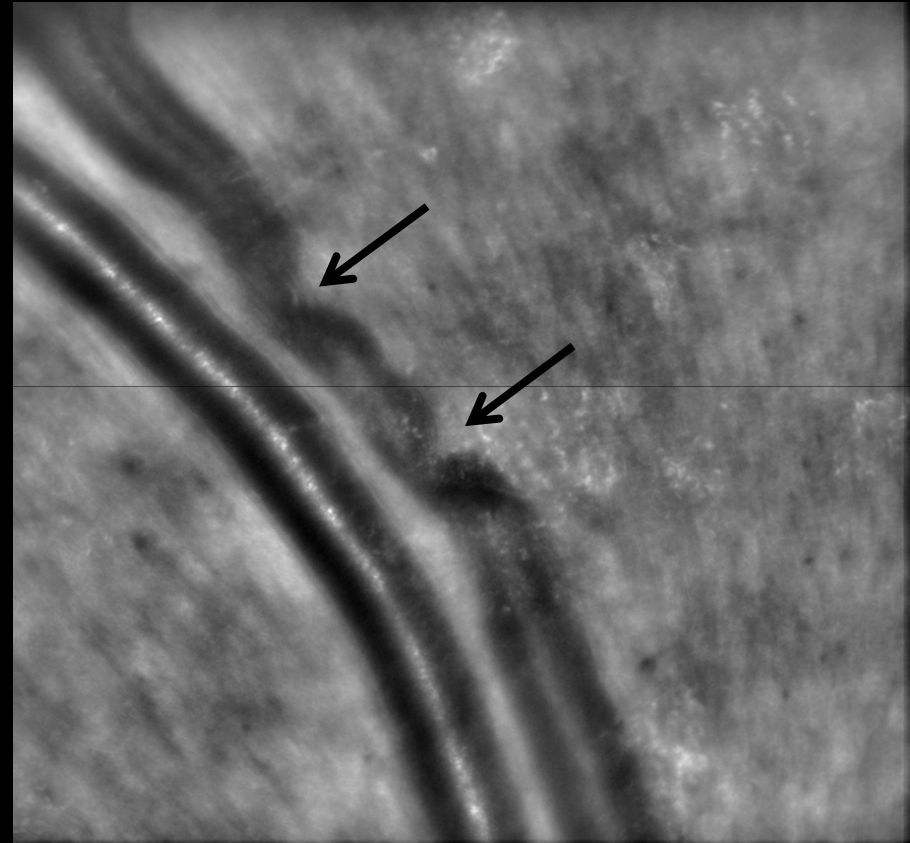
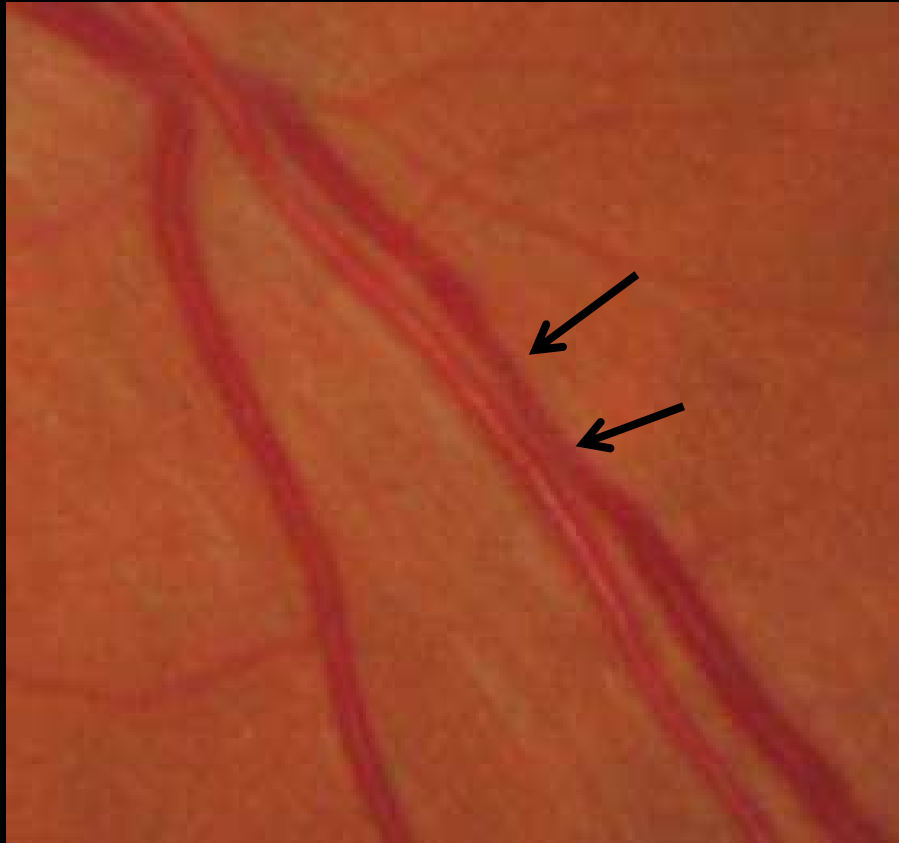
LA PAROI ARTÉRIOLAIRE EST-ELLE ÉPAISSIE DANS UN SIGNE DU CROISEMENT?



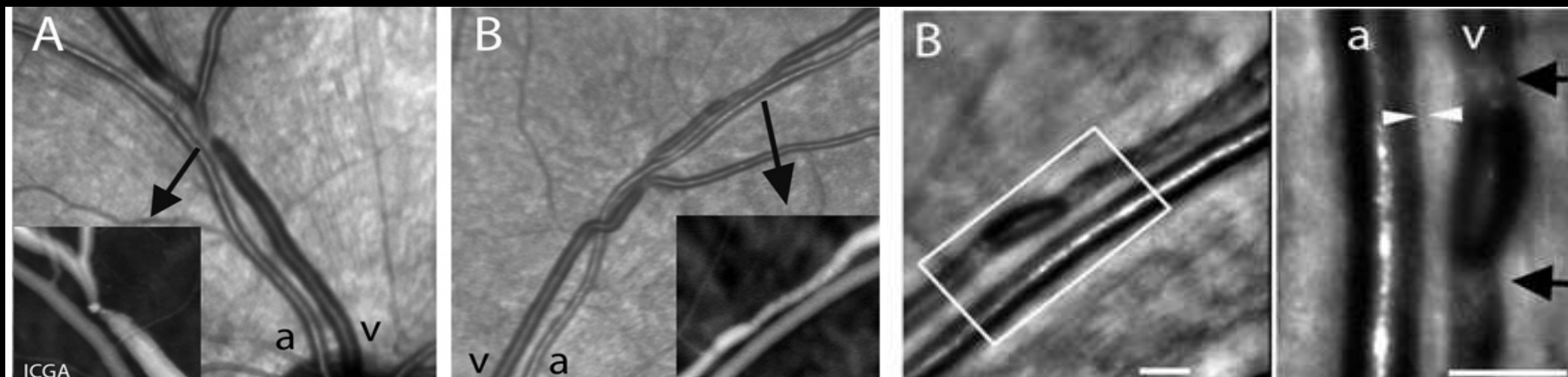
- La paroi artériolaire n'est pas épaissie



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
PHYSIQUE ENTRE ARTÈRE ET VEINE)

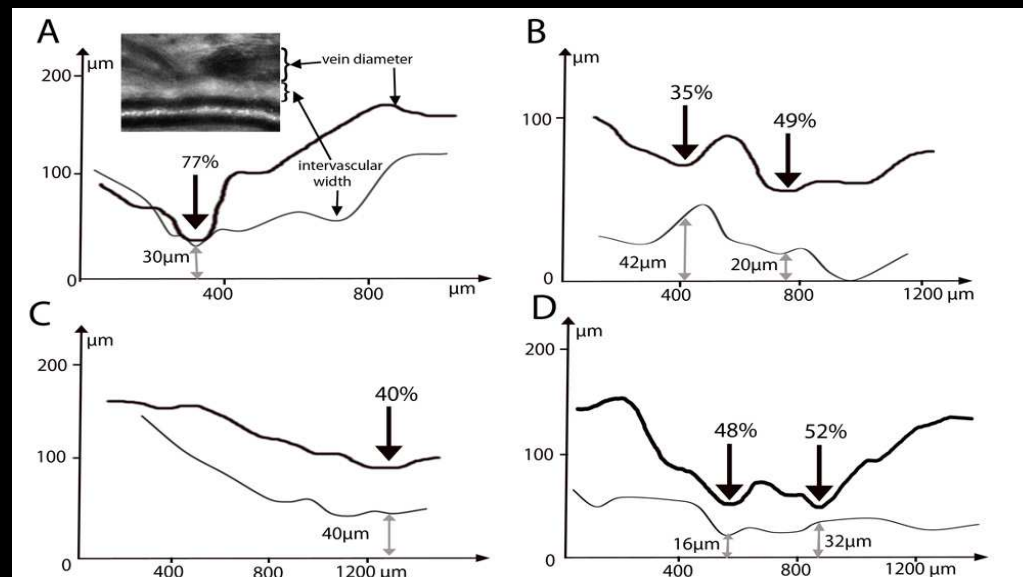
Brief Report

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

CONCLUSIONS AND RELEVANCE Arteriovenous nickings do not necessarily involve an arteriovenous compression. Instead, the topology of venous changes suggests a retractile process originating in the intervacular space. These findings have important implications for the understanding of retinal vein occlusions and of cerebrovascular aging.

JAMA Ophthalmol. 2015;133(8):947-950. doi:10.1001/jamaophthalmol.2015.1132
Published online May 21, 2015.

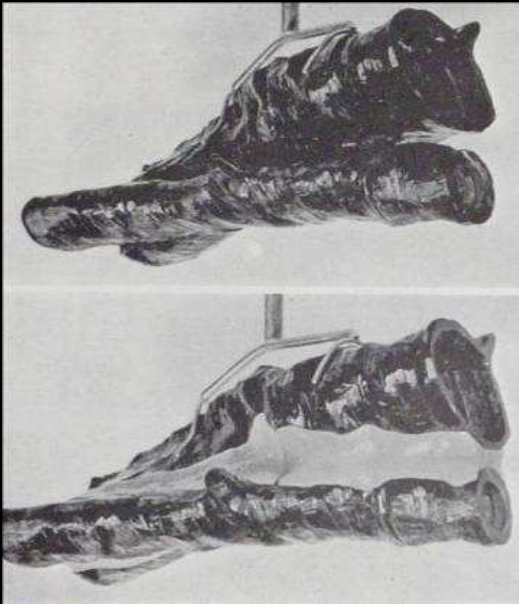


Paques et al, JAMA Ophth 2015

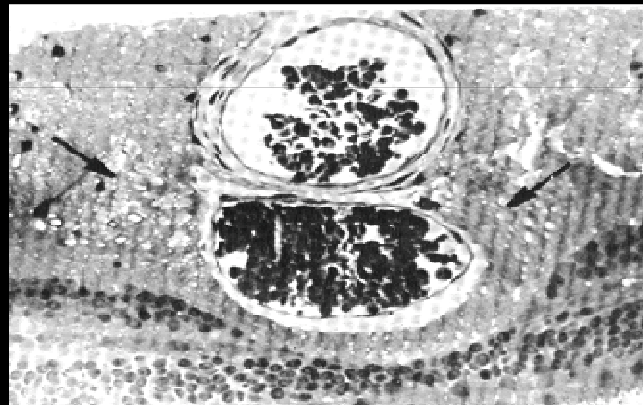
ALORS QUE SE PASSE-T-IL DANS UN SIGNE DU CROISEMENT?

L'histologie montre

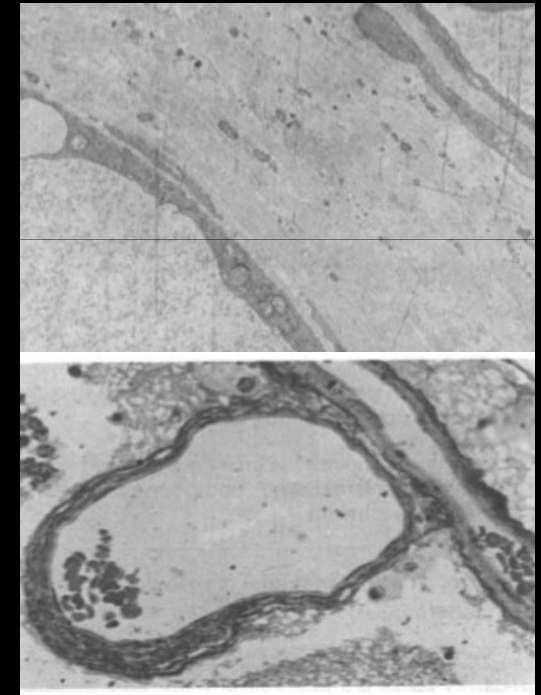
- *des modifications histologiques rétinienne autour du couple artérioveineux*
- *Mais que la veine n'est pas directement atteinte par l'artère*



Seitz et coll. 1964

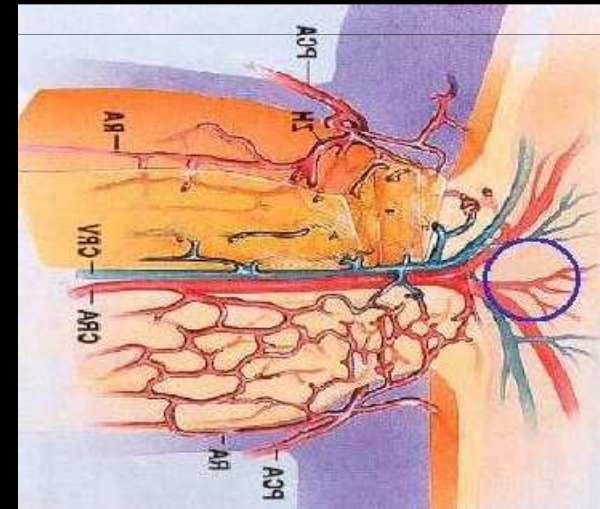
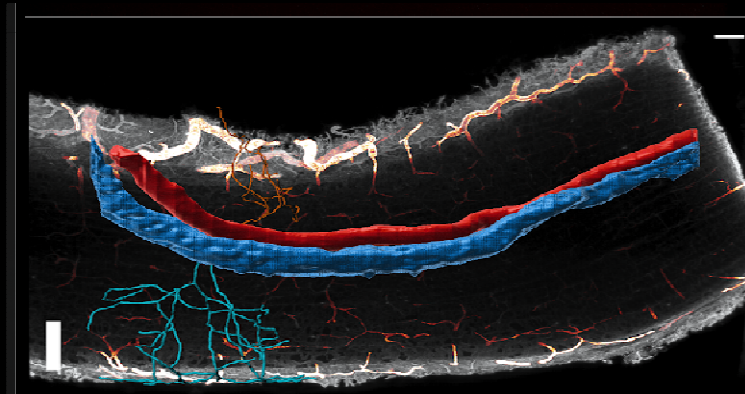


Kimura et al. Int Ophthalmol 2005



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

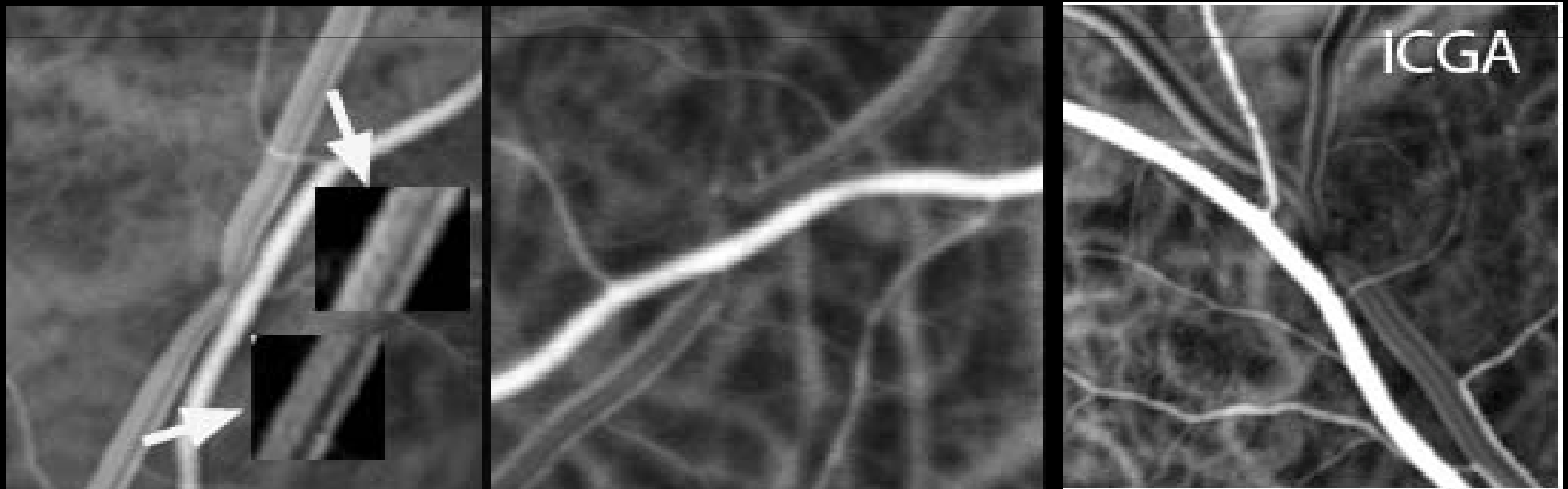
→ Fausse évidence de l'écrasement veineux par l'artère



« tout entre et tout sort par le nerf optique »

COURANT LAMINAIRE ET SIGNE DU CROISEMENT

- Le courant laminaire peut être préservé au niveau du croisement artériovéineux
- *Pas de turbulence du flux même si sténose serrée*



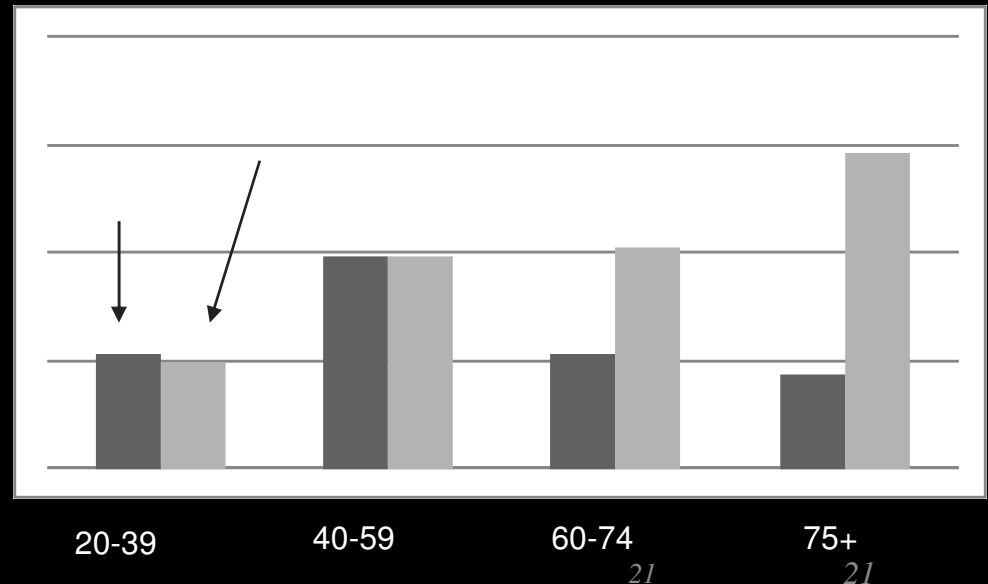
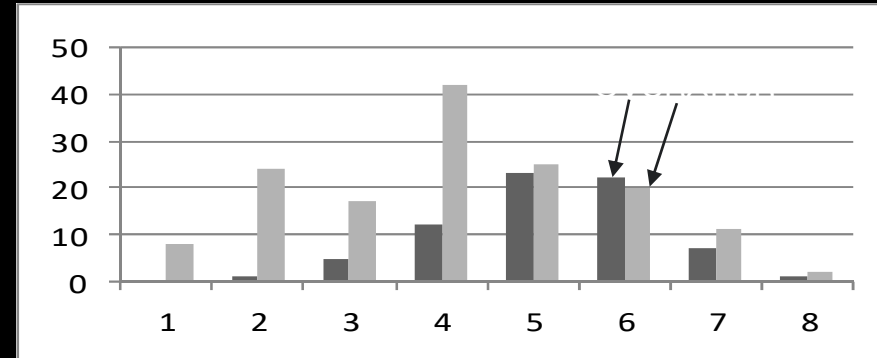
Incidence OVR

- 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

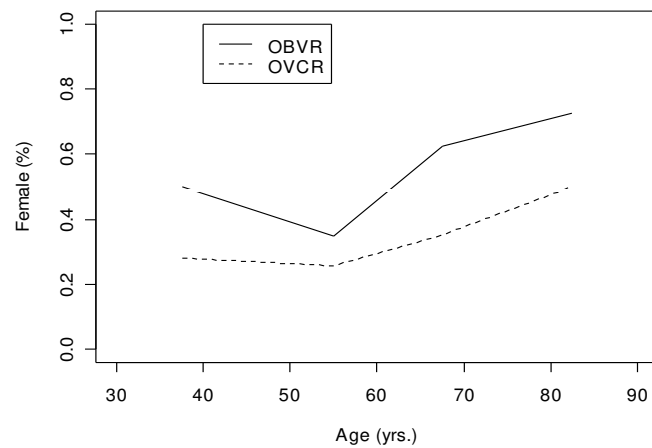
- Les plus de 75 ans représentent 7.3% de la population générale, mais 17% des OVCR
 - Incidence des OVR augmente tout au long de la vie
 - Age = premier facteur de risque



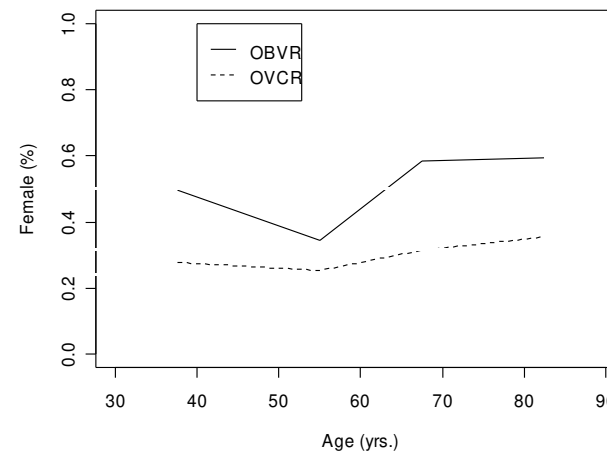
ES OVCR SONT PLUS FRÉQUENTES CHEZ LES HOMMES

	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

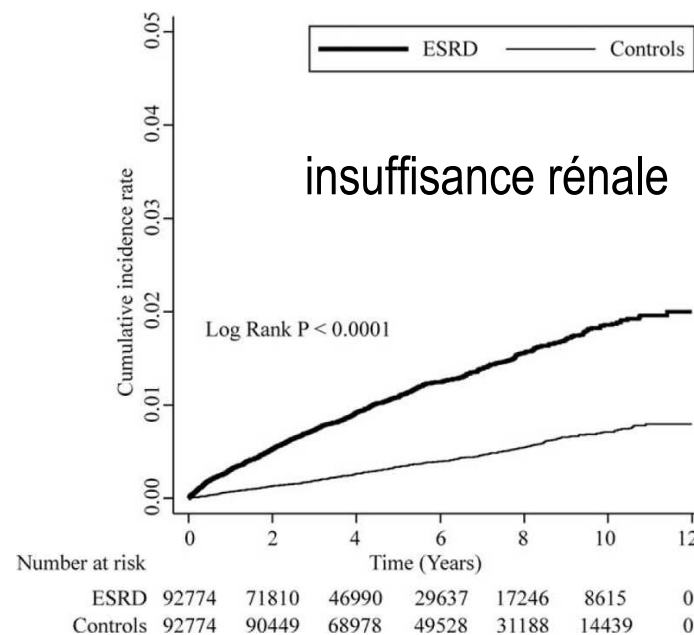
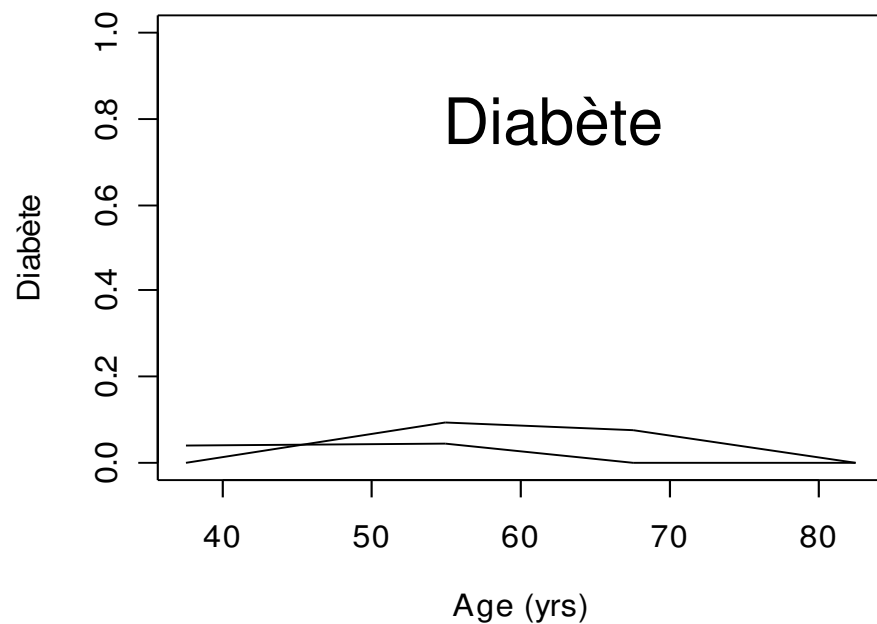
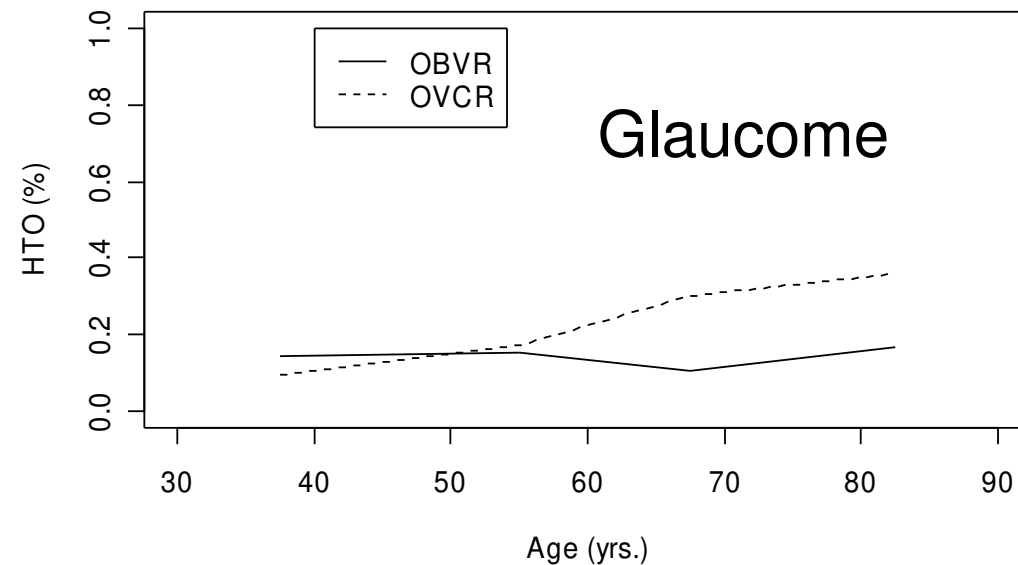
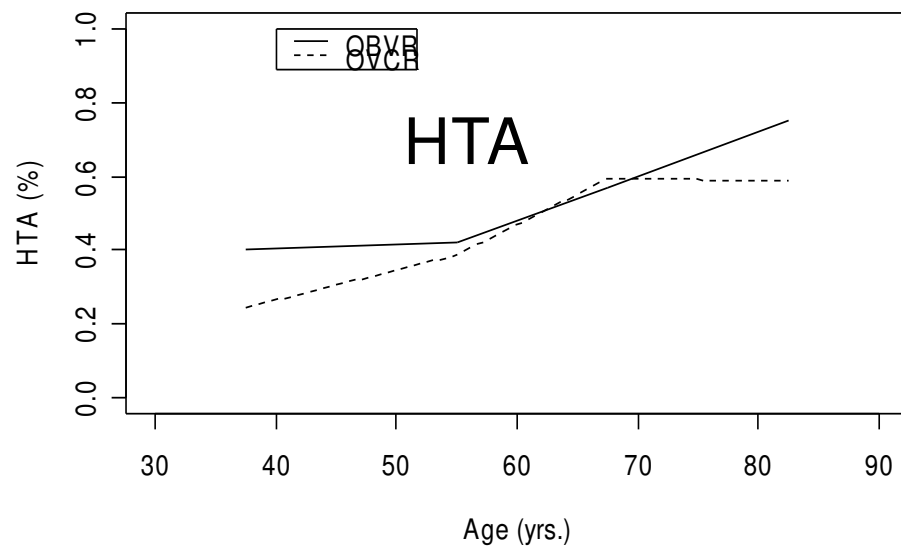


HTA

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.*

Y A T-IL DES ANOMALIES DE LA COAGULATION?

Y A T-IL DES ANOMALIES DE LA COAGULATION?

NON

OVR: 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

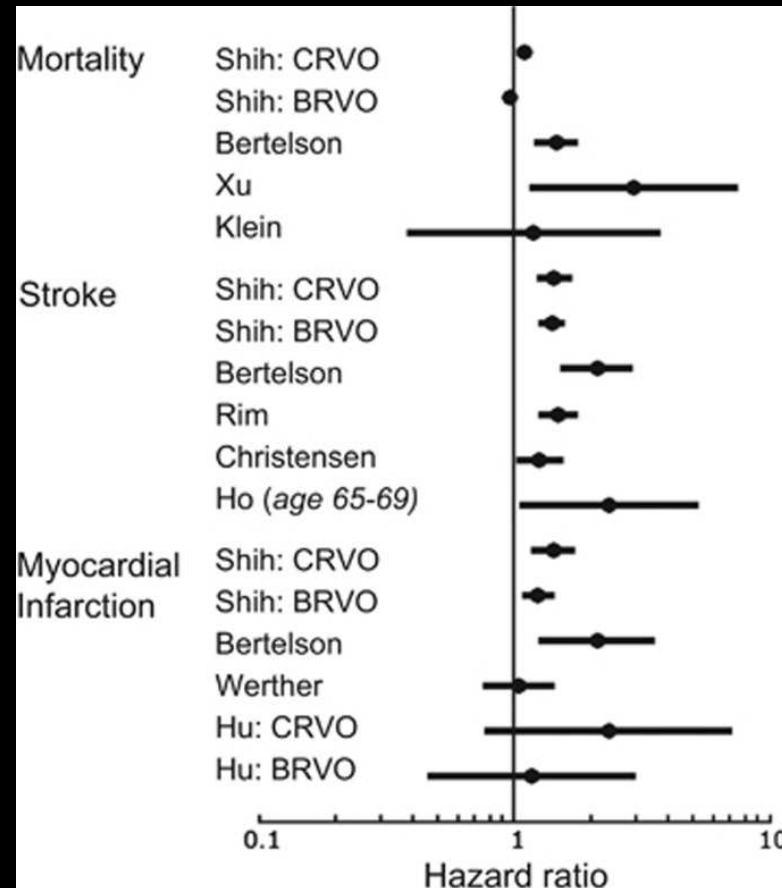
Of the 35 634 sampled patients (5965 sleep apnea patients vs 29 669 controls), 52 (0.15%) experienced RVO during a mean follow-up period of 3.72 years, including 13 (0.22%, all branch RVO) from the sleep apnea cohort and 39 (0.13%, 39 branch RVO and 10 central RVO) from the control group. Kaplan-Meier 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

Facteurs de risque des OVCR

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

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

L'OVR EST-ELLE RÉVÉLATRICE D'UN RISQUE PLUS ÉLEVÉ DE PATHOLOGIE VASCULAIRE?



Woo et al. Eye 2016

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

OVR et COVID

25% d'augmentation...

Retinal Vascular Occlusion and COVID-19 Diagnosis: A Multicenter Population-based Study

Jing-Xing Li ^{et al} *Retina* 2023

Abstract

Introduction: Several ocular diseases have been reported in patients with coronavirus disease 2019 (COVID-19), especially retinal vascular occlusion. This study aims to examine the risk of retinal vascular occlusion following COVID-19 diagnosis.

Method: This retrospective cohort study was based on 46 healthcare organizations in the United States using the TriNetX network. Individuals who had laboratory confirmation of COVID-19 from January 1, 2020 to December 31, 2021 were included. Multivariate analysis was adjusted on age, sex, race and comorbidities, and hazard ratio was calculated by Cox proportional hazard regression model.

Results: 1,460,634 paired individuals were enrolled for analysis. Patients with COVID-19 had a significantly higher risk of branch retinal vein occlusion (BRVO) (hazard ratio [HR] 1.27, 95% confidence interval [CI] 1.04-1.52) than those without COVID-19. The cumulative incidence rate of BRVO was also significantly higher in patients with COVID-19 compared with those without COVID-19 (log-rank $p = 0.014$). Within 12 weeks following COVID-19 diagnosis, transient effect of central retinal vein occlusion (HR 1.59, 95% CI 1.15-2.17) and BRVO (HR 2.11, 95% CI 1.51-2.95) were observed.

Conclusions: This large-scale multicenter study demonstrated retinal vein occlusion may be associated with COVID-19.

... Pas d'augmentation

Retinal vascular occlusion risks during the COVID-19 pandemic and after SARS-CoV-2 infection

Hyo Song et al. *Sci report* 2023

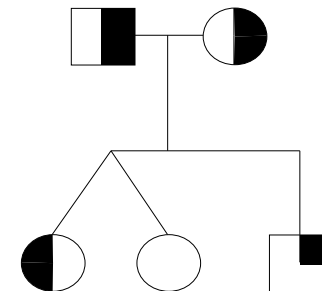
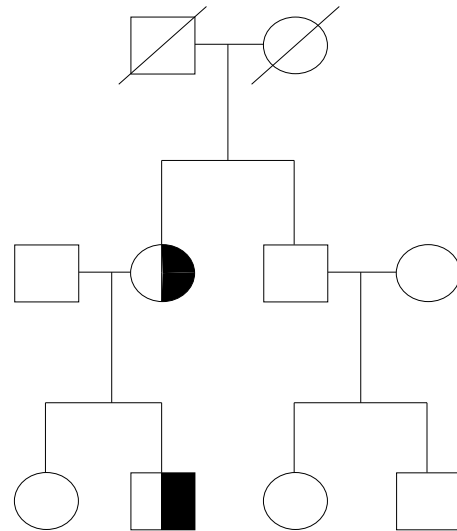
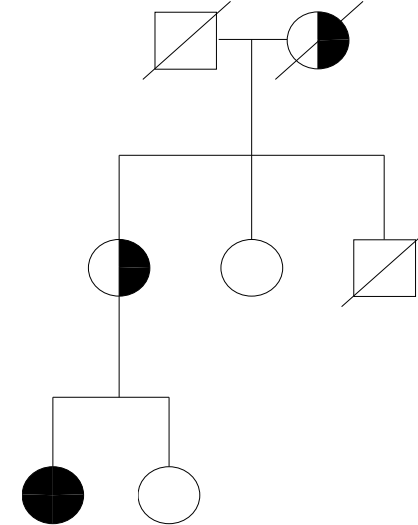
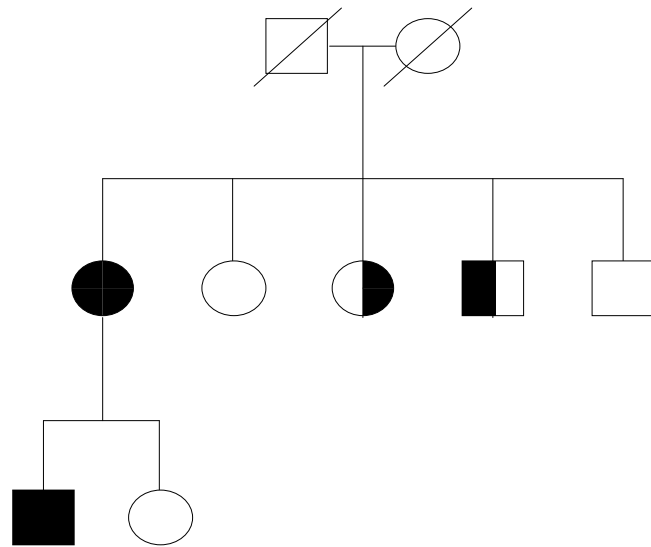
Abstract

The coronavirus disease 2019 (COVID-19) has been reported to affect vascular networks including the eye. However, evidence on the causal relationship between COVID-19 infection and retinal vascular occlusions remains limited.

This study aimed to determine the change in retinal vascular occlusion incidence during COVID-19 era and whether severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection induces retinal vascular occlusion.

Retinal vein occlusion (RVO) and retinal artery occlusion (RAO) incidences during 2018-2019 and 2020-July 2021 were compared, those in confirmed and suspected COVID-19 patients diagnosed from 2020 to January 2021 were calculated, and those in COVID-19 patients during 180 days prior and 180 days after diagnosis were assessed. Additionally, the standardized incidence ratio of RVOs in COVID-19 patients was analyzed. Incidence rates per 100,000 people/year of RVO during 2018-2019 and 2020-2021 was 102.0 and 98.8, respectively. RAO incidence rates during 2018-2019 and 2020-2021 were 11.7 and 12.0, respectively. In both confirmed and suspected COVID-19 patients, the incidence of RVO and RAO did not change significantly from 180 days before to after diagnosis in the adjusted model. RVO incidence slightly decreased while RAO incidence increased during the COVID-19 pandemic. SARS-CoV-2 infection did not significantly increase RVO or RAO incidence.

FORMES FAMILIALES D'OVR



Ne demandez plus de
« grand bilan »!

Sauf cas exceptionnel...
ou recherche

LE BILAN QUE JE DEMANDE

PIO

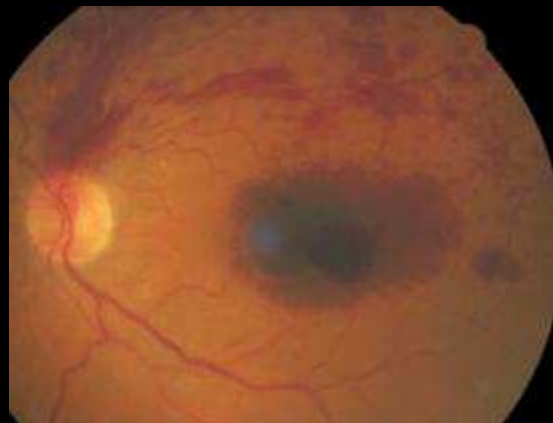
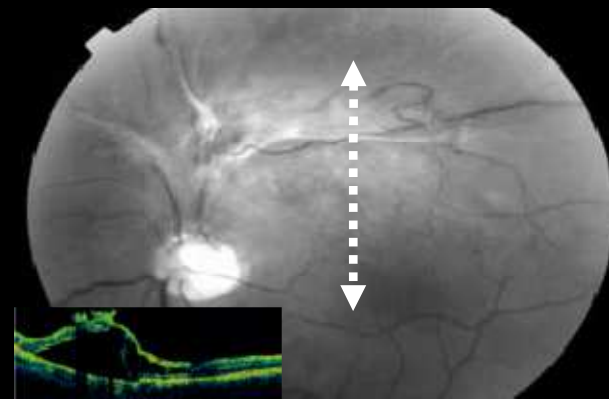
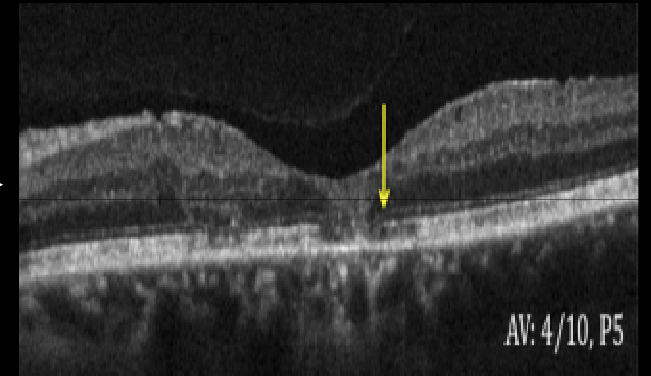
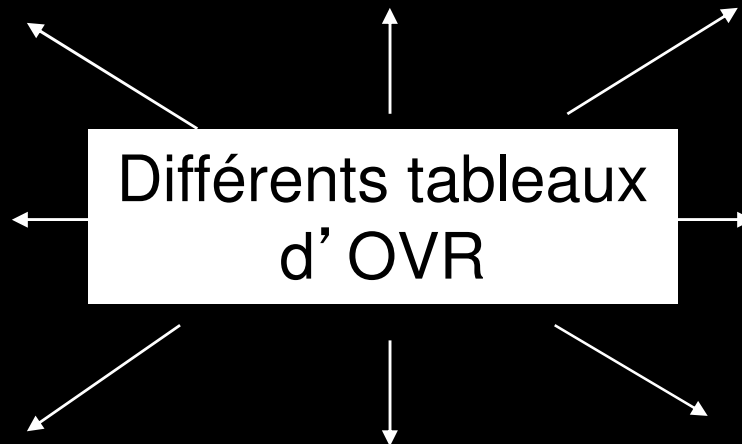
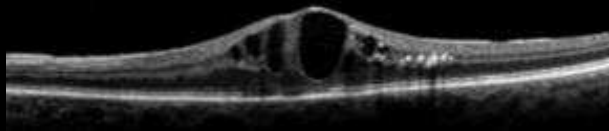
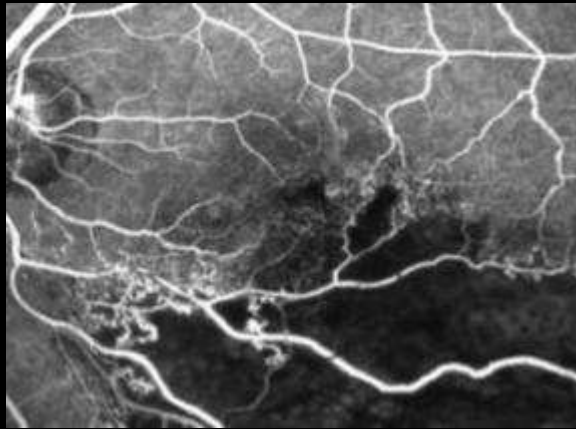
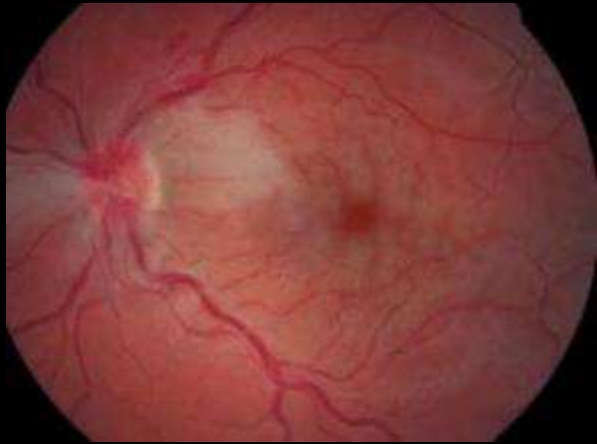
Pression artérielle

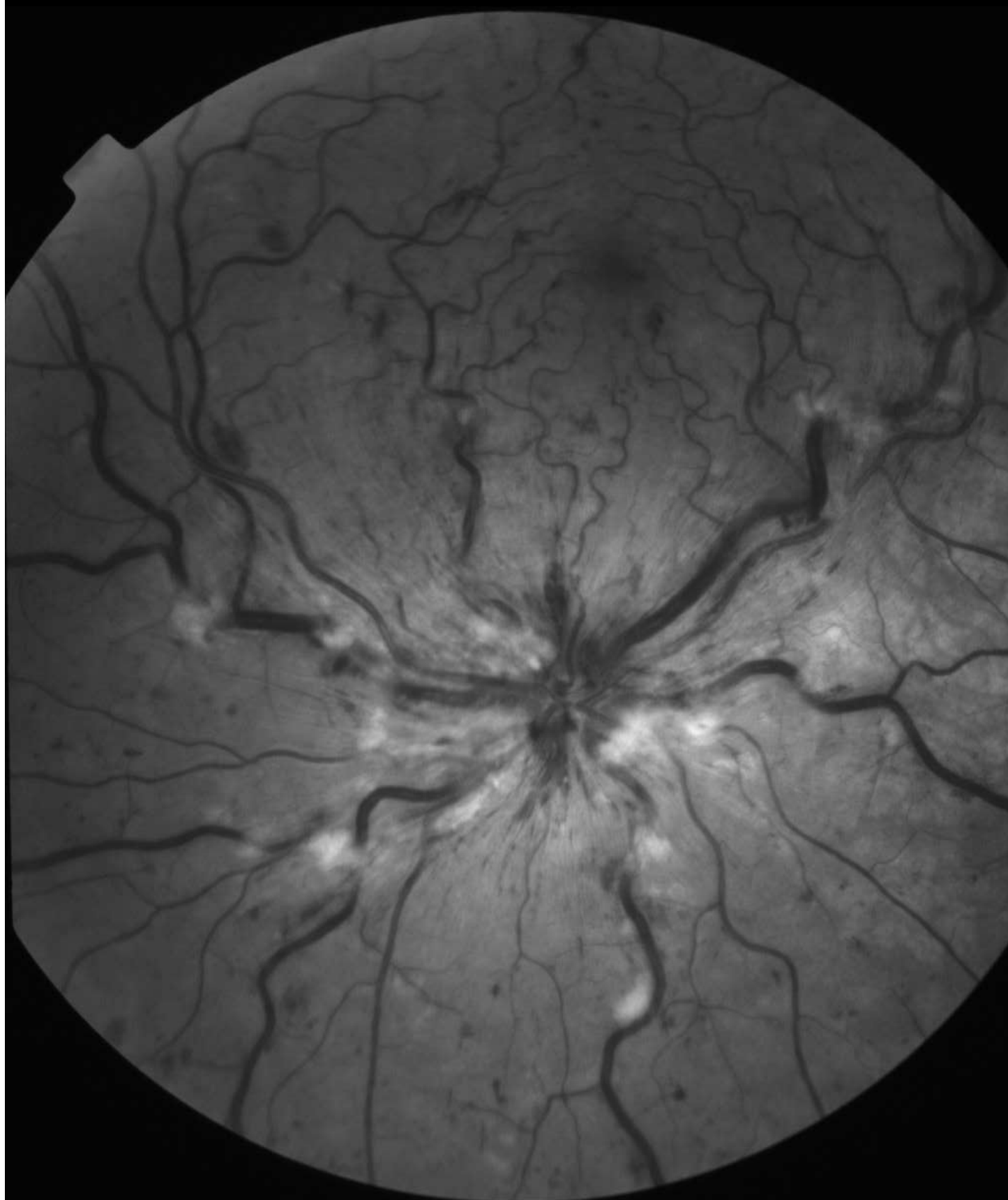
Glycémie

NFS

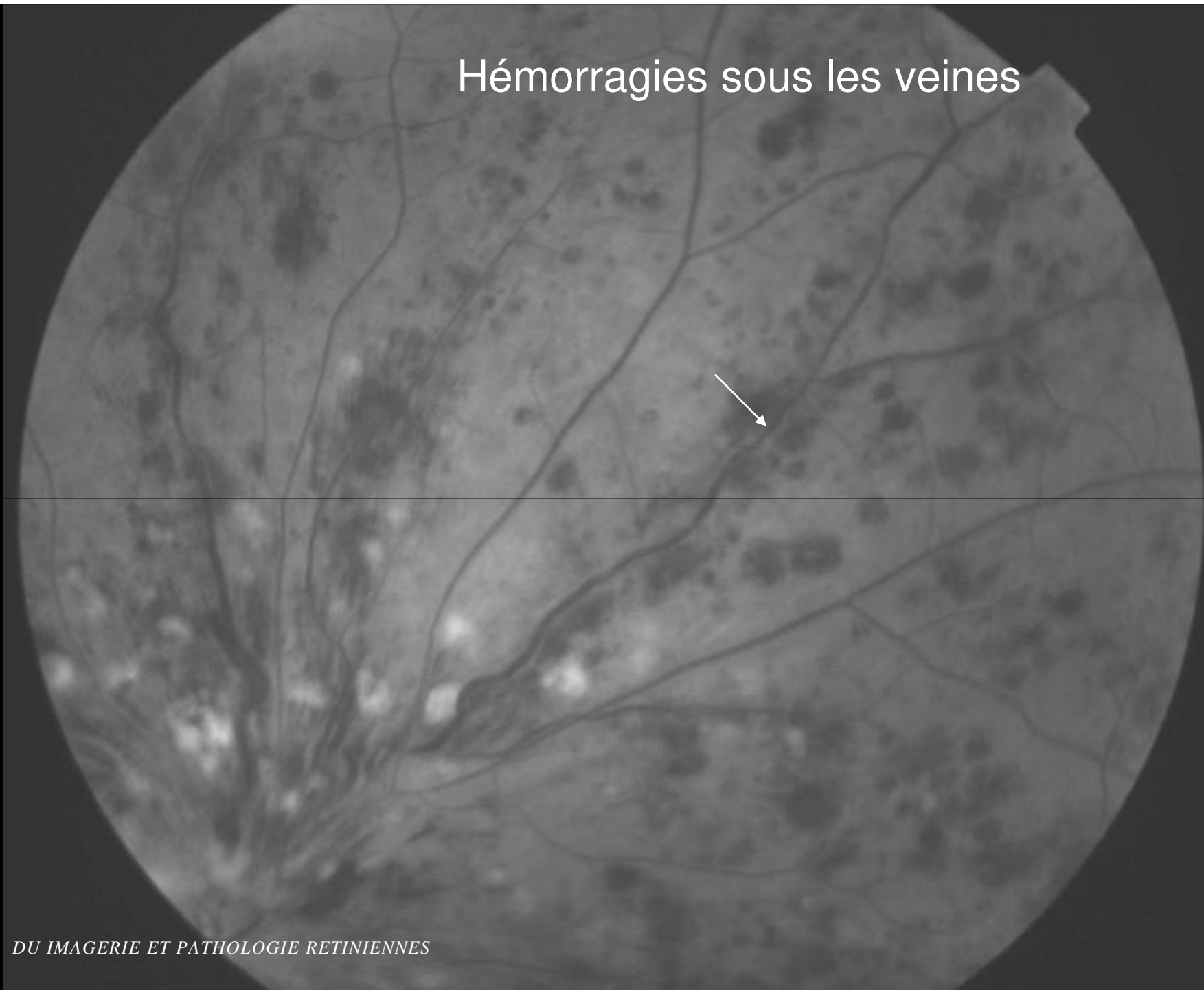
- Quel que soit l'âge... s'il n'y a AUCUN signe d'inflammation bien entendu
- Et c'est tout

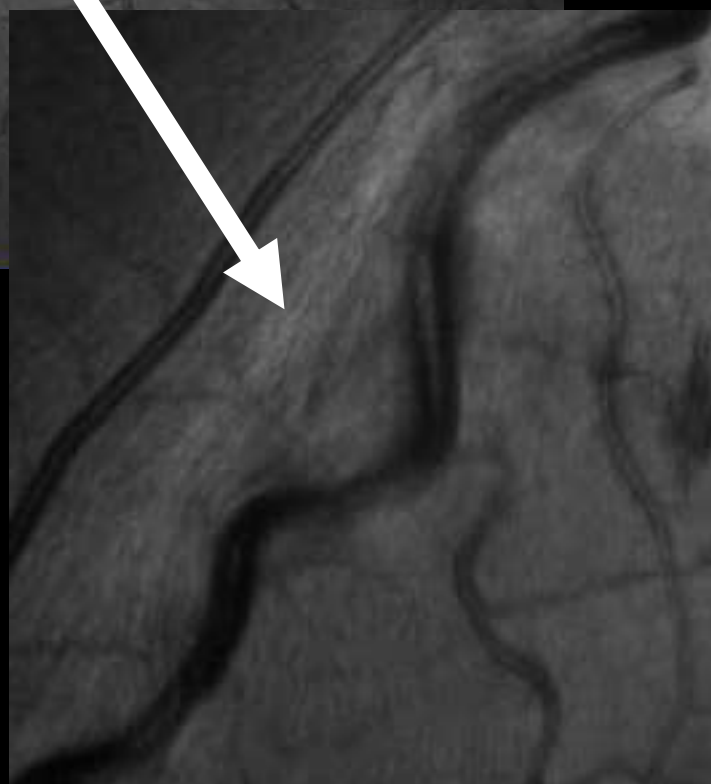
OVCR: ASPECTS CLINIQUES

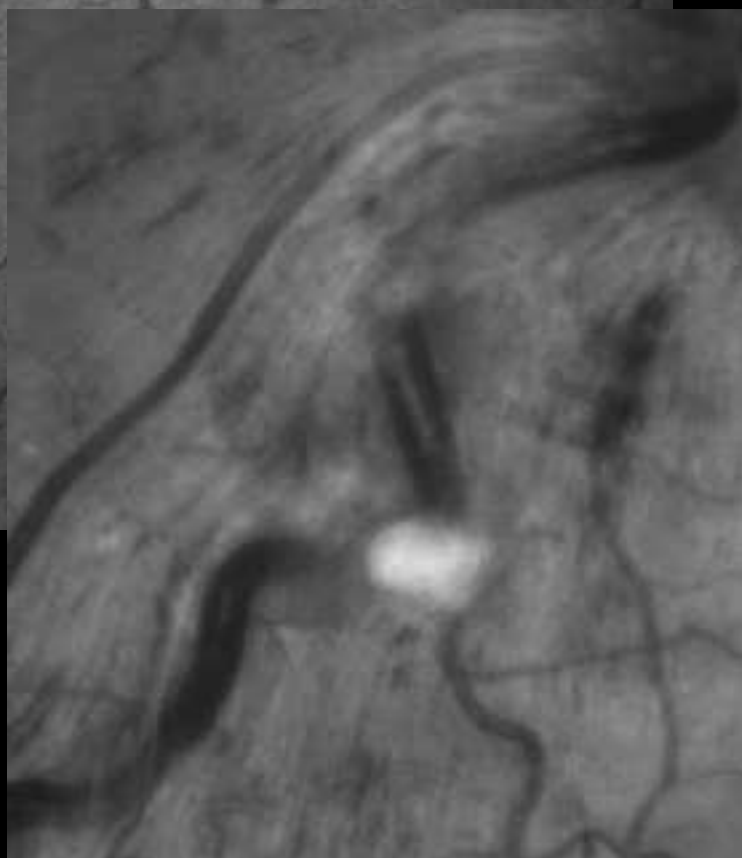




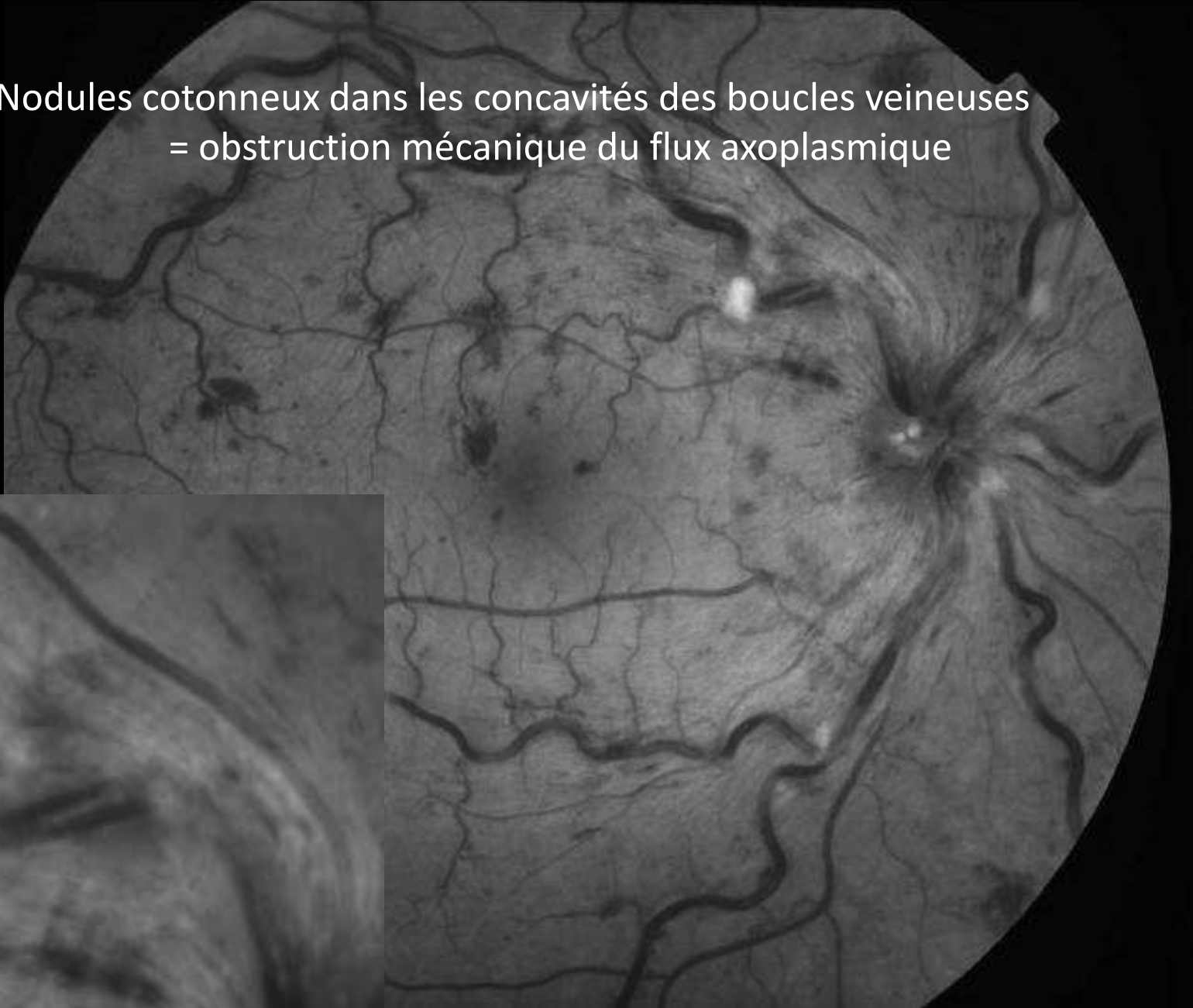
Hémorragies sous les veines



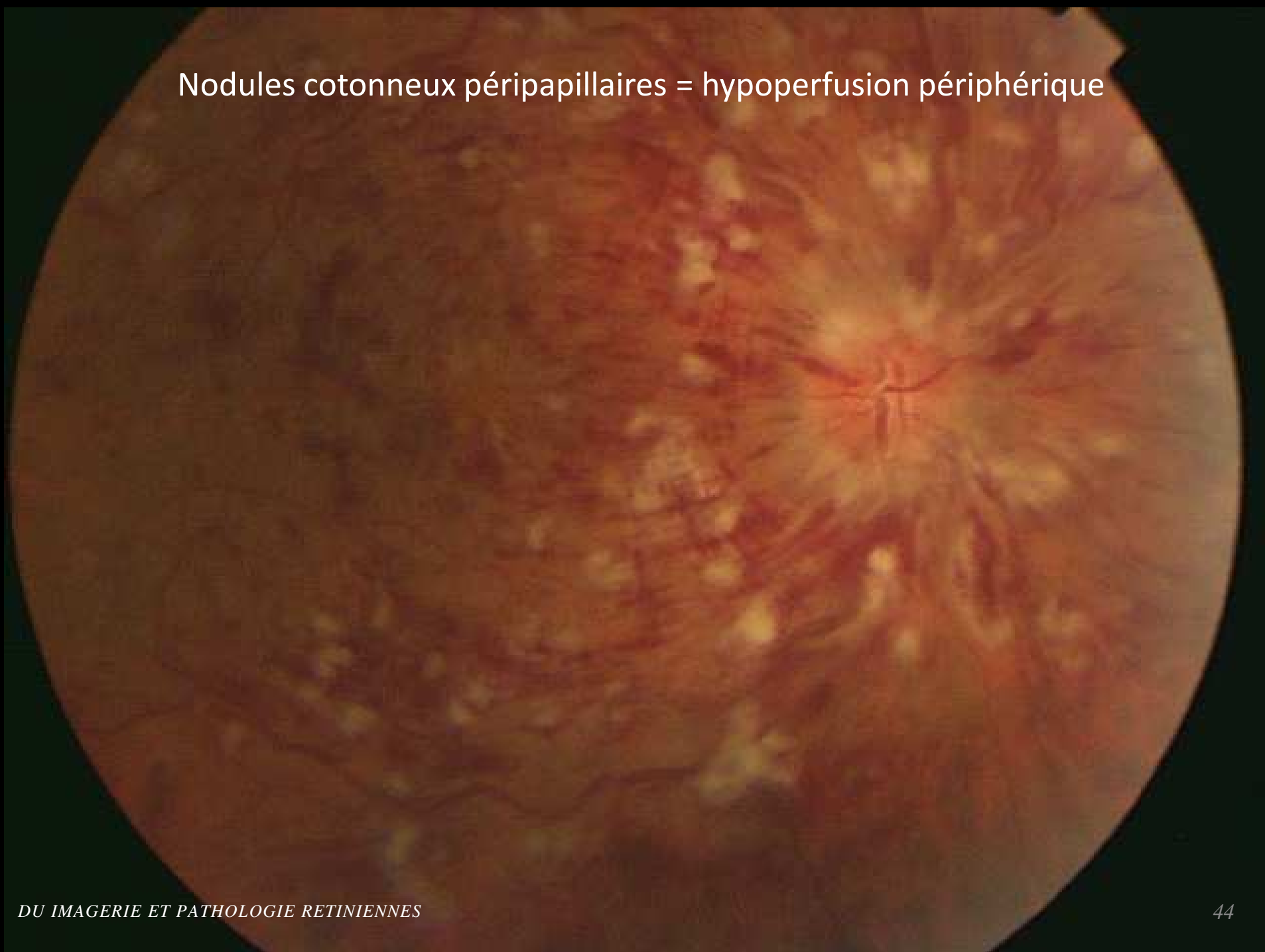




Nodules cotonneux dans les concavités des boucles veineuses
= obstruction mécanique du flux axoplasmique

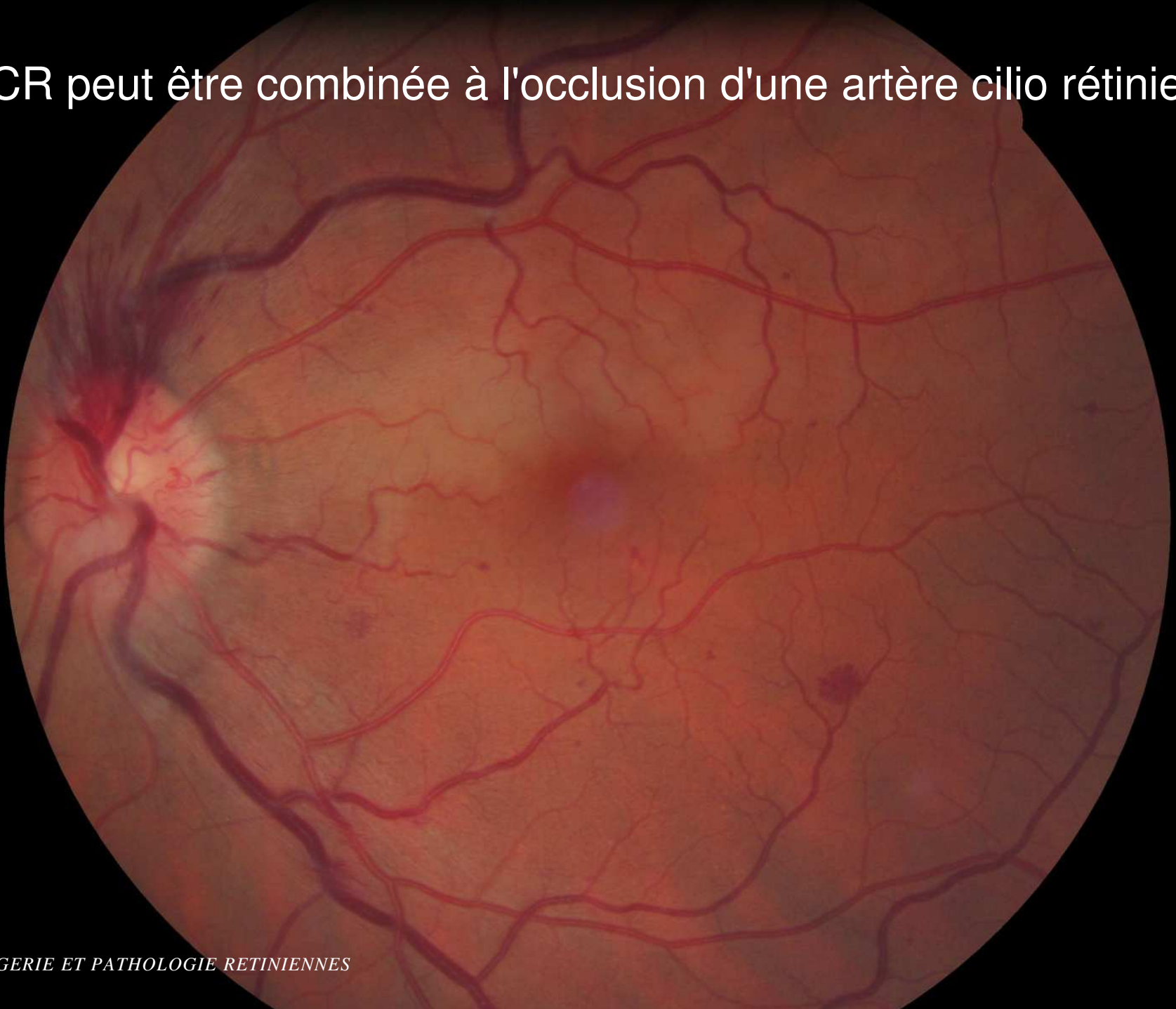


Nodules cotonneux péripapillaires = hypoperfusion périphérique



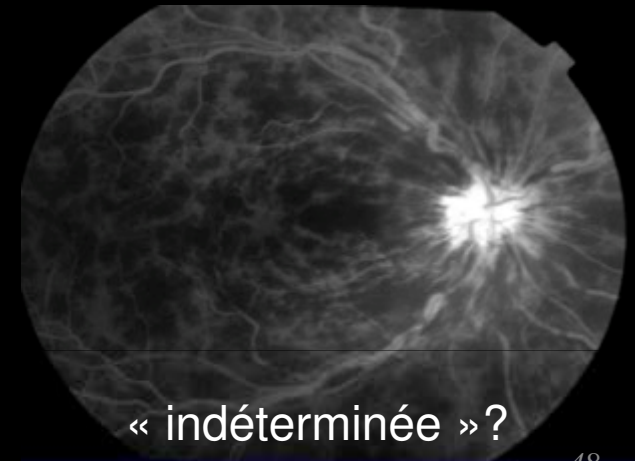
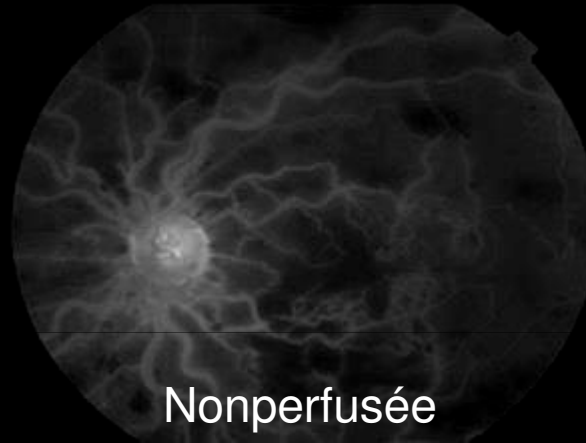
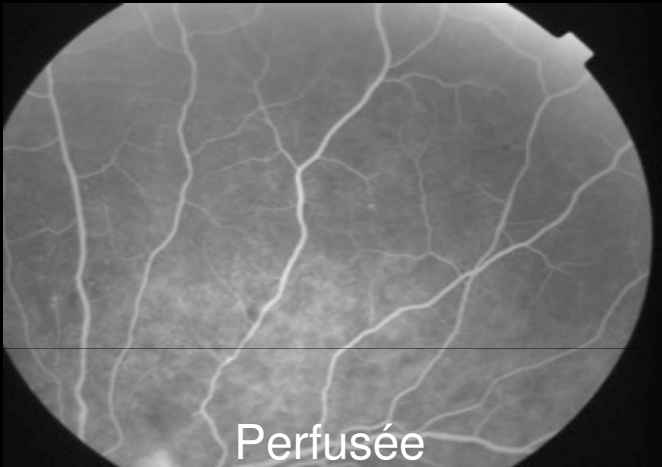


L'OVCR peut être combinée à l'occlusion d'une artère cilio rétinienne





CLASSIFICATION DES OVR



LIMITES DES CLASSIFICATIONS ACTUELLES

➤ Essentiellement basée sur des critères angiographiques

➤ « Perfusée / non-perfusée »

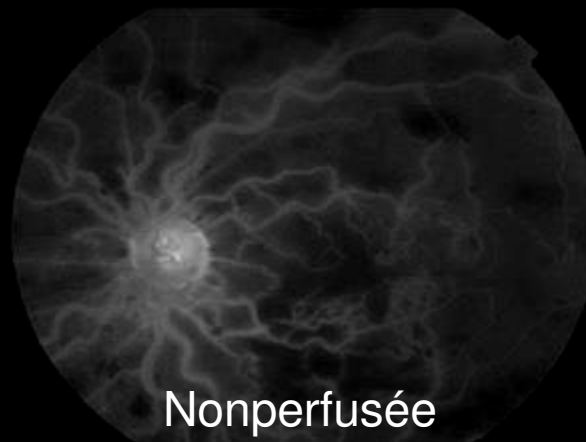
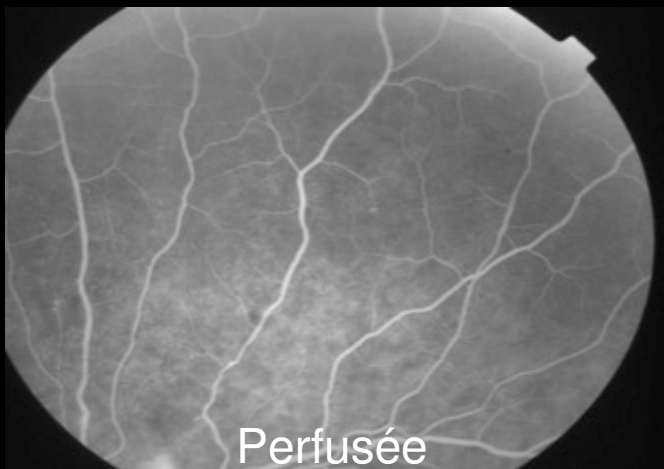
➤ « Ischemique / non-ischémique »

➤ 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





➤ 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

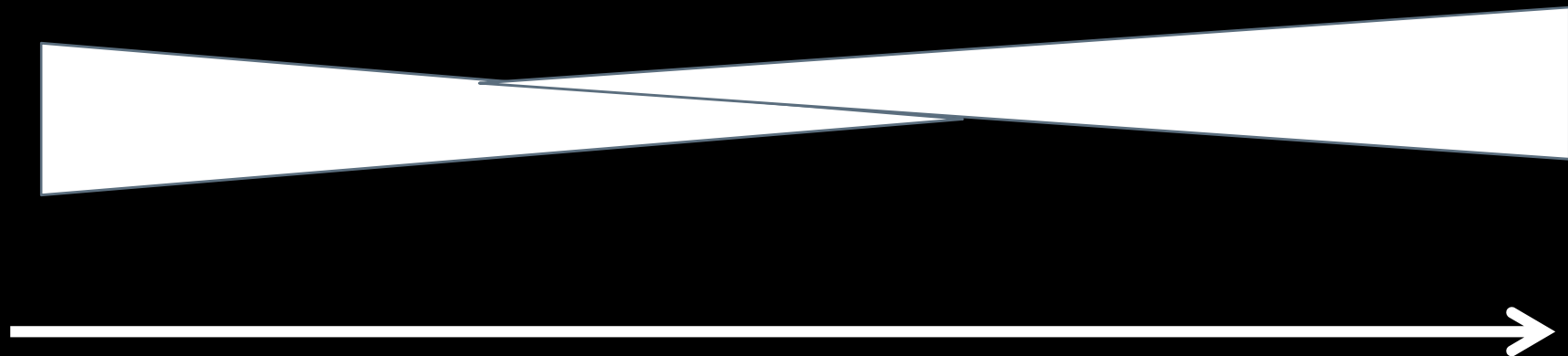
➤ Indépendamment de l'acuité visuelle, de la localisation, de l'angiographie

➤ OBVR est toujours du type B

➤ Les hémioclusions peuvent être de type A

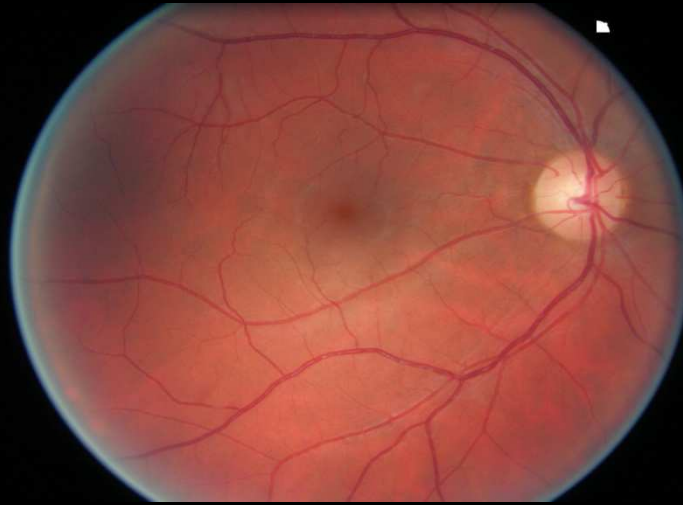
*Formes à retentissement artériel
(« type A » « PAMM »)*

*Formes
chroniques (« type B »)*



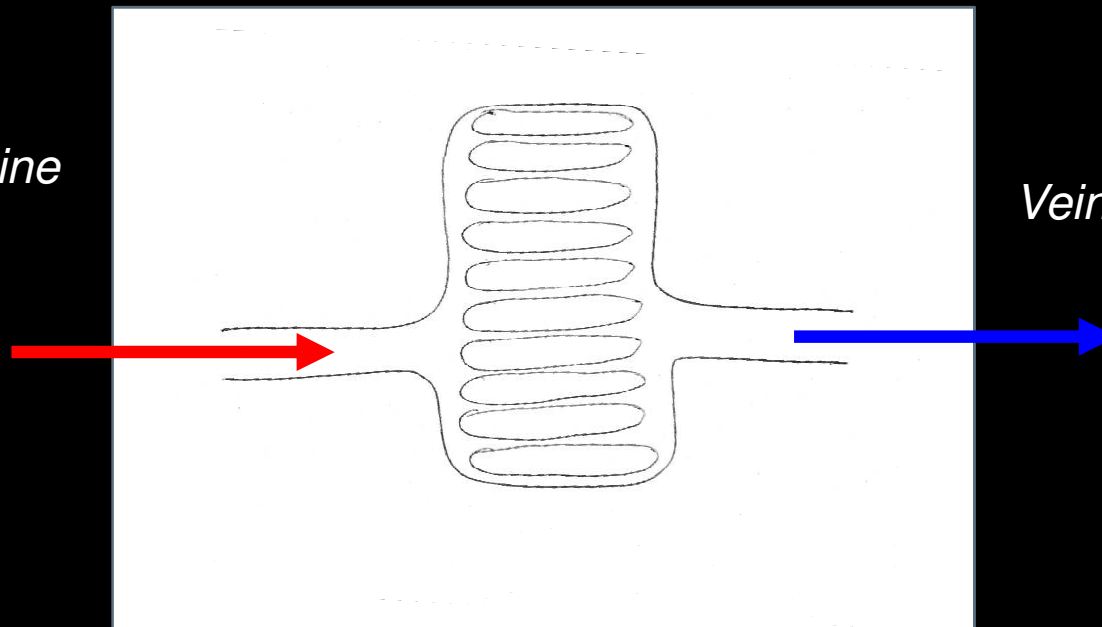
Survenue brutale

Survenue progressive



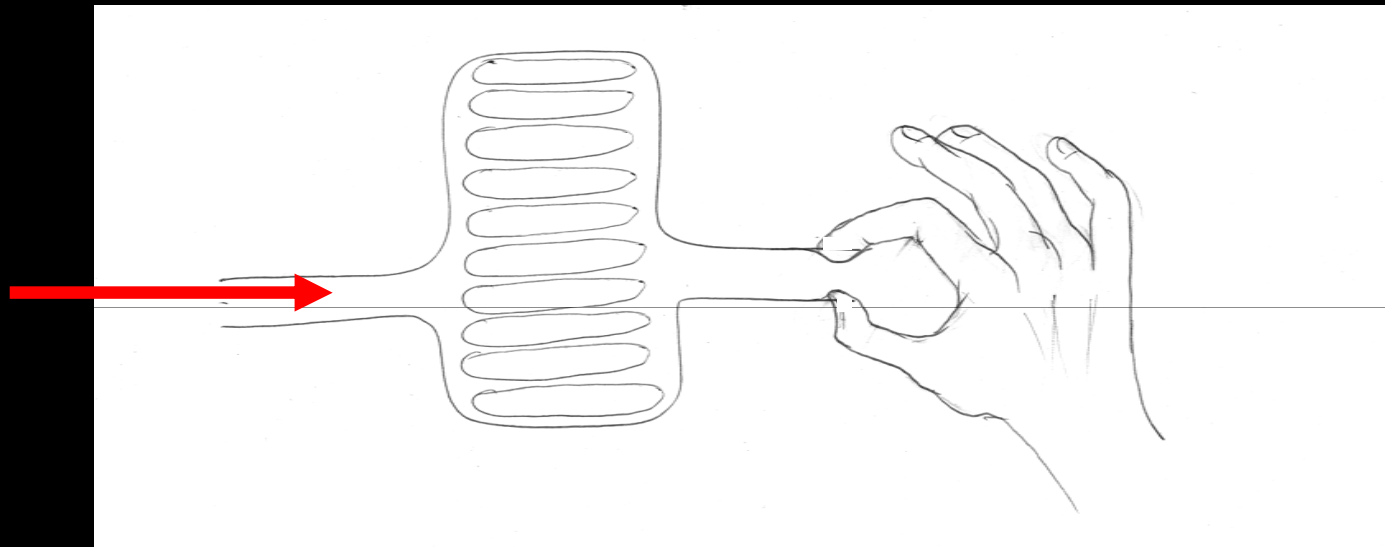
Capillaires

Artère centrale de la rétine

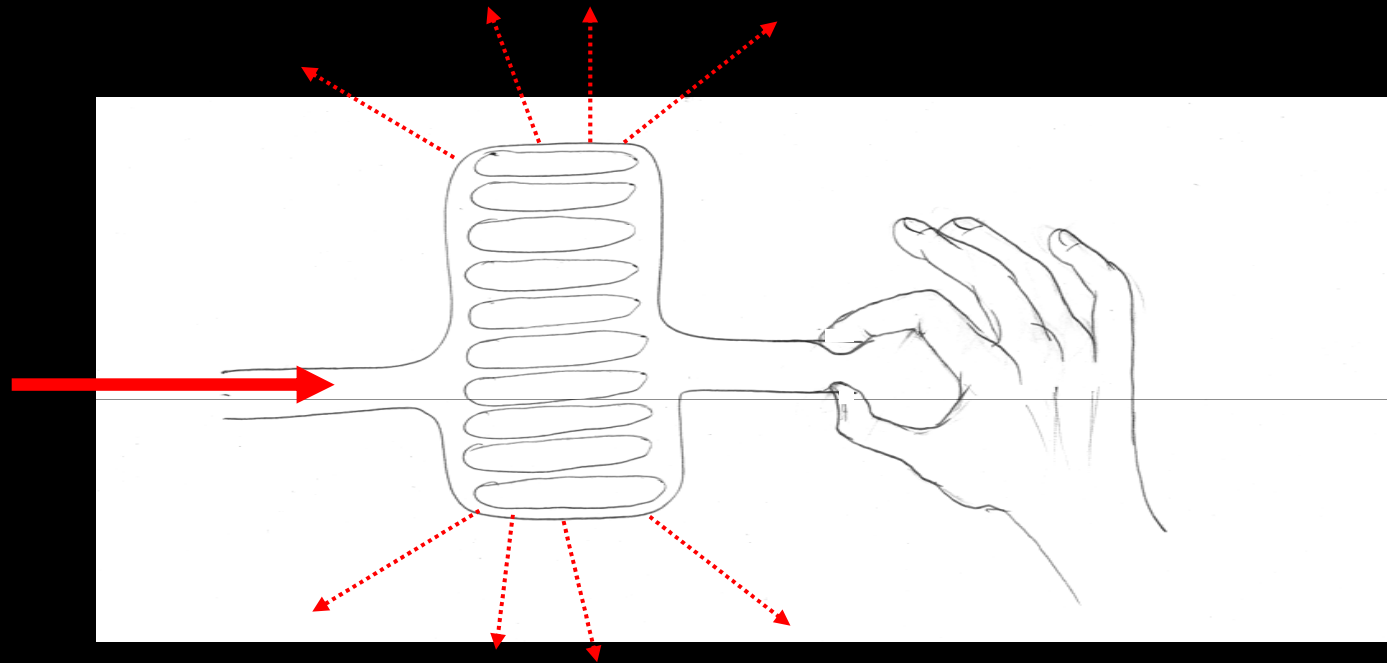


Veine centrale de la rétine

SURVENUE D'UNE OVR



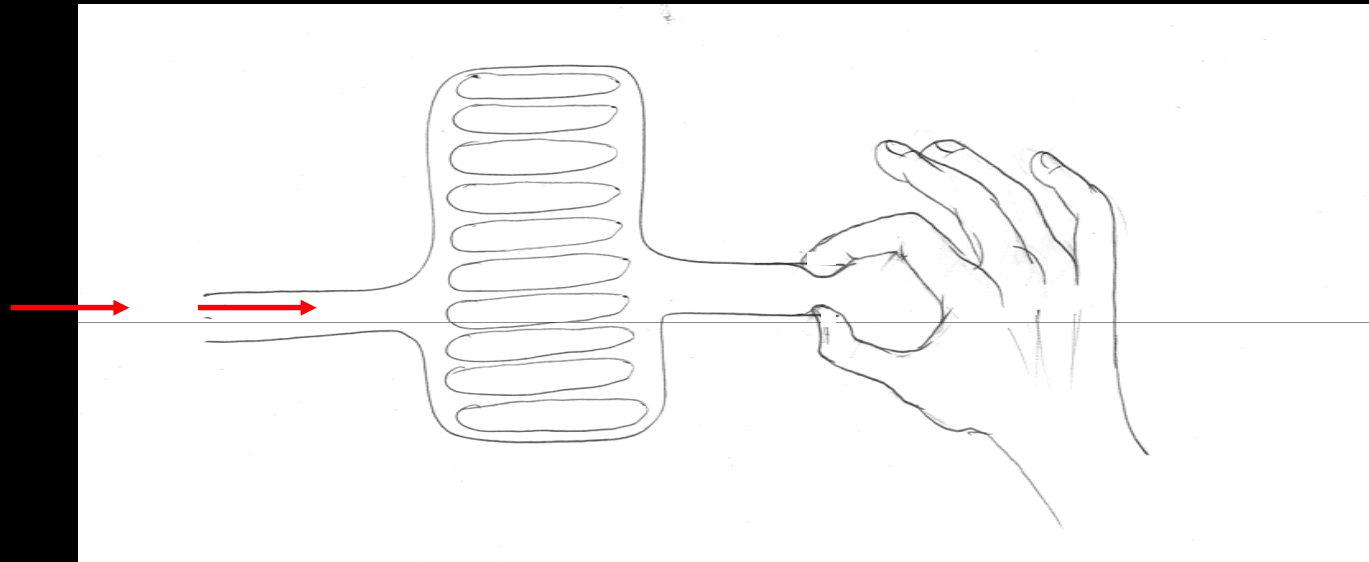
Si les capillaires cèdent



RUPTURE DE LA BARRIÈRE HÉMATORÉTINIENNE
→ HÉMORRAGIES ET ŒDÈME MACULAIRE



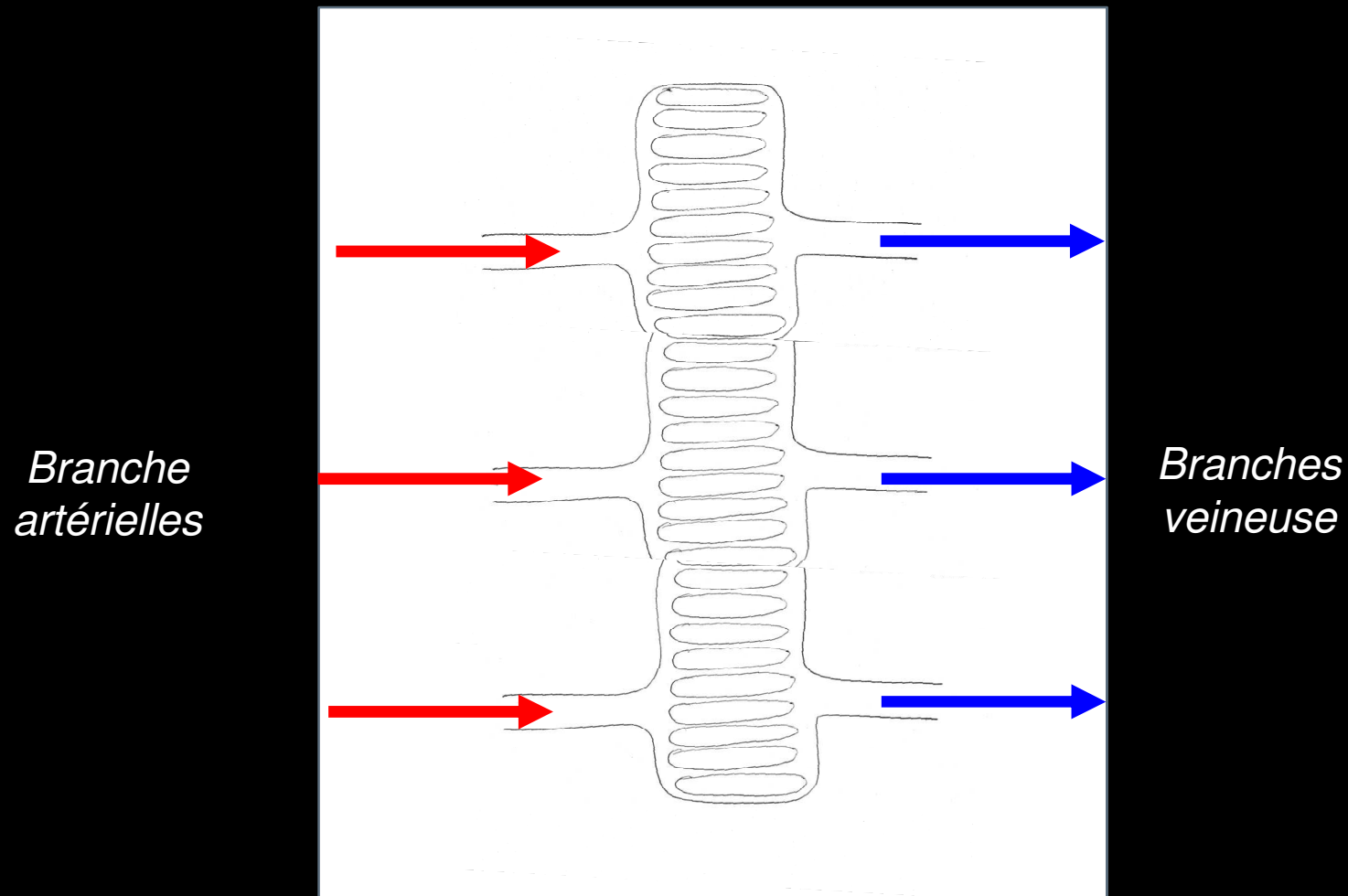
Si les capillaires résistent à la pression



➔ BAS DÉBIT GLOBAL

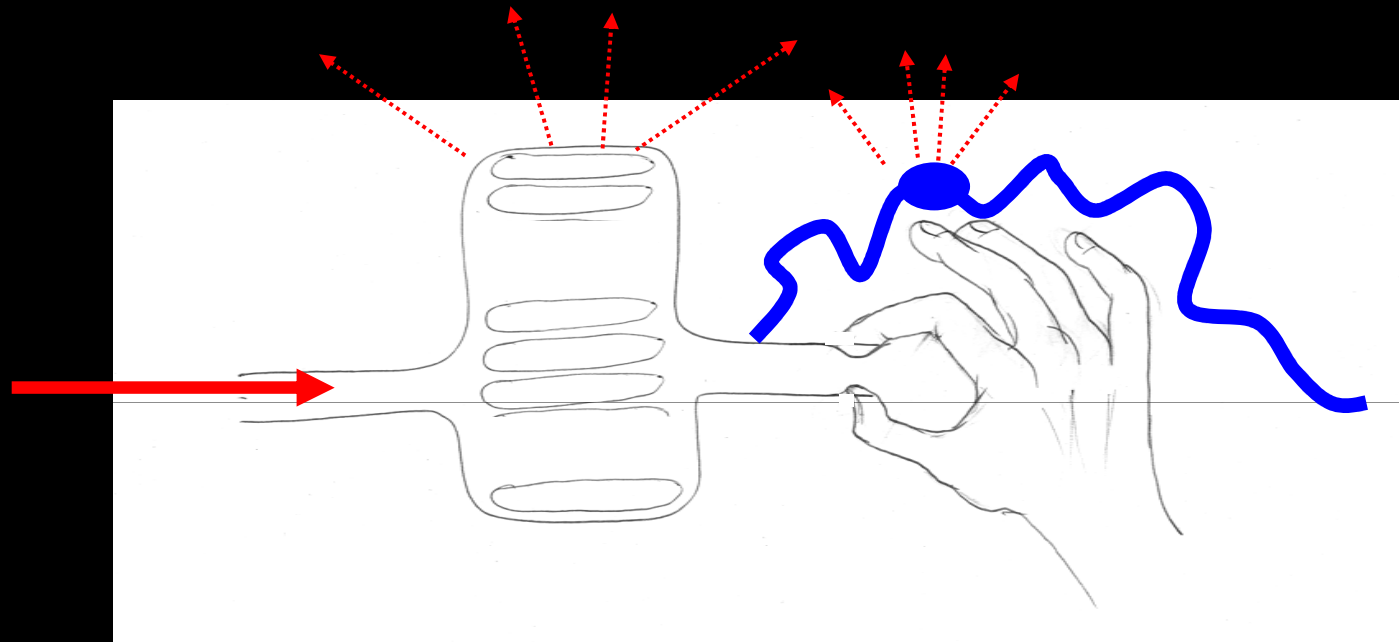


Qu'en est-il des OBVR?



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)

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



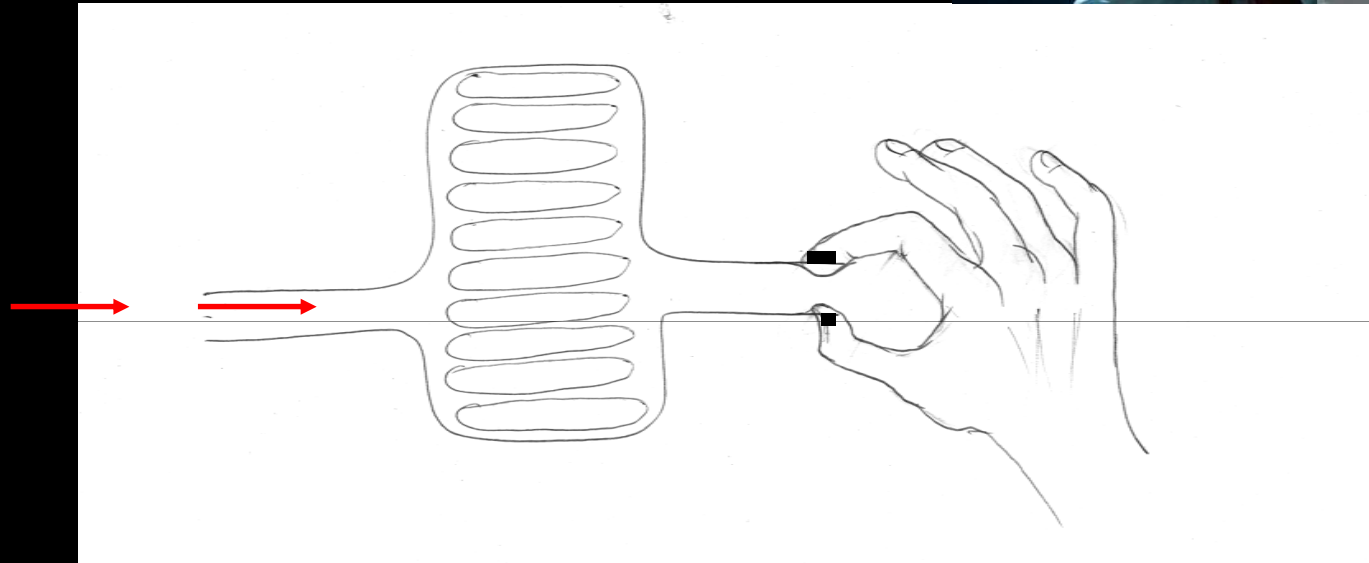
➔ NONPERFUSION, COLLATÉRALES, MACROANÉVRYSMES

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 forcément synonyme 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)

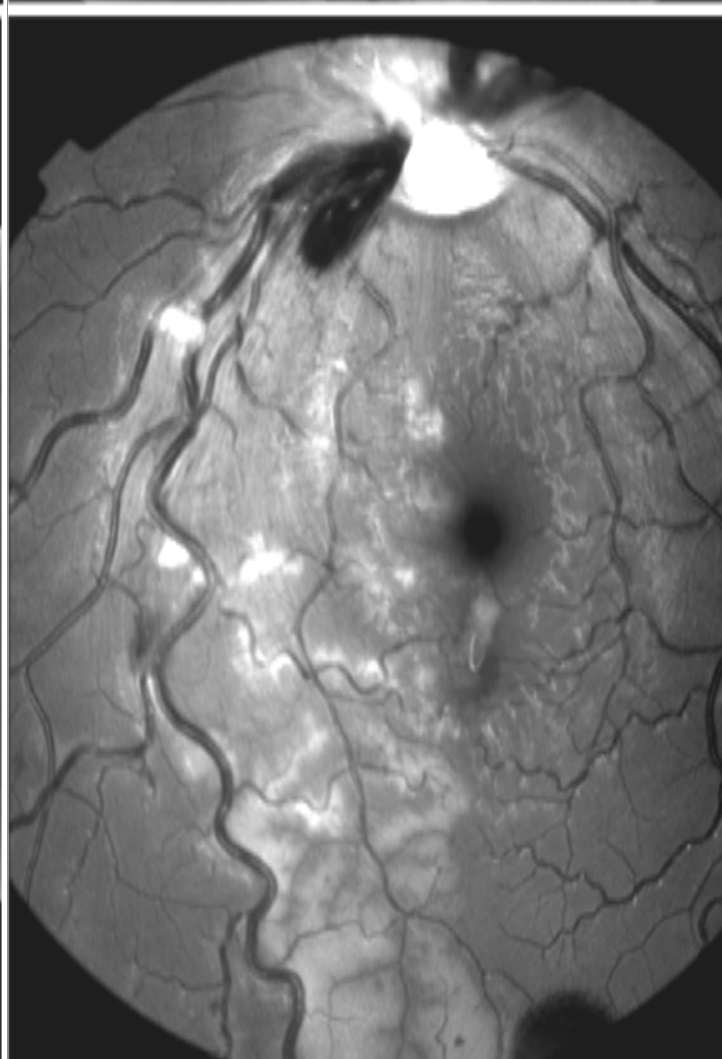
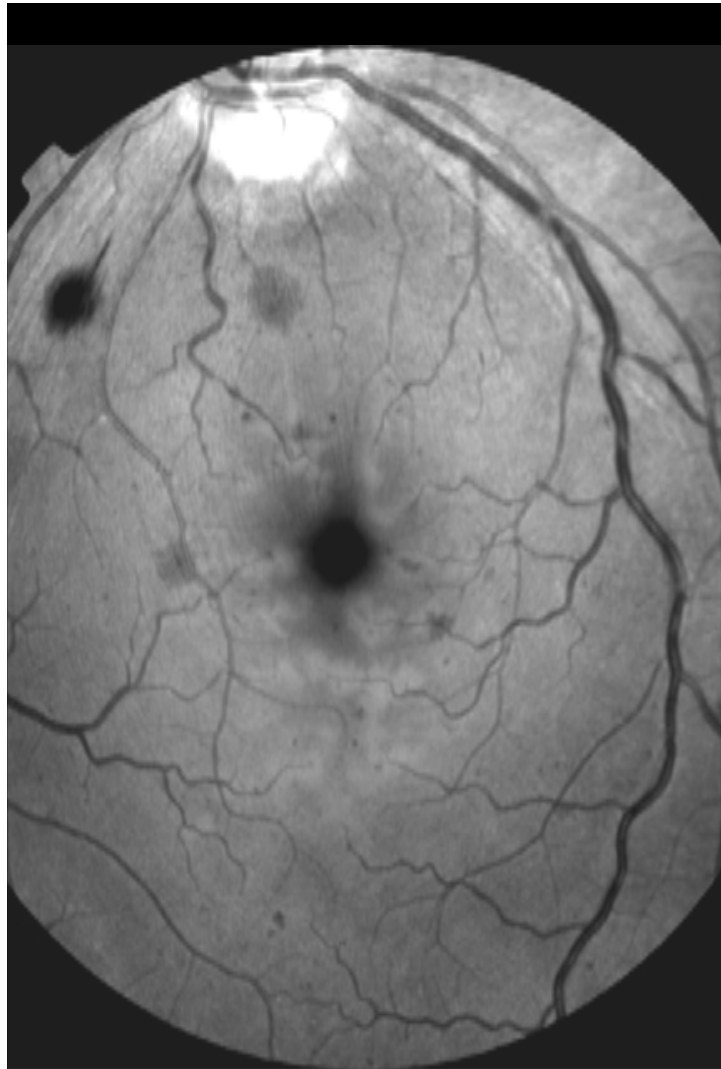
OVCR type A:

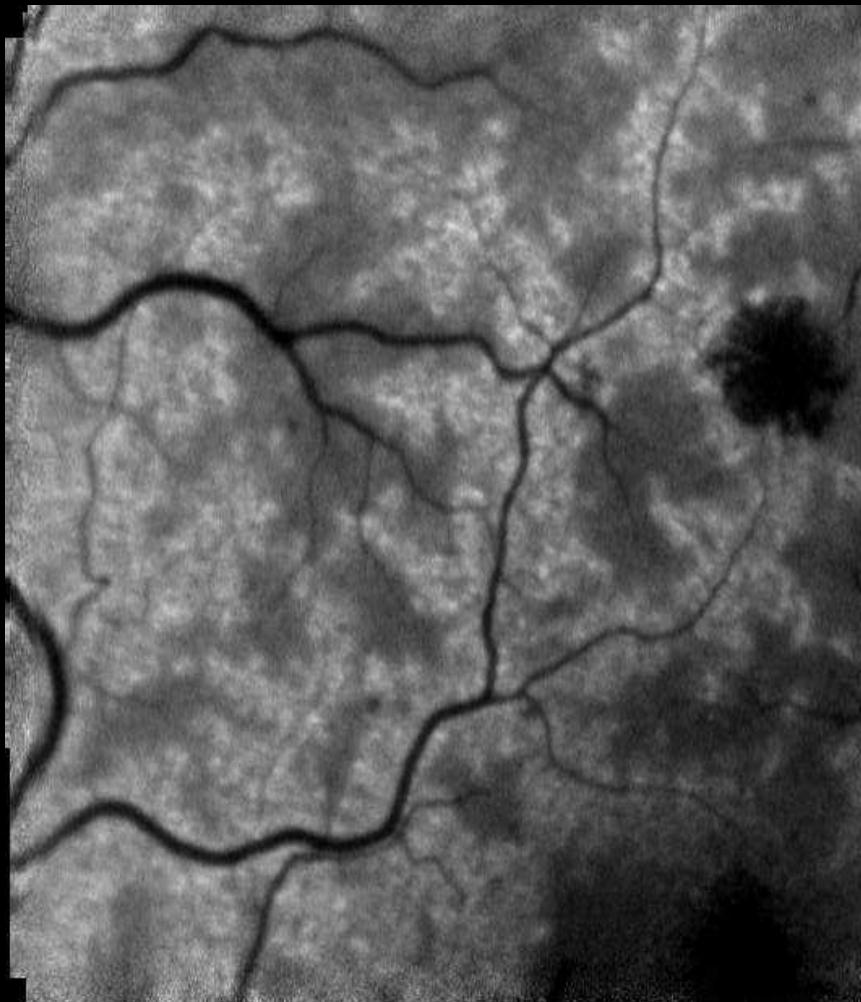
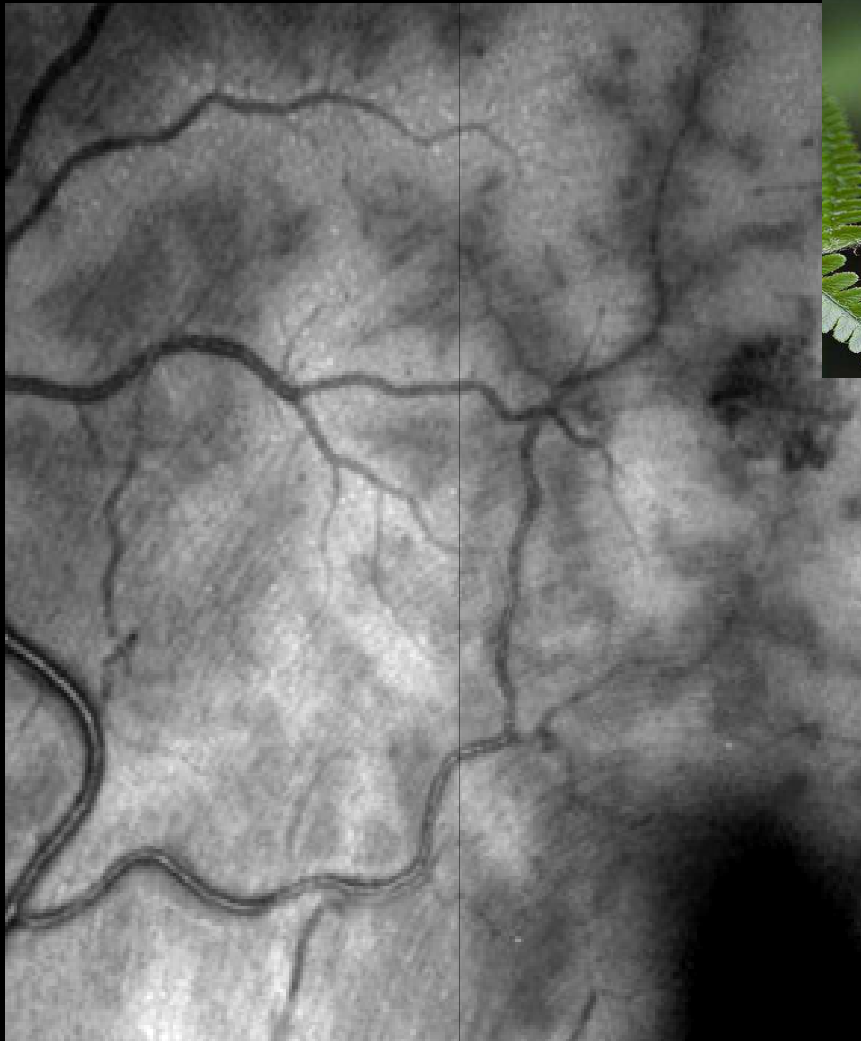
*Survenue brutale d'une OVCR
sur un réseau vasculaire sain*



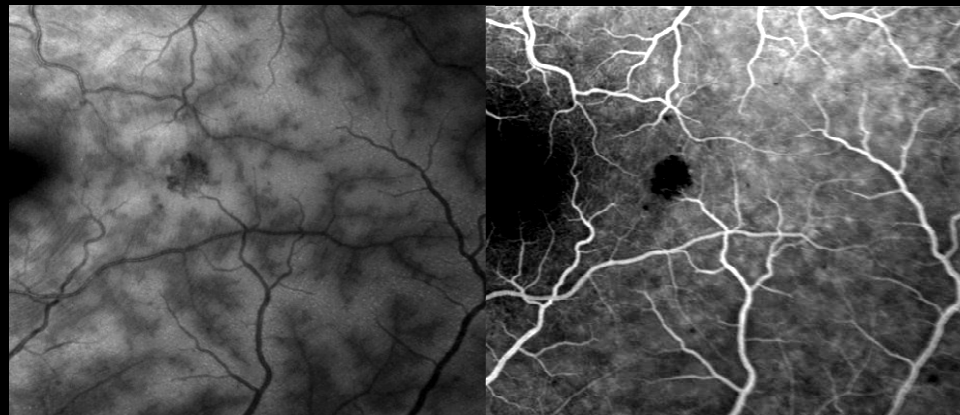
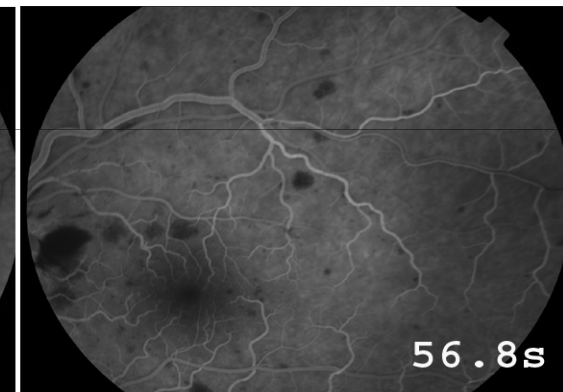
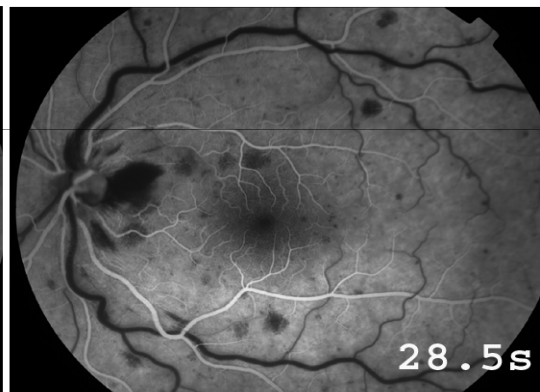
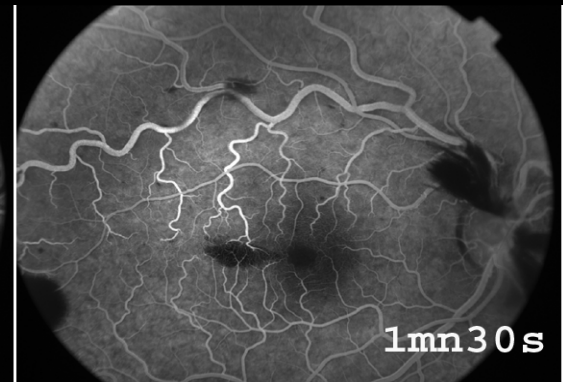
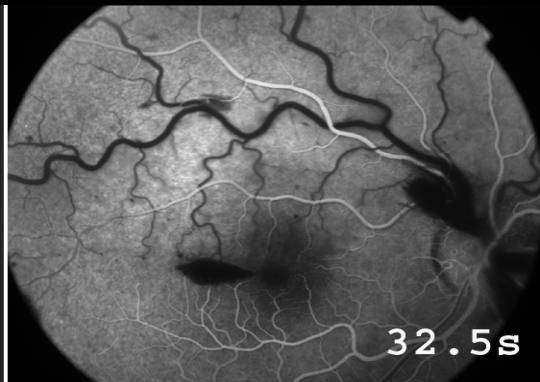
- ➔ BAS DÉBIT GÉNÉRALISÉ
- ➔ APPARITION DU BLANC PÉRIVEINULAIRE







PANRETINAL HYPOPERFUSION

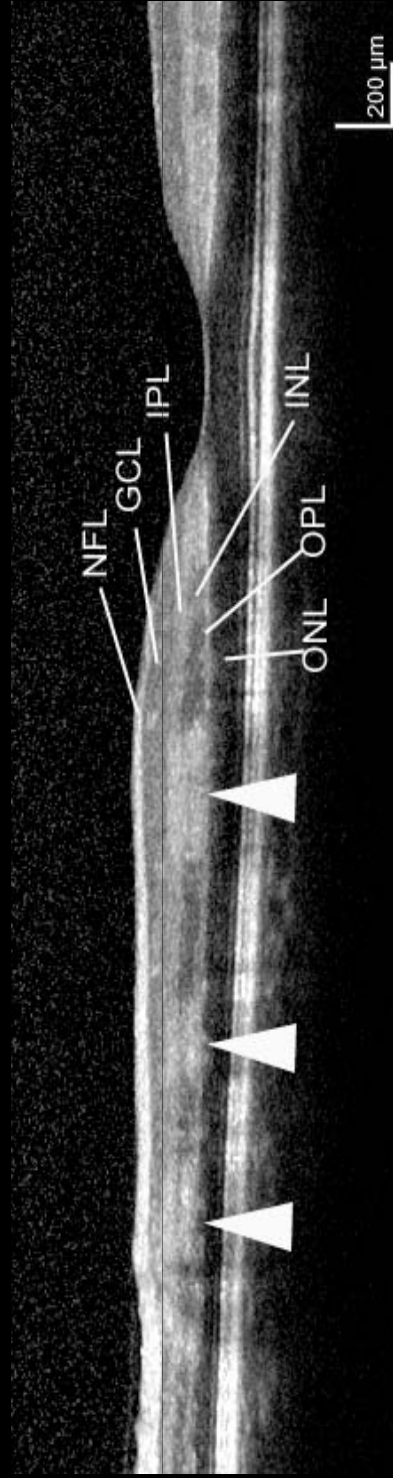
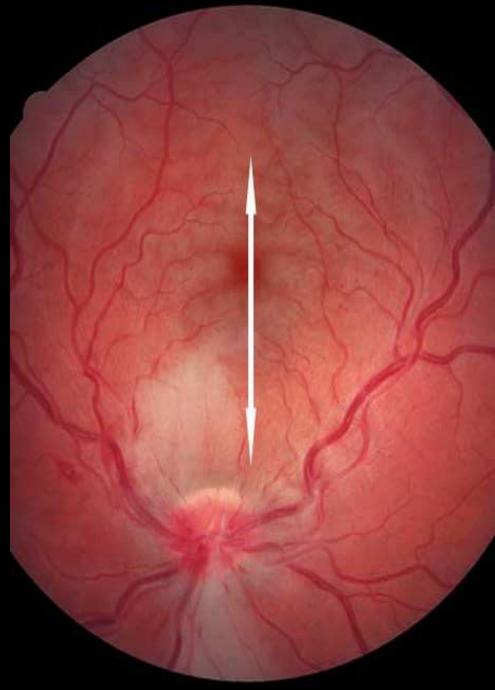




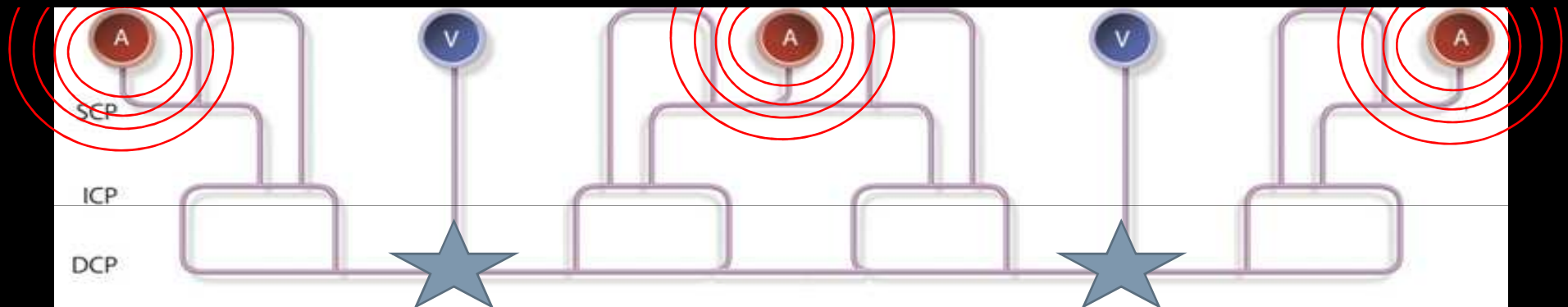


Le BPV représente une opacification ischémique aigue des neurones, secondaire à une hypoperfusion généralisée

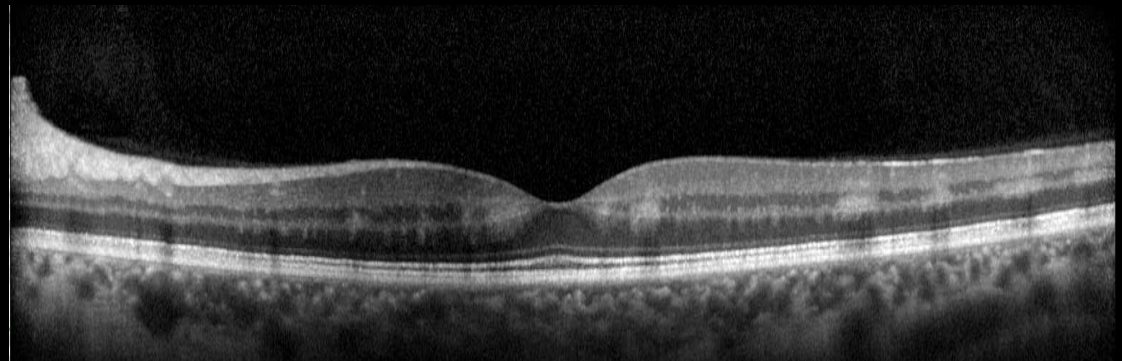
MacLeod Acta Ophthalmologica 2010

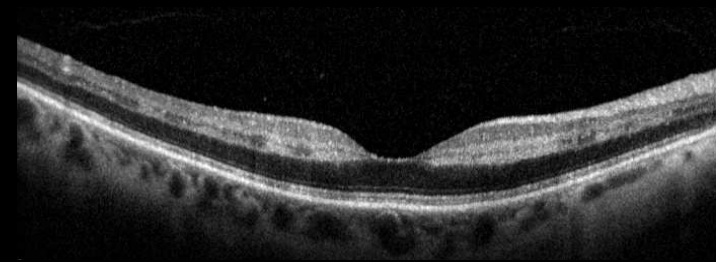
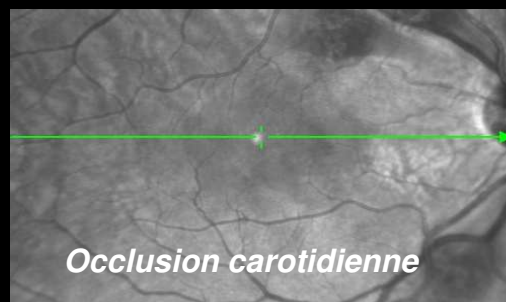
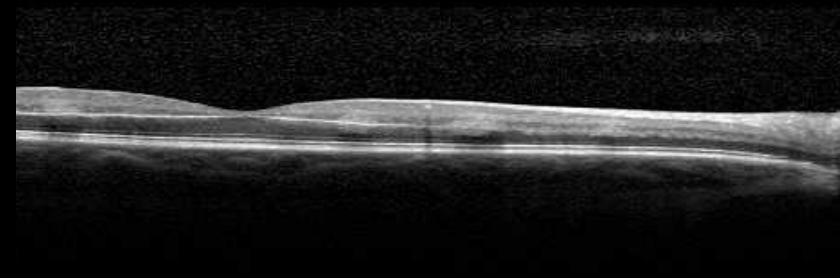
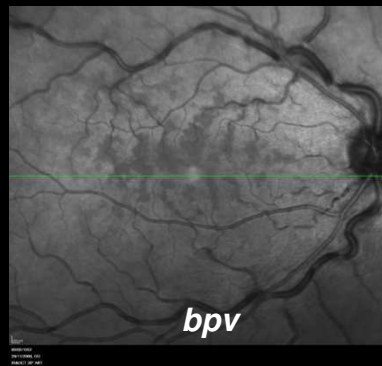
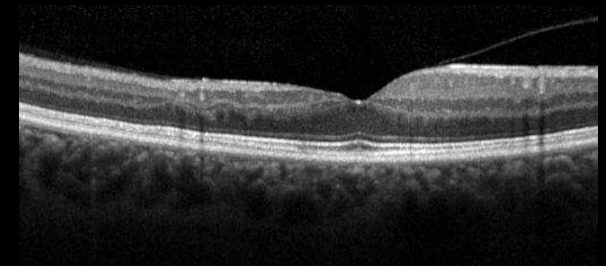
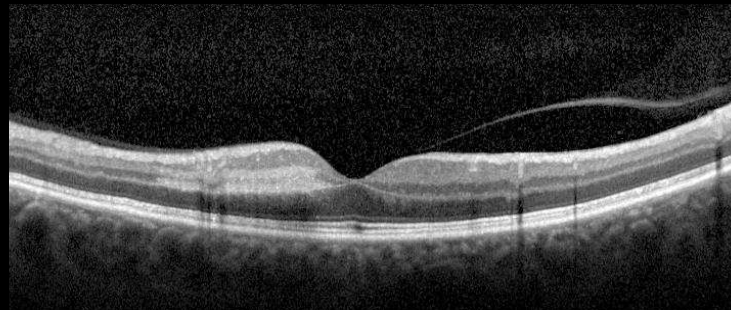
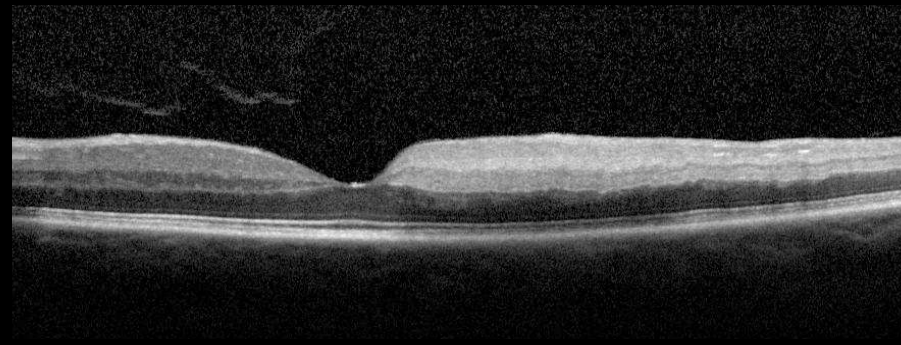
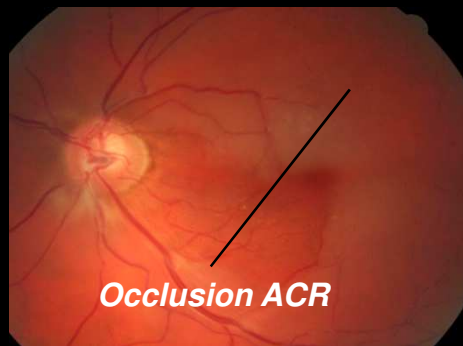


EFFECT OF DECREASED PERFUSION PRESSURE ON RETINAL METABOLISM: THE MICROVASCULAR ANATOMY EXPLAINS THE LOCALISATION OF PREVENUYLAR WHITENING (« PAMM »)



Area of retinal thinning





Thèses Feng Liang, Nadine Manasseh 2011

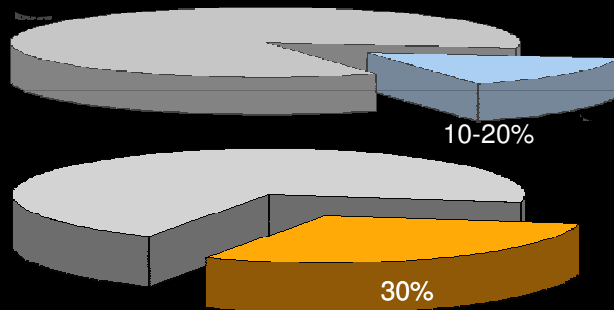
FACTEURS DE RISQUE SELON LE TYPE D'OVR

- La pression de perfusion oculaire est

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

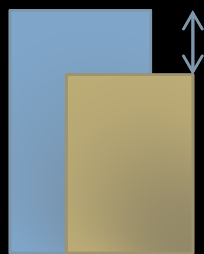
- Donc le débit sanguin rétinien
 - Augmente avec la pression artérielle
 - Diminue avec la pression intraoculaire

Type A



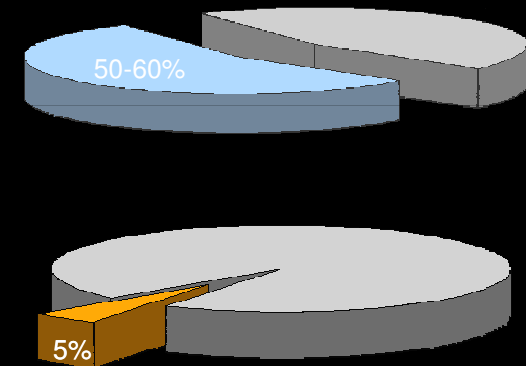
HTA

Glaucome



Glaucome
+
Hypotension
artérielle

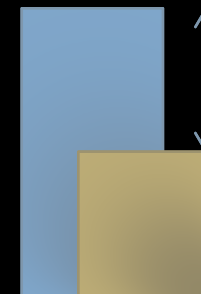
Type B



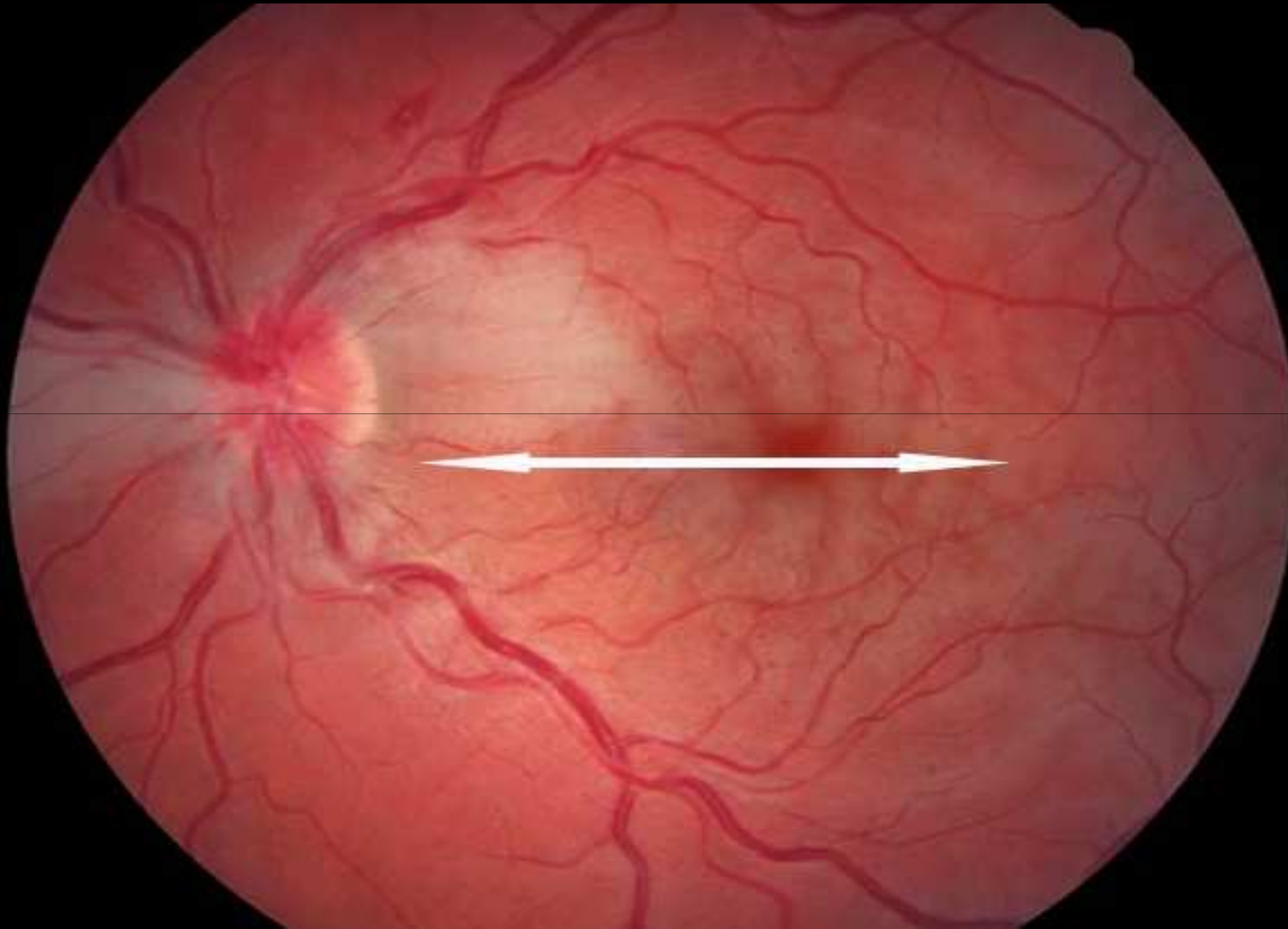
PA

Pression de
perfusion oculaire

PIO

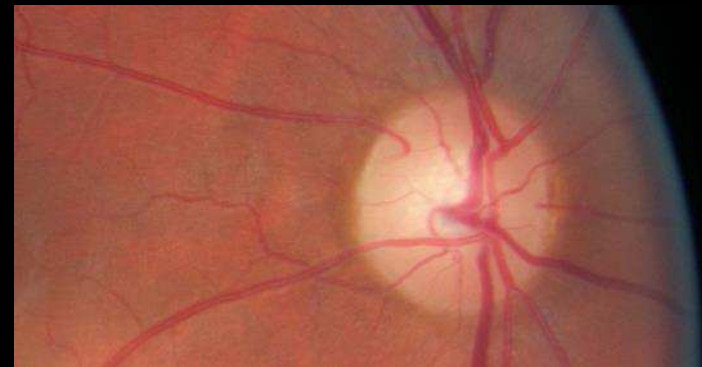


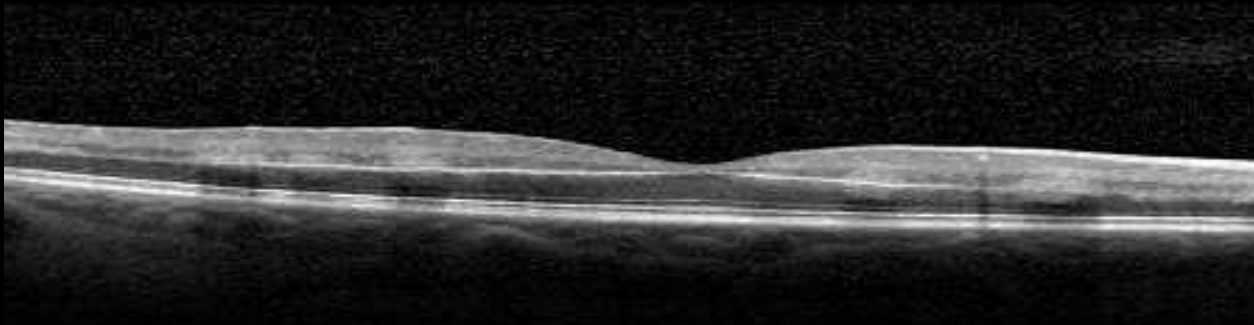
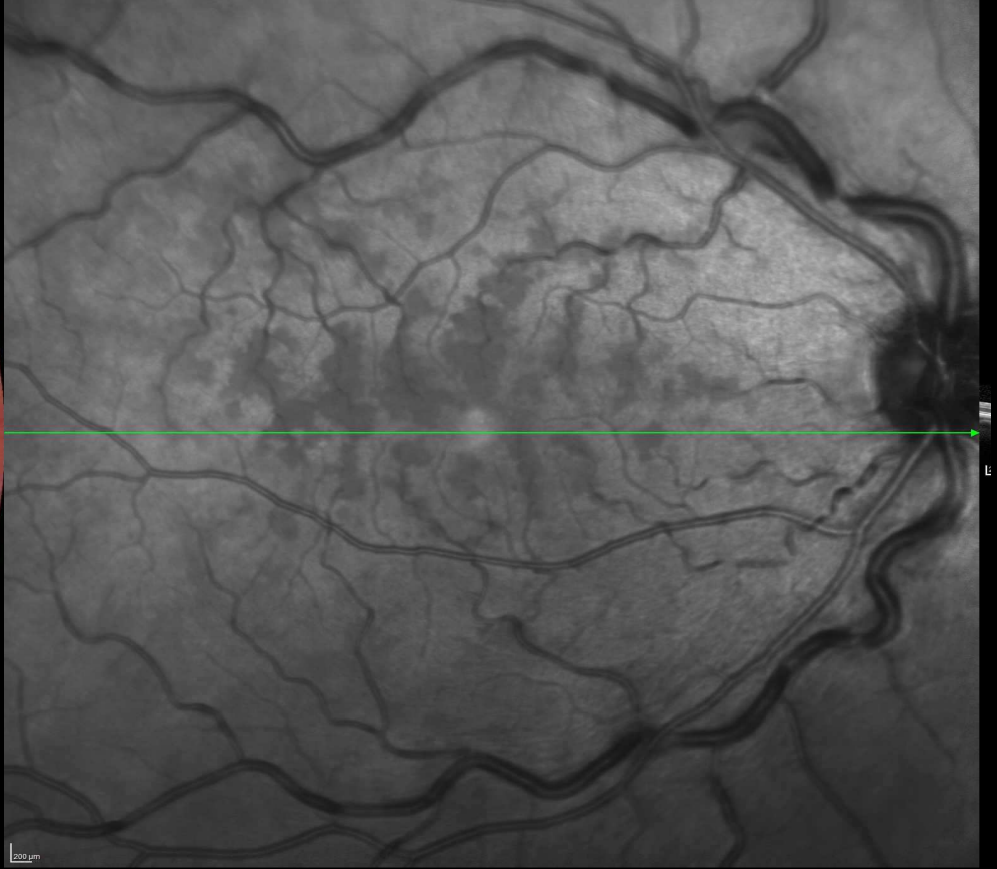
Patient 38 ans. BAV = CLD. PIO = 43 mmHg

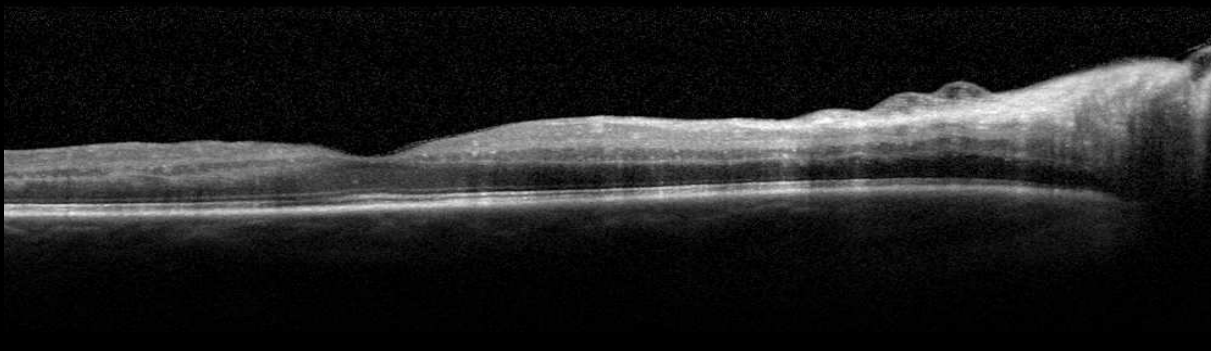
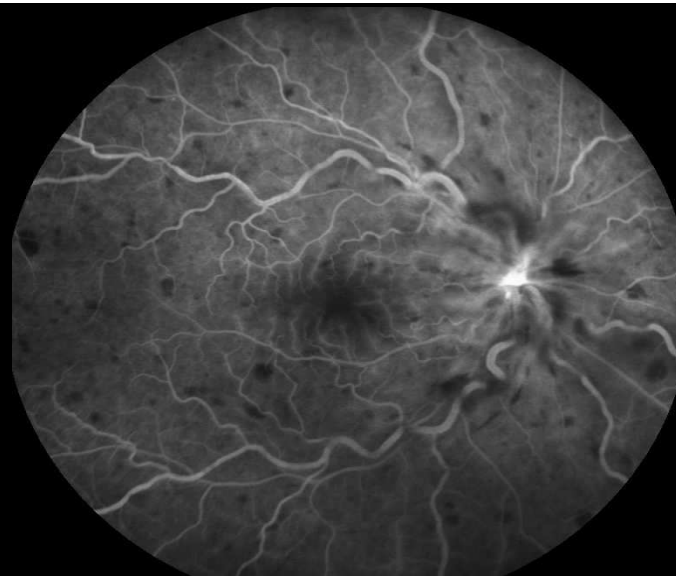


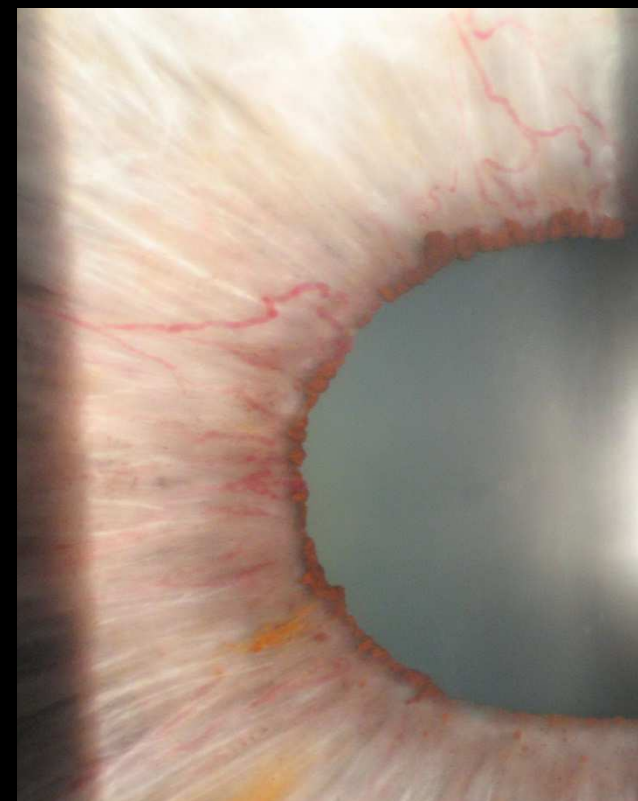
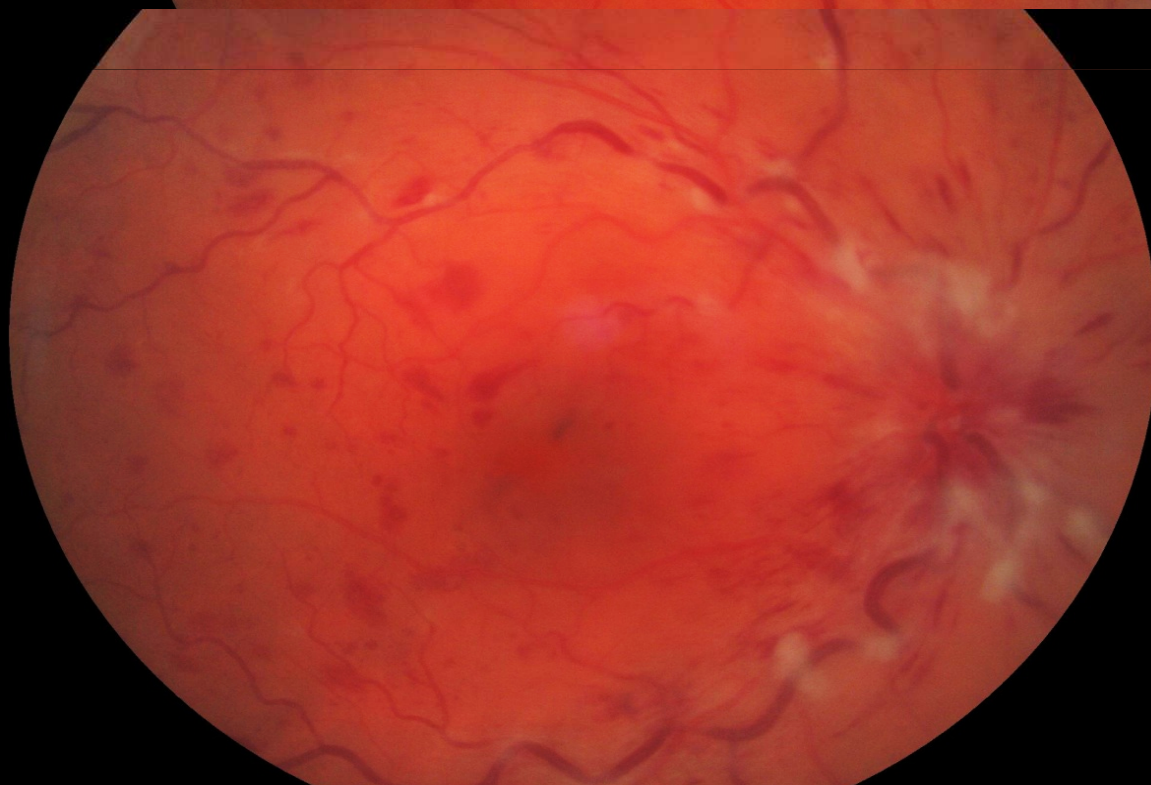
POURQUOI LES ARTERES CILIORÉTINIENNES S'OCCLUENT-ELLES AU COURS D'UNE OVCR?

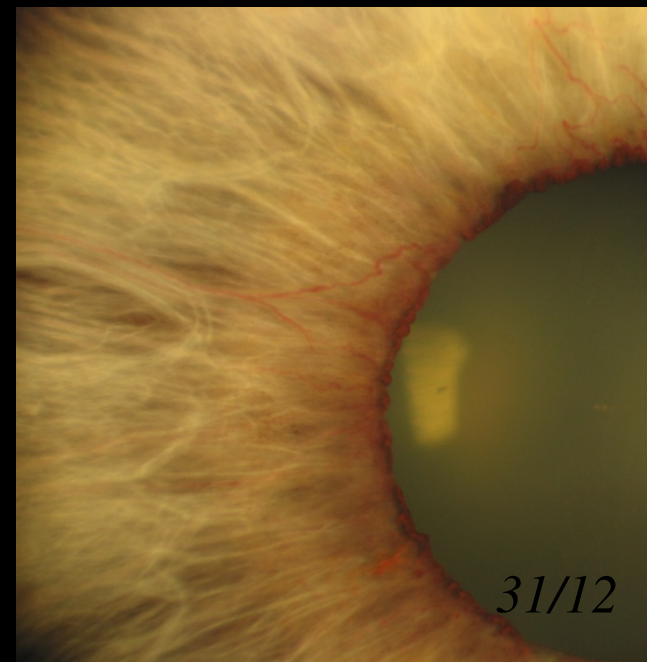
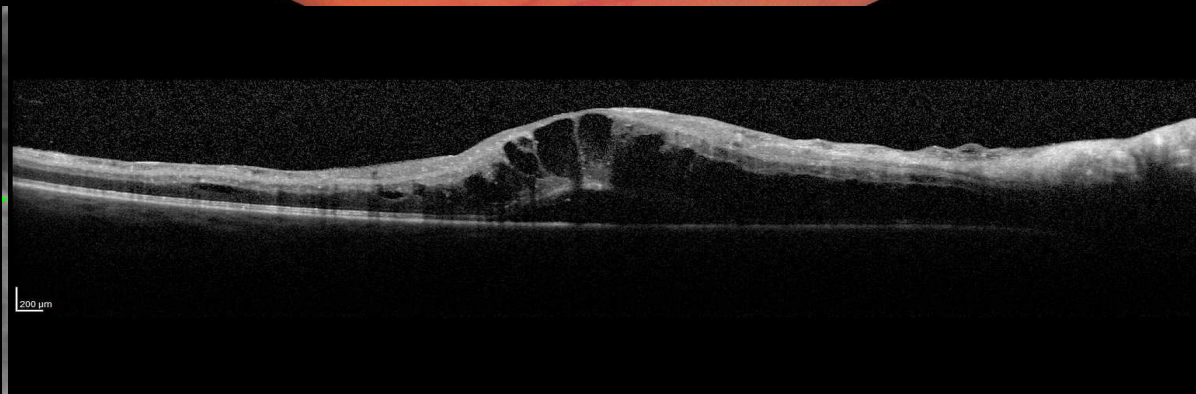
- Les ciliorétiniennes doivent traverser la couche des fibres optiques, qui est oedémateuses en cas d'OVR
- Le long trajet commun de l'artère et de la veine centrale met l'artère au contact d'une veine désaturée en oxygène, ce qui est un stimuli vasodilatateur

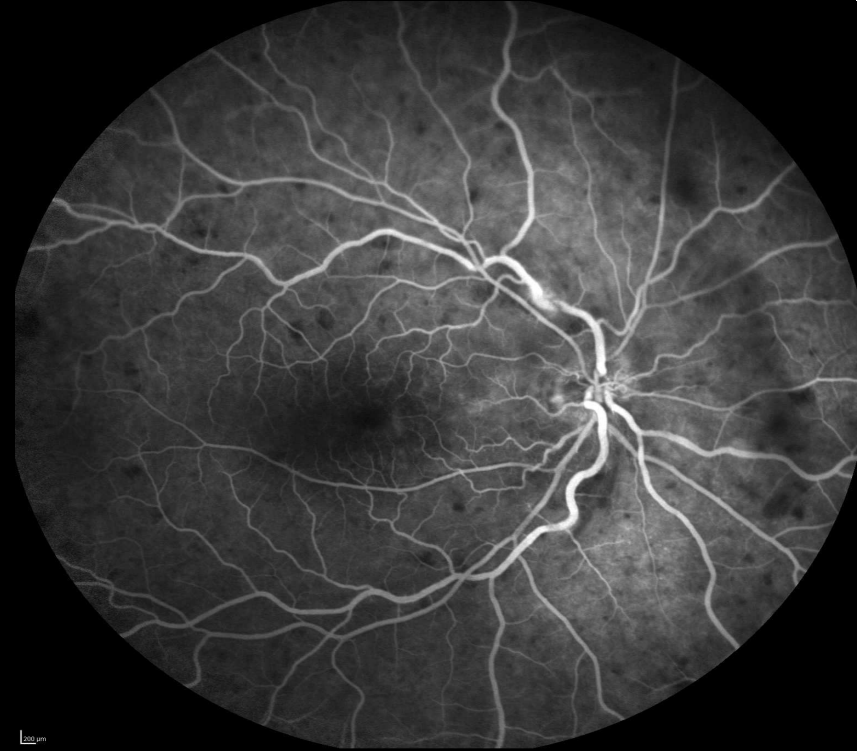
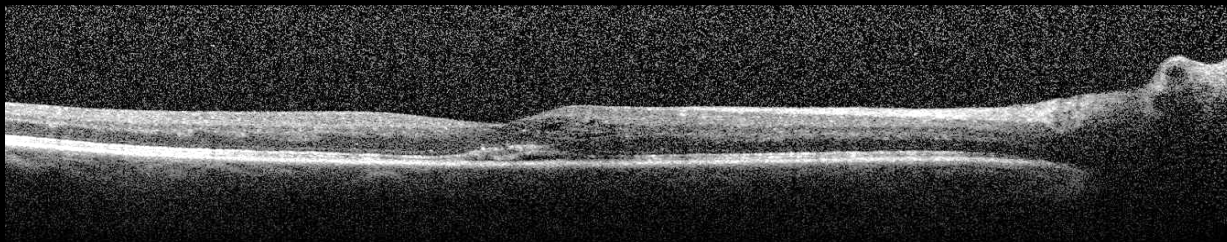
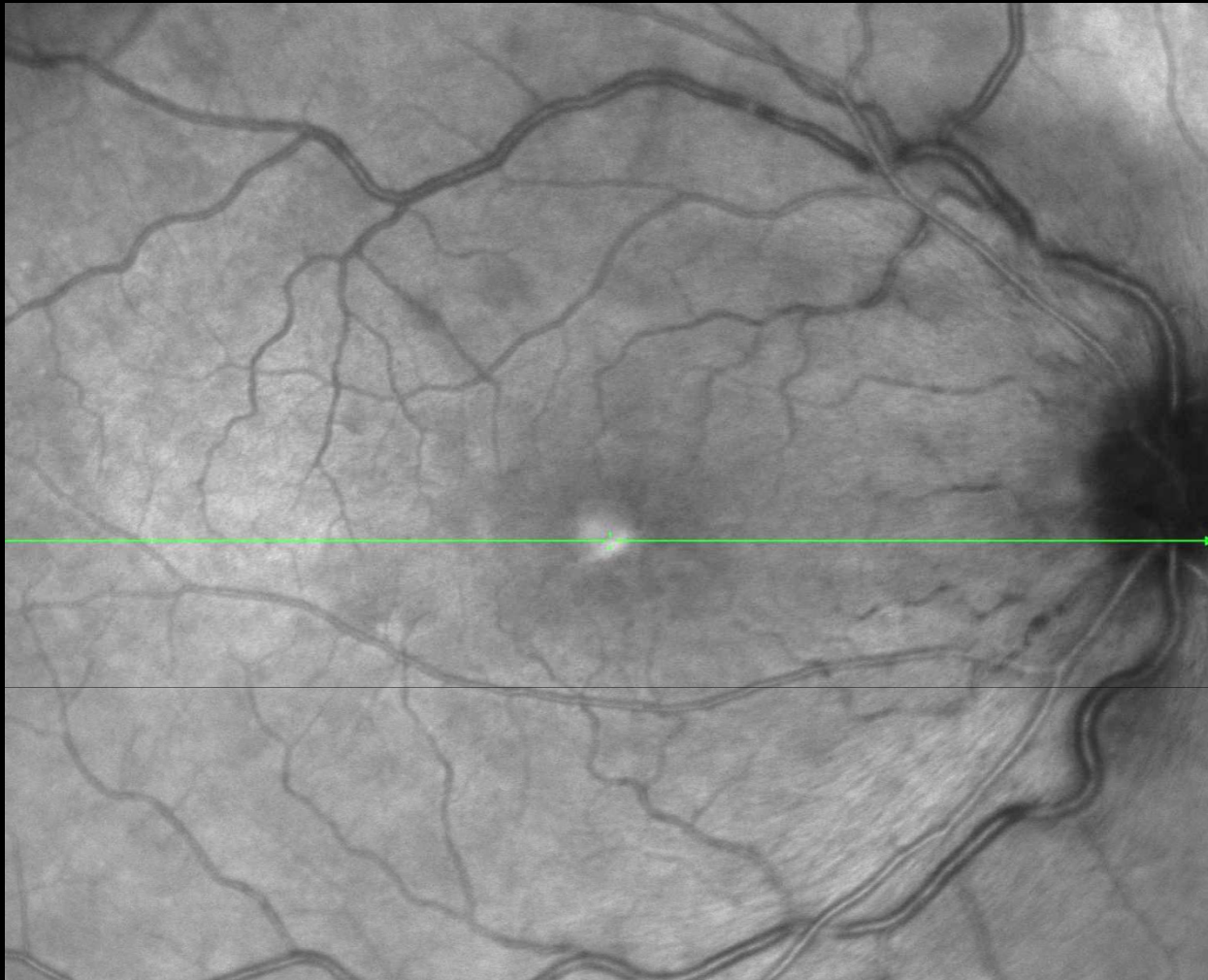






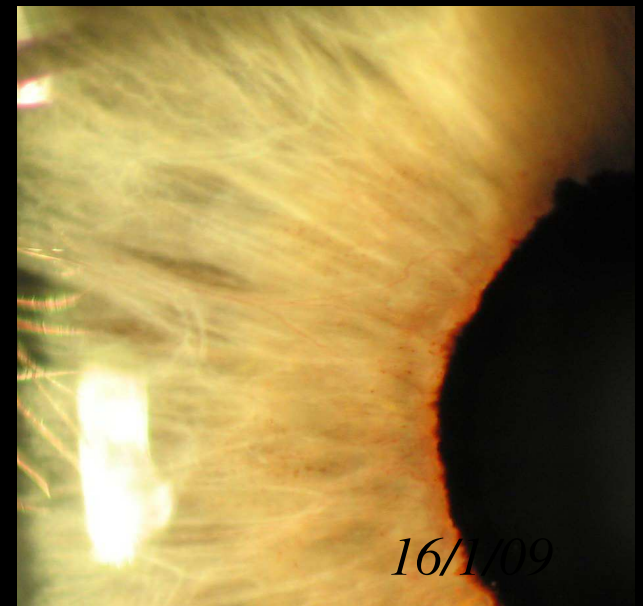




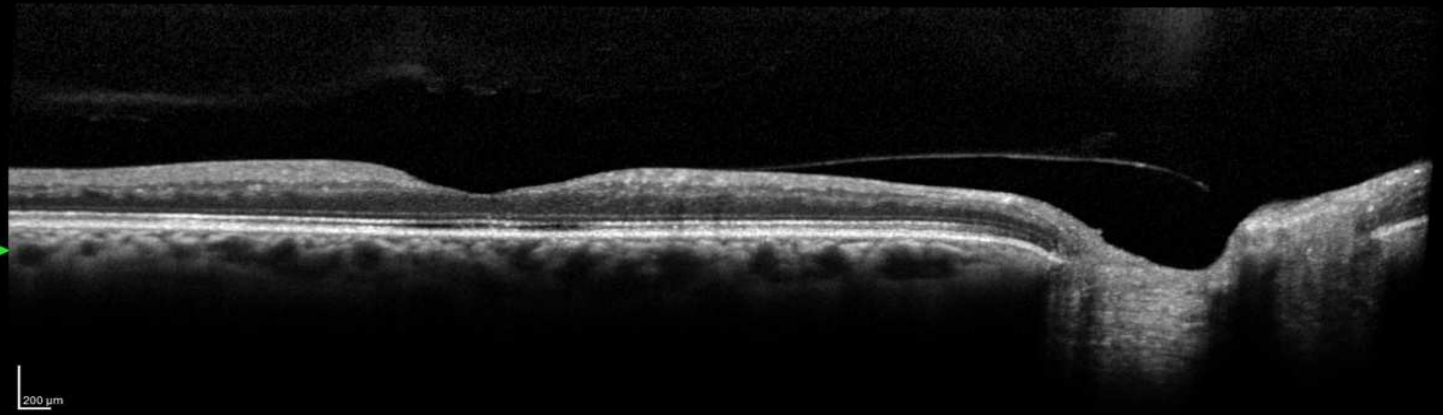
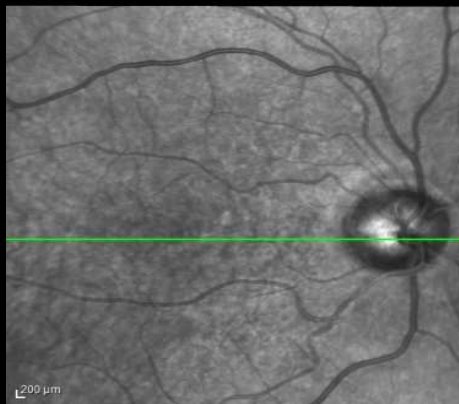
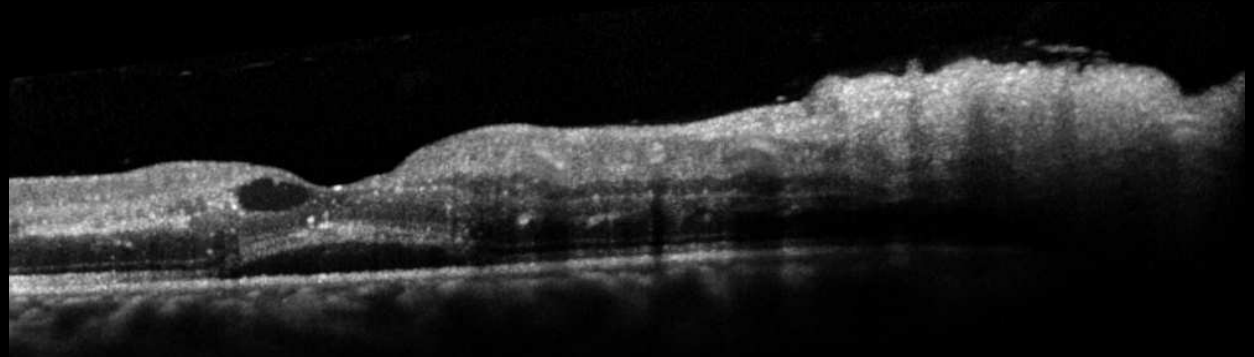


100 μ m
09/02/1992
16/01/2009, OD
FA 3.08.61 30° ART

HEIDELBERG
RETINA

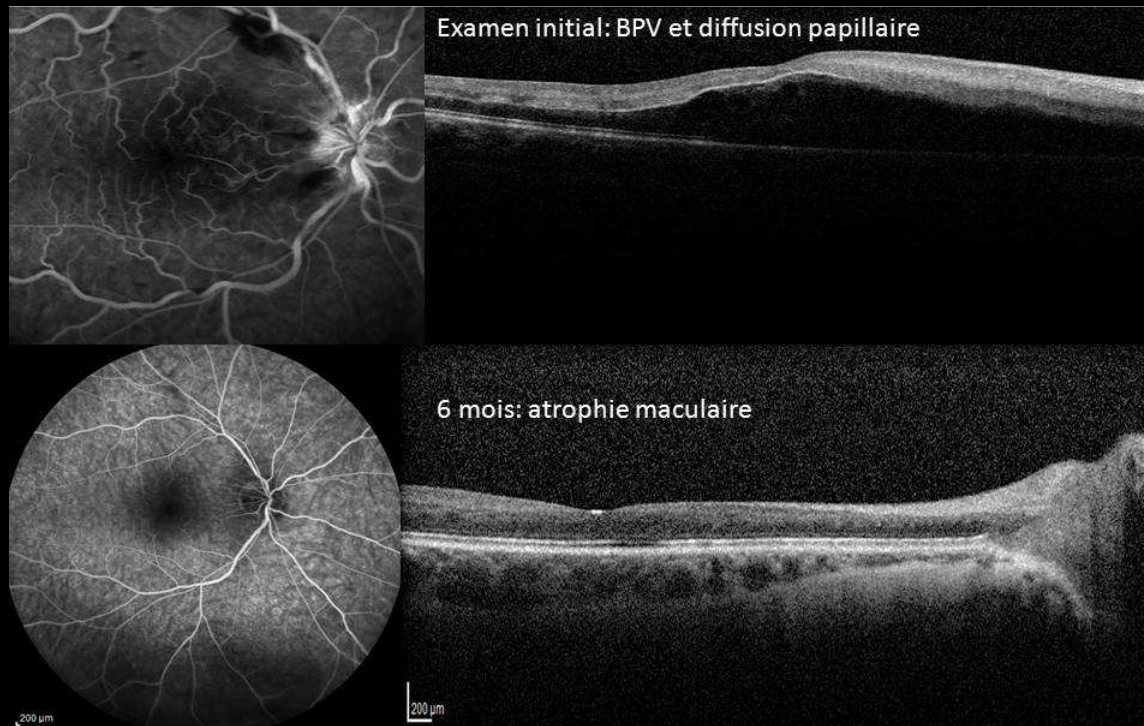


PVW may be subtle, and disc/macular edema more prominent





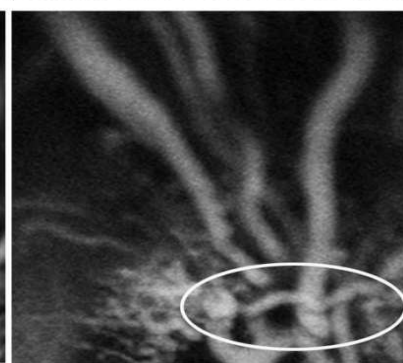
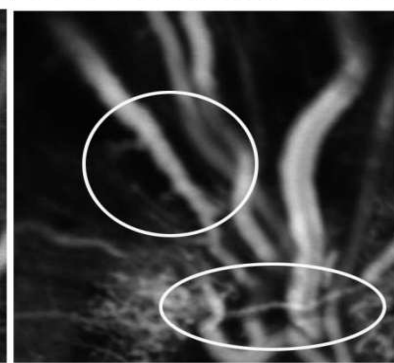
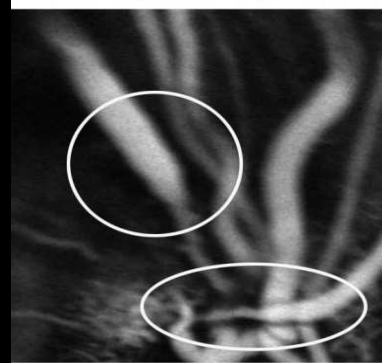
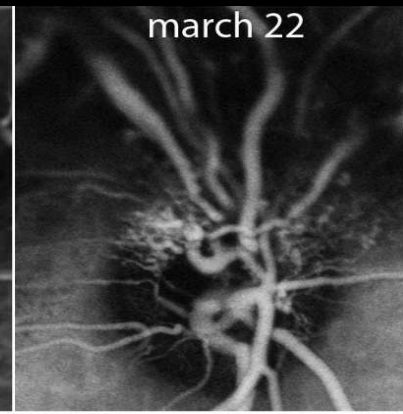
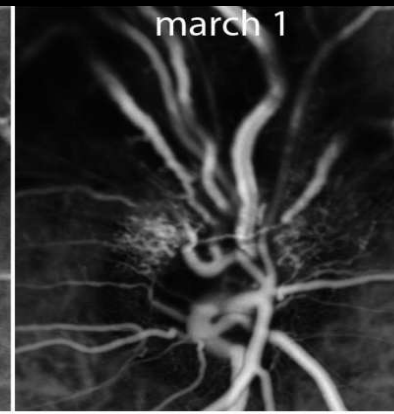
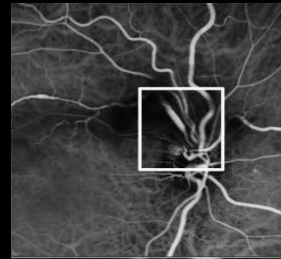
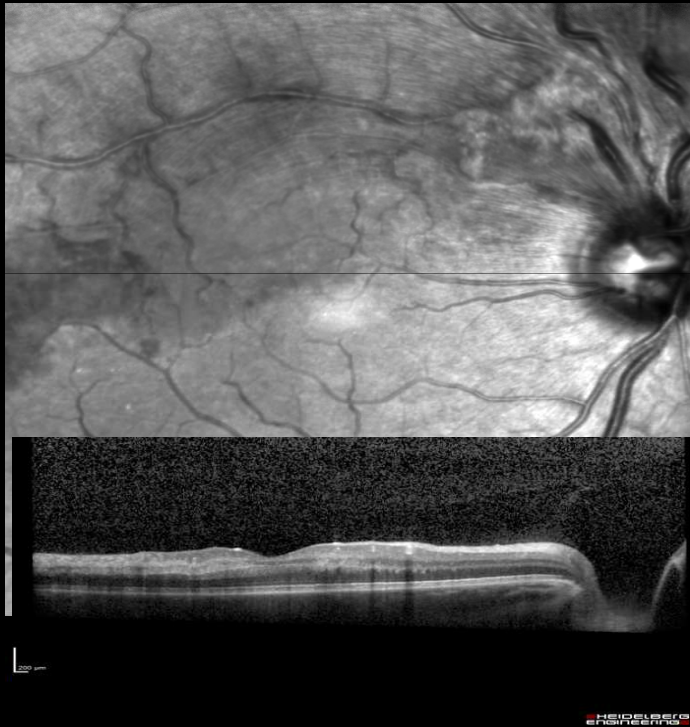
Le type A est souvent suivi d'un œdème papillaire



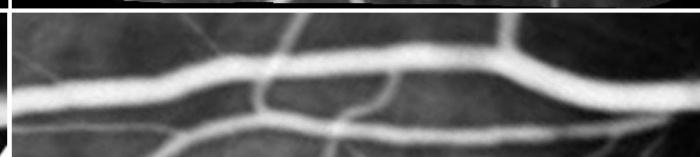
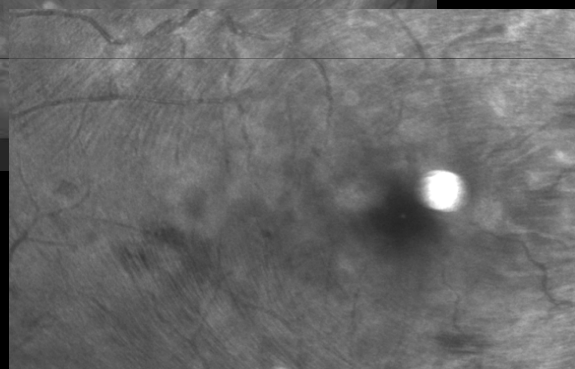
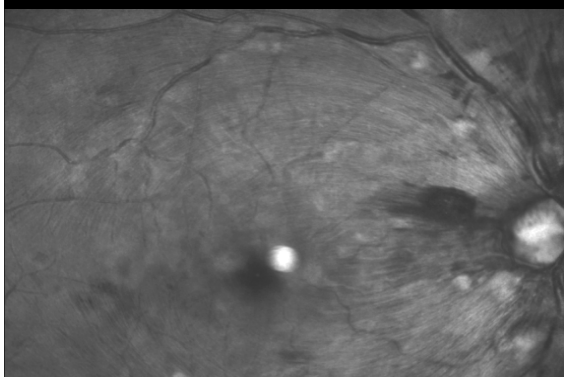
EVOLUTION DU TYPE A

- Debut: Veinoconstriction aiguë
 - 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 œdème maculaire par propagation
- Et evolution à long terme selon le degré de reperfusion de la veine centrale

EVIDENCE OF VEINOMOTRICITY DURING ACUTE CRVO

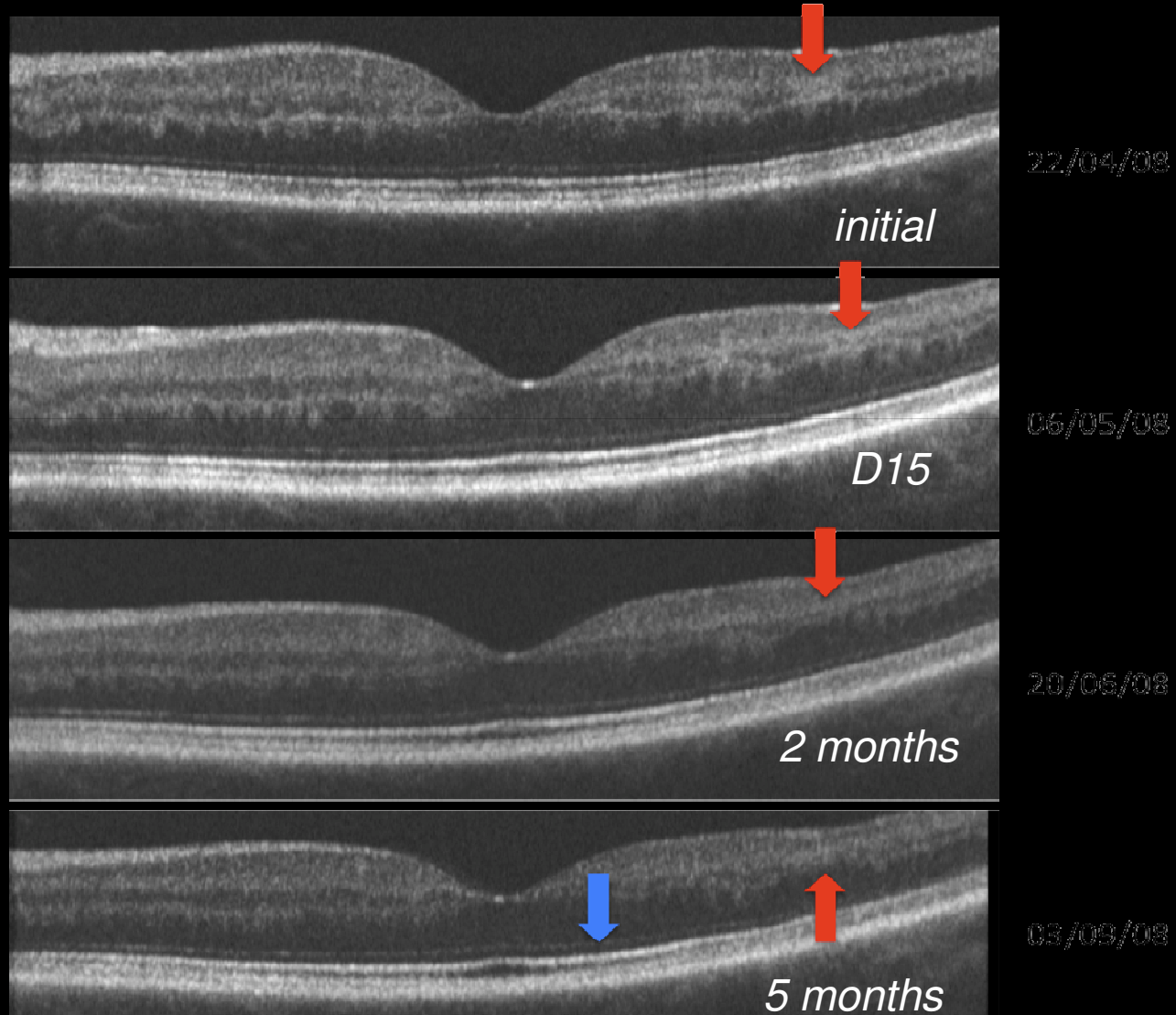


PARADOXICAL REDUCTION OF ARTERIAL *AND VENOUS* DIAMETERS IN A CASE OF CRVO

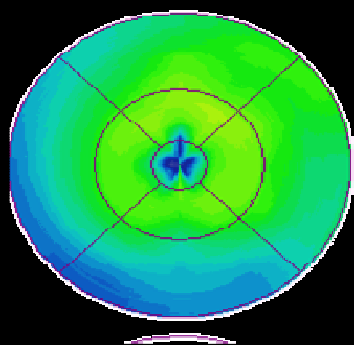
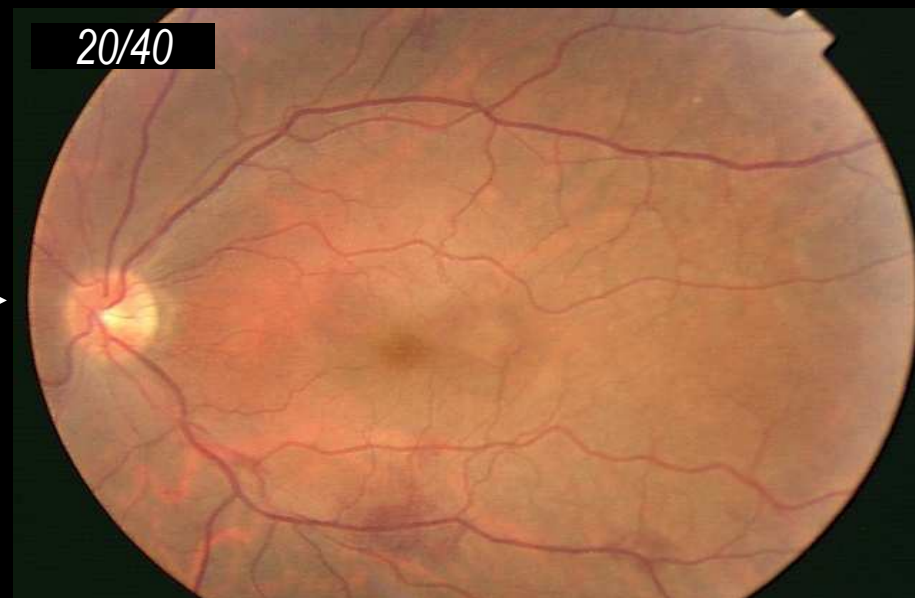


Vasoconstrictive process within the optic nerve, extending to retinal veins?

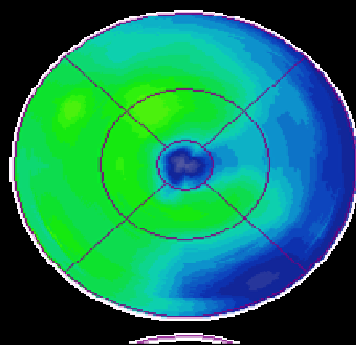
EVOLUTION DES LÉSIONS RÉTINIENNES



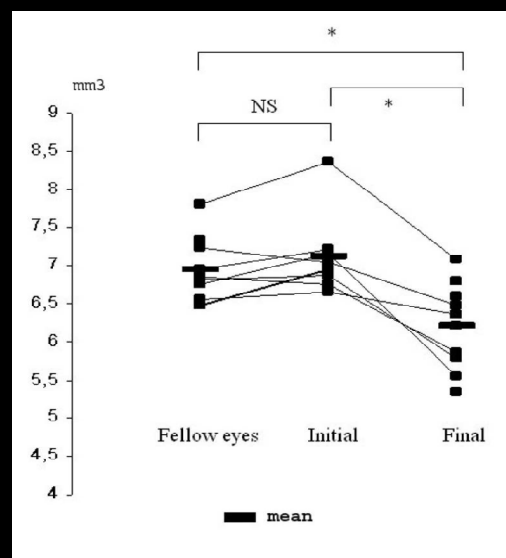
ANATOMICAL RESULTS



Fellow



Affected eye



Mean loss
of macular volume
10.3%

FUNCTIONAL RESULTS

At last visit:

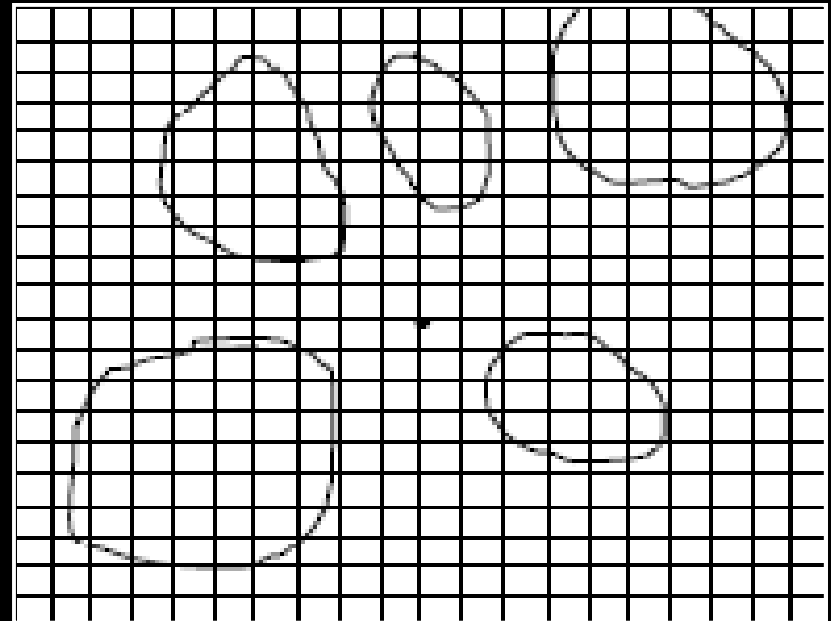
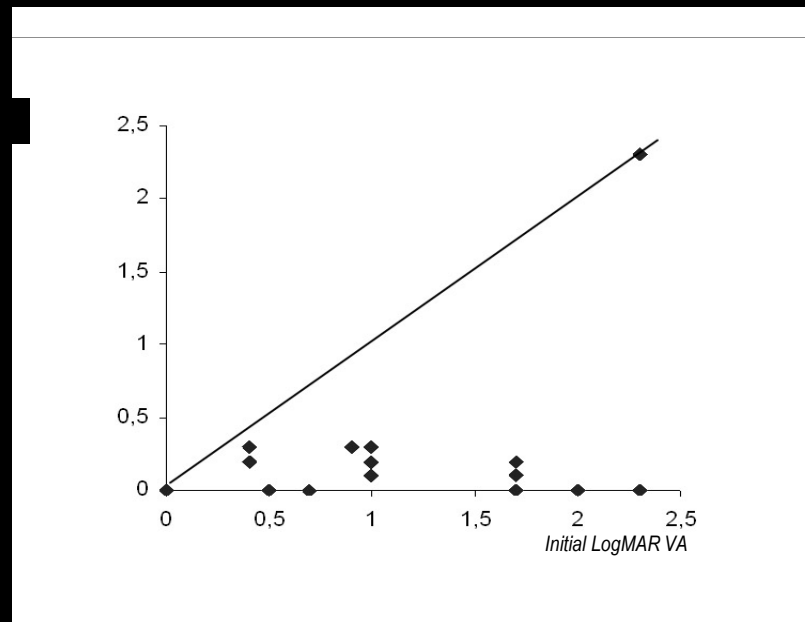
median VA 20/200 → 20/25

13 out of 20 complained of pericentral scotomas

1 had developed preretinal newvessels

No chronic macular edema

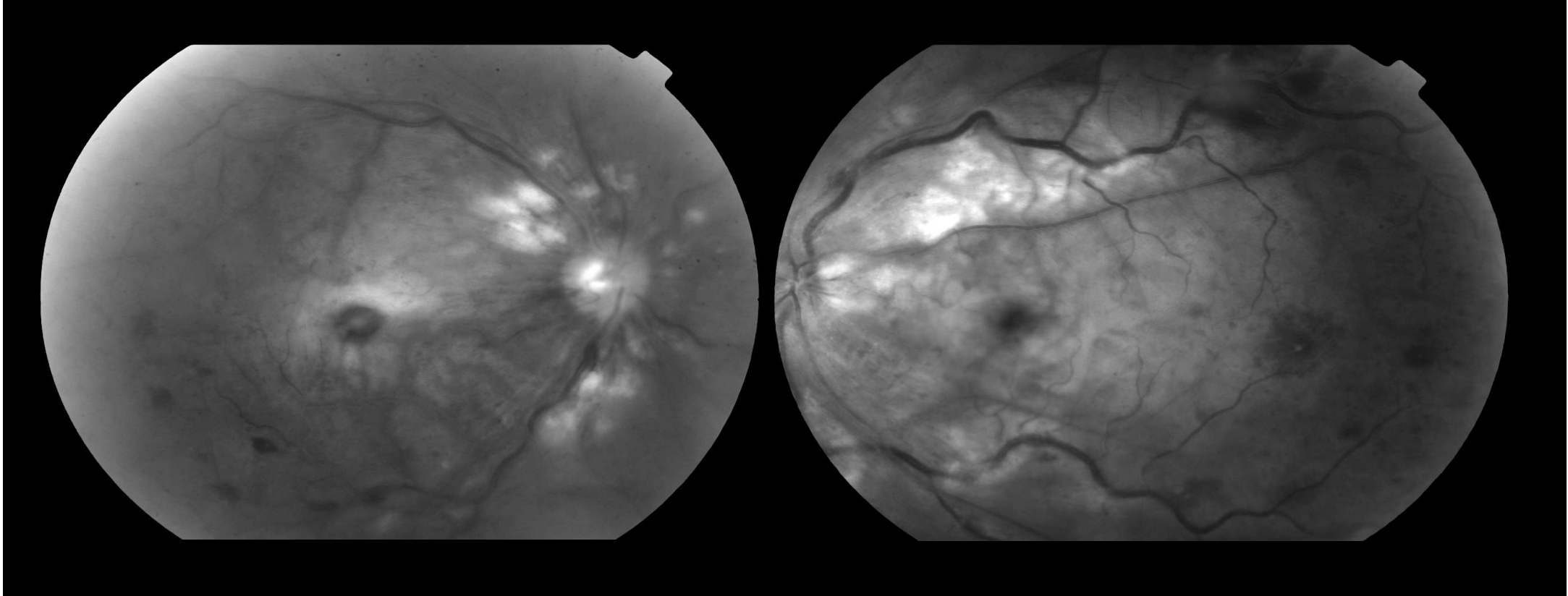
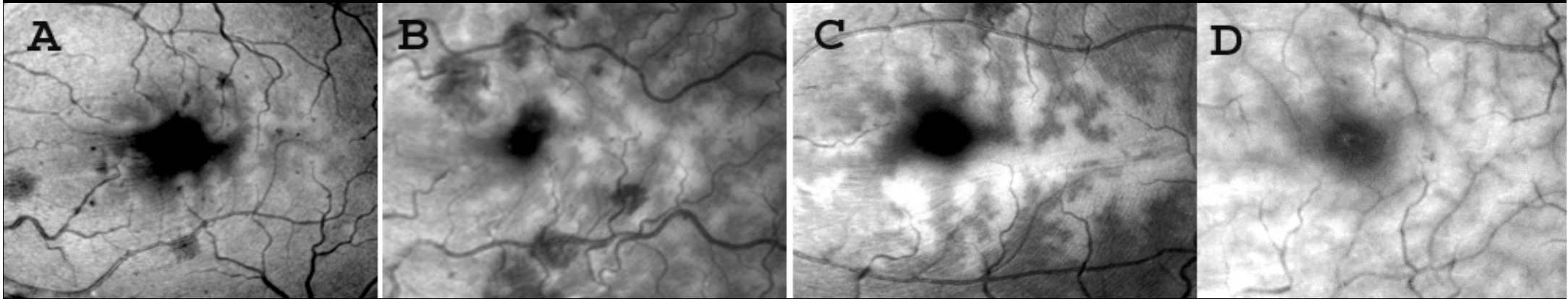
Final LogMAR VA

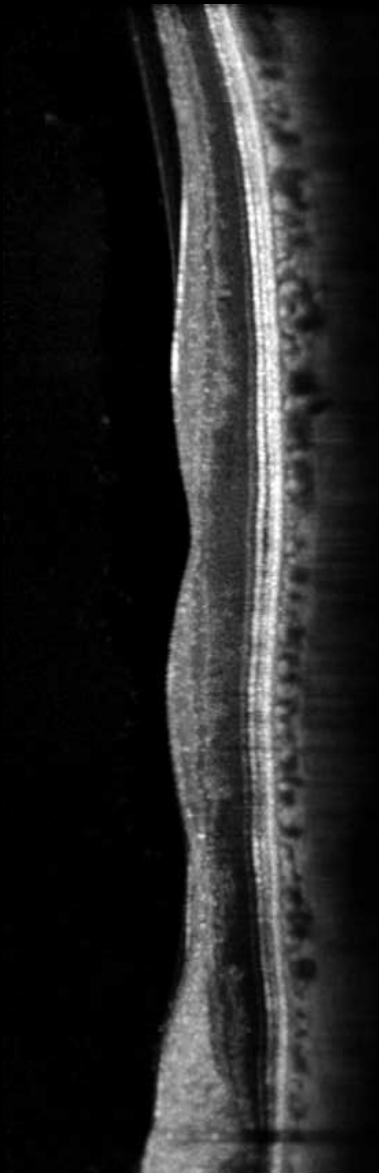


QUELQUES EXEMPLES DE TYPE A



CONTINUUM DE PVW À OCCLUSION DITE « COMBINÉE »





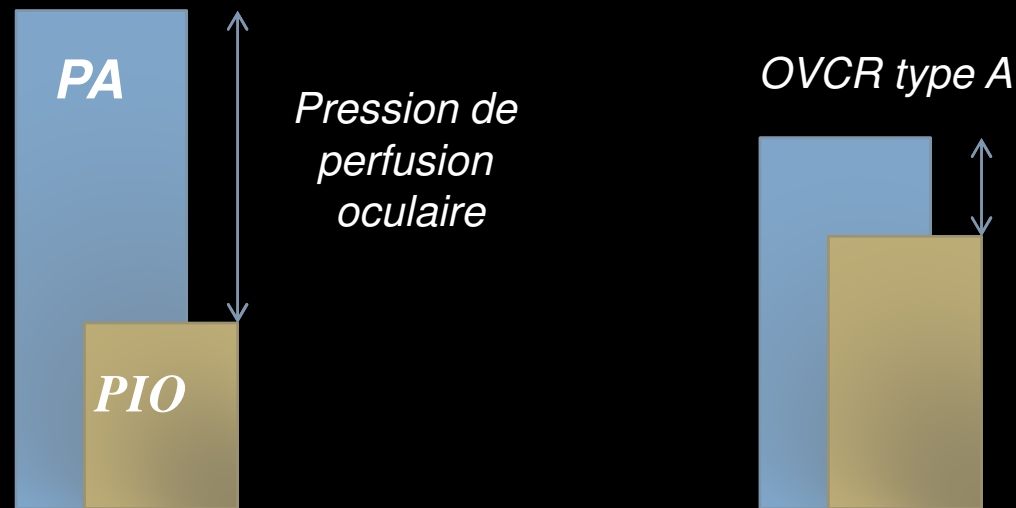
TRAITEMENT DU TYPE A

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?







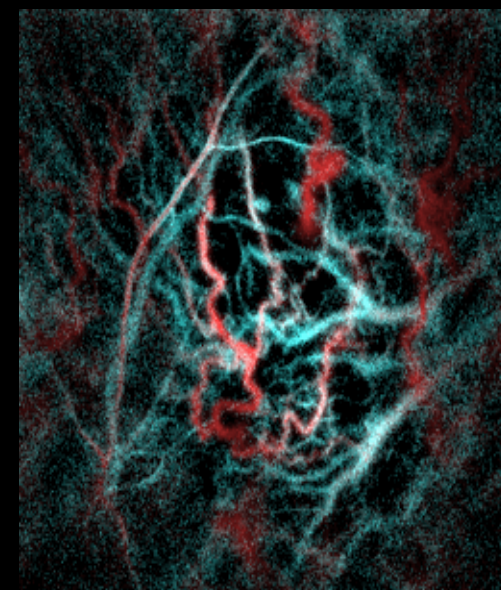
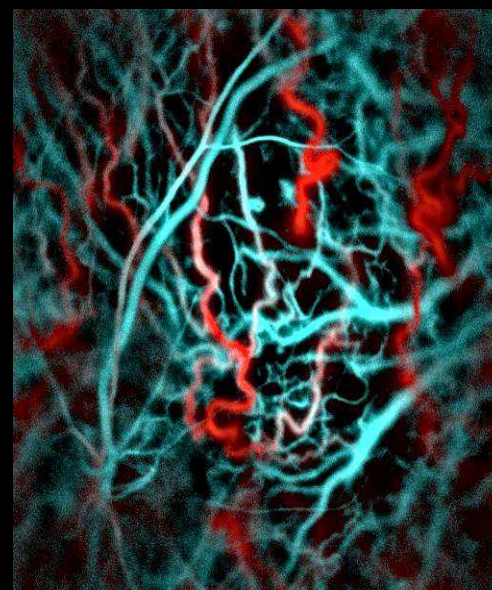
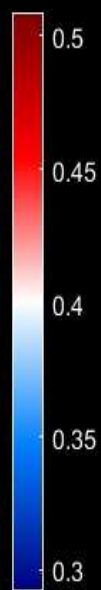
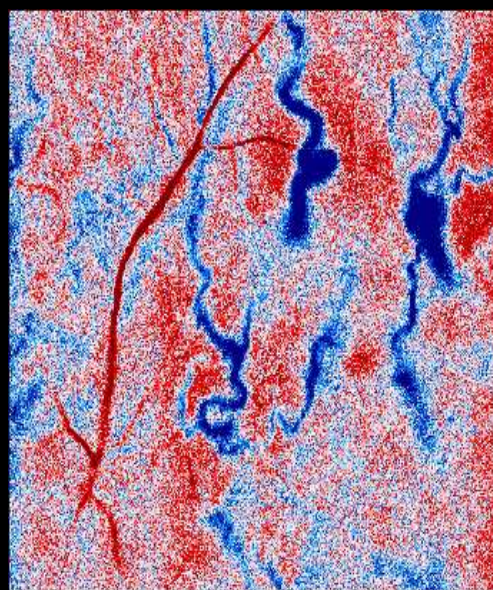
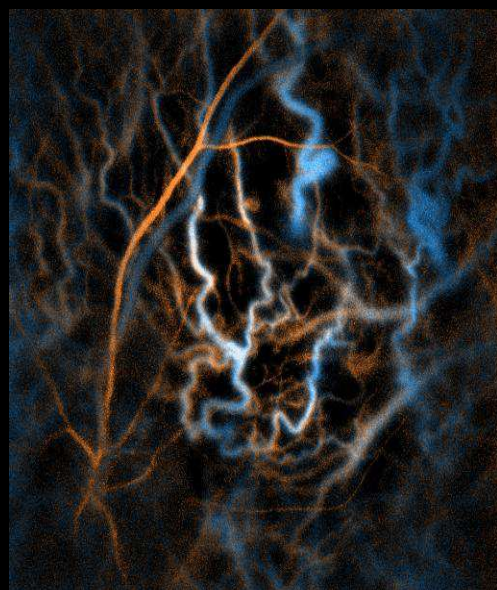
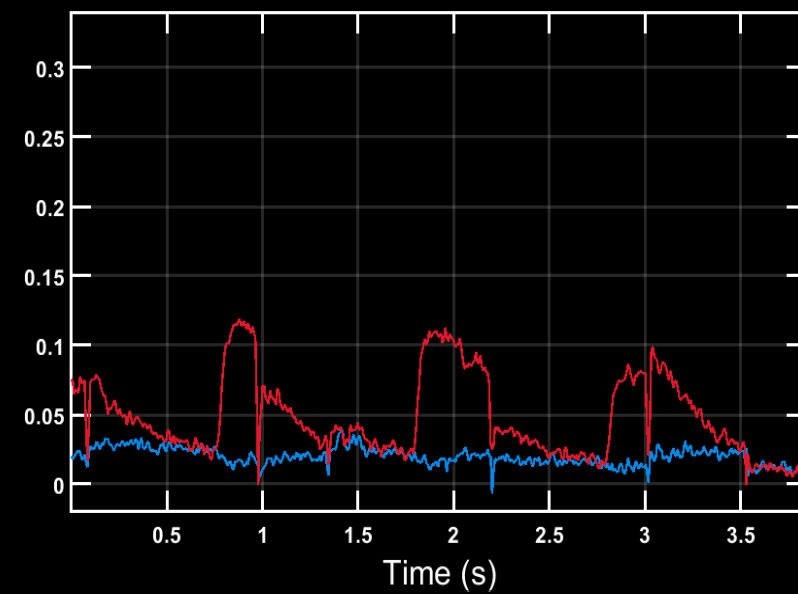
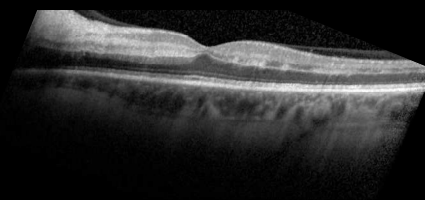
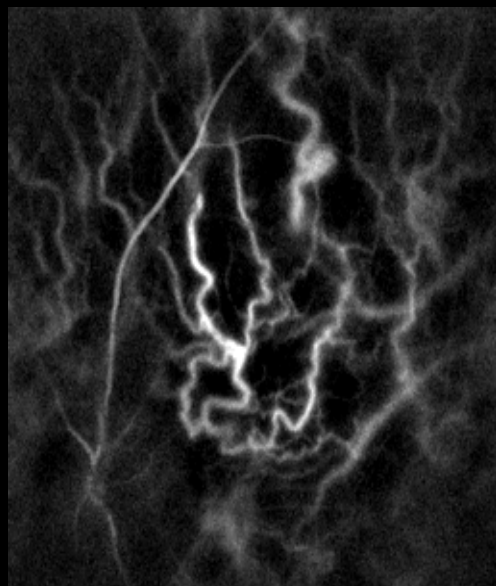
200 μm

200 μm

200103
CAJ0170
OS

- $IR = 0.80$
- $IP = 1.77$
- $IR = -0.04$
- $IP = -0.03$

Type A

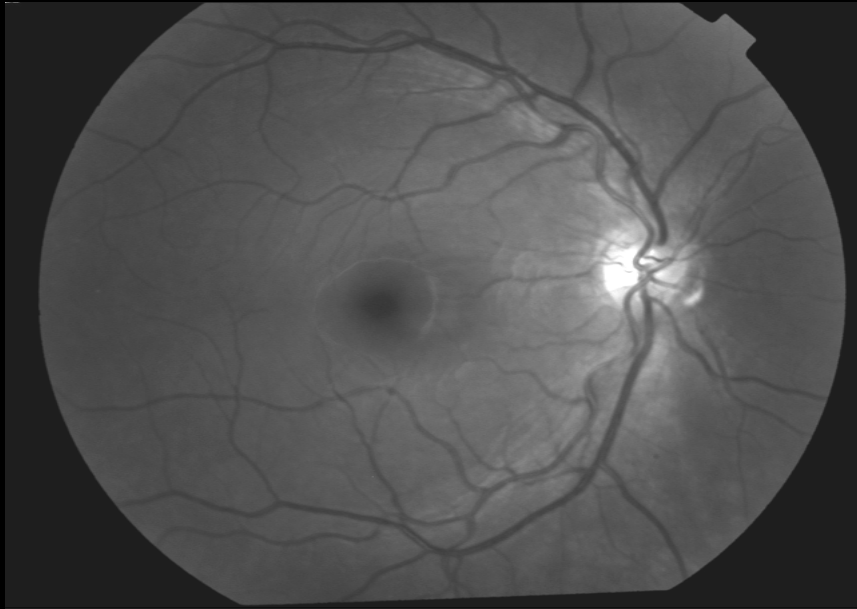


QUE SE PASSE-T-IL AU COURS DES
OVCR DE TYPE B?

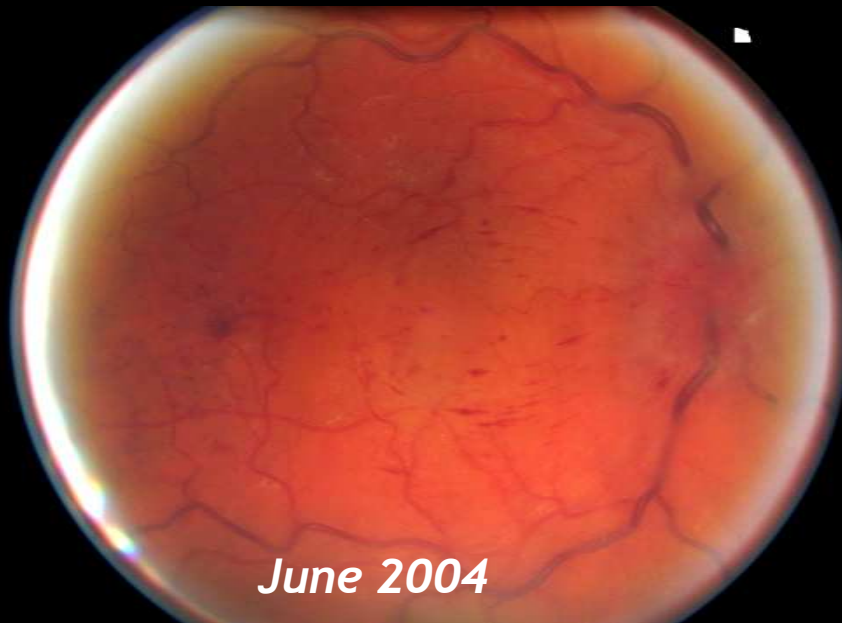
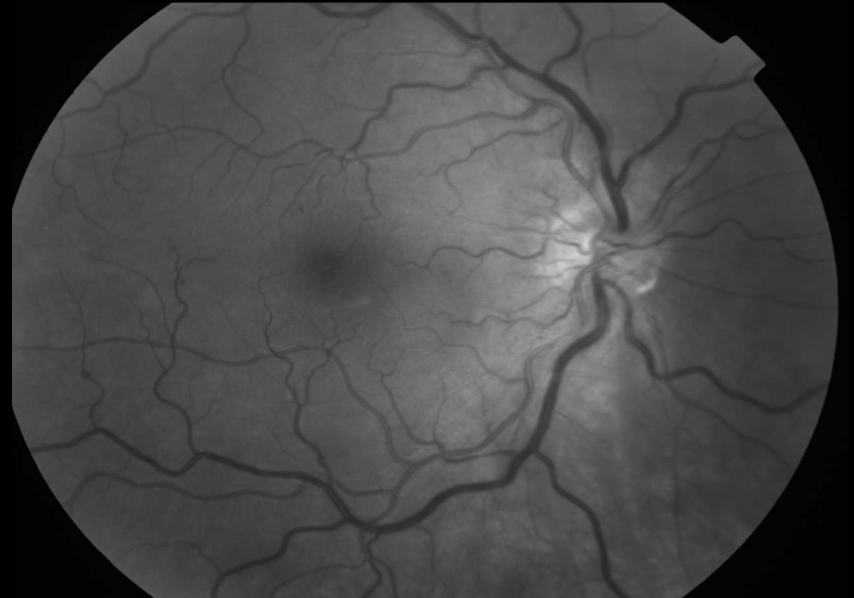




June 2003

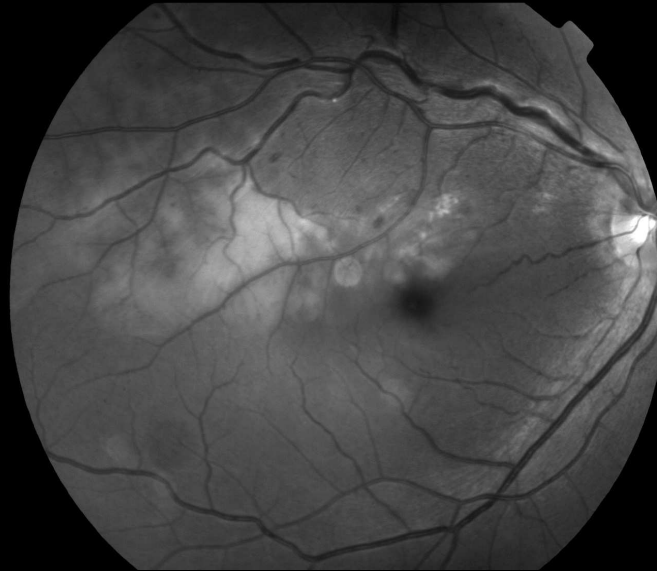


March 2004



June 2004

➤ Les héli-occlusions peuvent être de type A ou B



➤ Les OBVR sont (virtuellement) toutes des types B



OCCLUSIONS DE BRANCHE VEINEUSE

OD



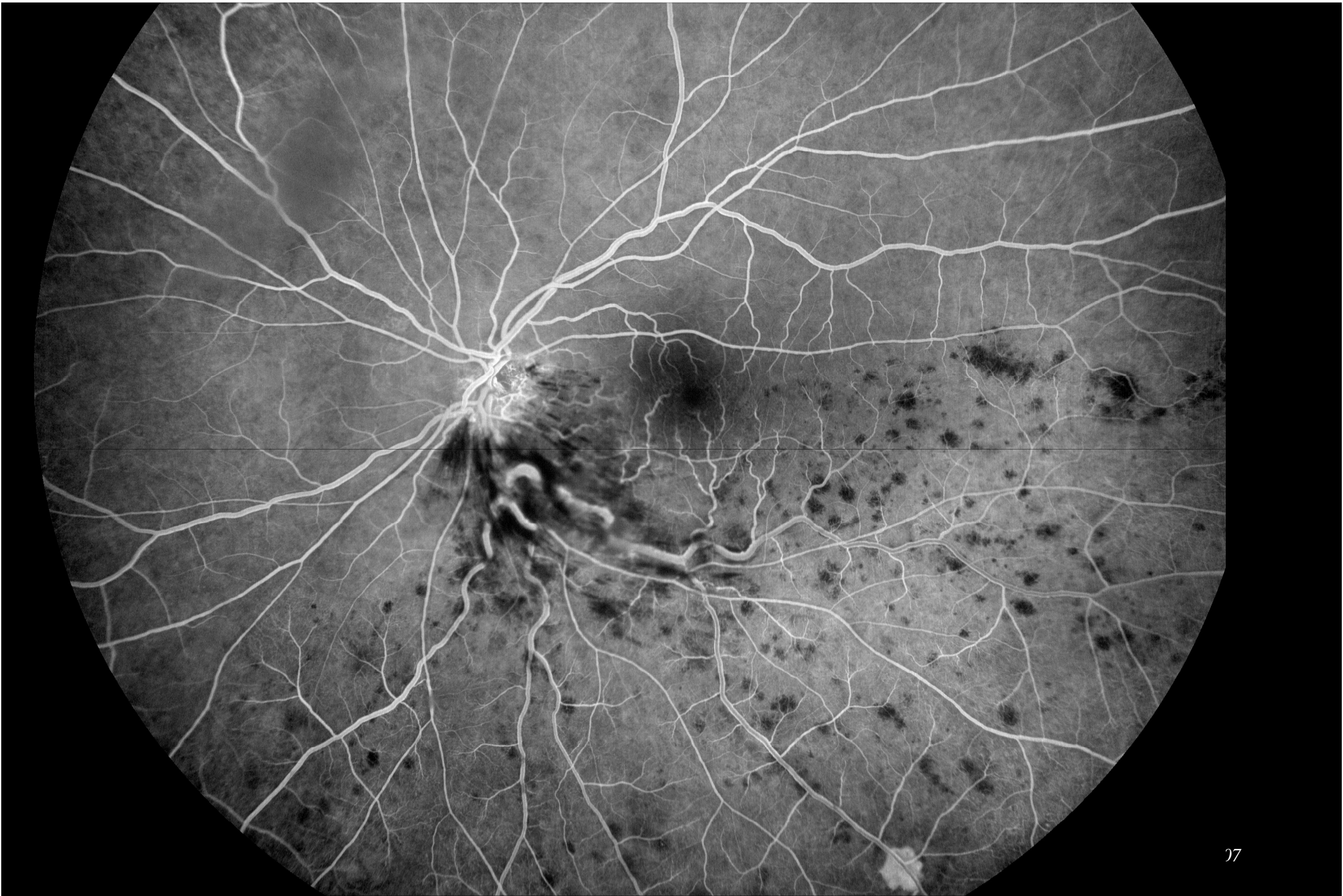
DU IMAGE

104



DU IMAC



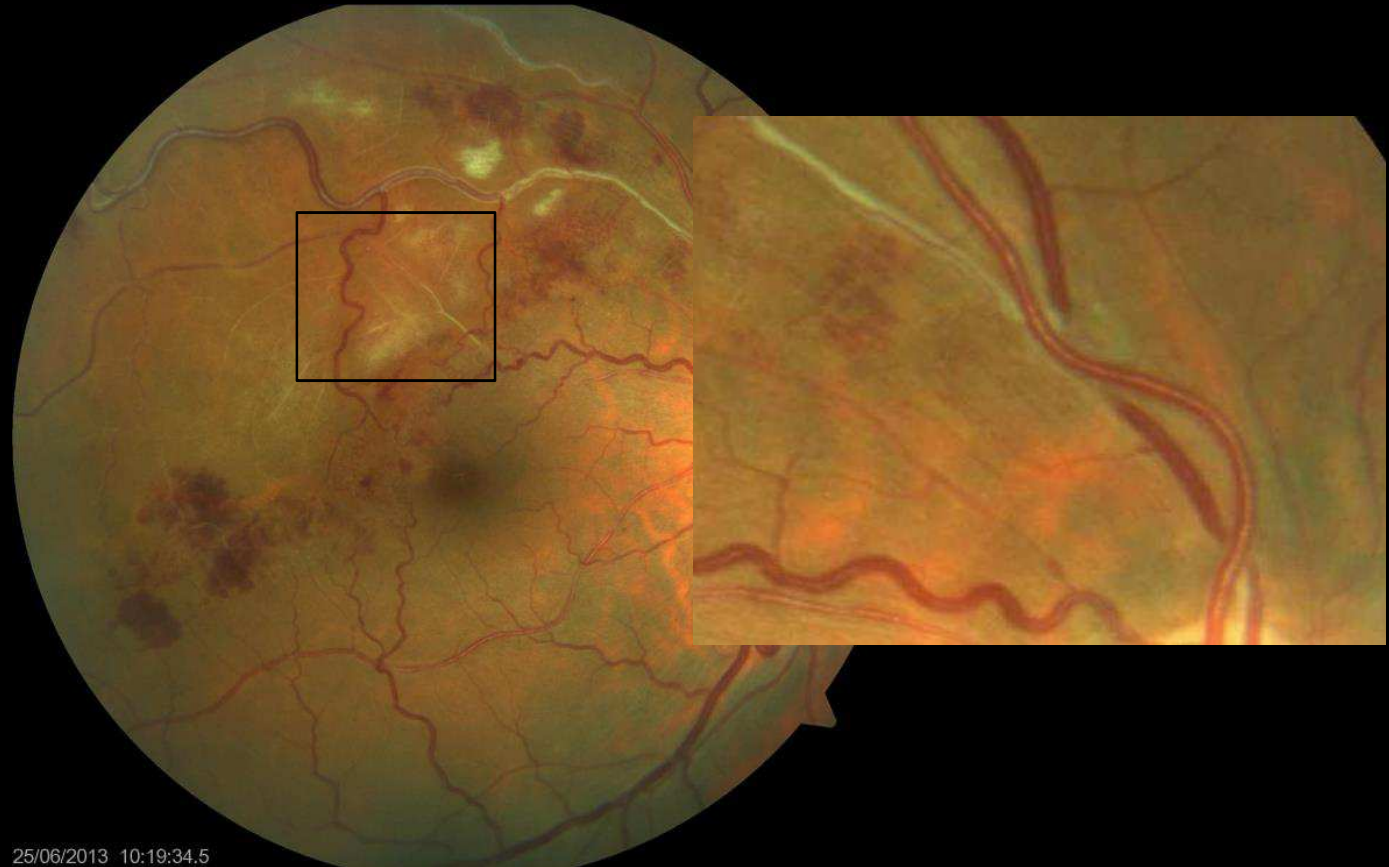




DU 1A



LE LIEU DE SURVENUE DES OBVR EST ACCESSIBLE, MAIS LE MÉCANISME EN CAUSE EST INCERTAIN

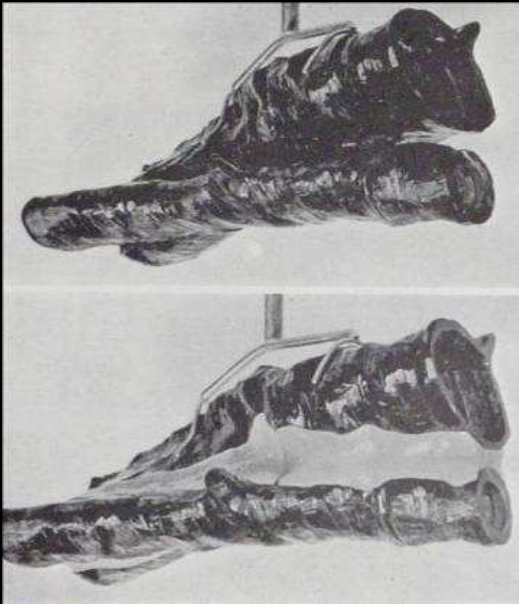


25/06/2013 10:19:34.5

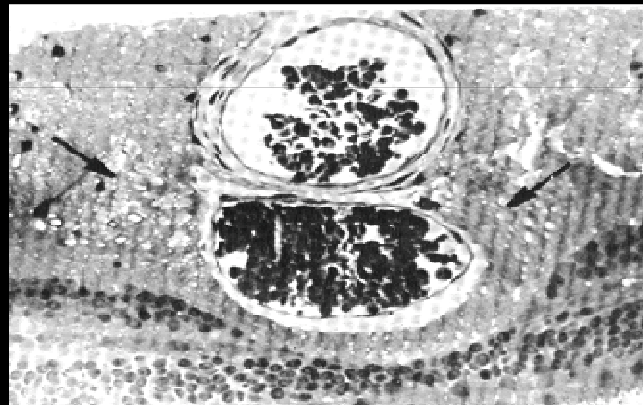
ALORS QUE SE PASSE-T-IL DANS UN SIGNE DU CROISEMENT?

L'histologie montre

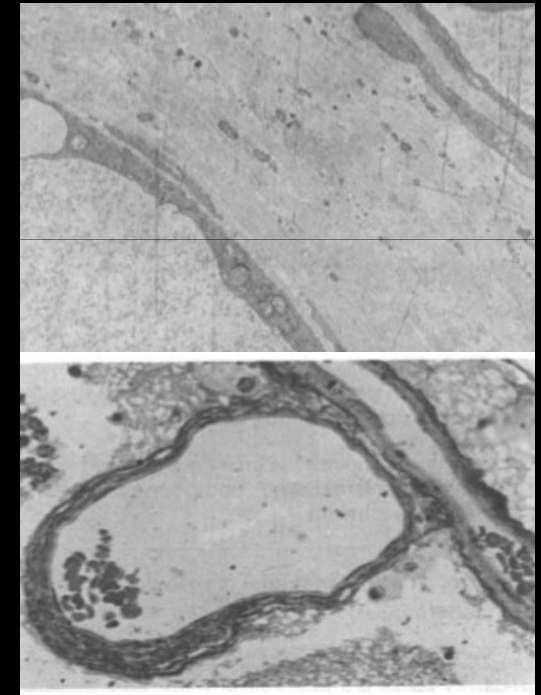
- *des modifications histologiques rétinienne autour du couple artérioveineux*
- *Mais que la veine n'est pas directement atteinte par l'artère*



Seitz et coll. 1964

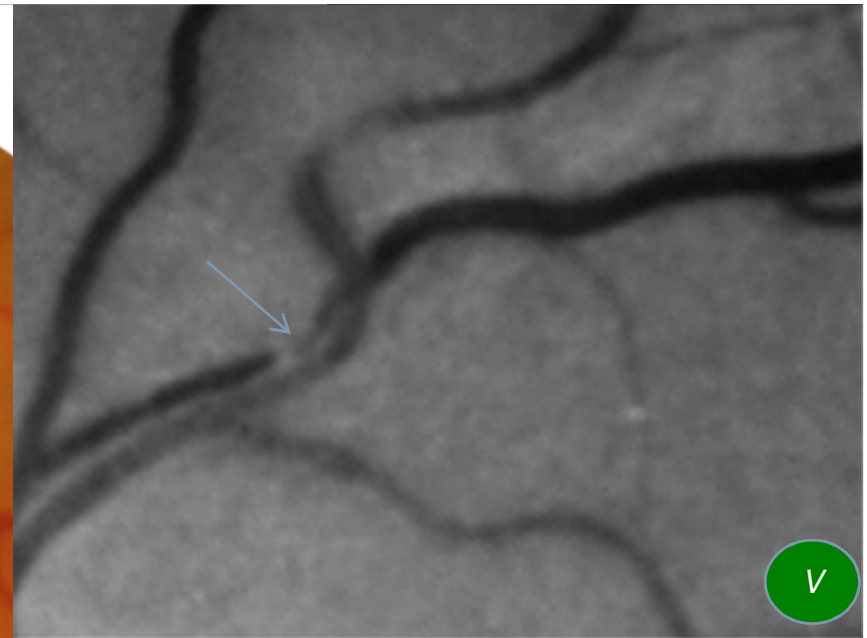


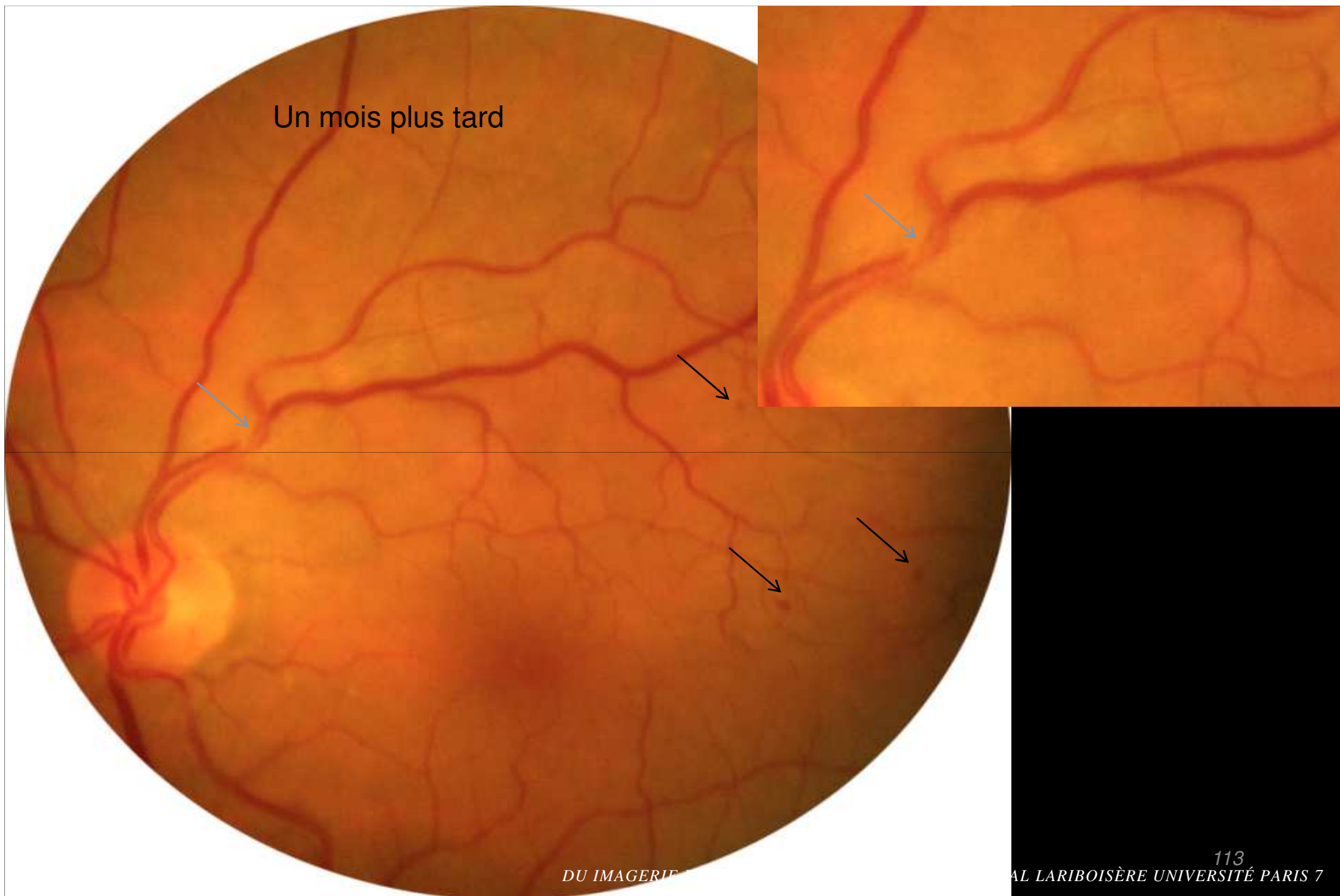
Kimura et al. Int Ophthalmol 2005



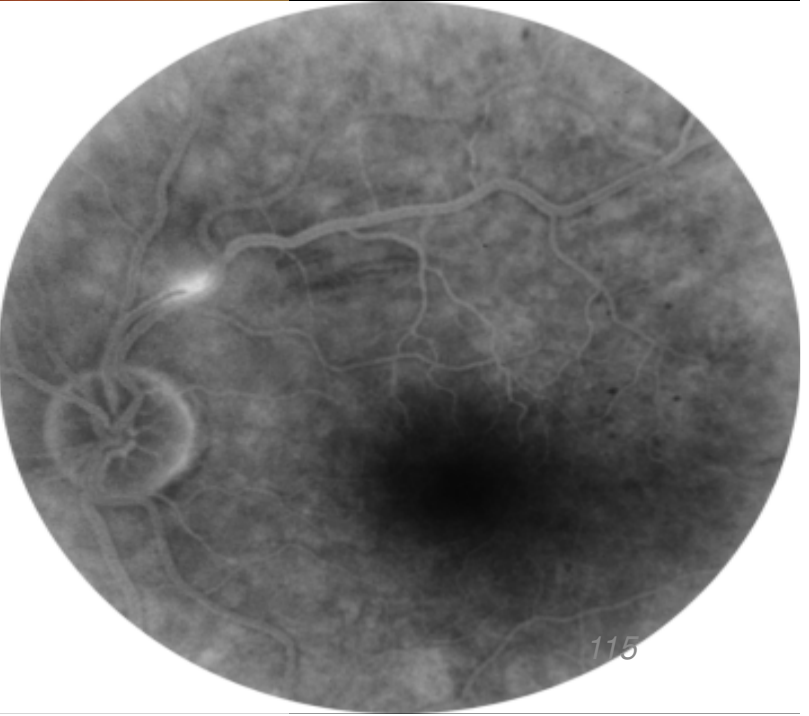
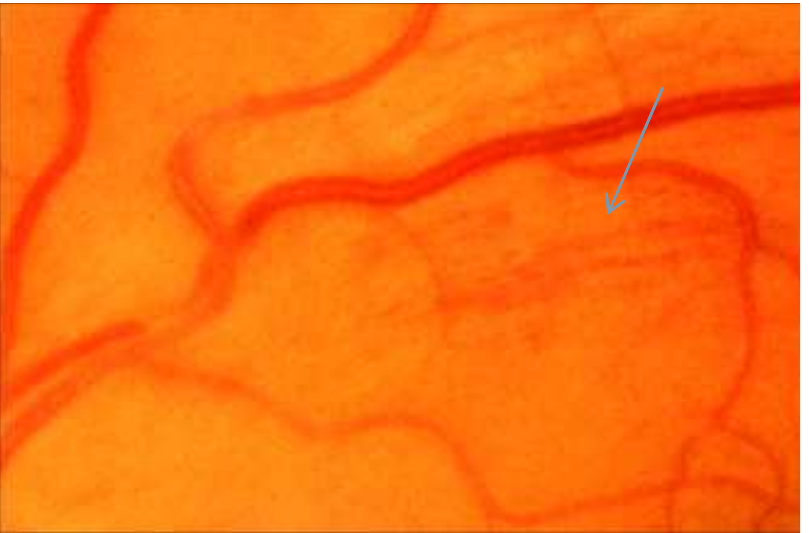
- *Jefferies et al.*
- *Aust NZ J Ophth 1993*

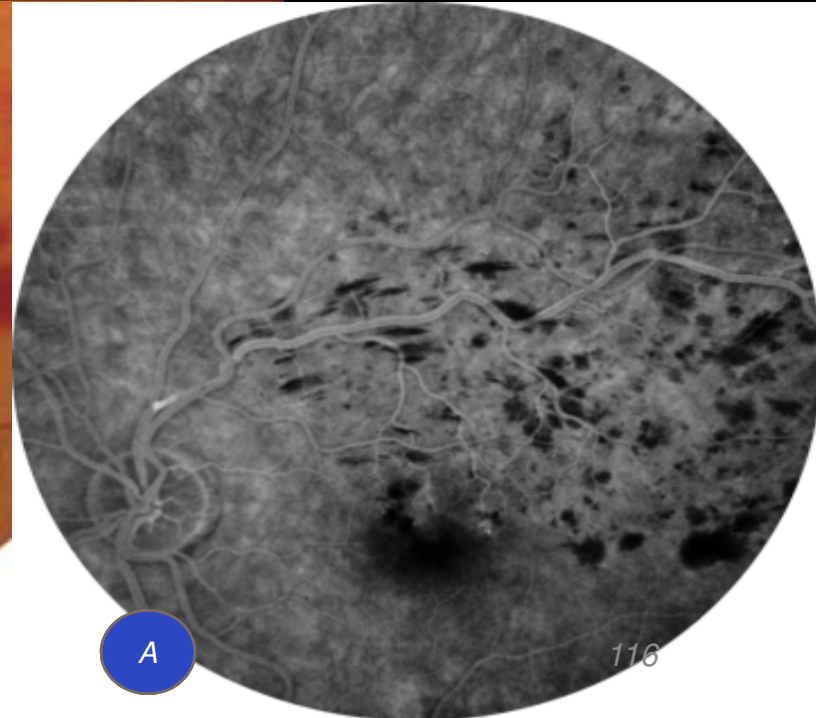
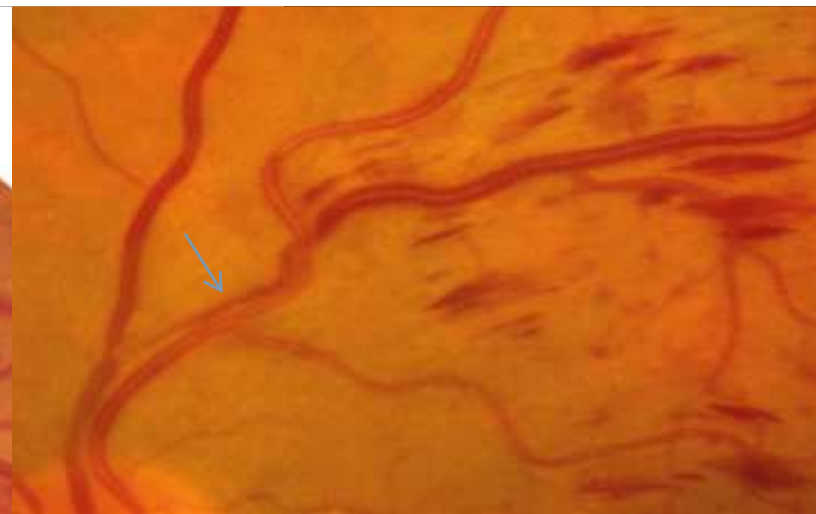
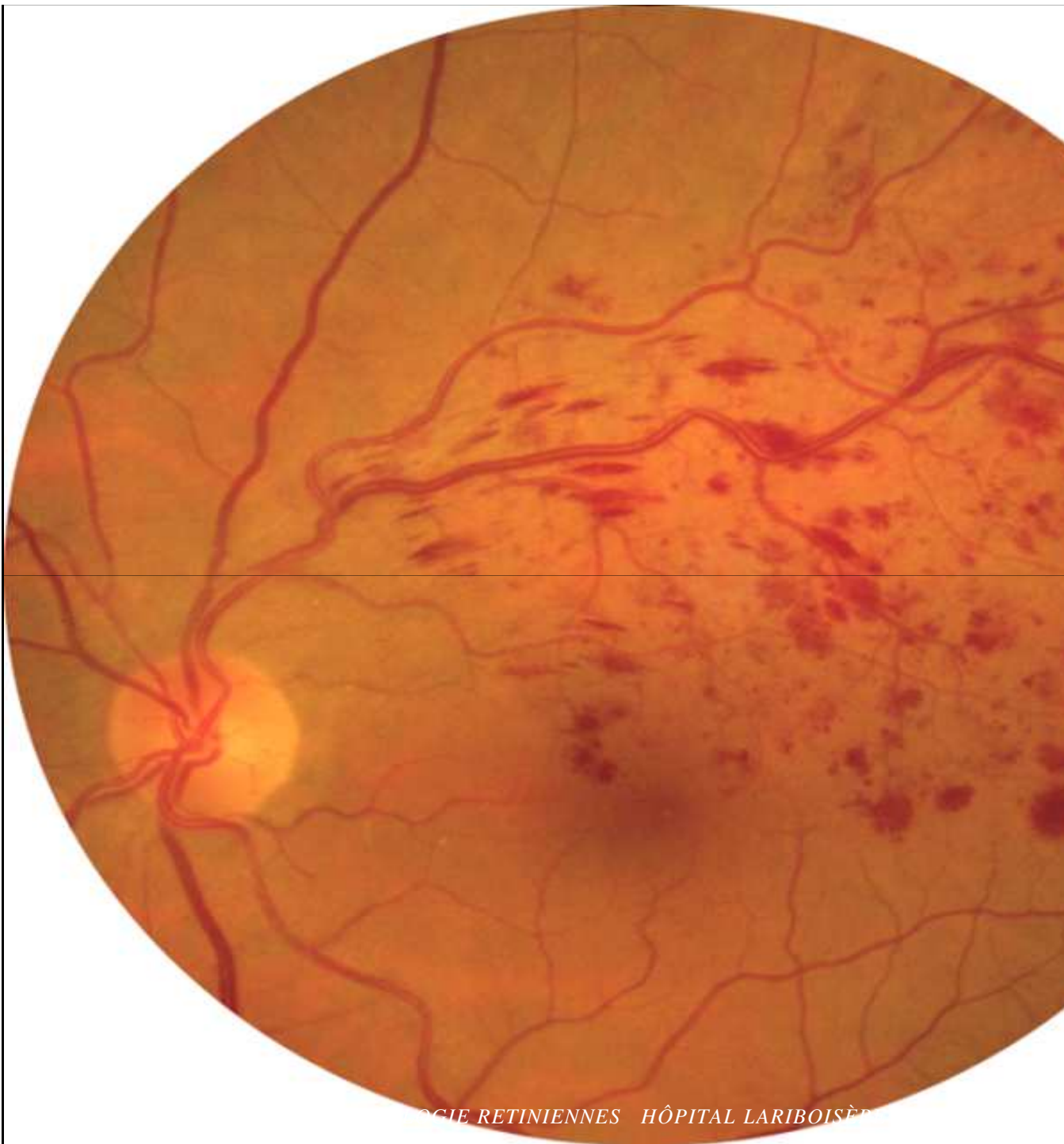
→ Fausse évidence de l'écrasement veineux par l'artère











A

116

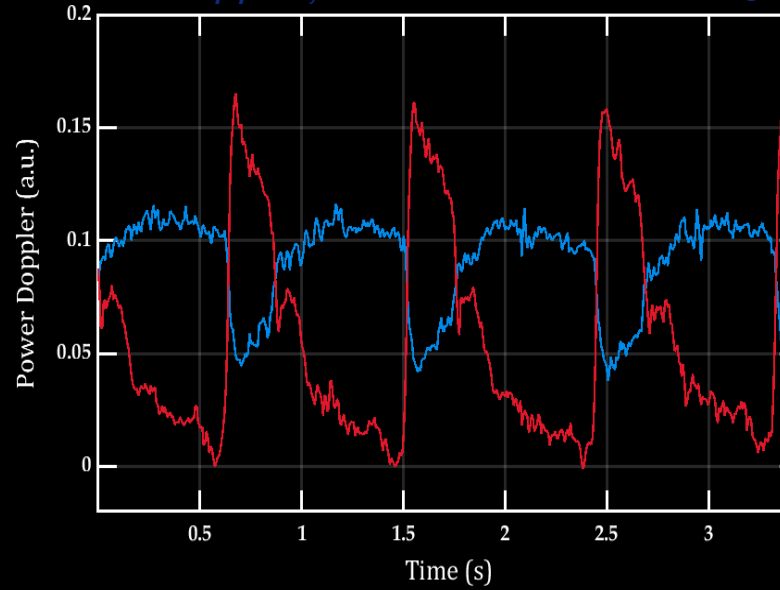
191025
WAL0129
OS

- $IR = 0.91$
- $IP = 2.28$
- $IR = -0.97$
- $IP = -0.54$

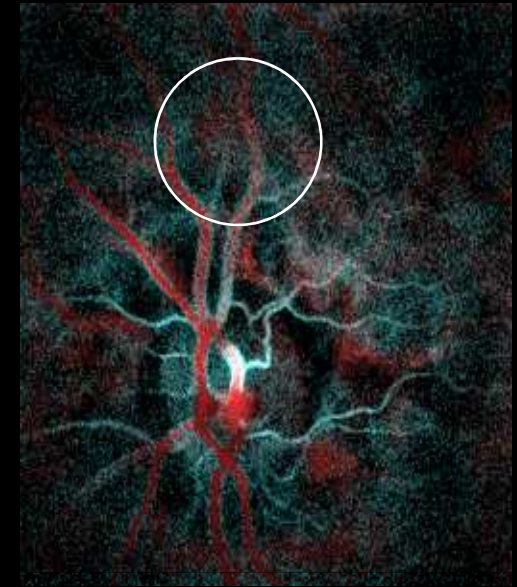
*Power Doppler movie
(x0.8)*



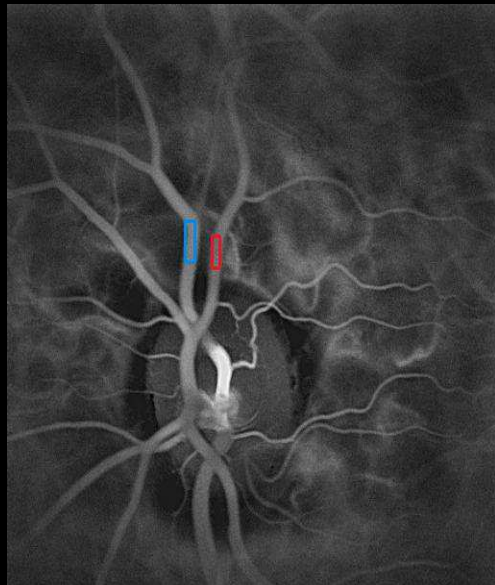
Power Doppler, 2-37 kHz



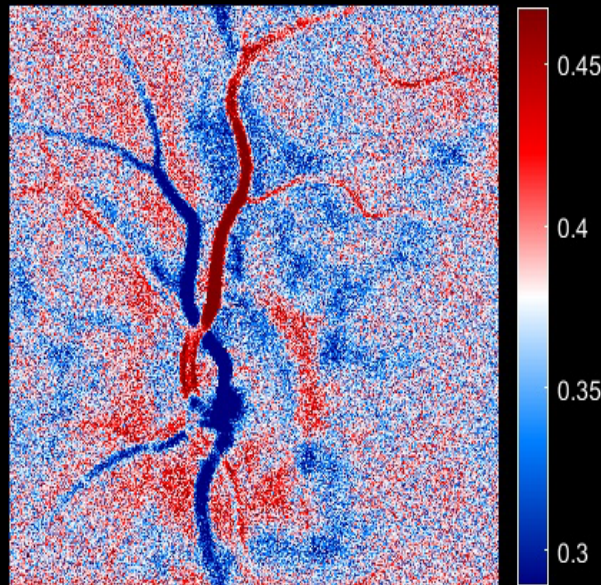
Power Doppler variations



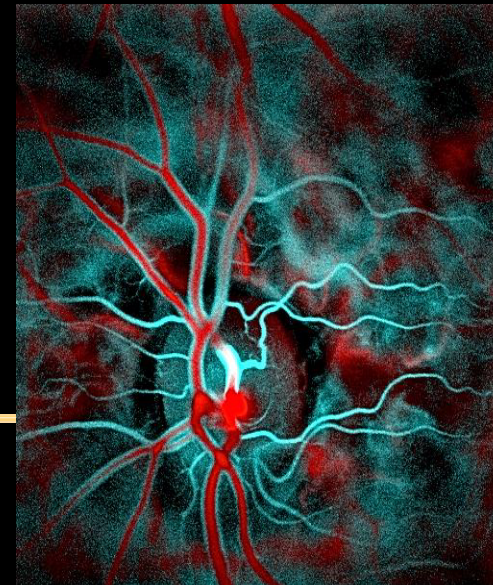
Systole / Diastole



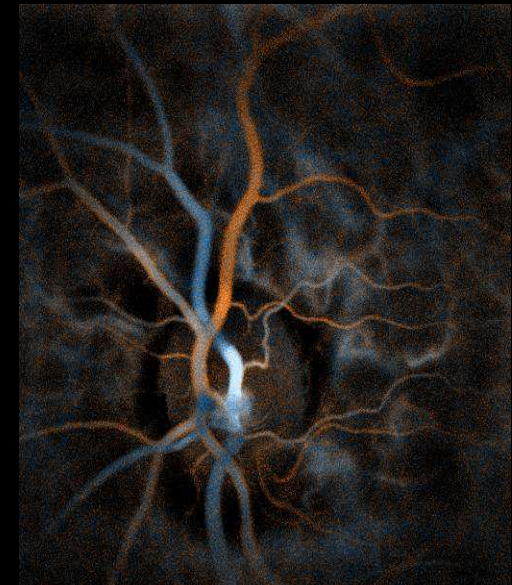
Resistivity map



Low flow / high flow



Composite movie



APRÈS LA PHASE AIGUE

EVOLUTION

- Quel que soit le type d'OVR ou son étendue, si elle ne guéri pas, un remodelage micro vasculaire peut apparaître:

- Occlusion capillaire
- Collatérales
- Macroanévrismes

NON PERFUSION

OCCLUSION CAPILLAIRE

➤ Définit la forme dite « non perfusée »

➤ la présence de capillaires occlus est très fréquente comme la montré l'imagerie grand champ

➤ *Il n'y a pas de seuil clairement définissable entre « forme non perfusée » et « formes perfusées »*

➤ Cause de l'occlusion capillaire?

➤ Seuls facteurs de risque connus:

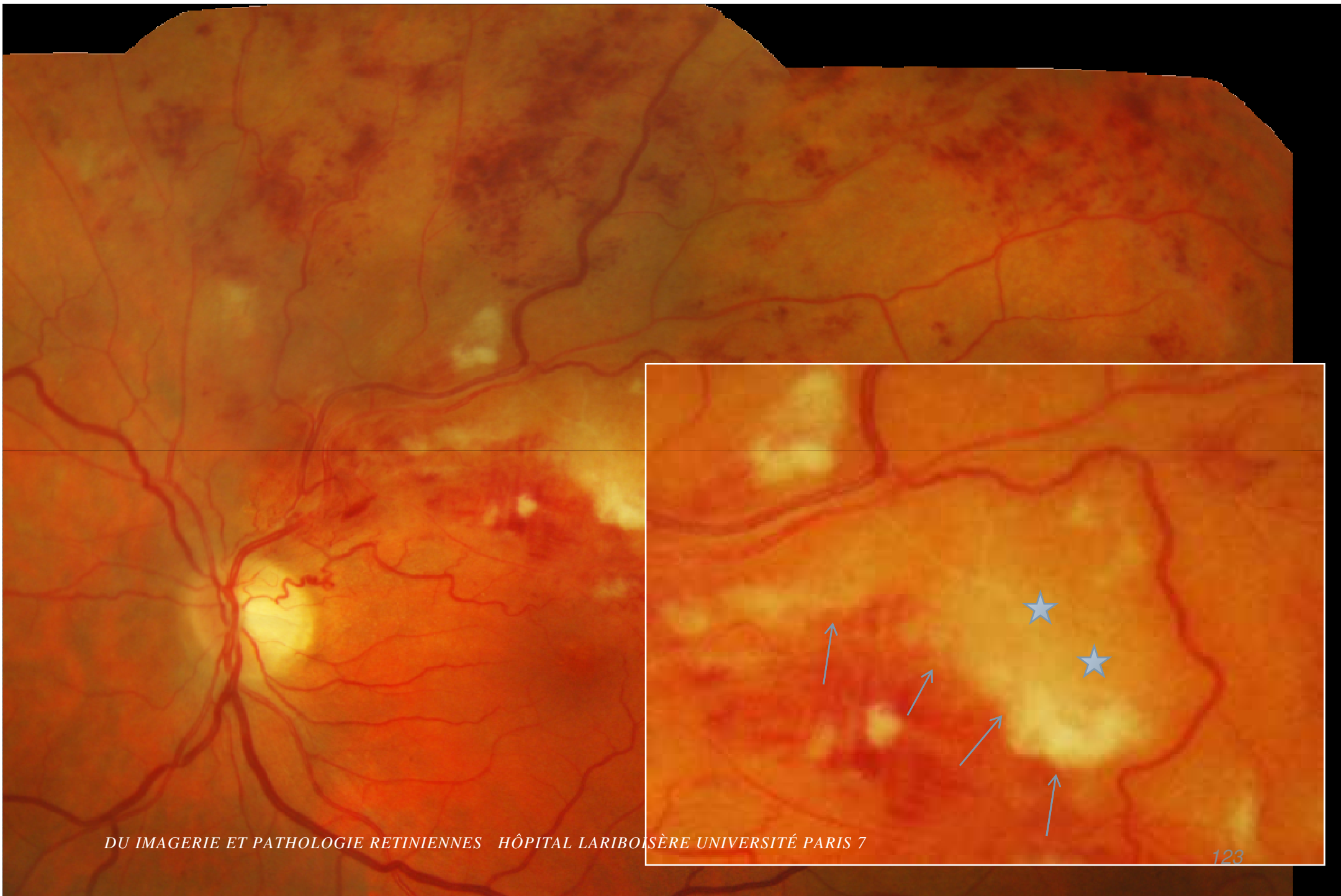
- Age
- Diabète

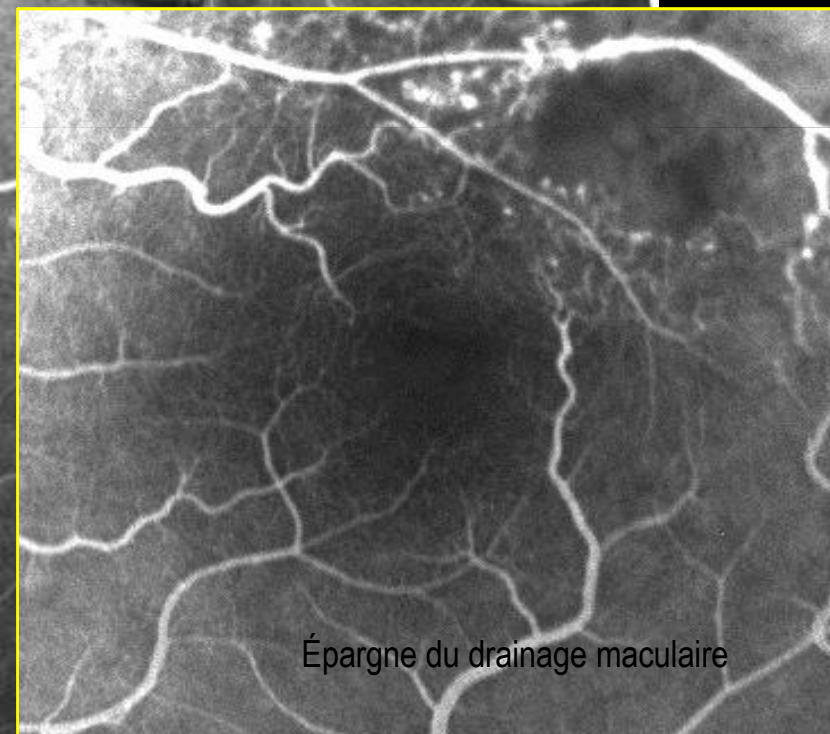
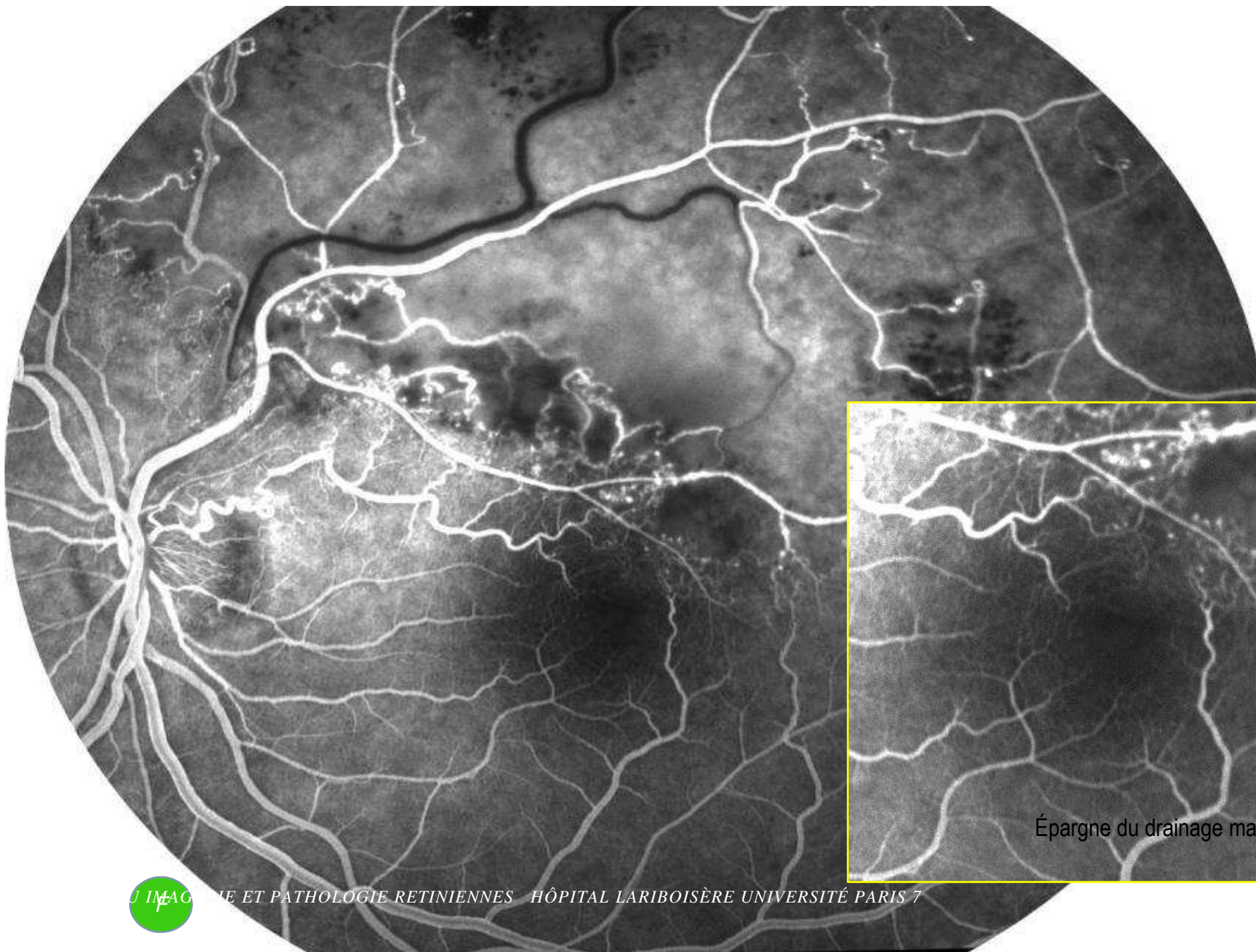
➤ Conséquences:

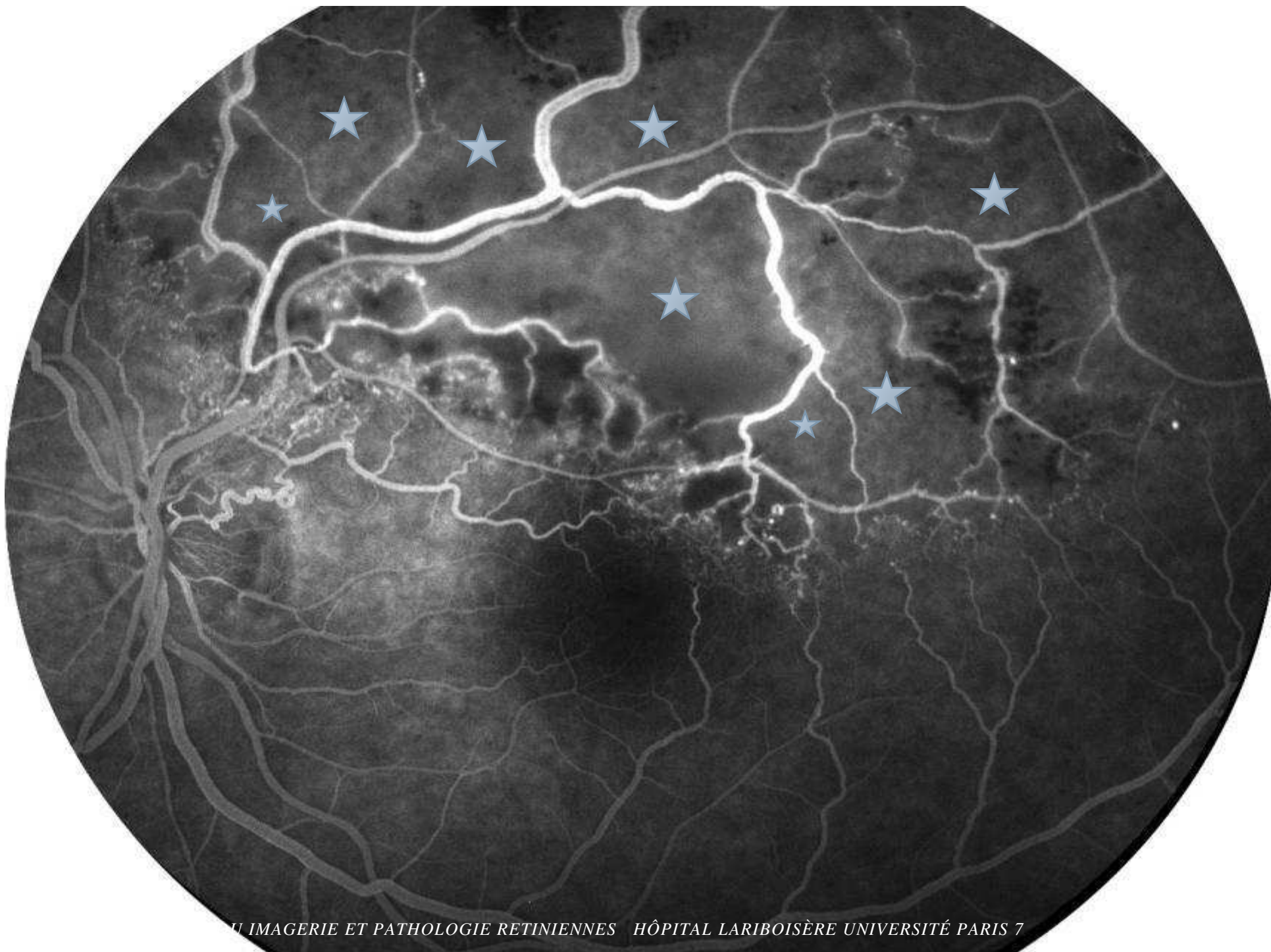
- Baisse acuité visuelle
- Synthèse de facteurs de croissance néo-vasculaires stimulée par l'ischémie neuronale

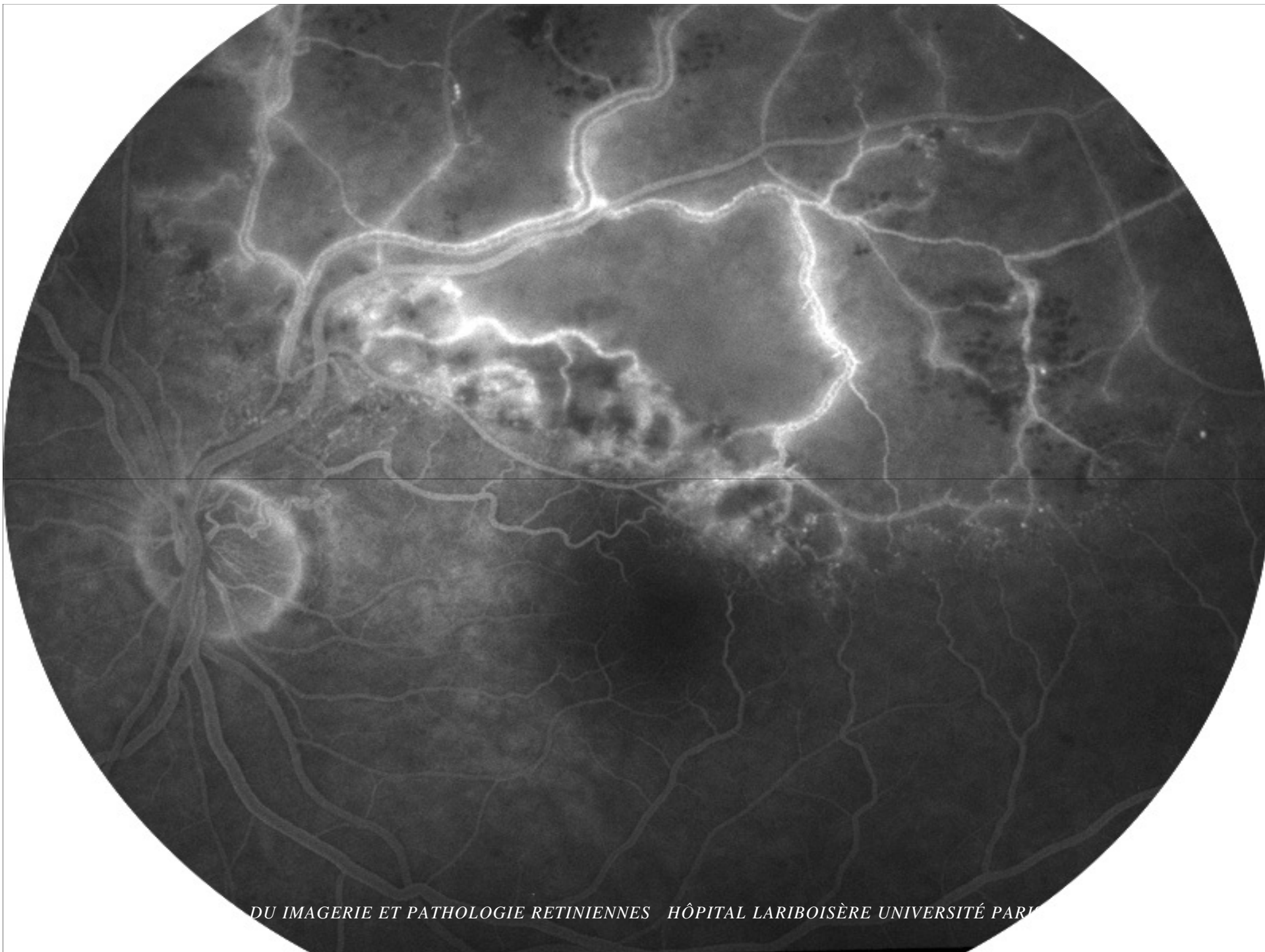


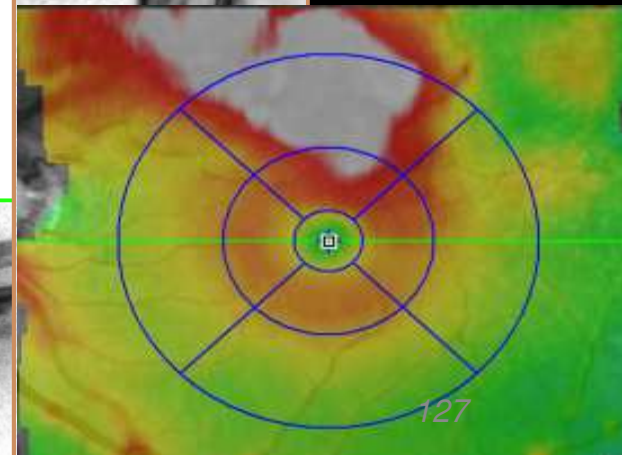
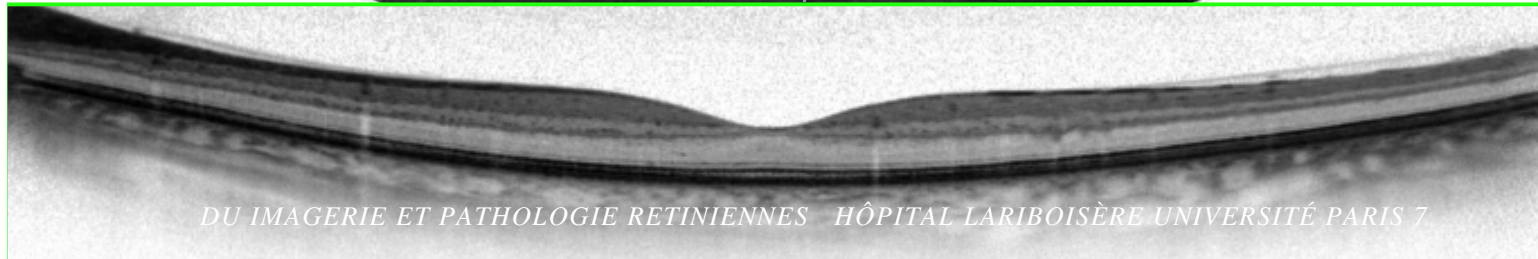
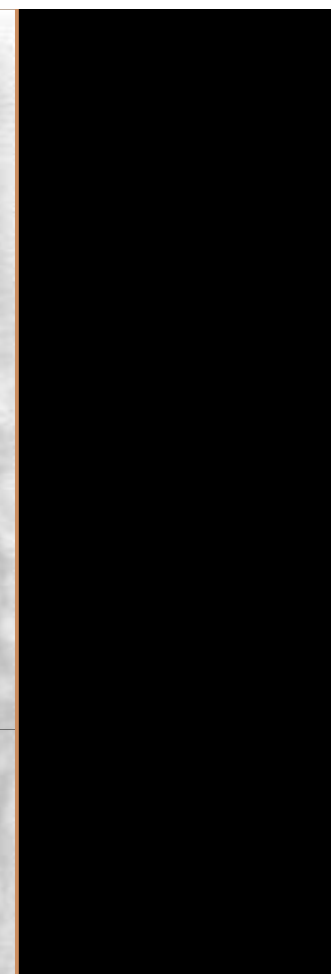
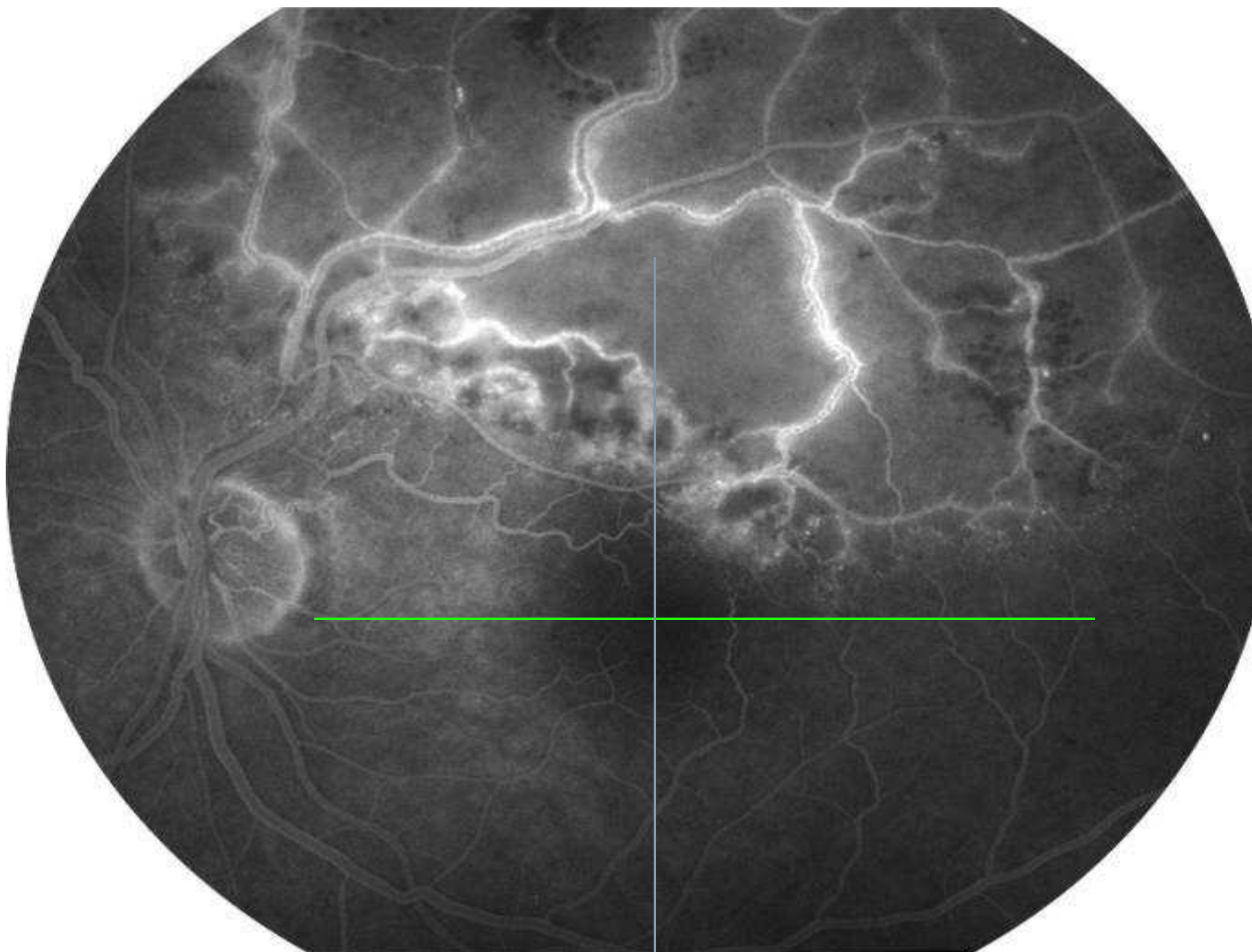




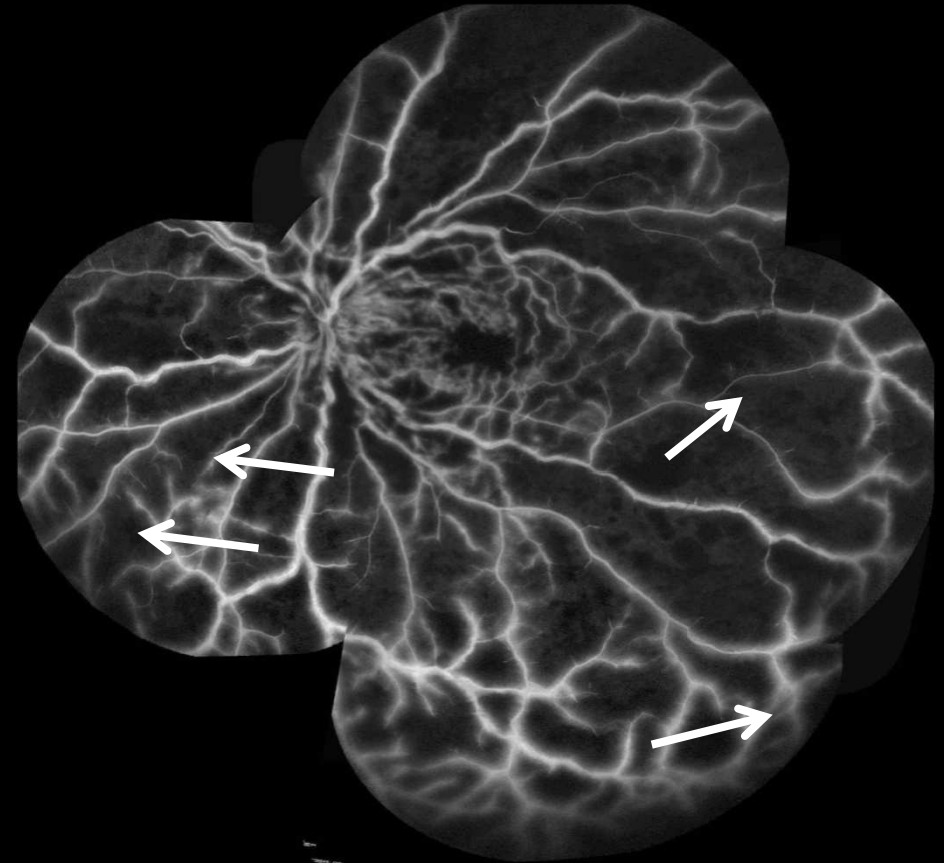
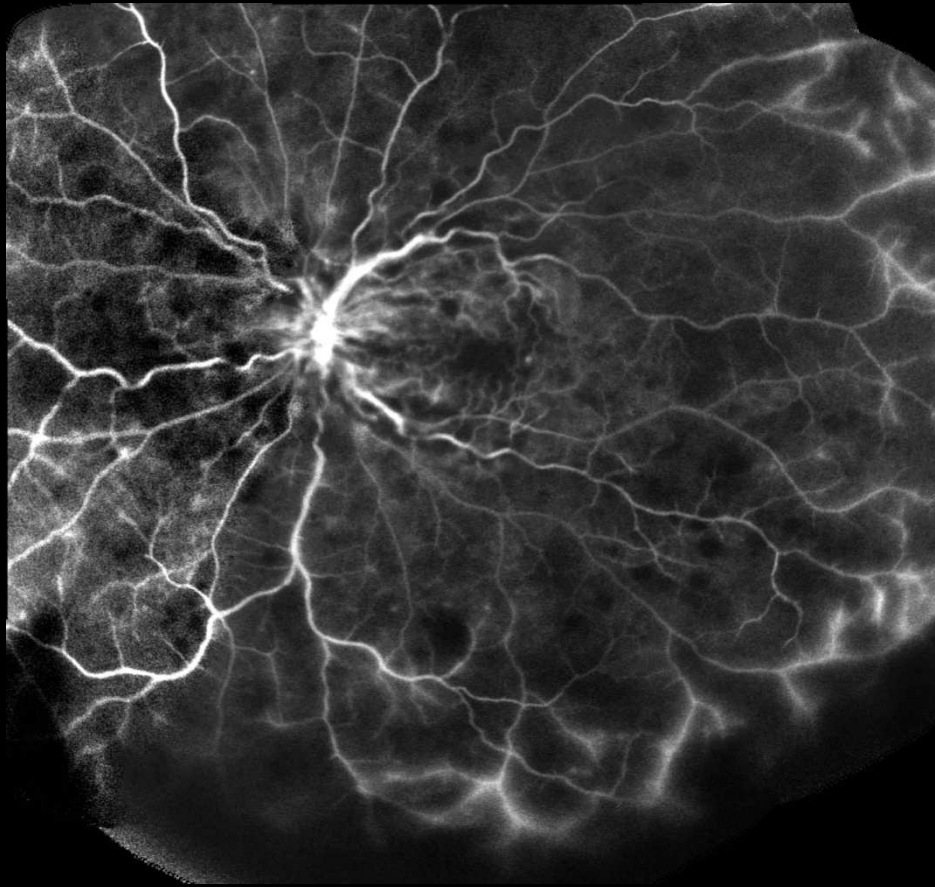




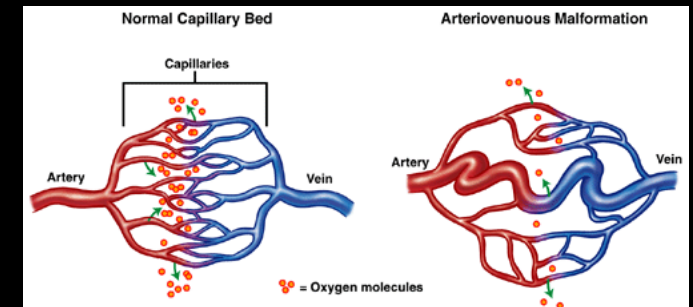




... Mais le plus souvent commence par la périphérie



DÉVELOPPEMENT DE NONPERFUSION CAPILLAIRE:
UN EFFET SHUNT?

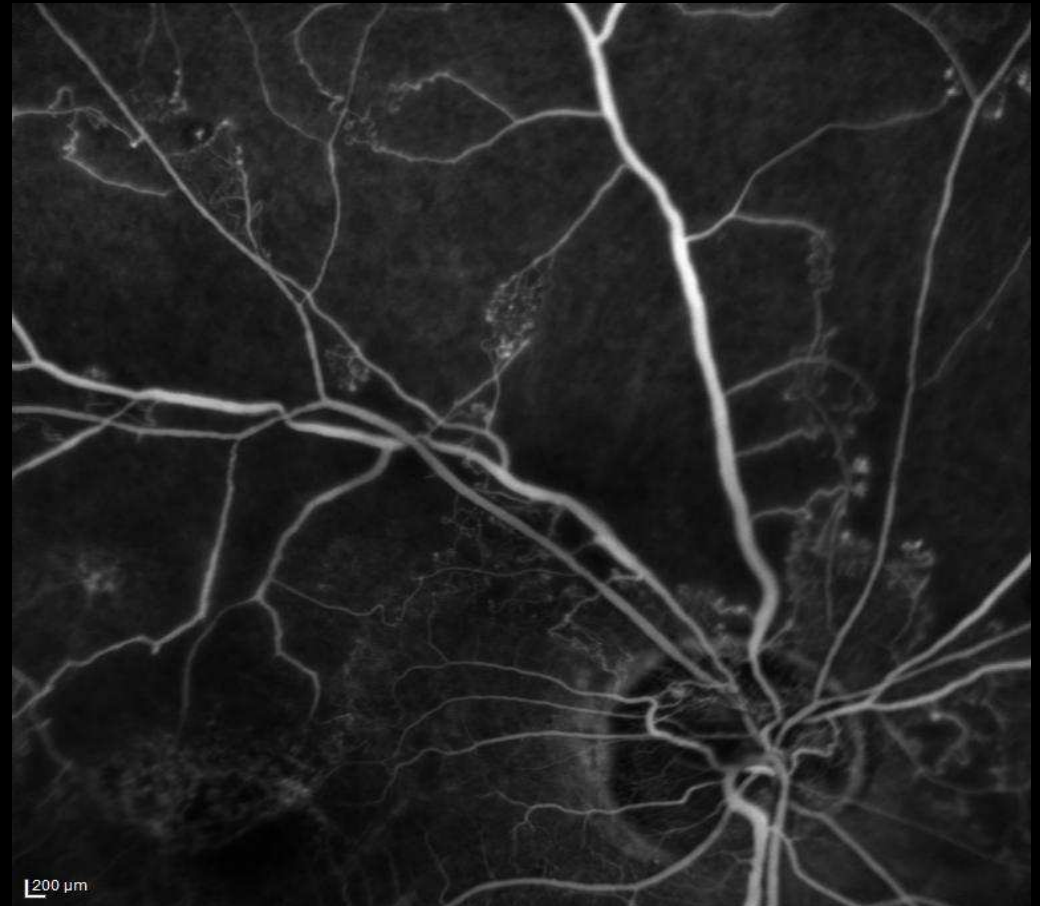






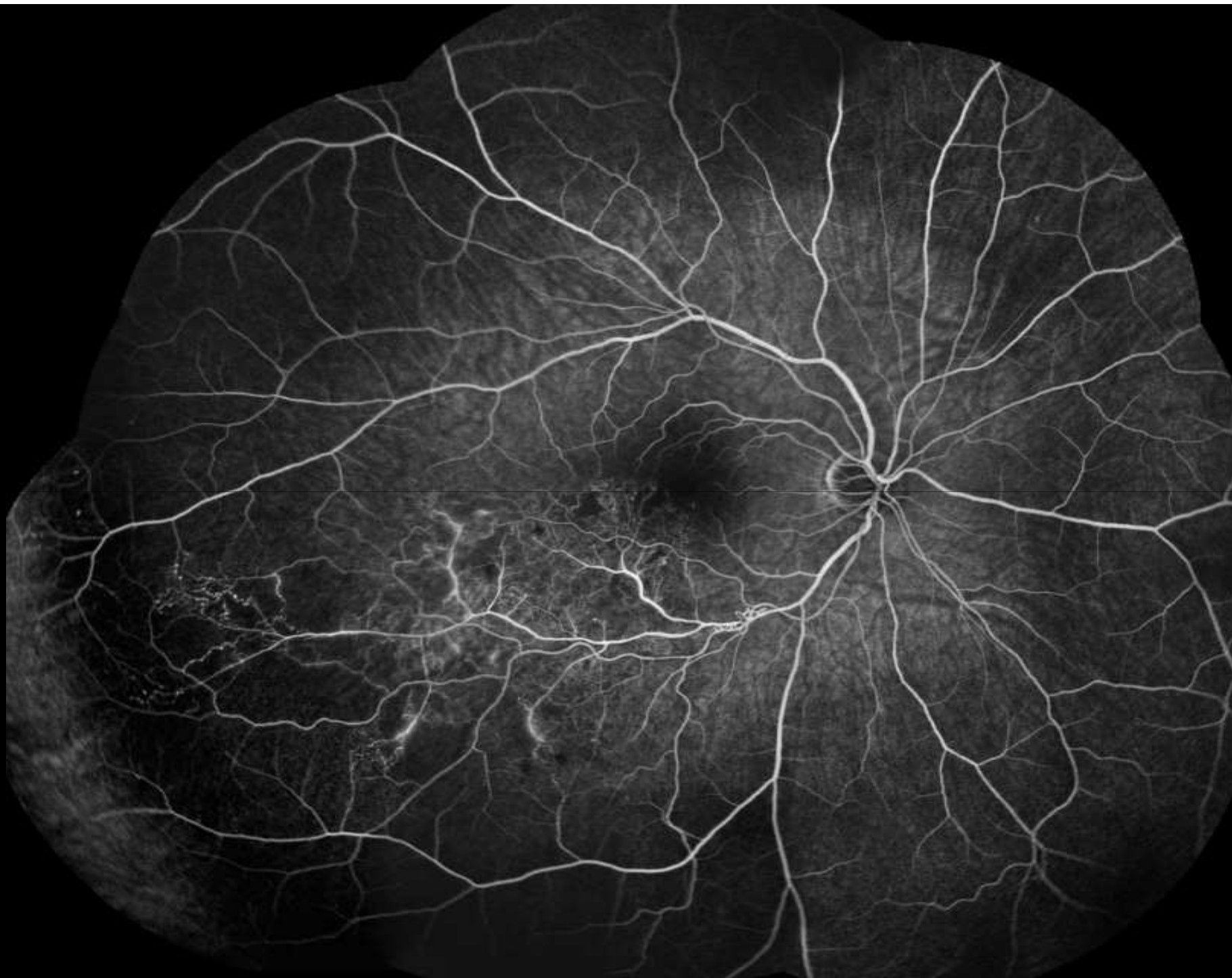
21/02/2014, OD
FA 1:32.26 55° ART(11) 207177.1

HEIDELBERG
ENGINEERING

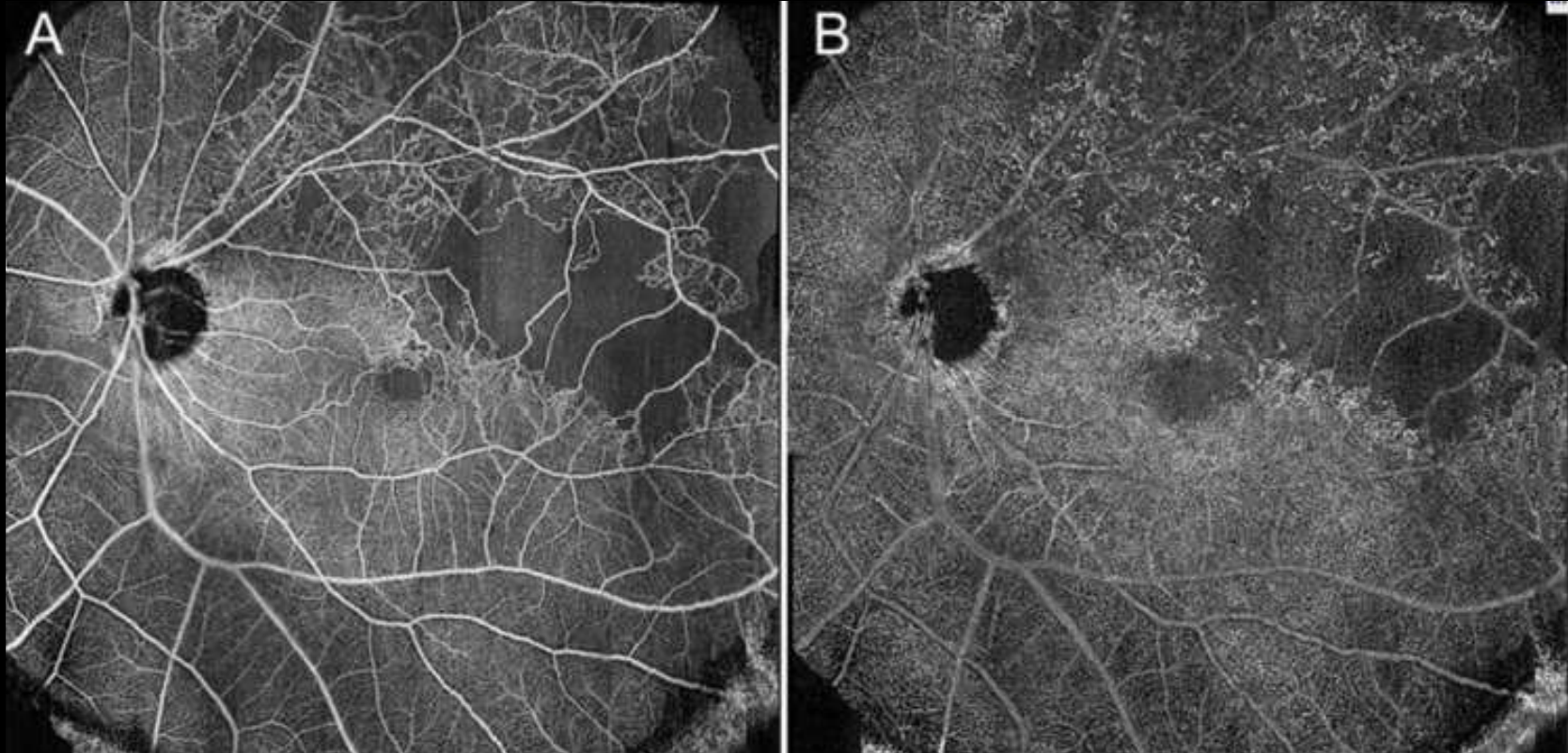


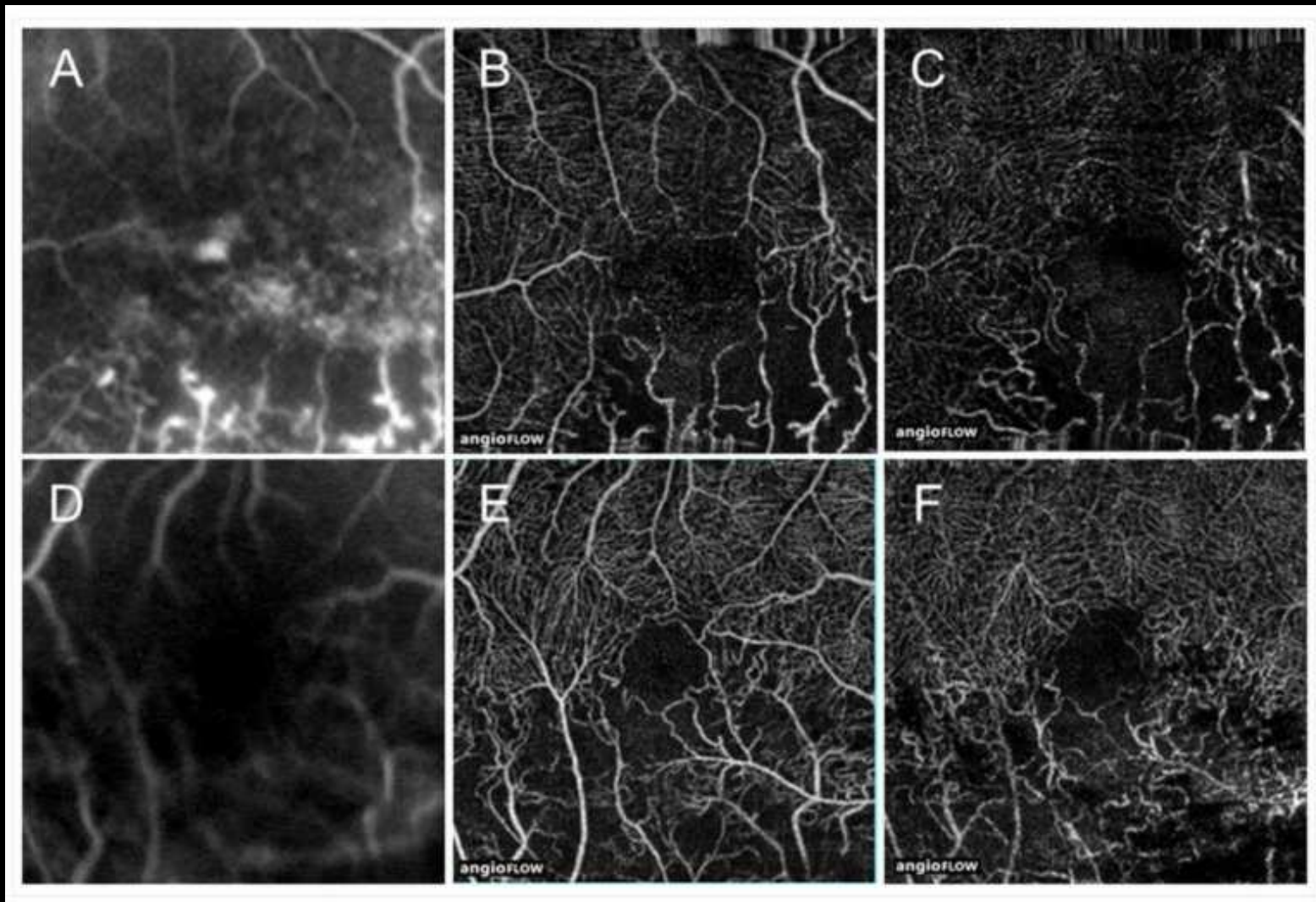
21/02/2014, OD
FA 1:02.15 30° ART(11)

HEIDELBERG
ENGINEERING

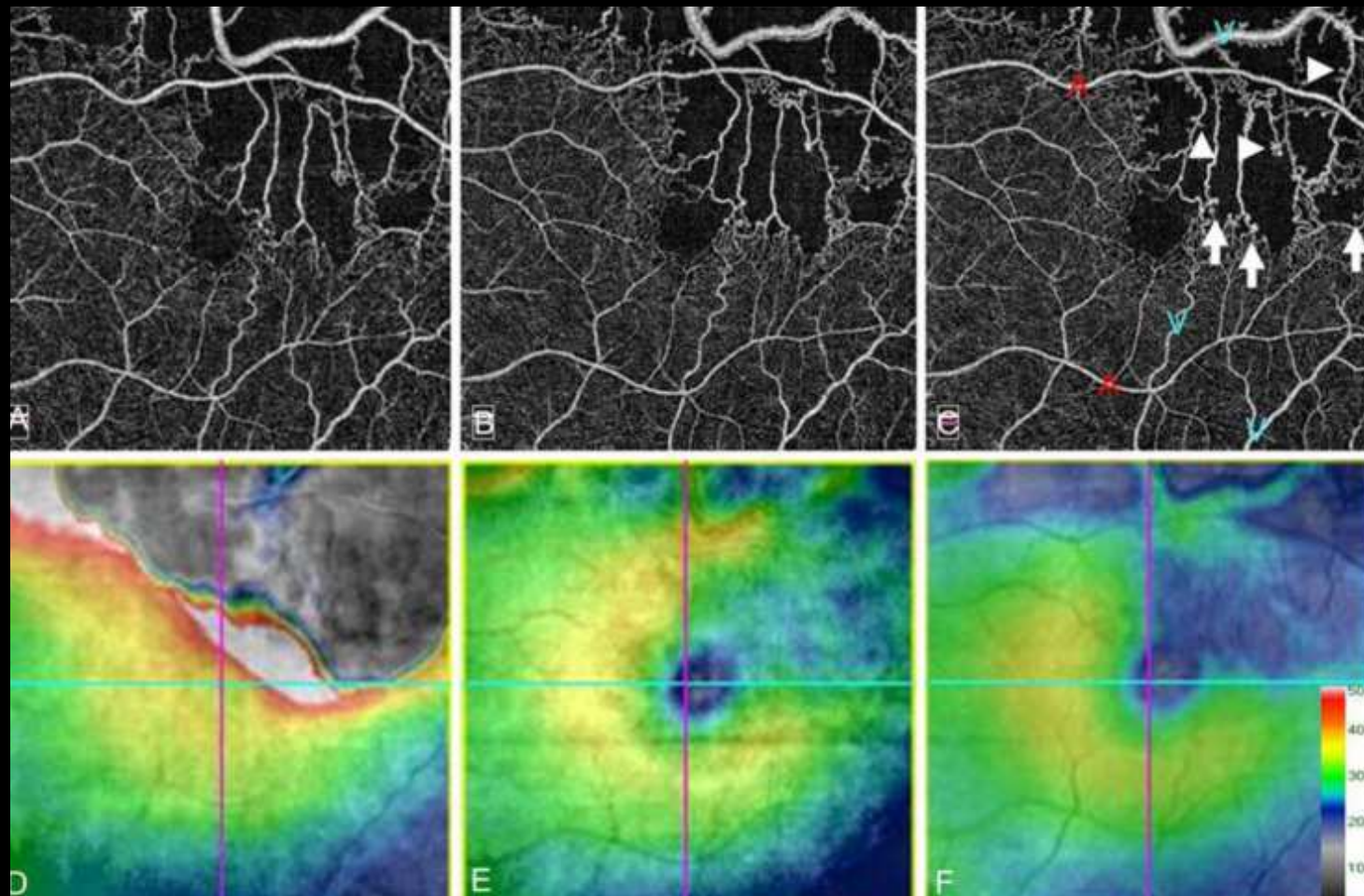


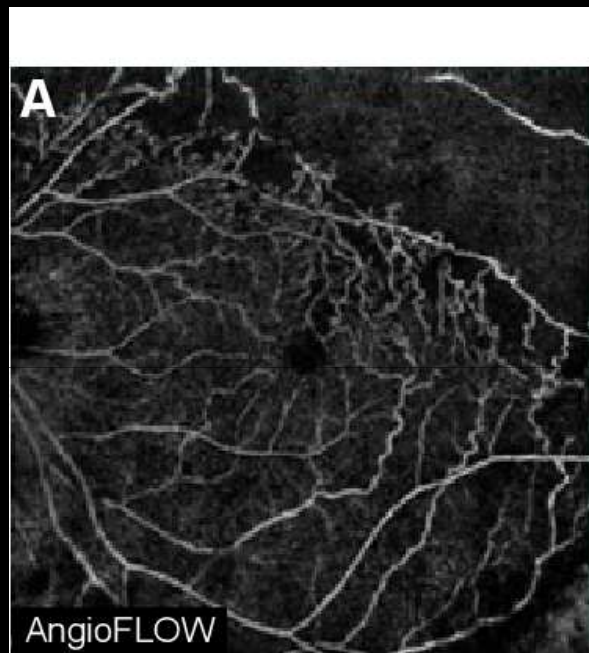
LA NONPERFUSION PRÉDOMINE DANS LES COUCHES PROFONDES



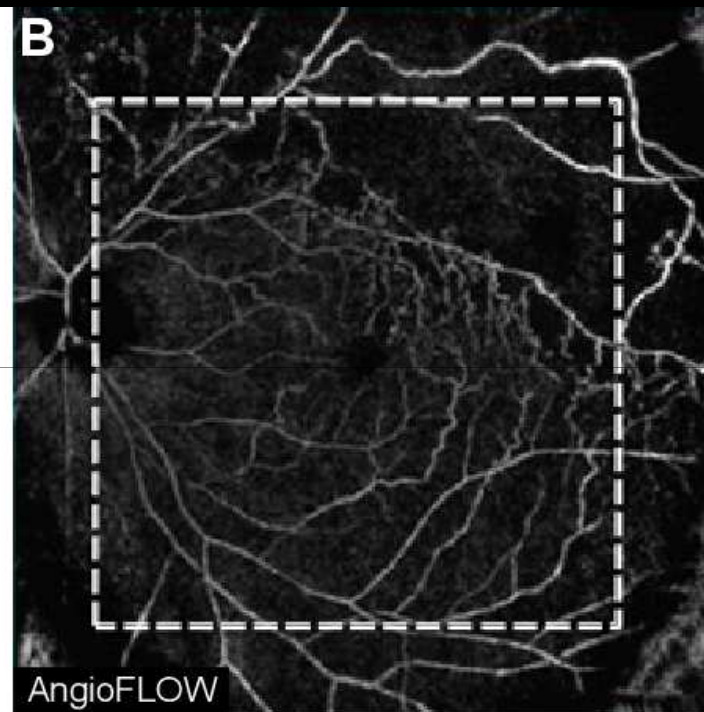


DU IMAGERIE ET PATHOLOGIE RETINIENNES

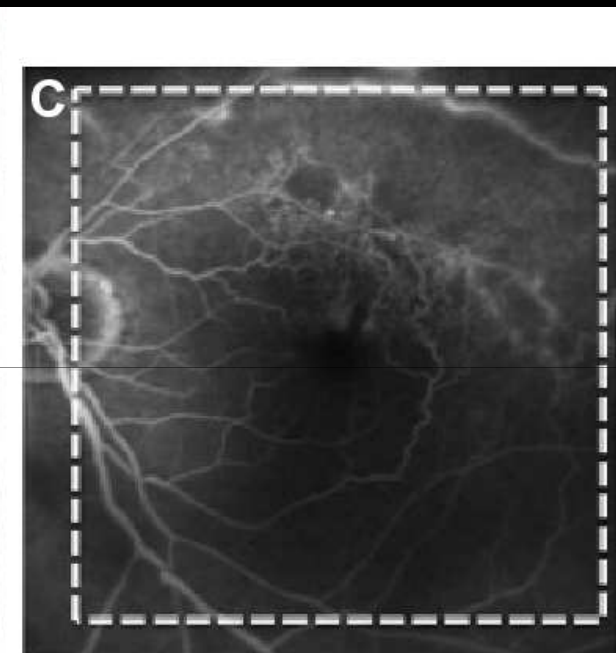




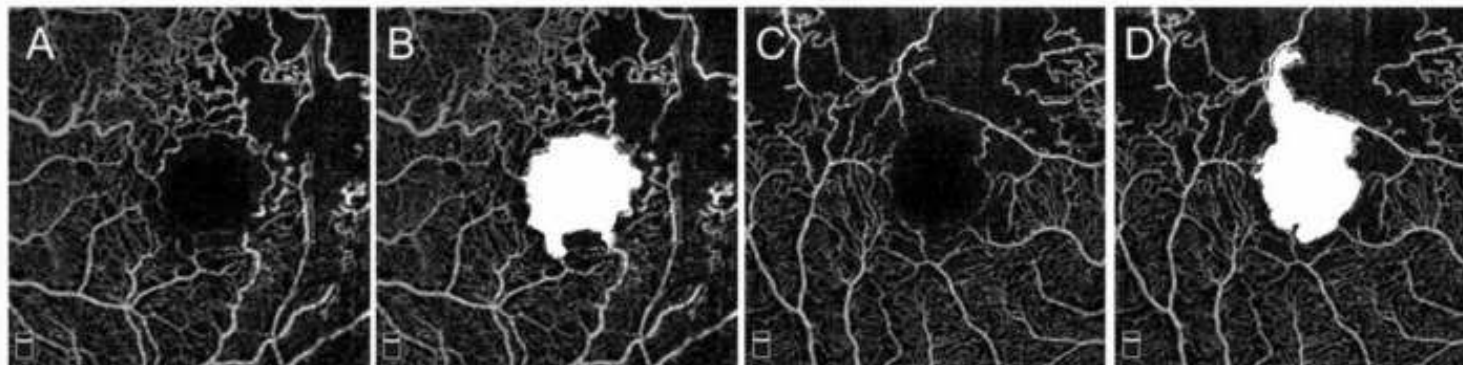
8×8 mm



8×8 mm with EFI technique

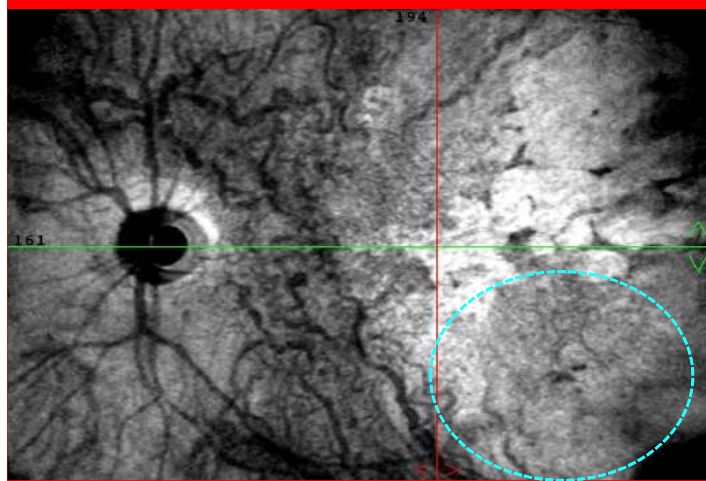


FAG (30°) using HRA2

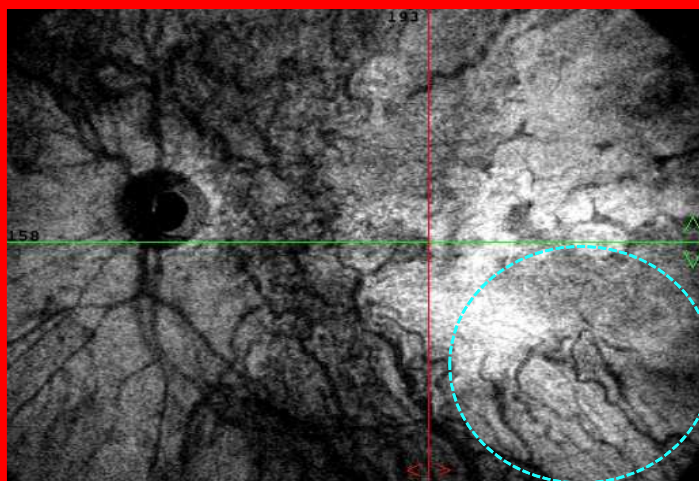


$$\text{Acircularity Index} = \frac{\text{Perimeter of the FAZ}}{\text{Perimeter of the circle with equal area}}$$

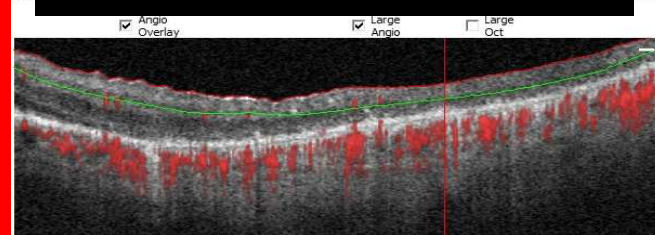
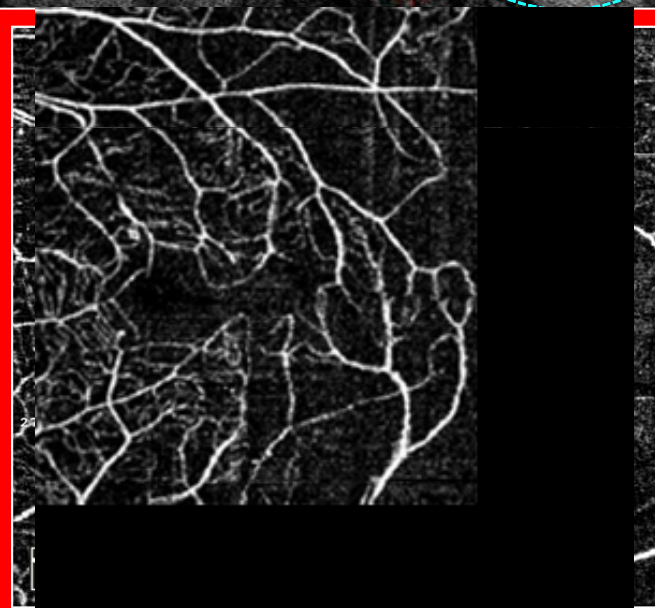
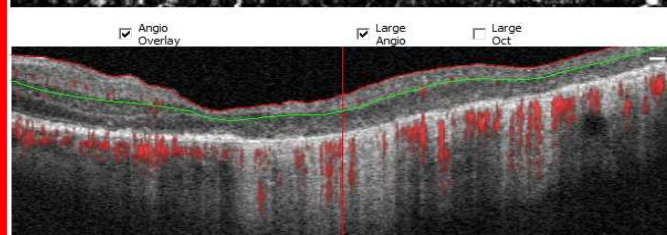
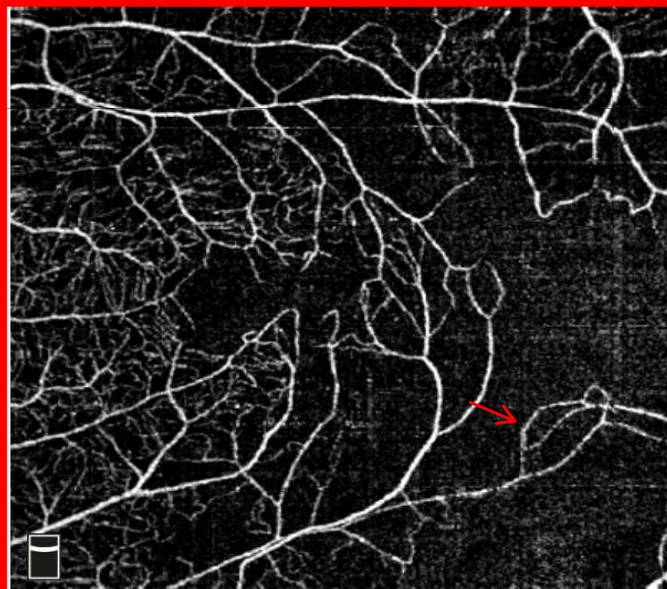
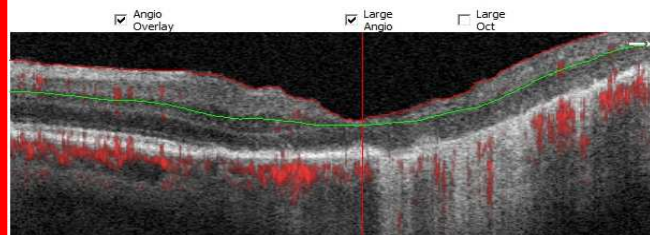
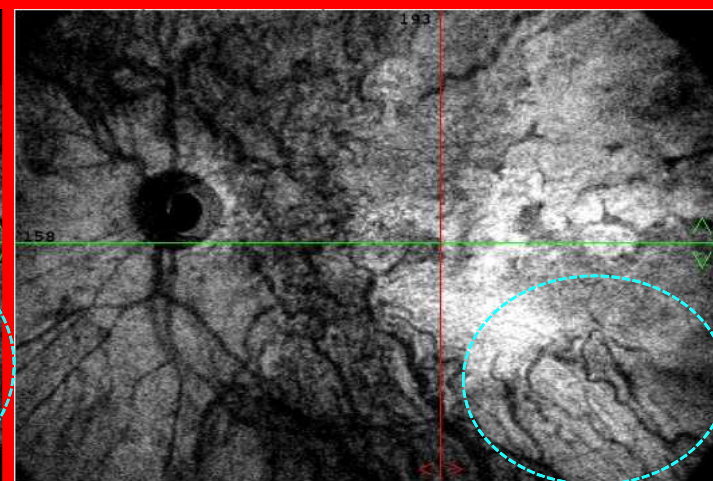
16/06/2017



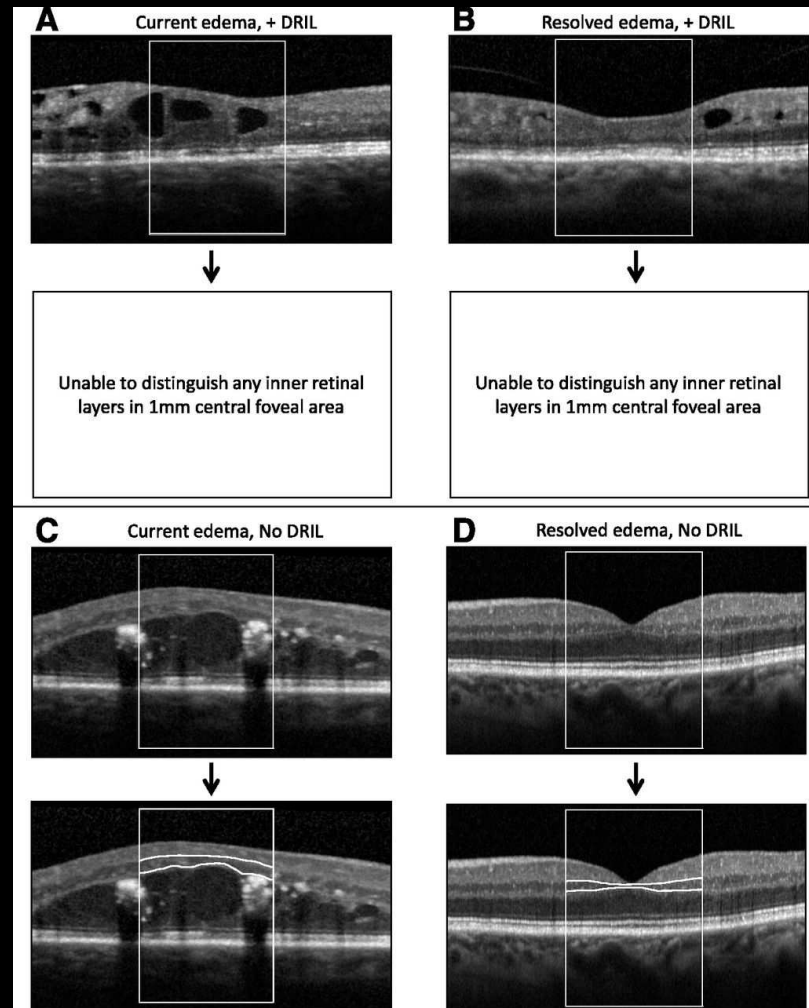
25/07/2017

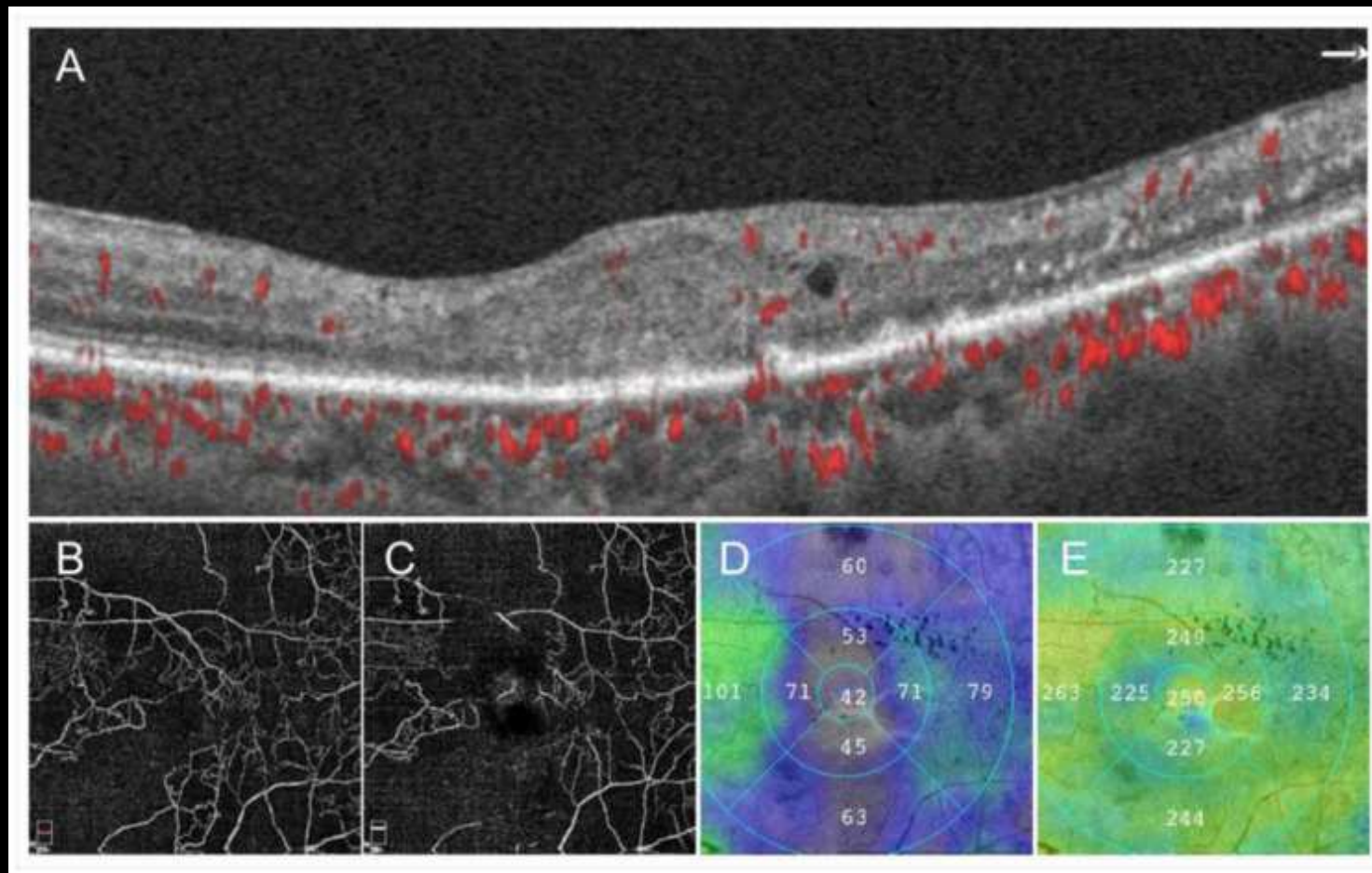


05/09/2017



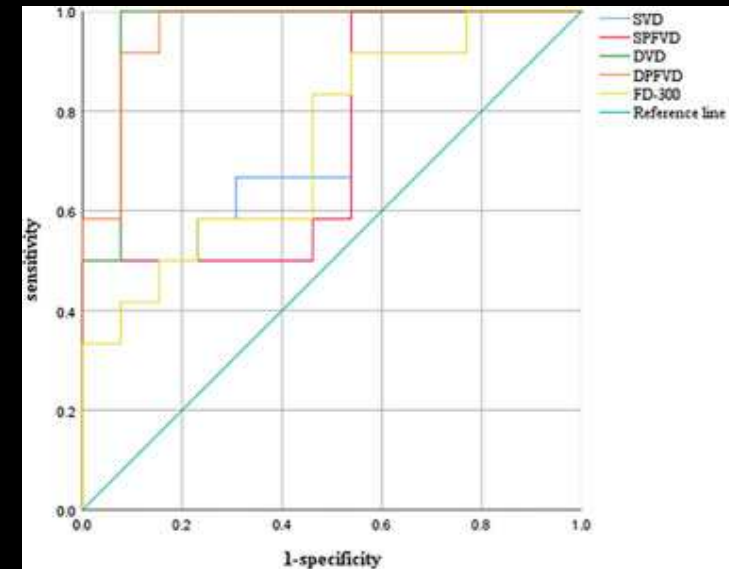
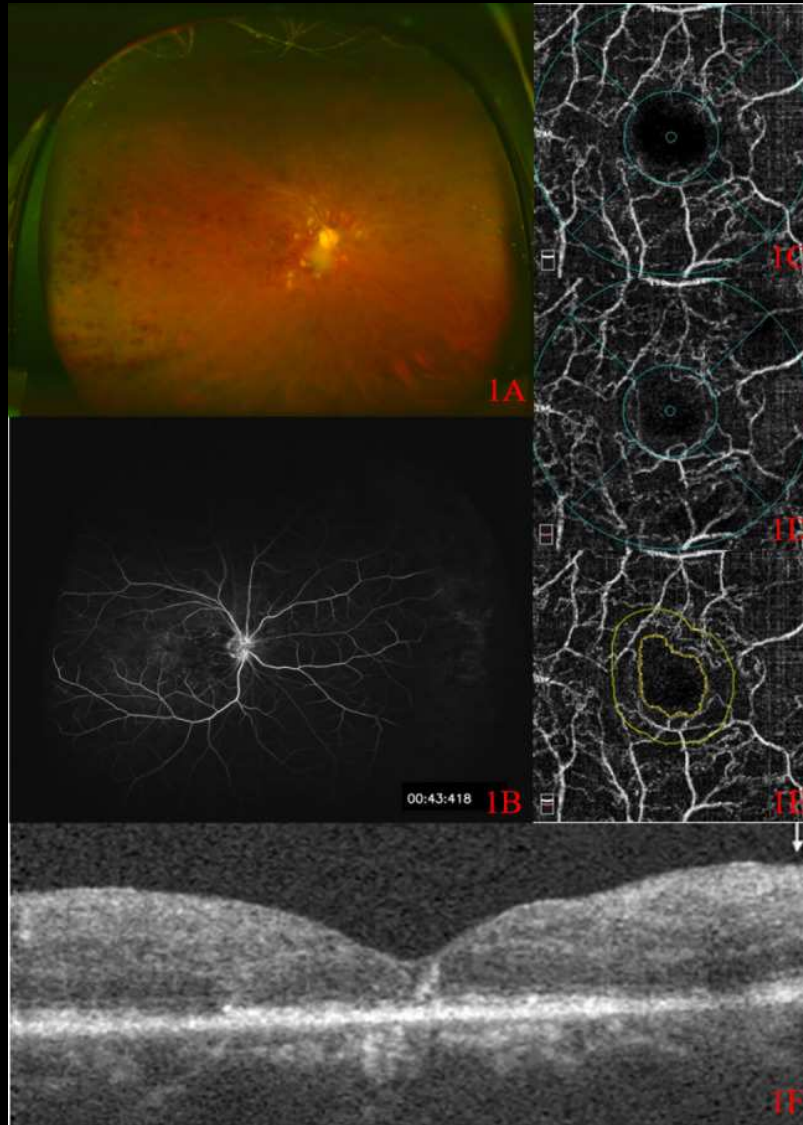
DRIL: DISORGANIZATION OF INNER RETINAL LAYERS





Hirano Y, Suzuki N, Tomiyasu T, et al. Multimodal Imaging of Microvascular Abnormalities in Retinal Vein Occlusion. J Clin Med. 2021;10(3):405. Published 2021 Jan 21. doi:10.3390/jcm10030405

AU COURS DES OVCR, LA PERFUSION MACULAIRE EST CORRÉLÉE À LA PERFUSION PÉRIPHÉRIQUE



An W, Zhao Q, Yu R, Han J. The role of optical coherence tomography angiography in distinguishing ischemic versus non-ischemic central retinal vein occlusion. *BMC Ophthalmol.* 2022 Oct 28;22(1):413. doi: 10.1186/s12886-022-02637-y. PMID: 36307778; PMCID: PMC9617433.

Cavalleri M, Sacconi R, Parravano M, Costanzo E, Pezzella M, Bandello F, Querques G. Optical Coherence Tomography Angiography in Central Retinal Vein Occlusion: Macular Changes and Their Correlation with Peripheral Nonperfusion at Ultra-Widefield Fluorescein Angiography. *Ophthalmologica.* 2022;245(3):275-284. doi: 10.1159/000522503. Epub 2022 Feb 10. PMID: 35144261.

EVOLUTION DE L'OCCLUSION CAPILLAIRE

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

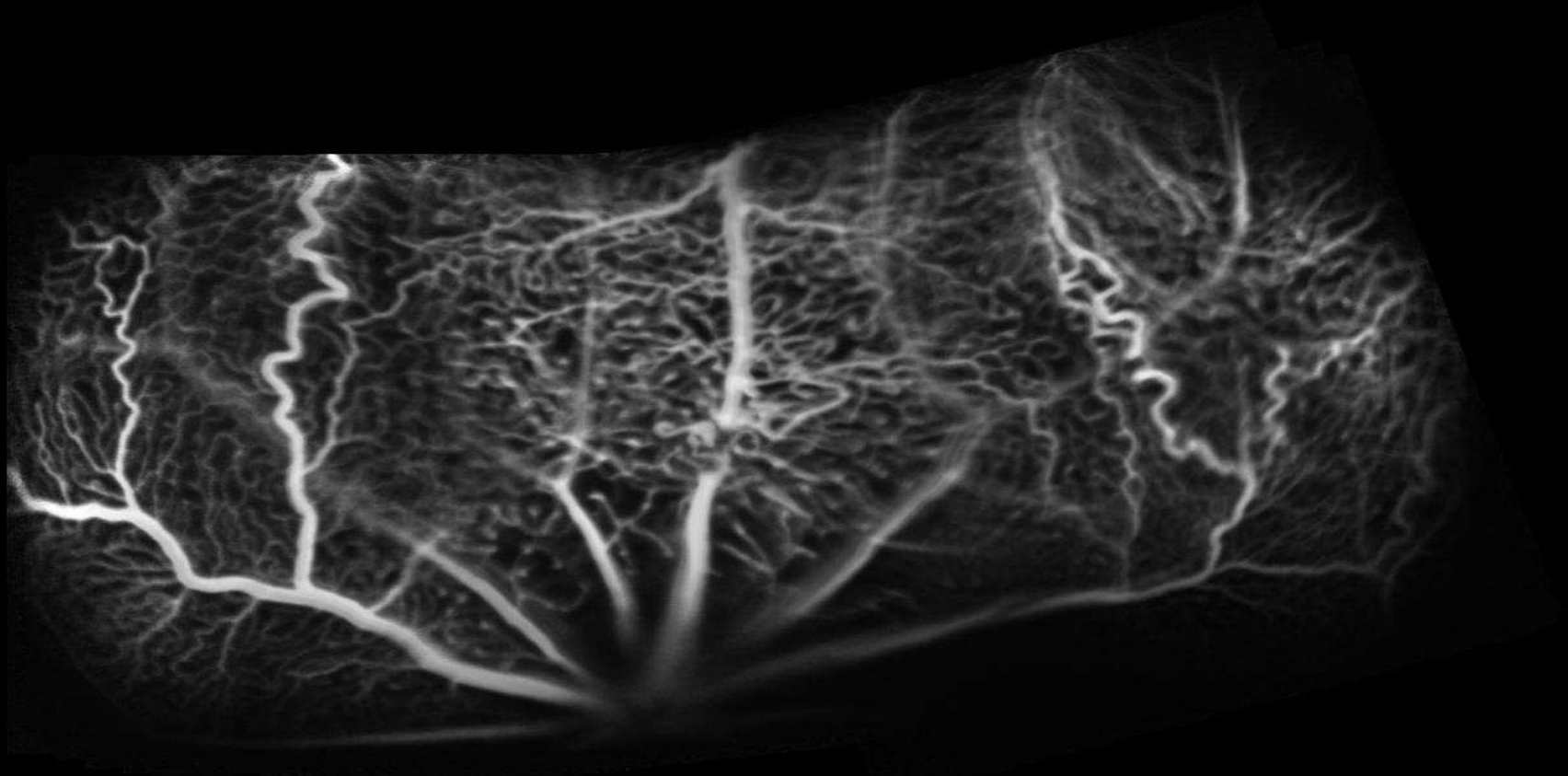
➔ le risque de rubéose est maximal au début de l'évolution puis décroît sans s'annuler

COMMENT DÉPISTER LA NON PERFUSION? ET PLUS PRÉCISÉMENT LE RISQUE DE GLAUCOME NÉOVASCULAIRE AU COURS D'UNE OVCR?

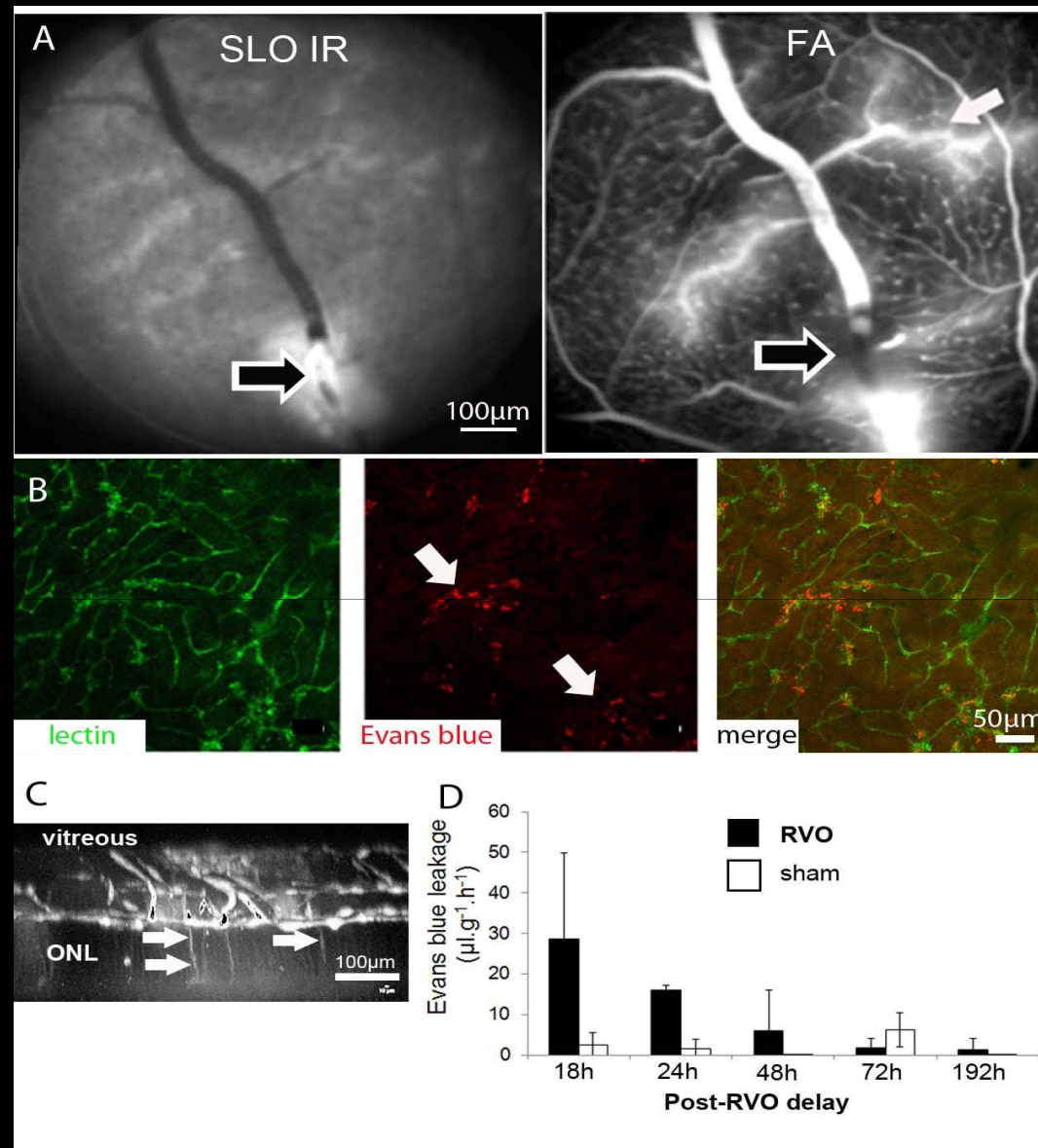
L'acuité visuelle et l'OCT sont des excellents indicateurs de la présence d'une non perfusion étendue

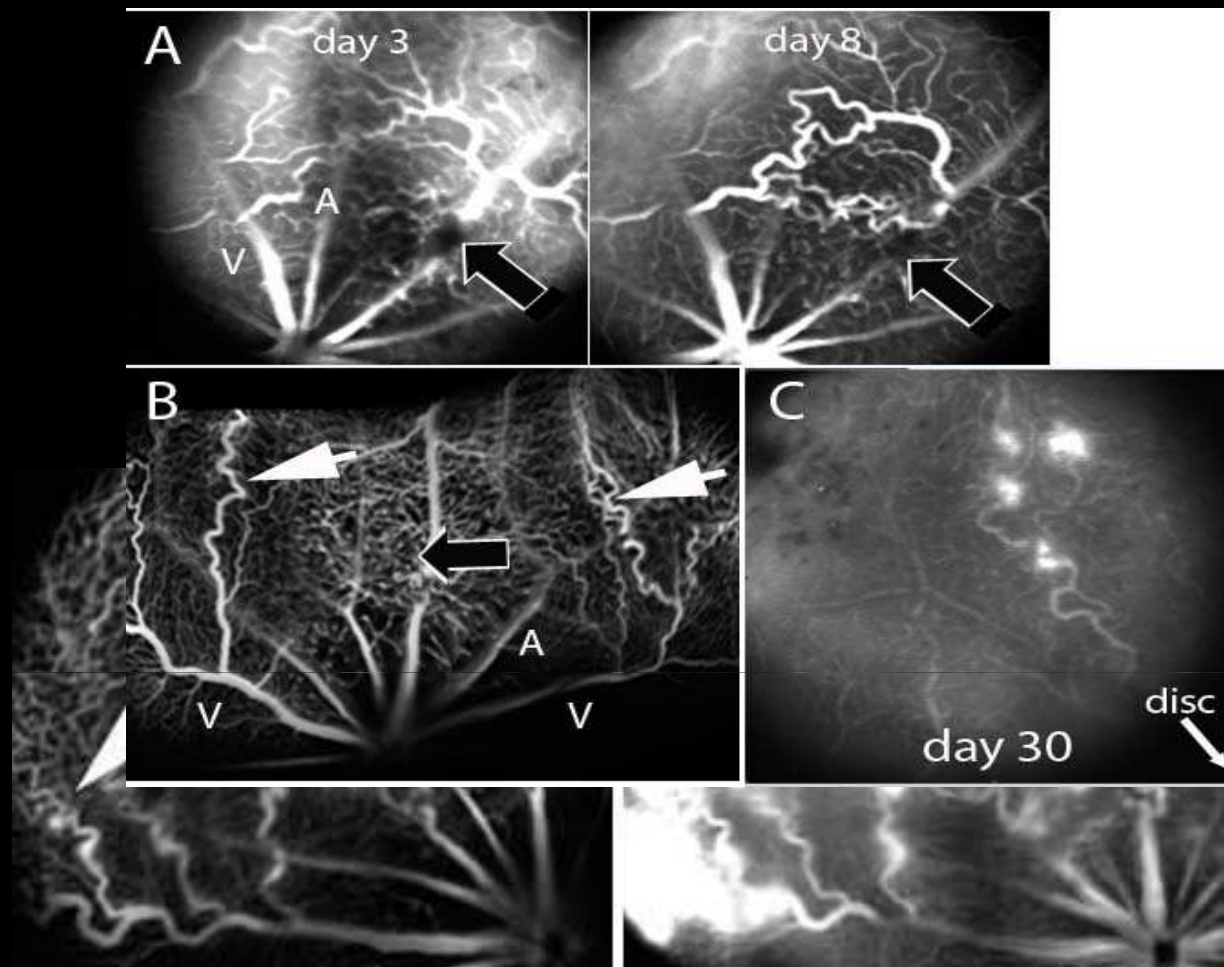
- Sans doute parce qu'une non perfusion étendue affecte la macula au moins à un certain degré
- Une acuité visuelle $> 2/10$ est peu souvent compliquée d'une rubéose
- Au contraire, une acuité à « voit bouger la main » est beaucoup plus à risque de développer une rubéose
- Et une non perfusion s'accompagne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

ETUDES EXPÉRIMENTALES



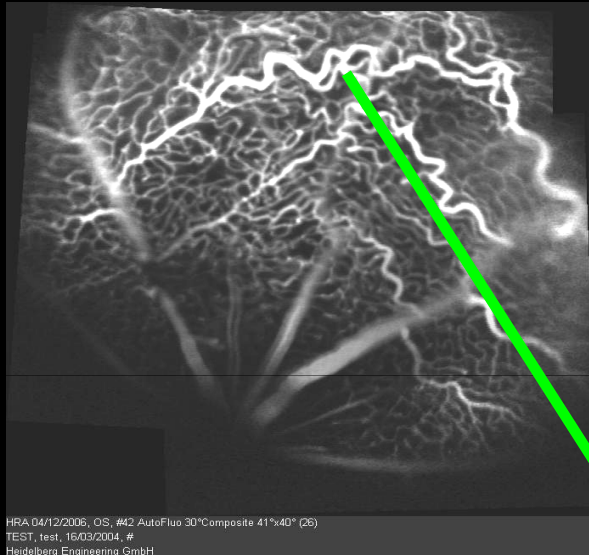
HRA 17/07/2014 , OS, AutoFluo 30°Composite 71°x41° (31)
Occlusions, Veineuse, 12/05/2008, #
Heidelberg Engineering GmbH



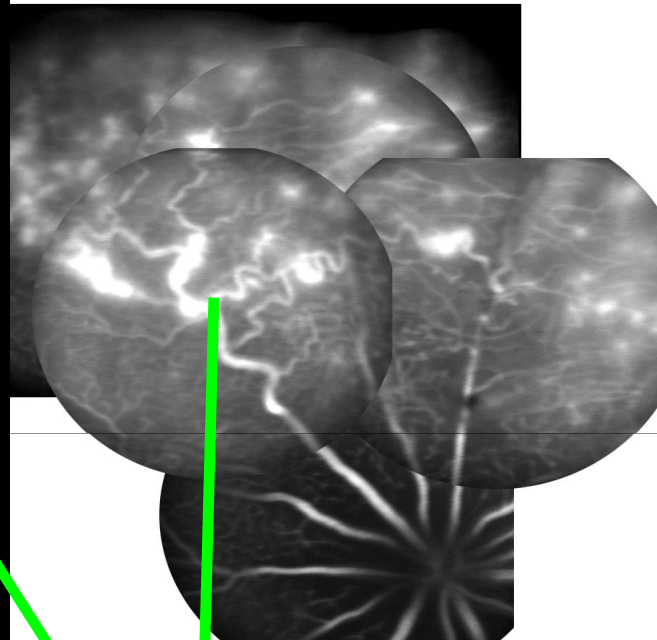


L'OCCLUSION VEINEUSE CHEZ LA SOURIS PEUT REPRODUIRE CERTAINS ASPECTS CLINIQUES DU RMV

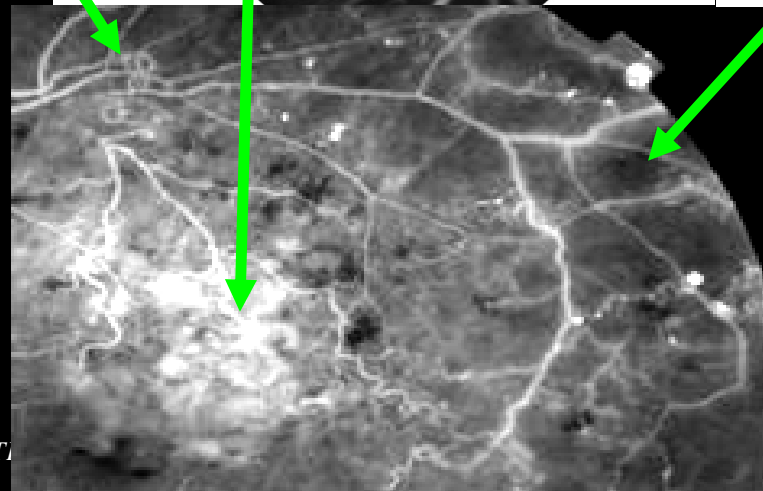
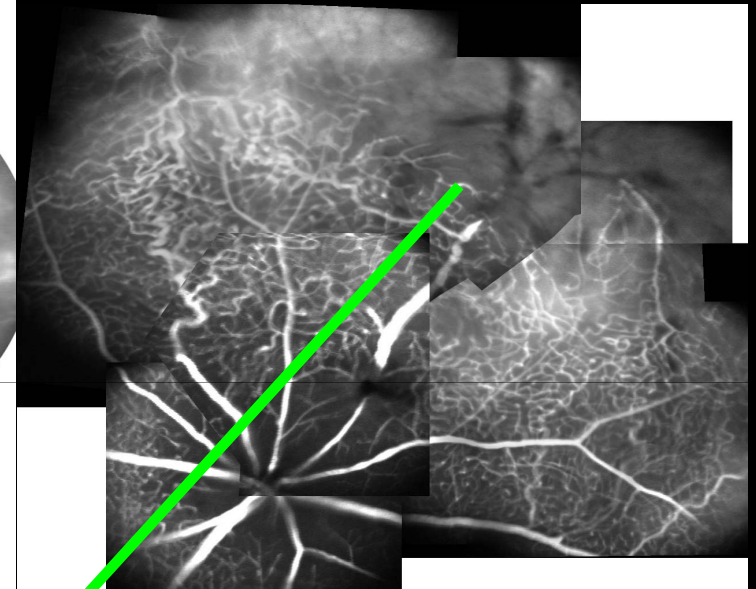
Circulation collatérale



Rupture de la BHR



Occlusion capillaire



NÉOVASCULARISATION DES OVR

Néovaisseaux:

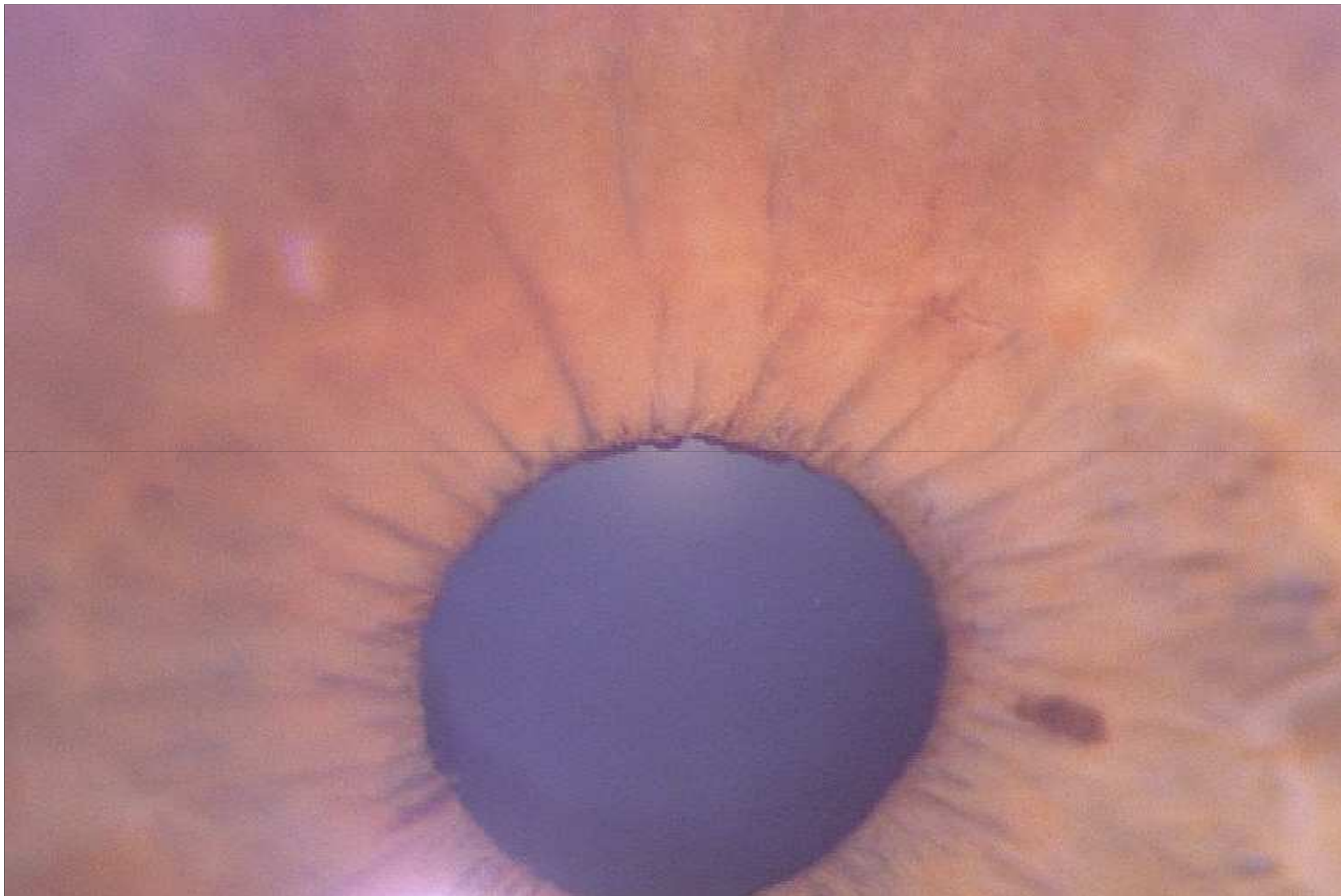
Prépapillaires

Prérétiniens

Iriens : risque de glaucome néovasculaire

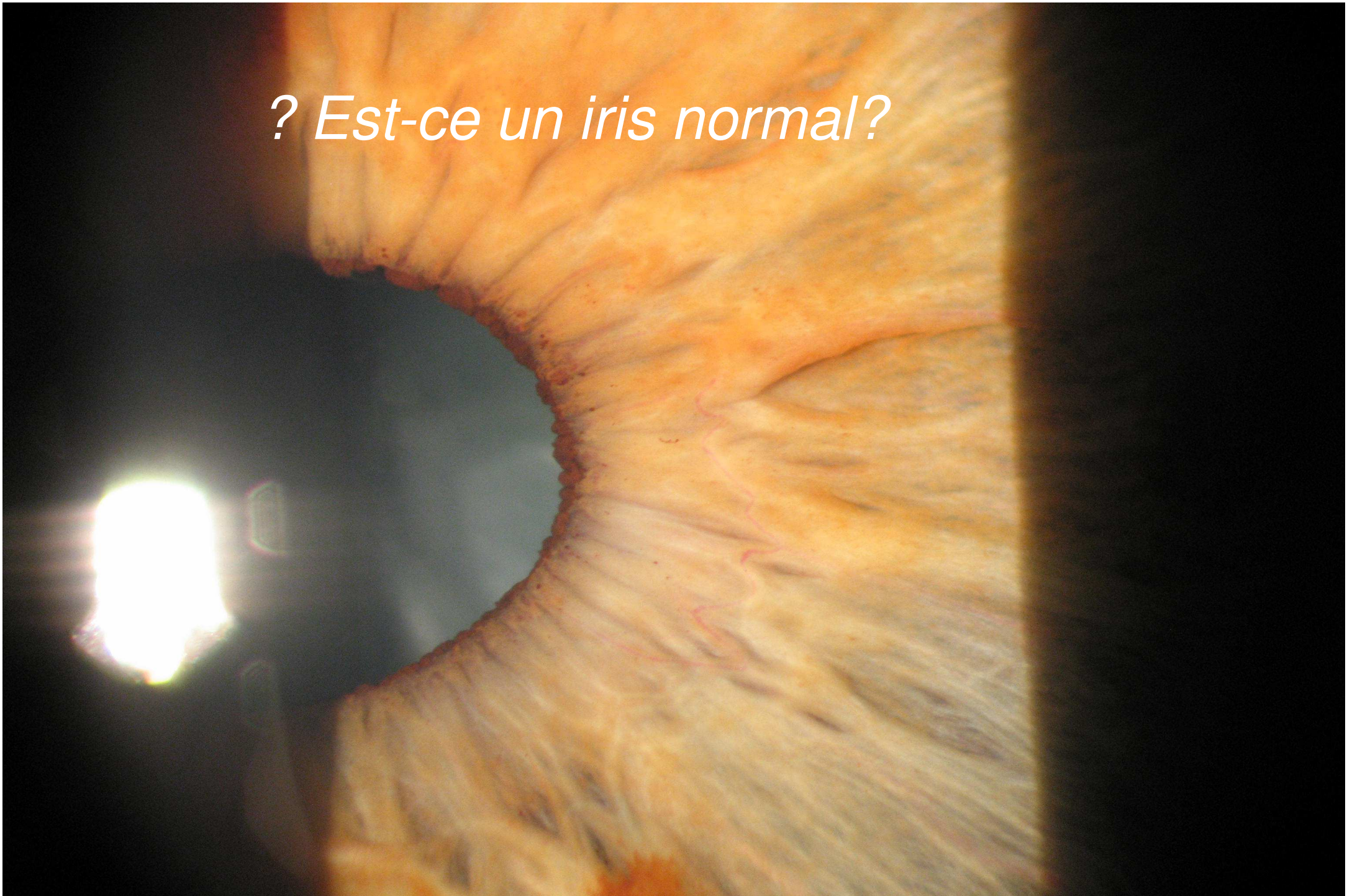
Seulement si OVCR

Importance de l'examen de l'iris

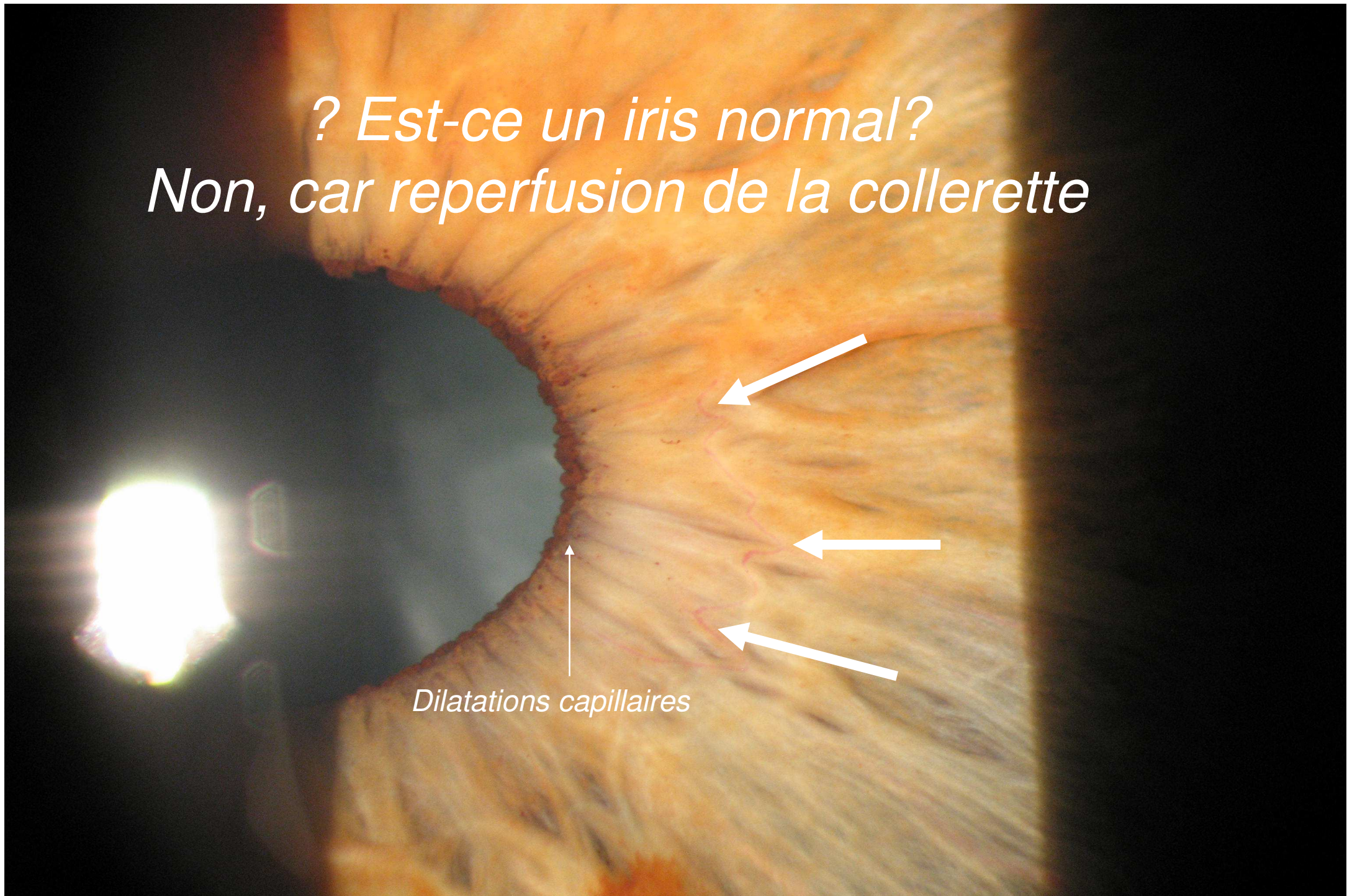




? *Est-ce un iris normal?*



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



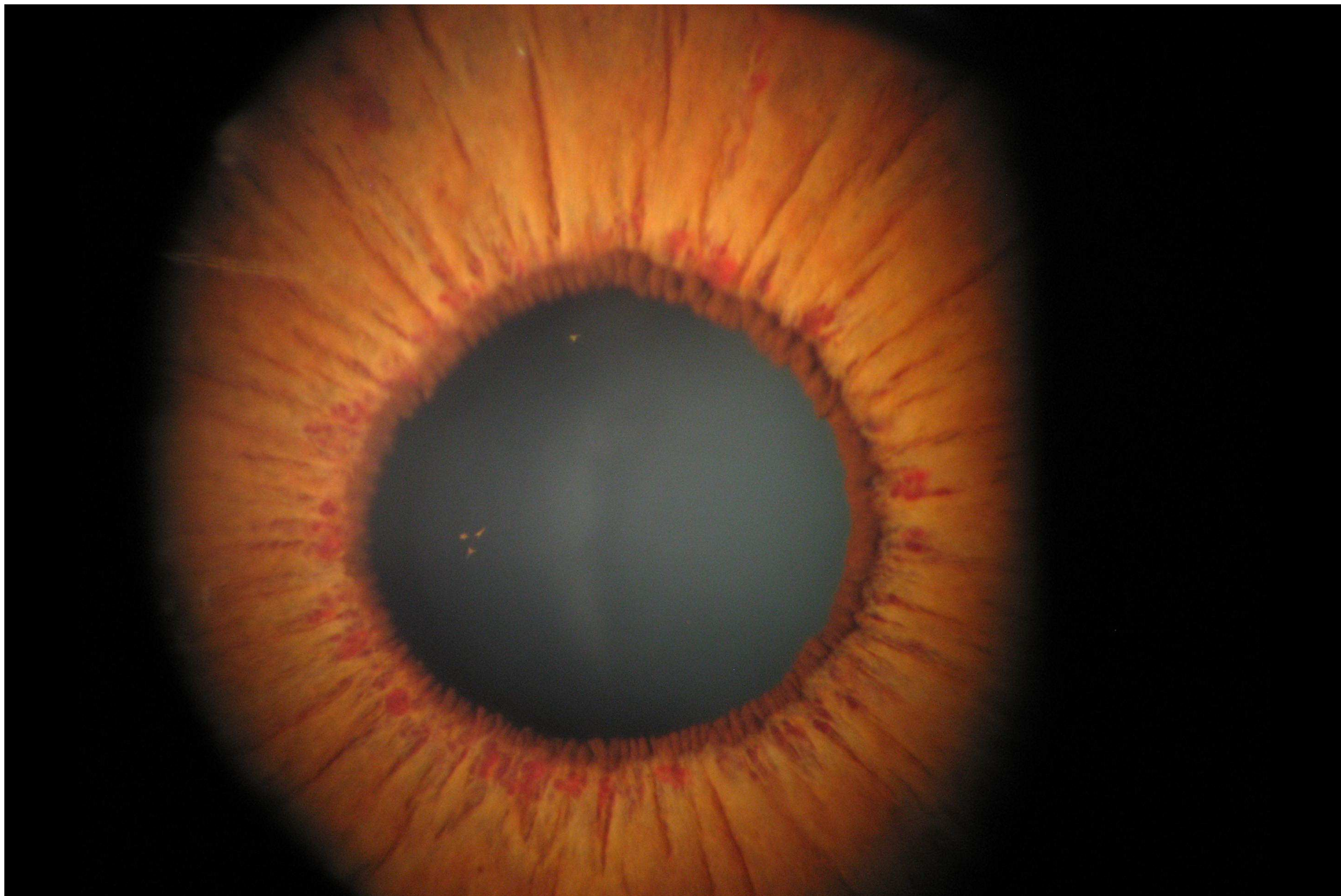


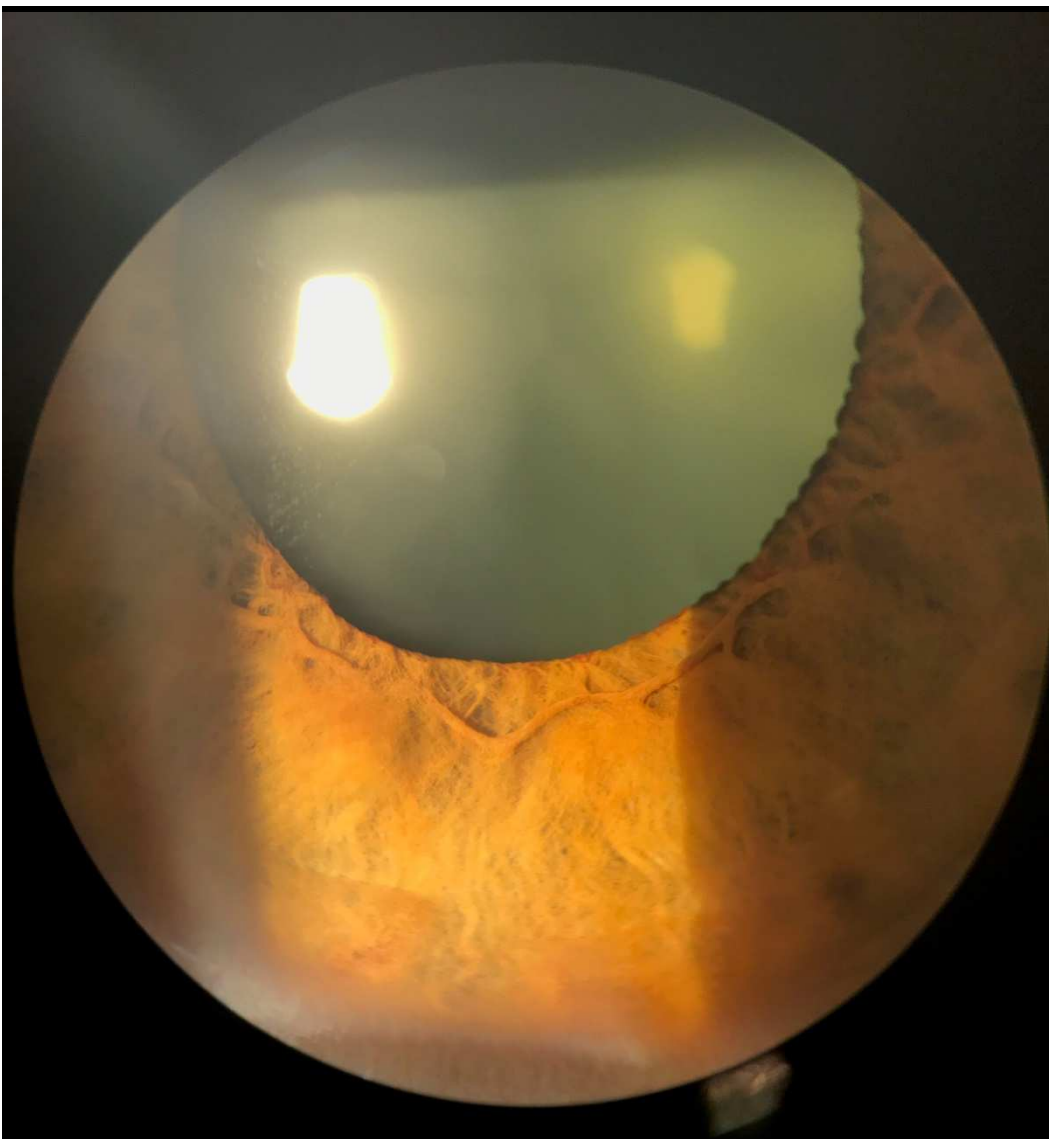
Dilatation vasculaire irienne



POUR RECHERCHER UNE RUBÉOSE IRIENNE,
IL FAUT UN IRIS NON DILATÉ
ET UN FORT GRANDISSEMENT

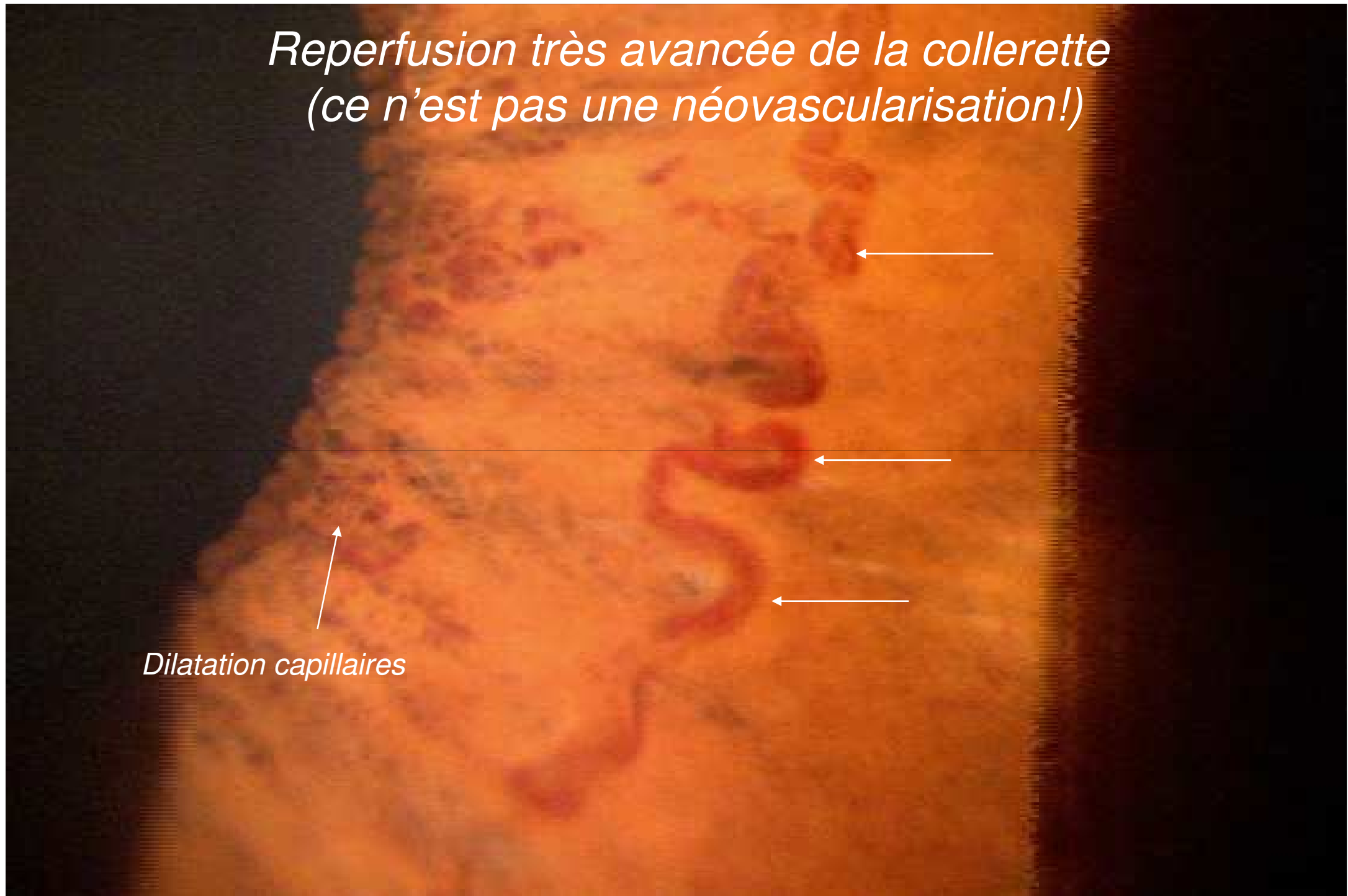






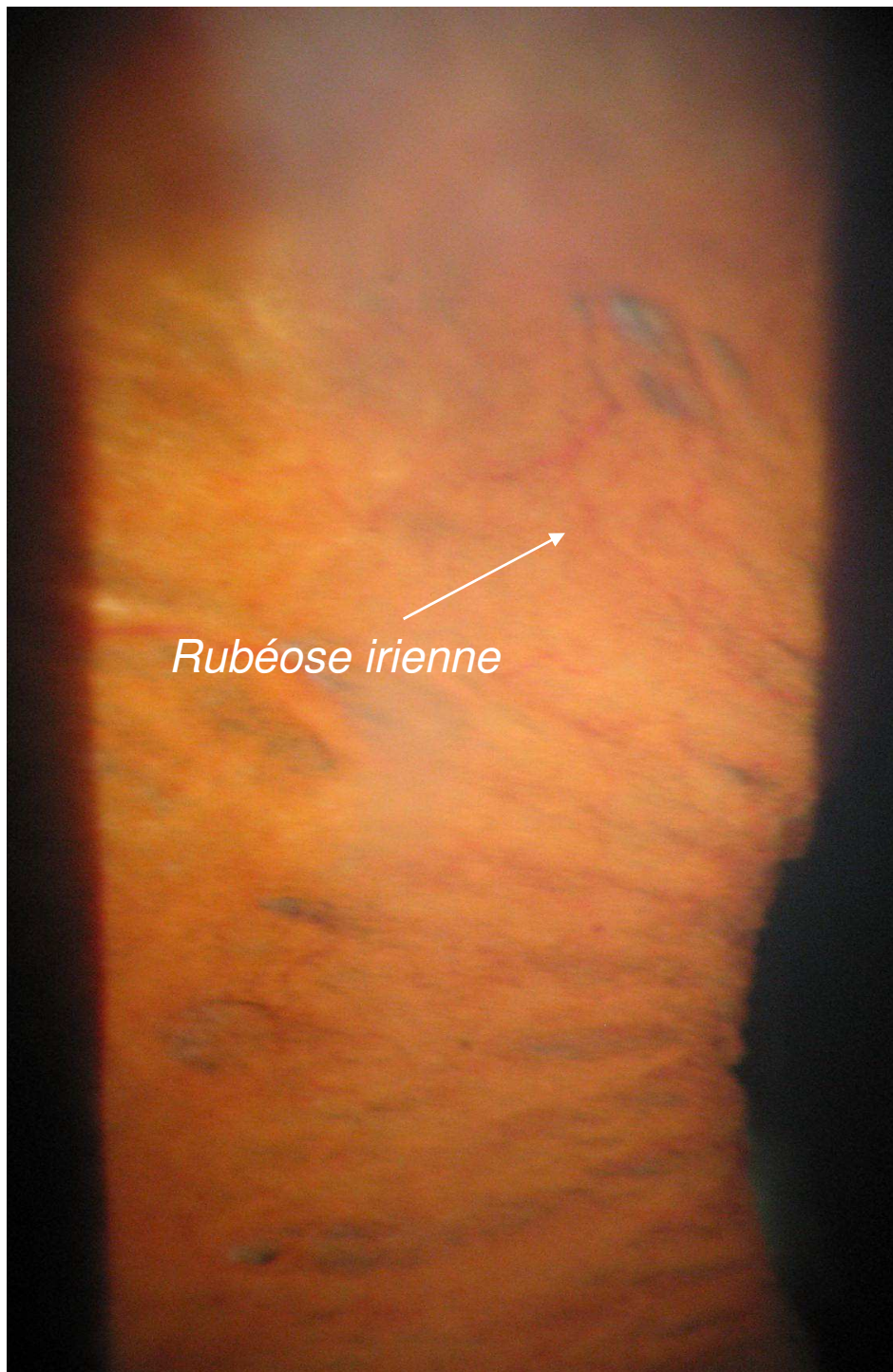
*Reperfusion très avancée de la collerette
(ce n'est pas une néovascularisation!)*

Dilatation capillaires



Vasodilatation irienne sur iris pigmenté





Rubéose irienne

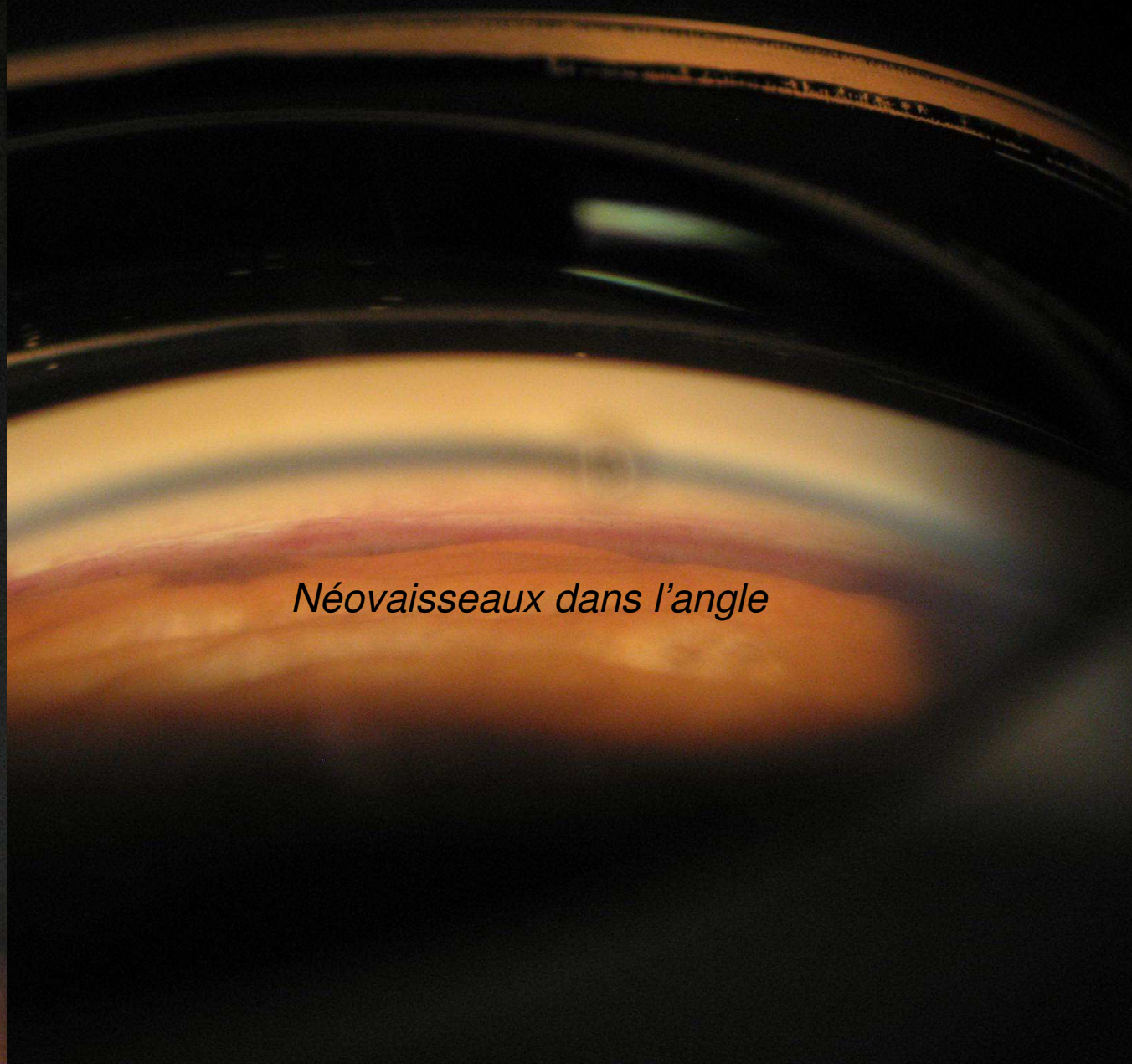


Néovaisseaux dans l'angle

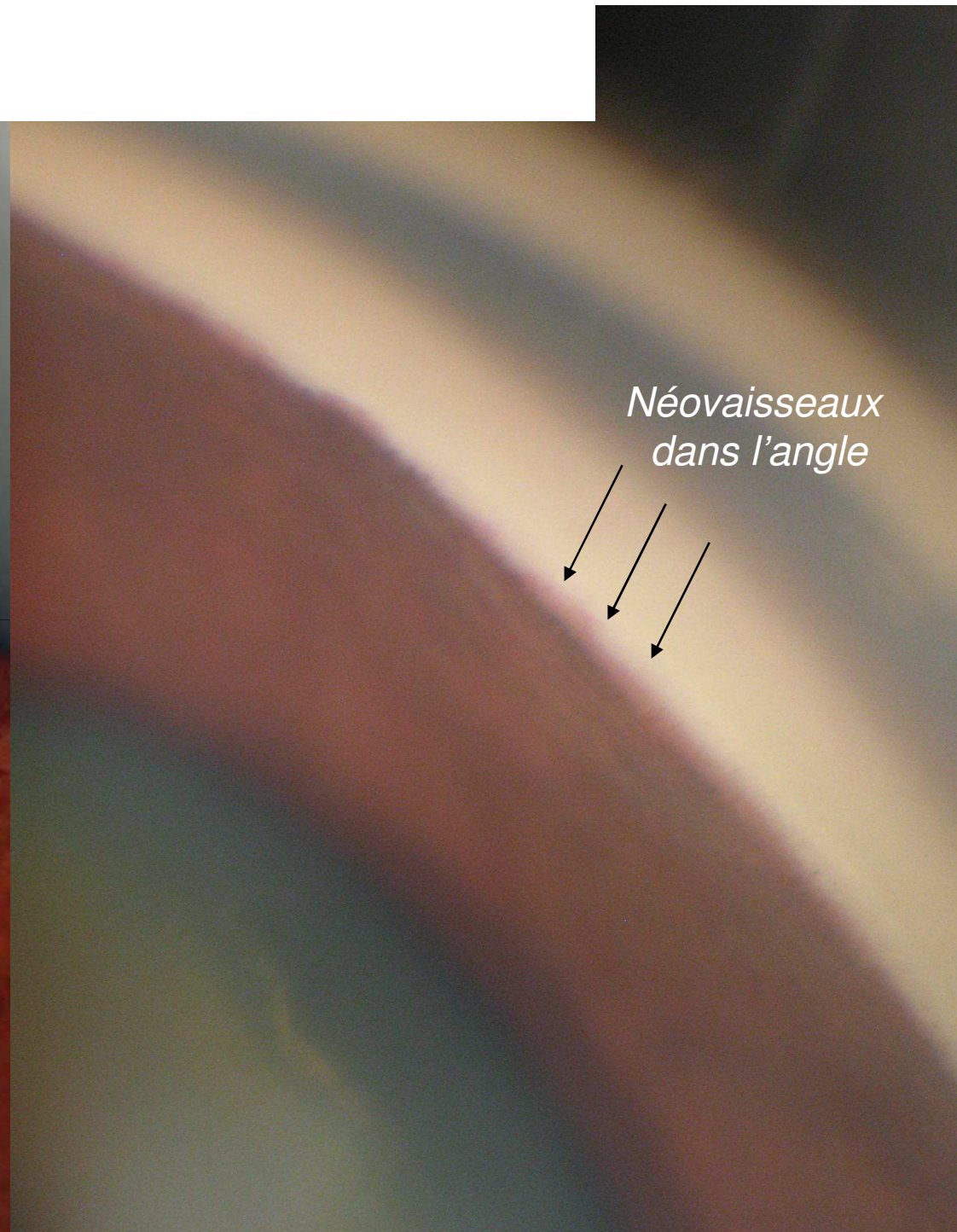
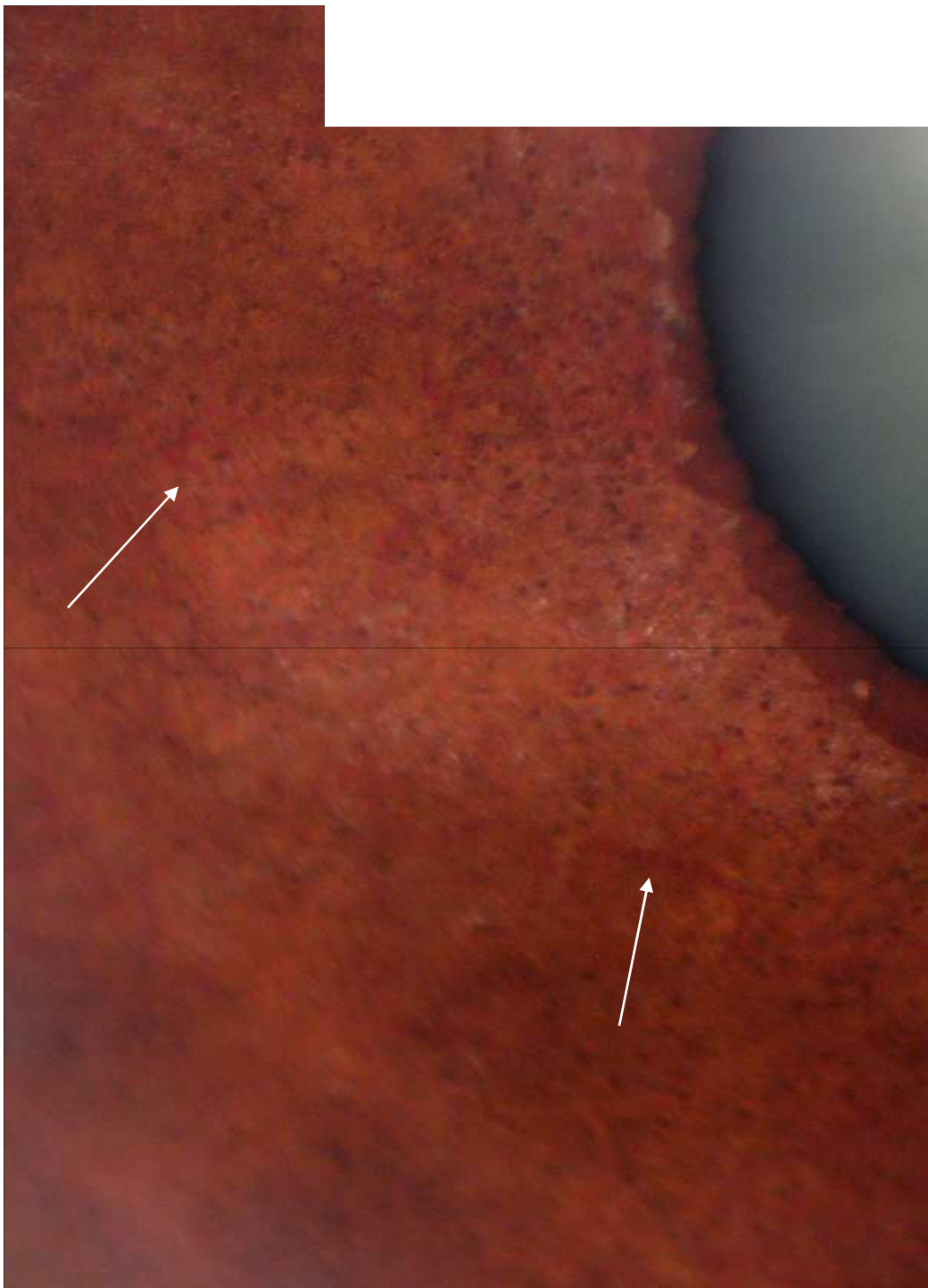
Attention aux pièges
la rubéose peut commencer dans l'angle



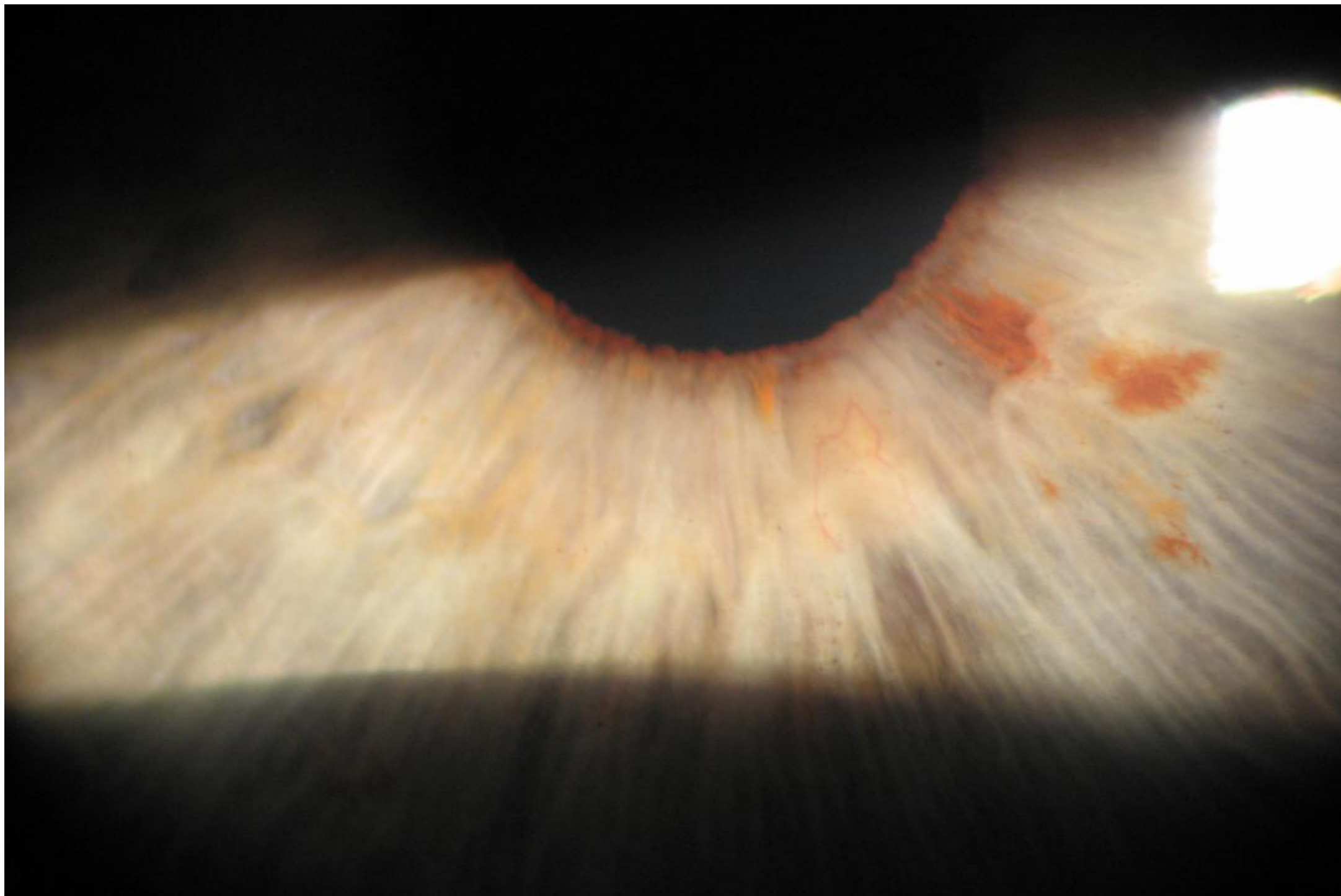
Vasodilatation irienne

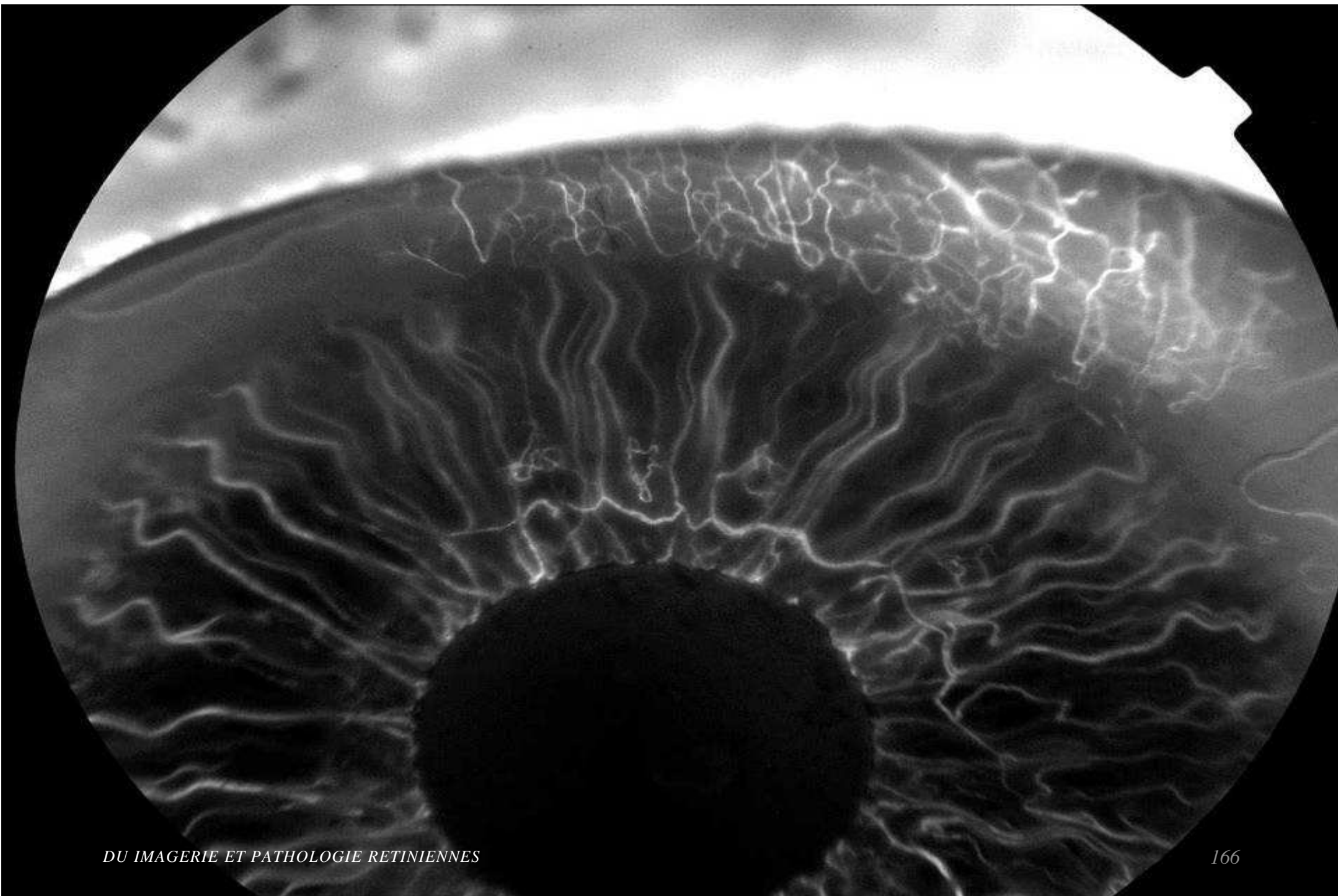


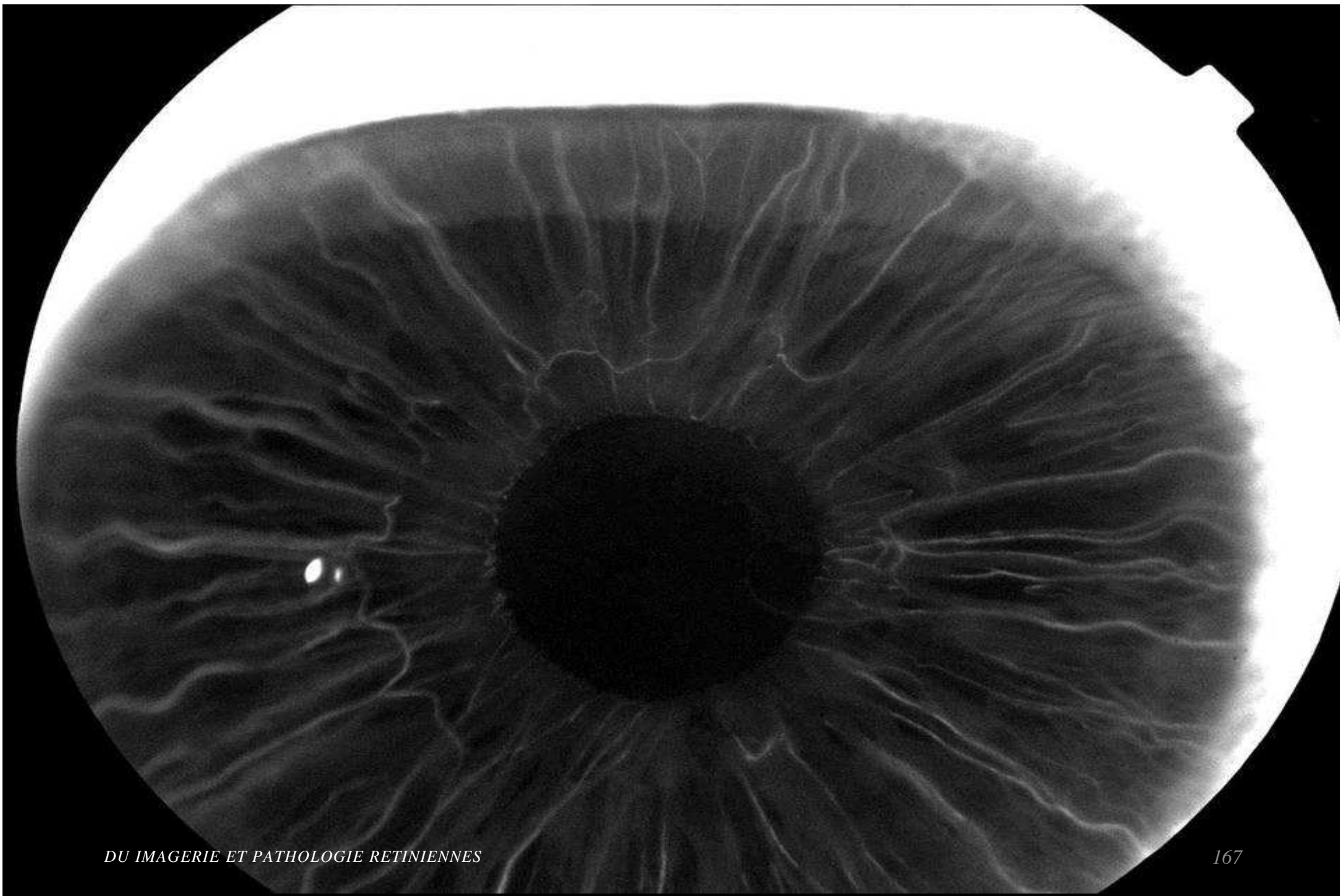
Néovaisseaux dans l'angle











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

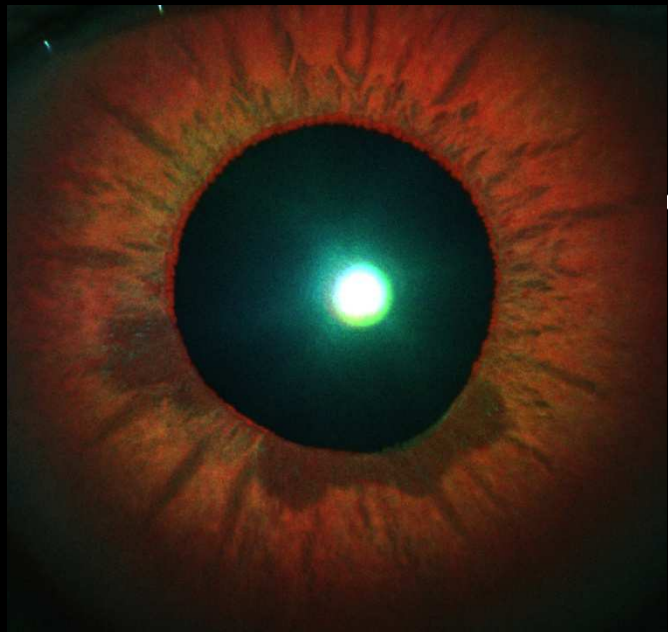
- Perfusion par plasma seul

Hayreh 1978

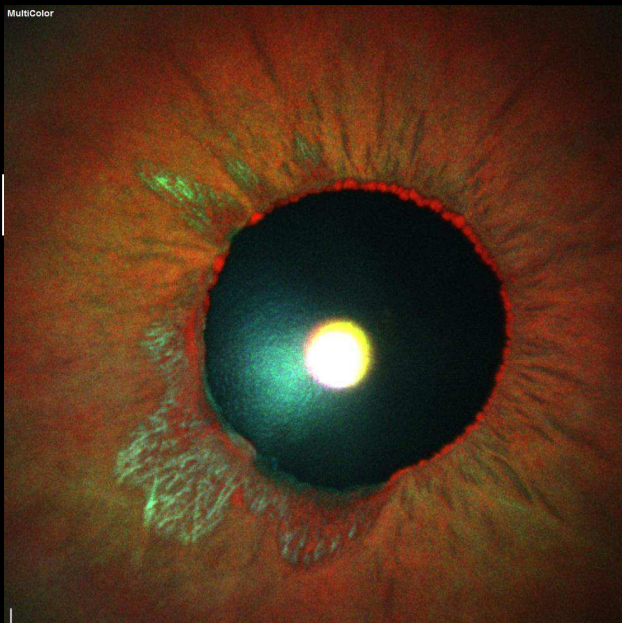
L'IRIS EST UN BIOSENSEUR DE LA PRÉSENCE DE FACTEURS ANGIOGÉNIQUES

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

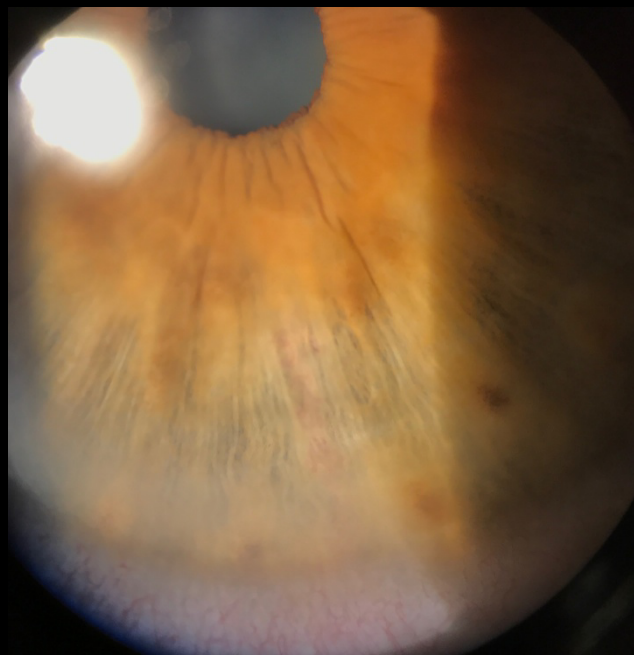
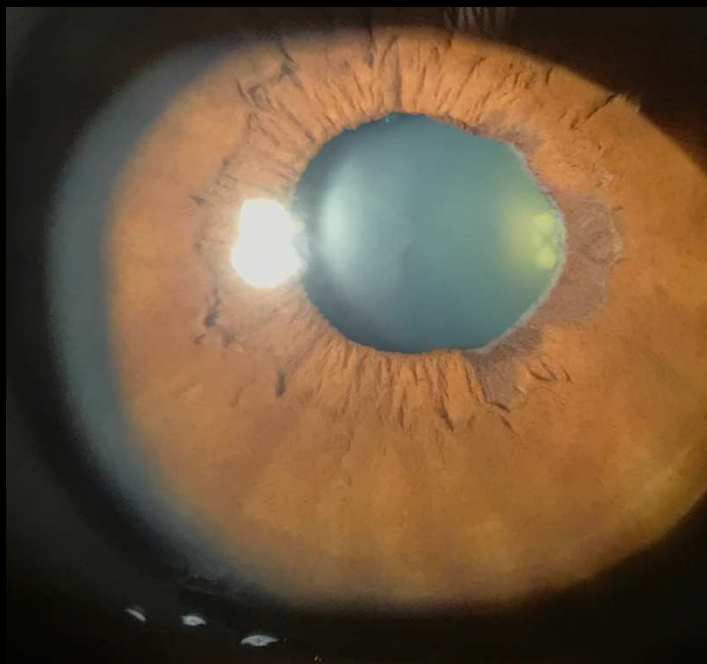
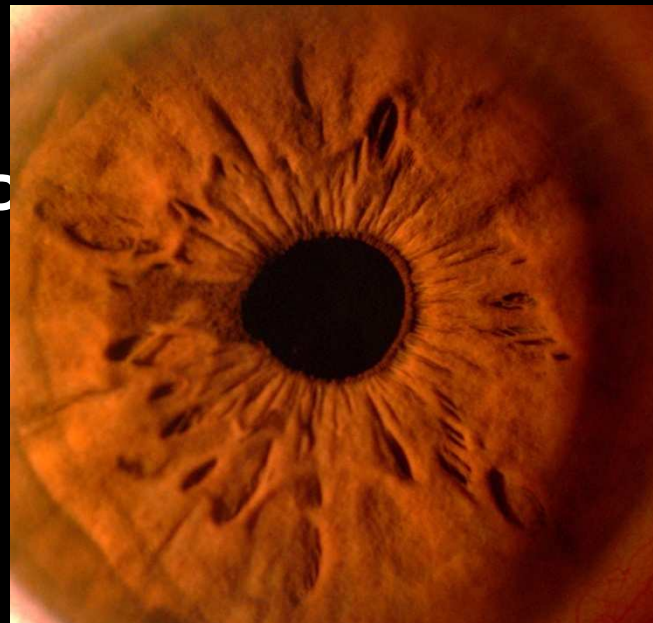
- Diffusion antérieure de facteurs vasodilatateurs et angiogéniques
 - VEGF
 - Boyd 2002
- Dilatation des vaisseaux stromaux
- Augmentation de la pression de perfusion de segments de la collerette déjà perfusés
- Signe précoce de vasodilatation des vaisseaux iriens, pouvant précéder la rubéose
- (≠ néovaisseaux)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



N



P

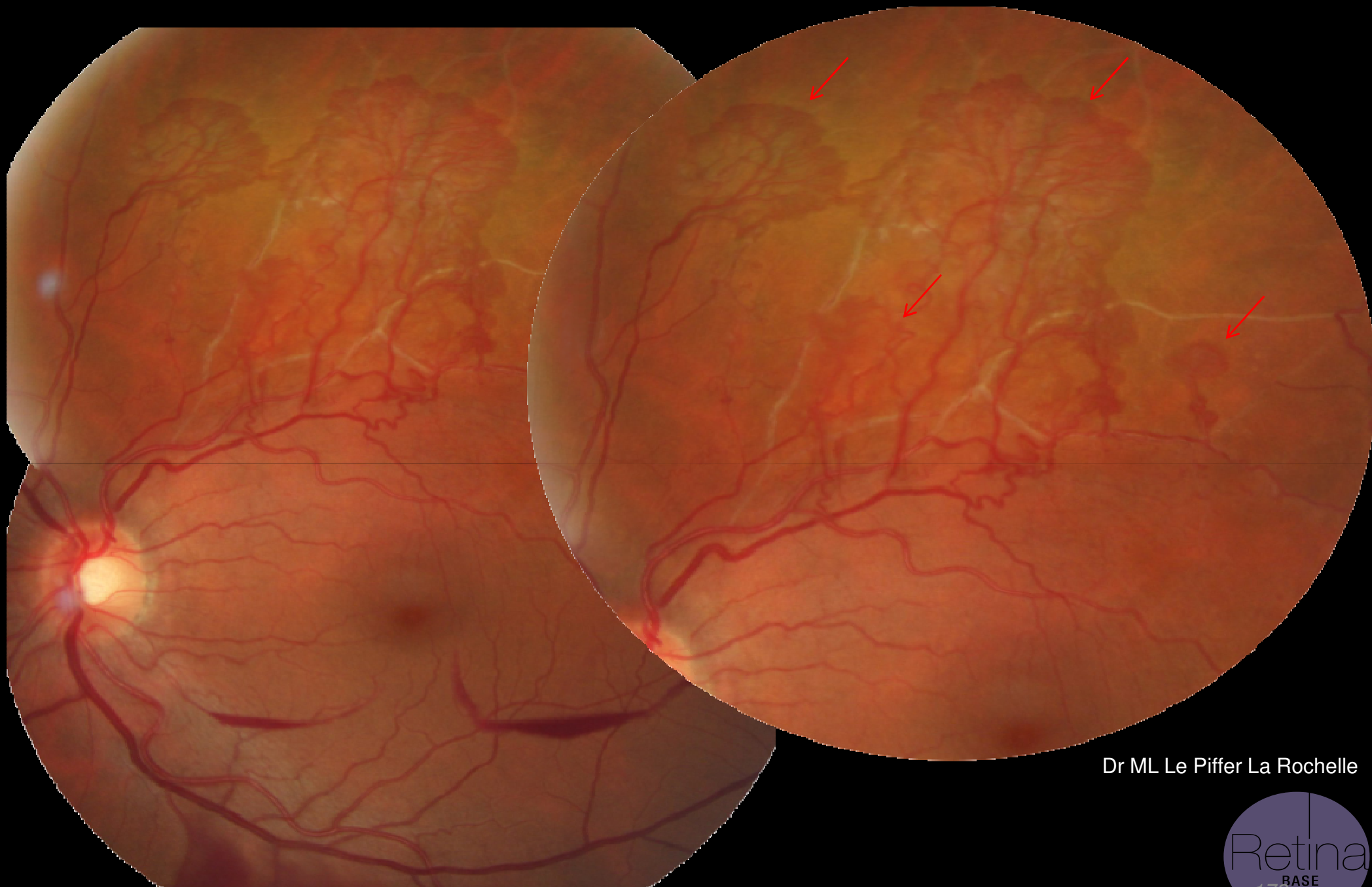


LA RUBÉOSE IRIENNE EXISTE-T-ELLE AU COURS DES OBVR?

- Non
- (Mais des néovaisseaux du segment postérieur sont possibles)

NÉO-VASCULARISATION

- Survient en cas de non perfusion (40 à 50% des OBVR)
 - Sévérité proportionnelle à la surface non perfusée
 - Néo-vascularisation sur la papille si non perfusion approche 1/4 de la surface rétinienne
 - Apparaît rarement avant 4 mois
- 20 à 30% des OBVR auraient des NVx avec hémorragie intravitréenne, sans laser.
- La néovascularisation est néanmoins inconstante
 - Rôle possible du DPV



Dr ML Le Piffer La Rochelle





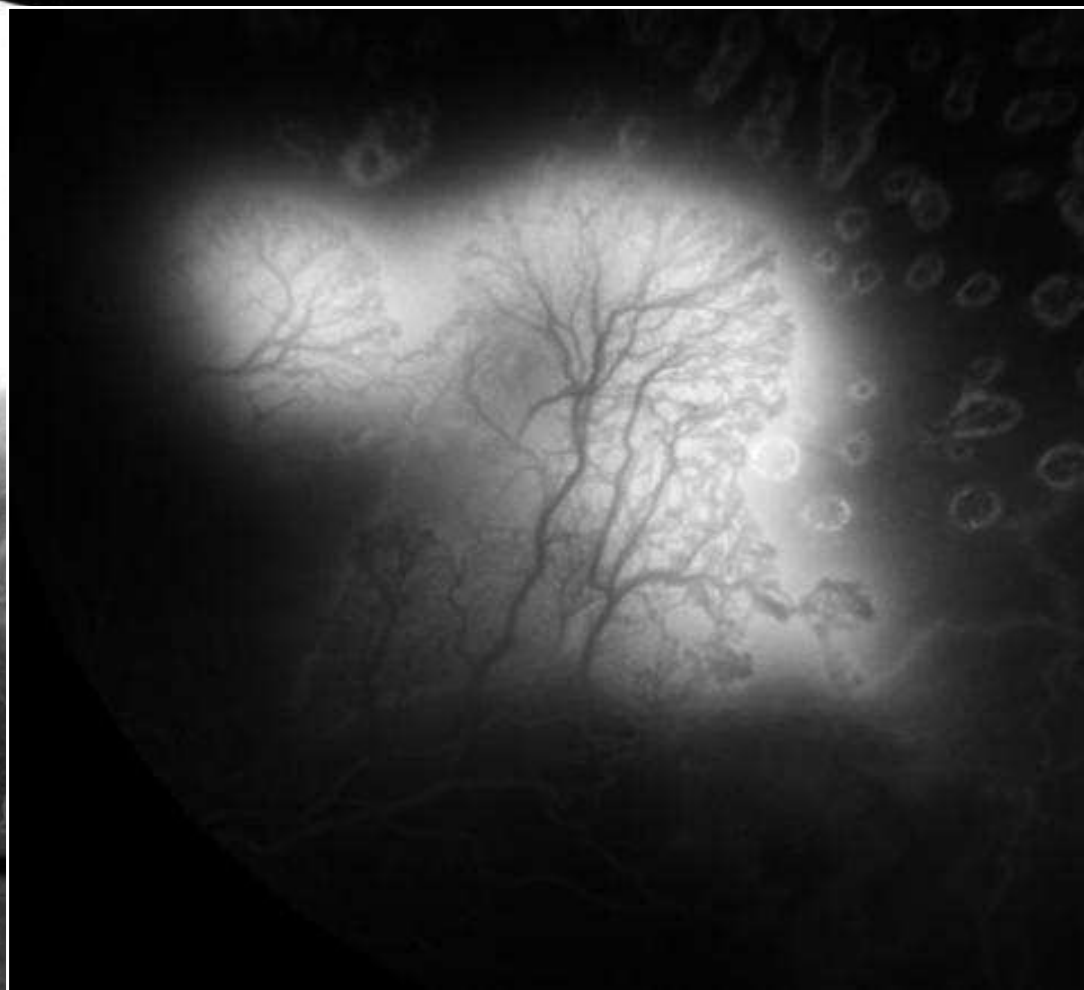
Dr ML Le Piffer La Rochelle



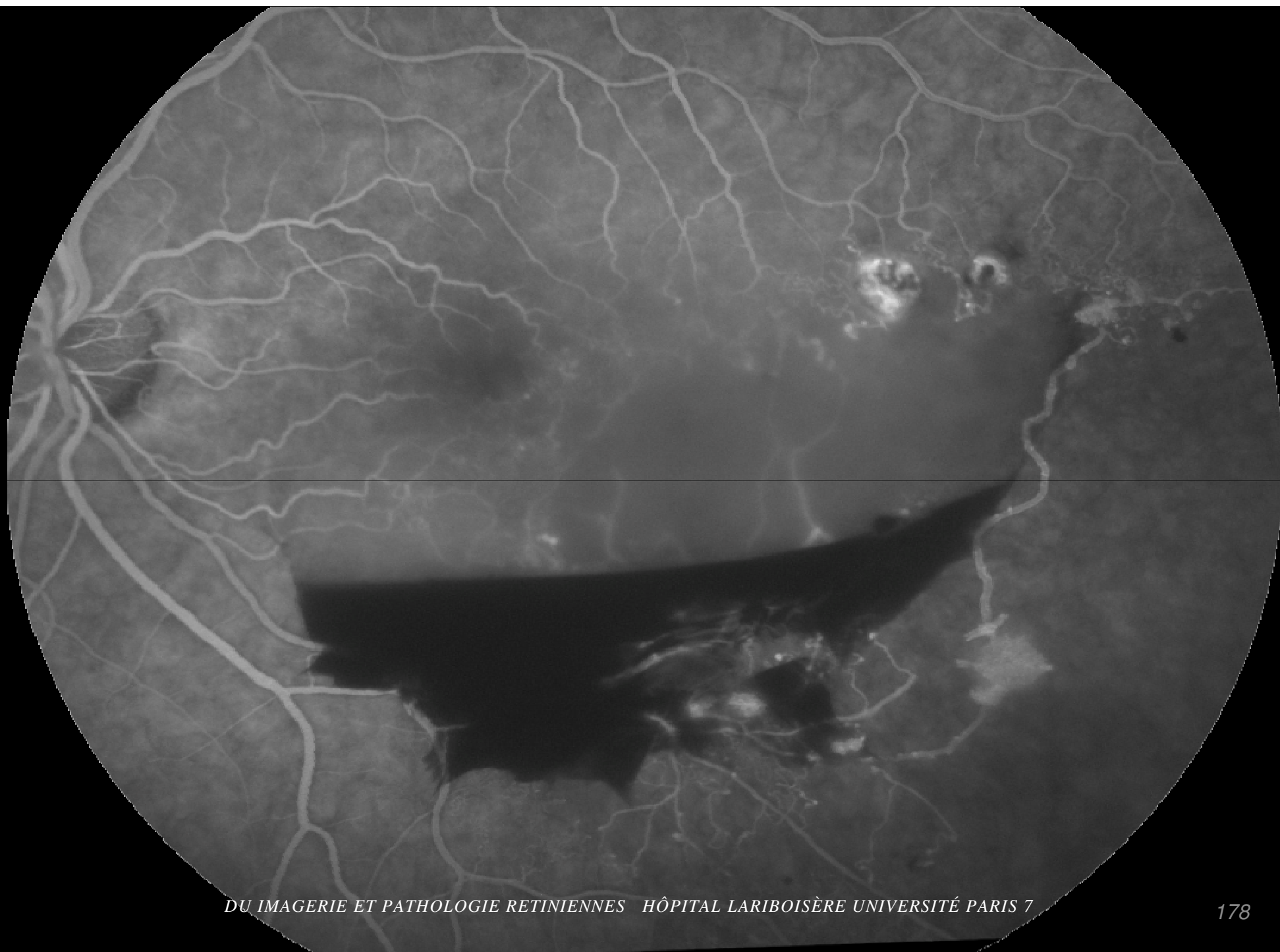


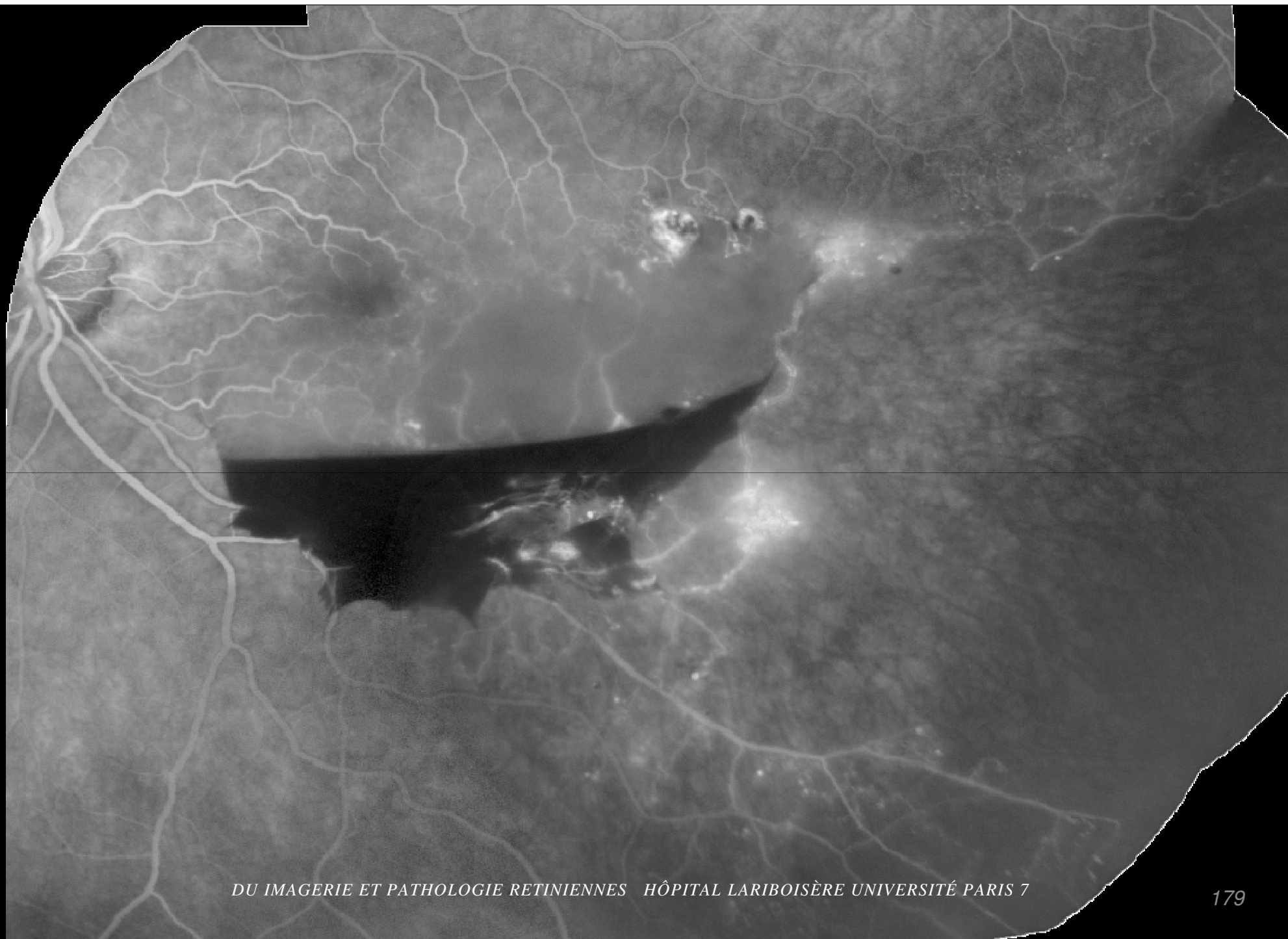
Dr ML Le Piffer La Rochelle

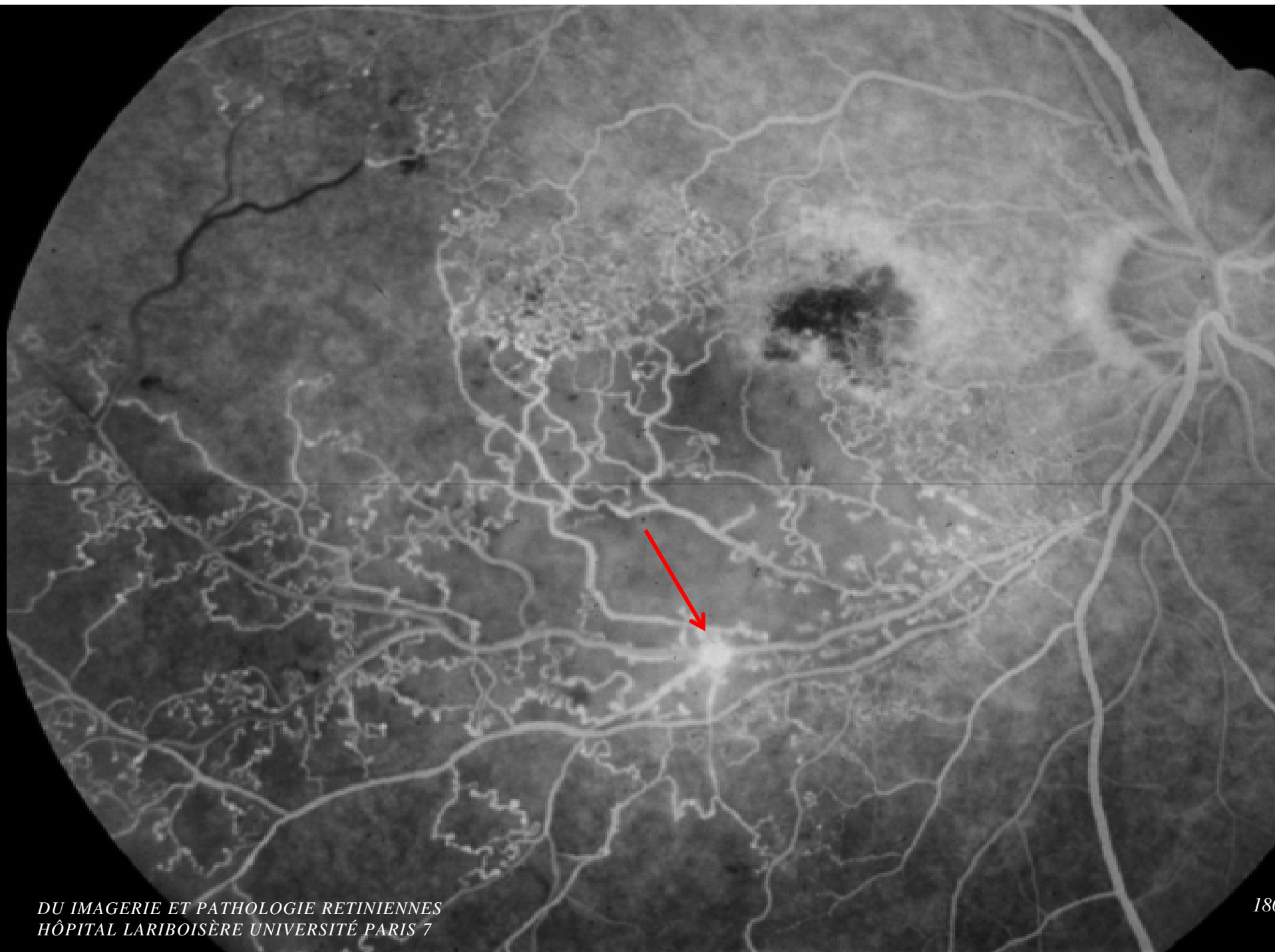






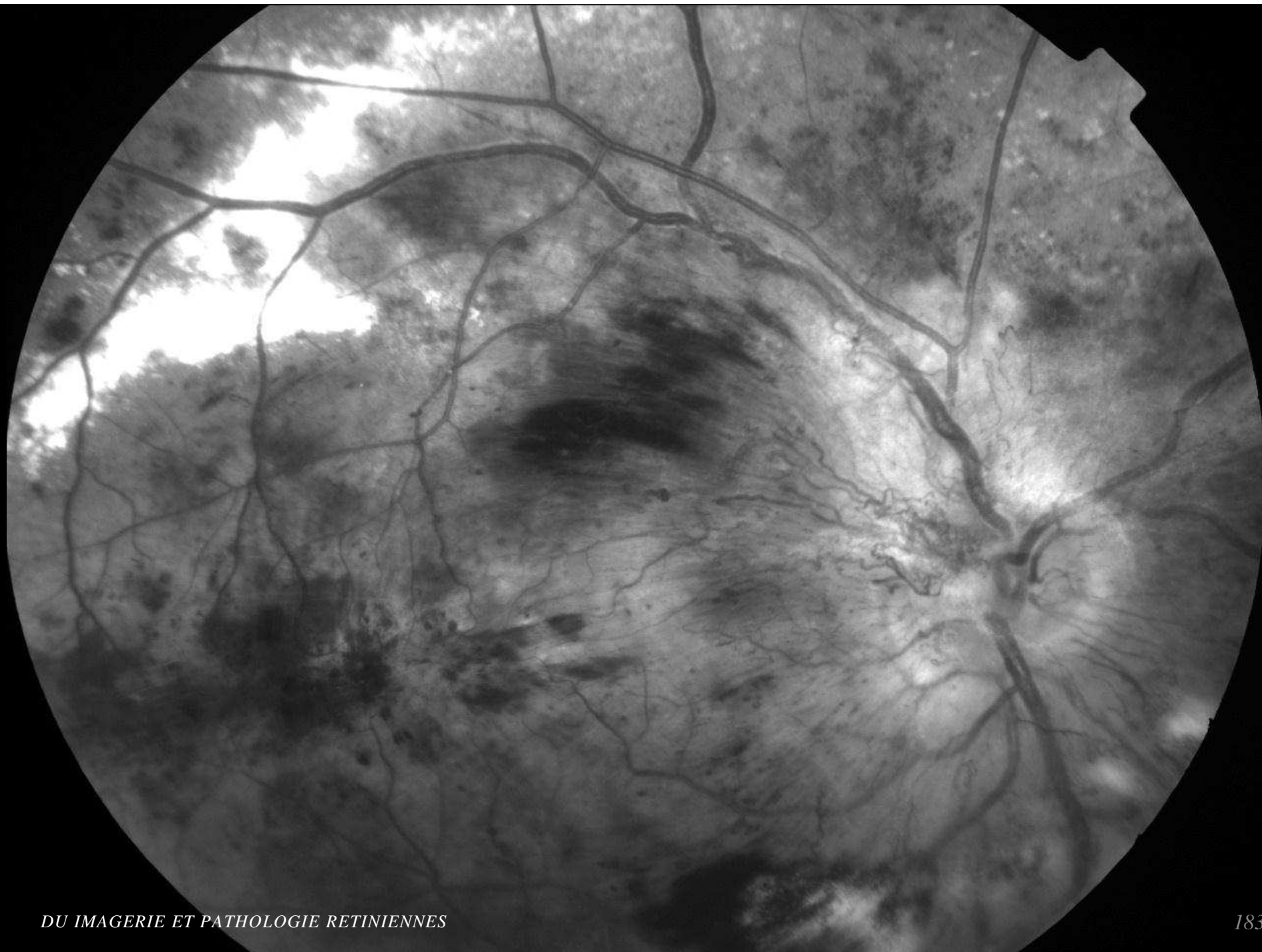


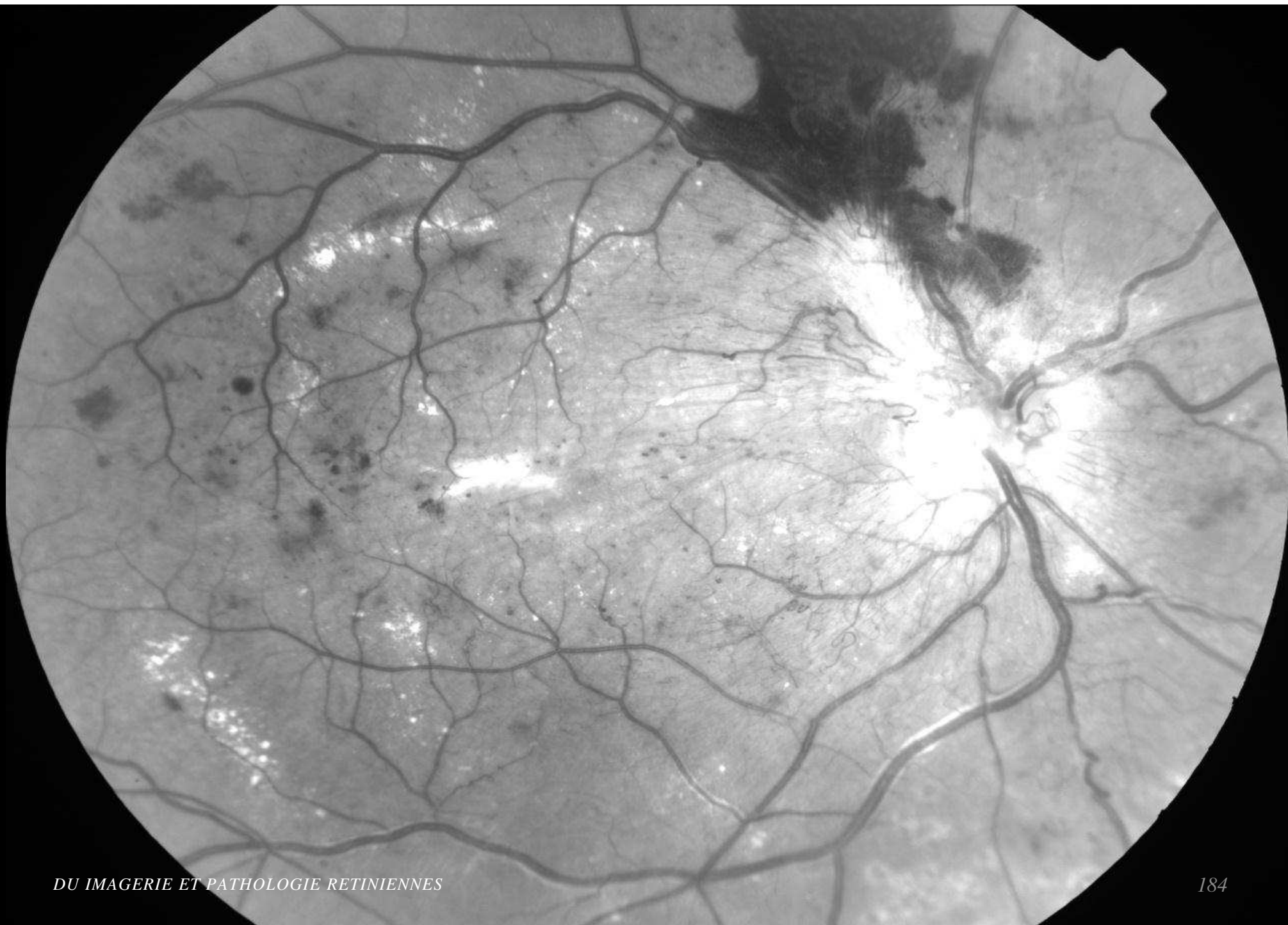




CAS PARTICULIER: L'OVCR DU DIABÉTIQUE

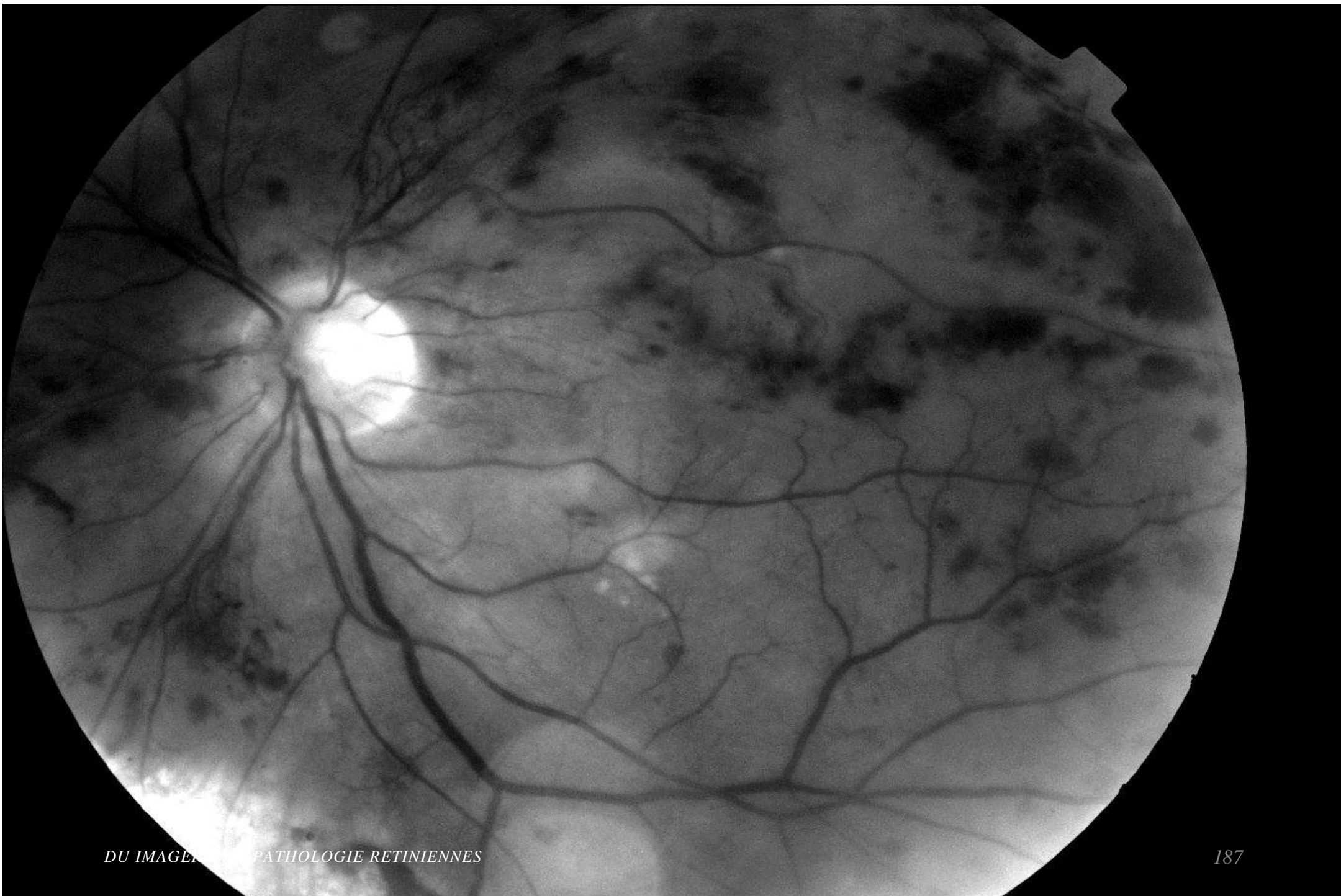


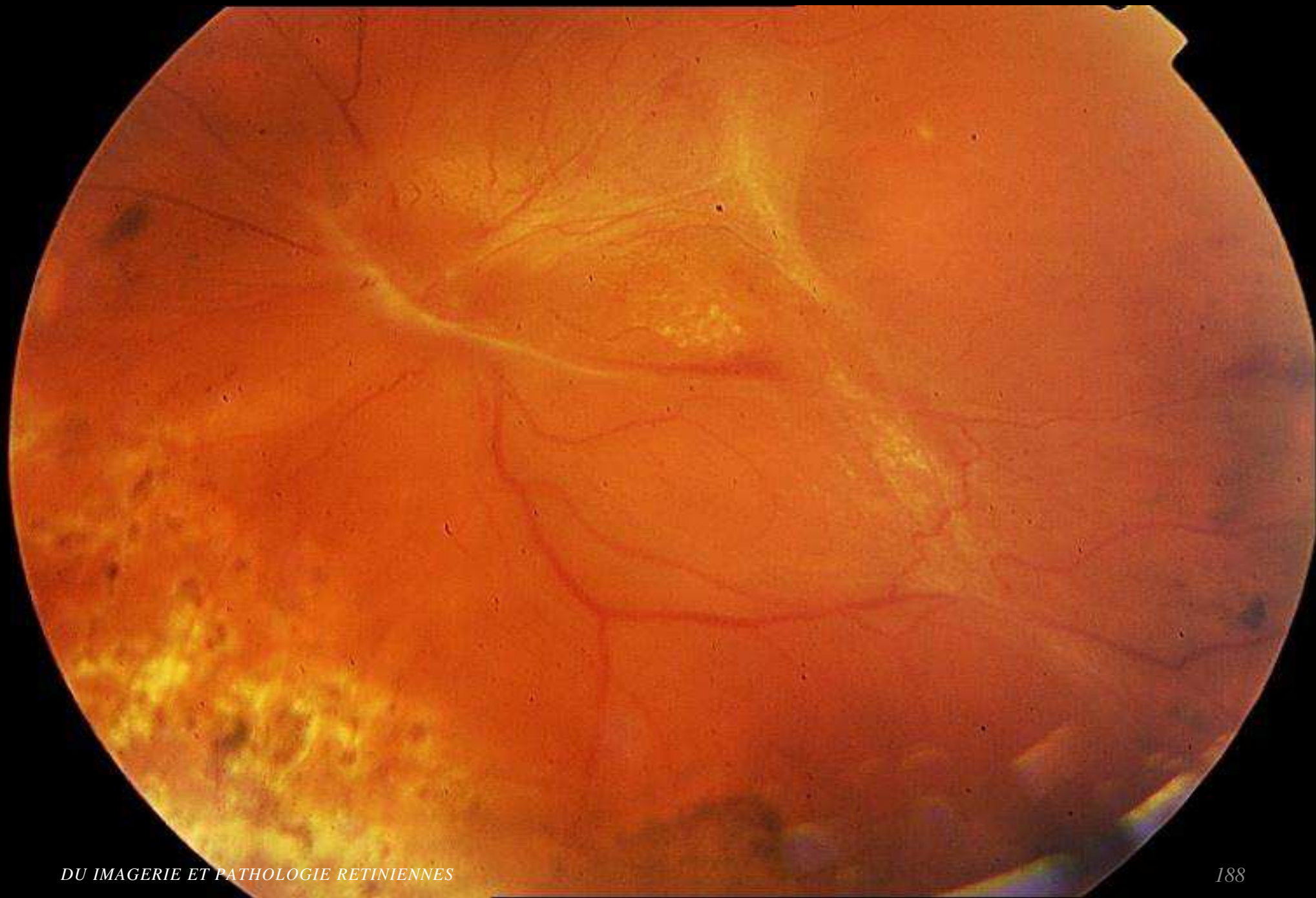


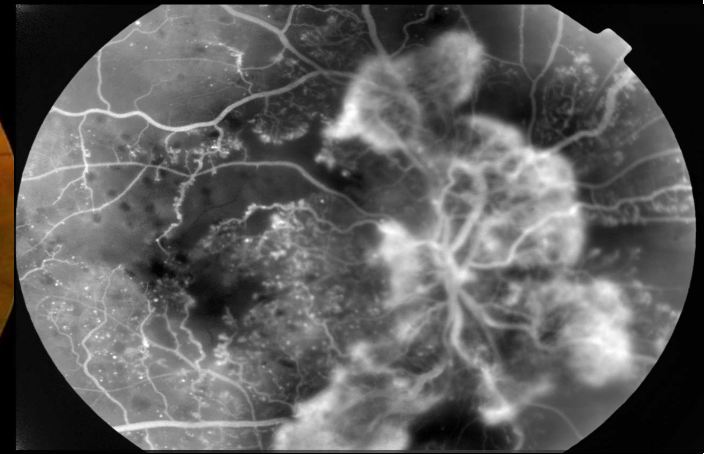












- Le diabète aggrave les OVR

NP → HW ! (v MP / JFG)

TOTAL POPULATION (106 PATIENTS)

	No new vessels (n=70)	New vessels (n=36)	p
Age (yrs) $\pm$SD	61.2$\pm$13.3	65.8$\pm$12.8	0.054
Age $\geq$ 65 yrs (%)	39	61	0.028
Men, % patients	51	58	0.502
Duration of the disease (months) $\pm$SD	27$\pm$24	31$\pm$25	0.181
History of ocular hypertension (%)	37	39	0.861
Hypotensive therapy (%)	44	53	0.409
Lipid lowering medications (%)	40	31	0.342
Current smokers (%)	7	8	0.827
Familial history of diabetes (%)	36	33	0.808
Known or unknown diabetes (%)	17	28	0.203
Body mass index (kg/m ²) $\pm$ SD	25.3 $\pm$ 4.6	27.2 $\pm$ 3.2	0.001
Waist circumference (cm) $\pm$ SD	93 $\pm$ 12	100 $\pm$ 12	0.006
Systolic blood pressure (mmHg) $\pm$SD	143$\pm$21	146$\pm$17	0.354
Diastolic blood pressure (mmHg) $\pm$SD	84$\pm$11	81$\pm$10	0.118
HbA1c (%) $\pm$ SD	5.9 $\pm$ 0.5	6.5 $\pm$ 1.5	0.034
HbA1c $\geq$ 6% (%)	36	58	0.053

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

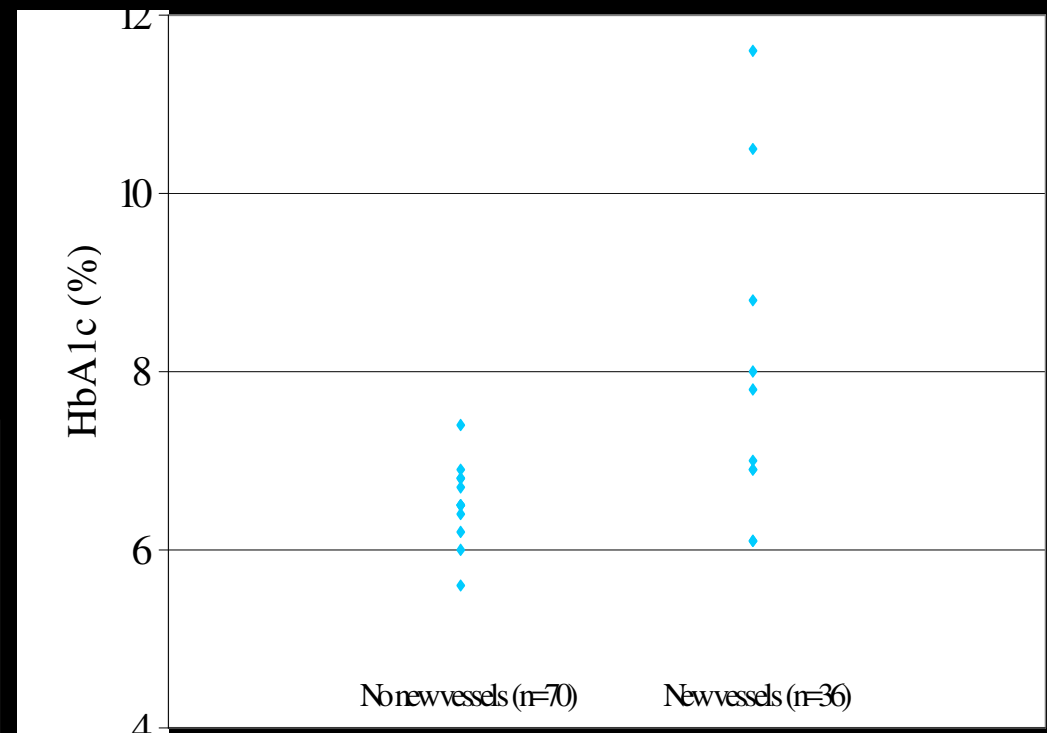
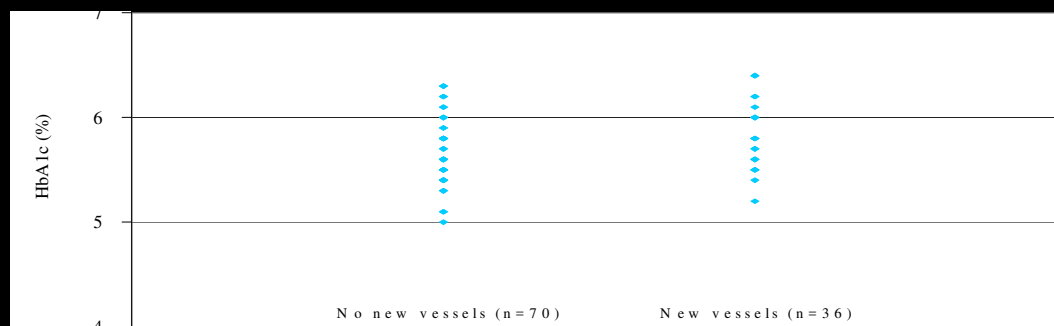
NON DIABETIC PATIENTS (N=84)

	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

HbA1c AND NEW VESSELS IN NON DIABETIC VS 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

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); et dans cette DVP 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

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

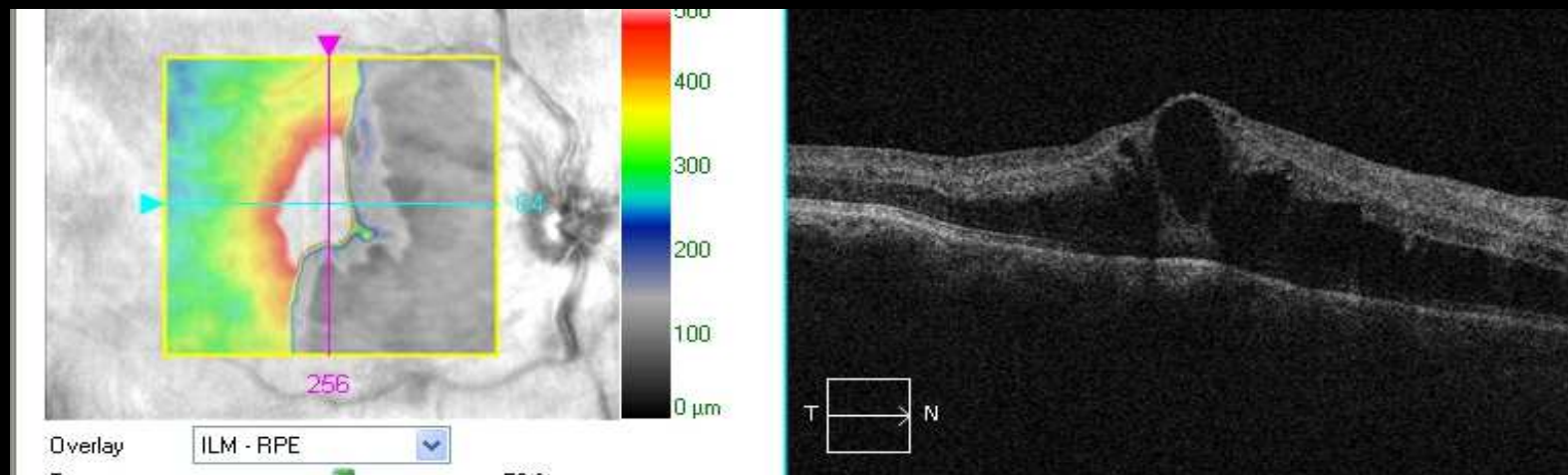
- Notion de dépopulation capillaire précoce dans la DVP, justement là où se forment les collatérales (*Dupas et coll, soumis*)
- Un suivi plus rapproché des patients diabétiques, surtout s'ils sont déséquilibrés, paraît raisonnable

L'œdème maculaire

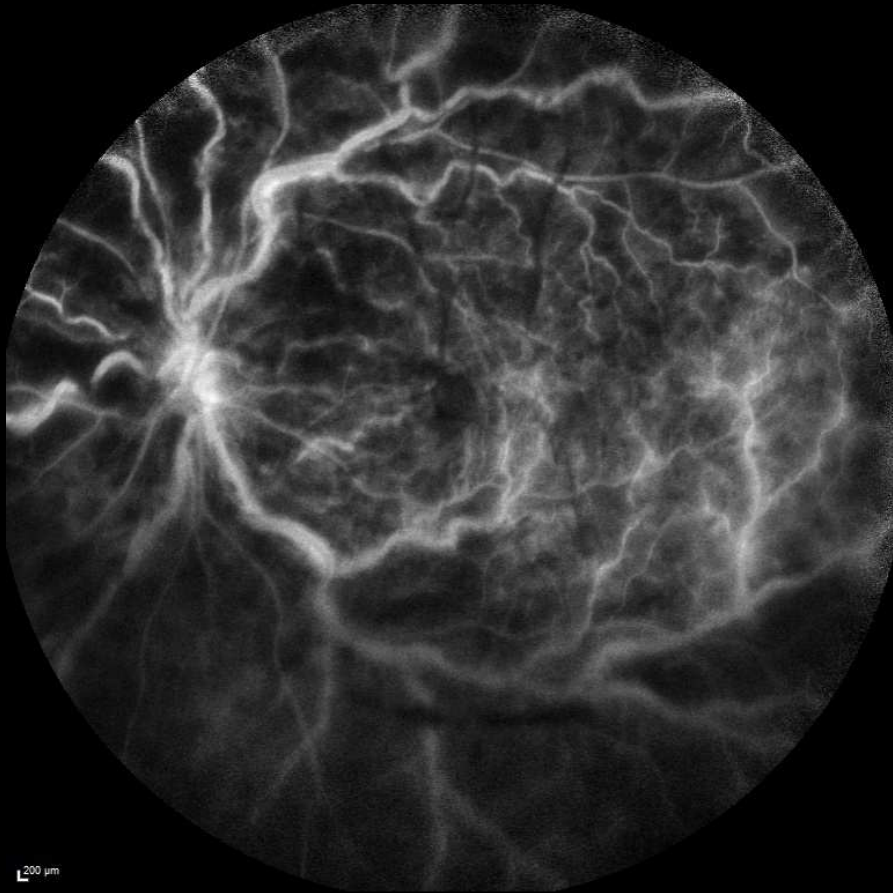


Mode de révélation le plus fréquent d'une OVR

La plupart des OVR font ou feront un œdème maculaire, dont les mécanismes peut être multiples



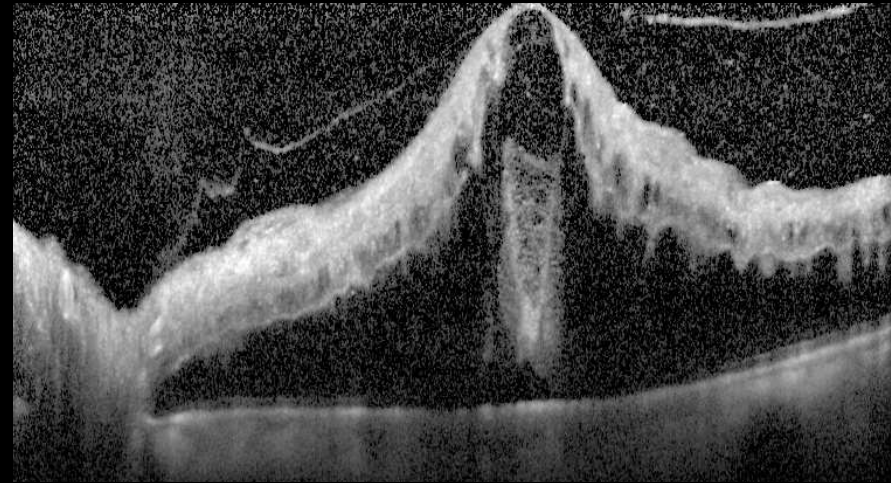
Merci Th Desmettre

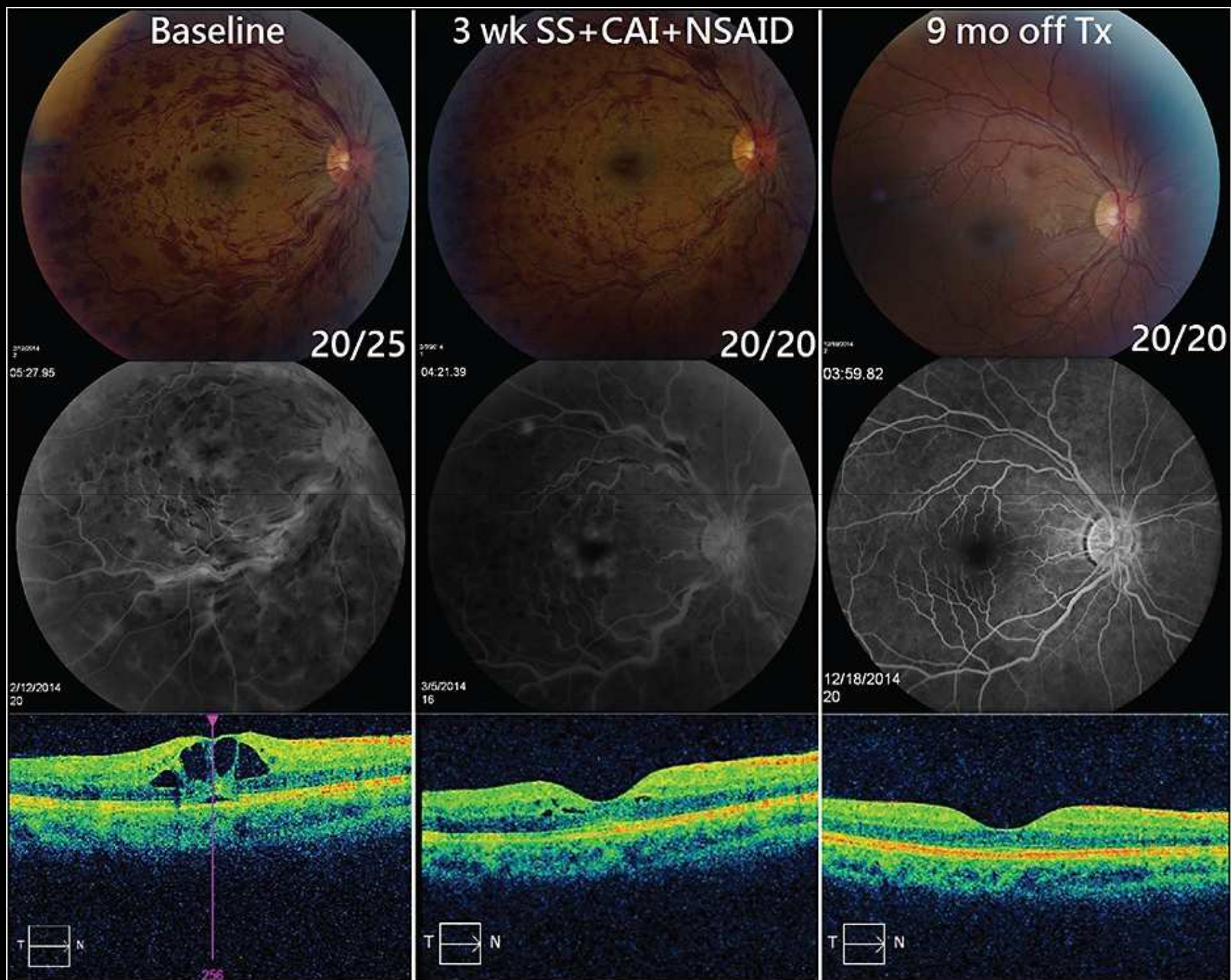


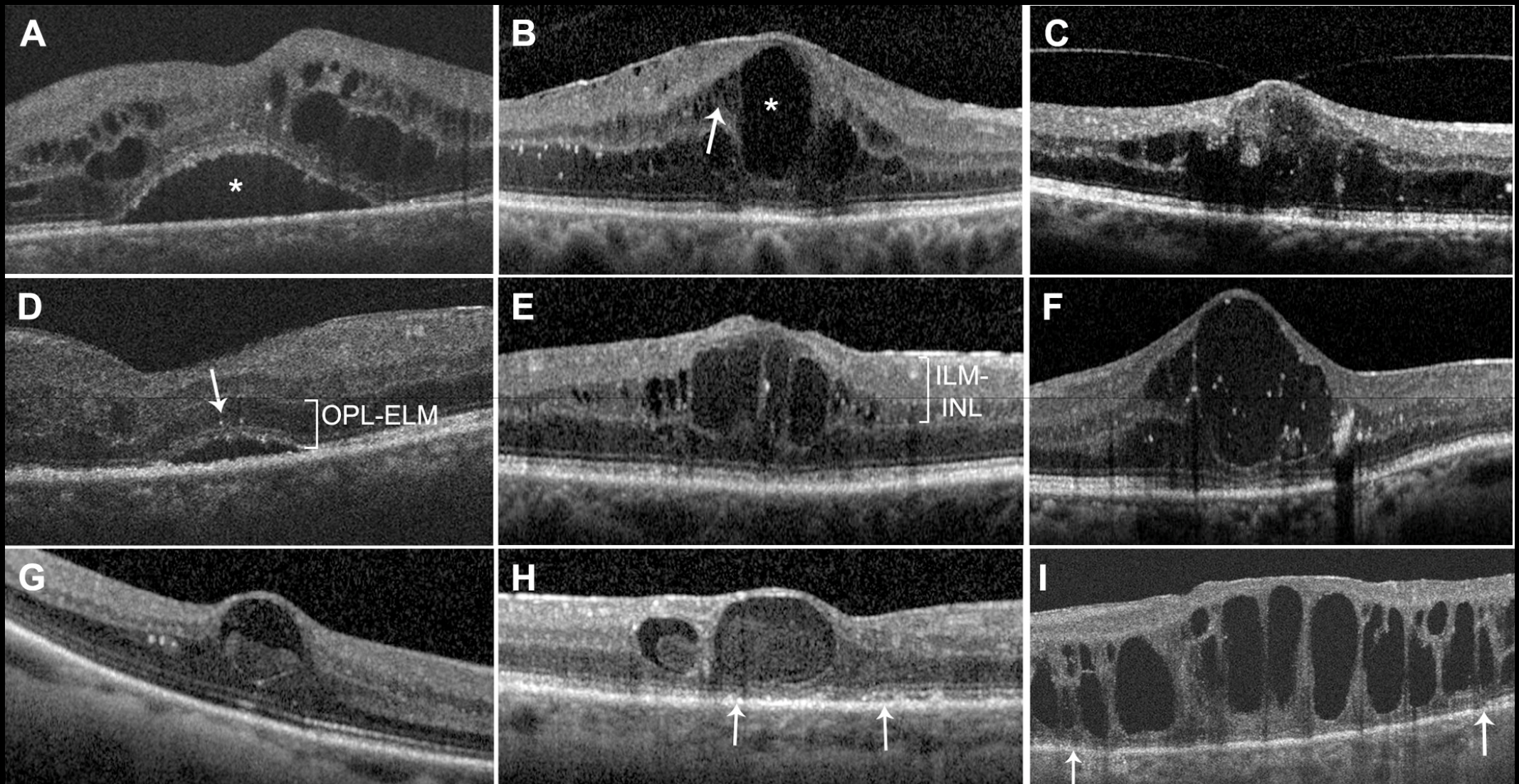
200 μm

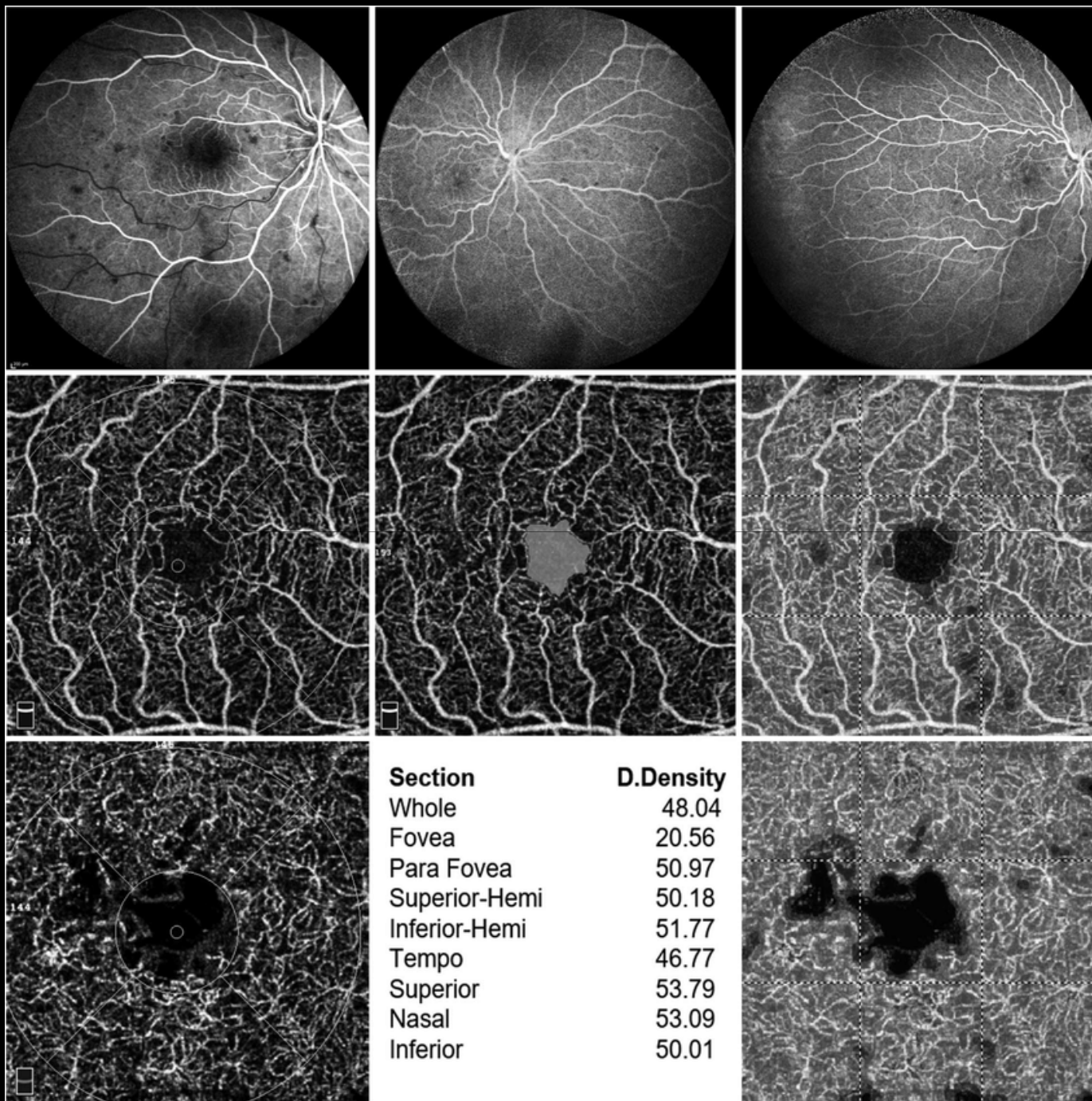
31/01/2017, OS
FA 4:13.08 55° ART(16)

HEIDELBERG
ENGINEERING

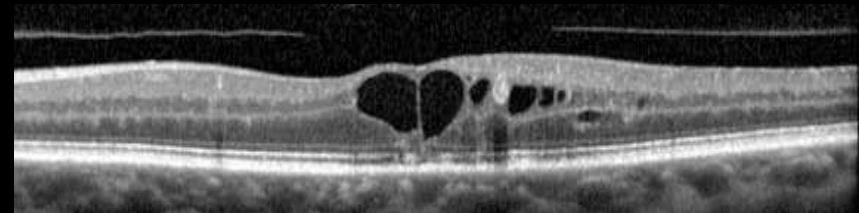
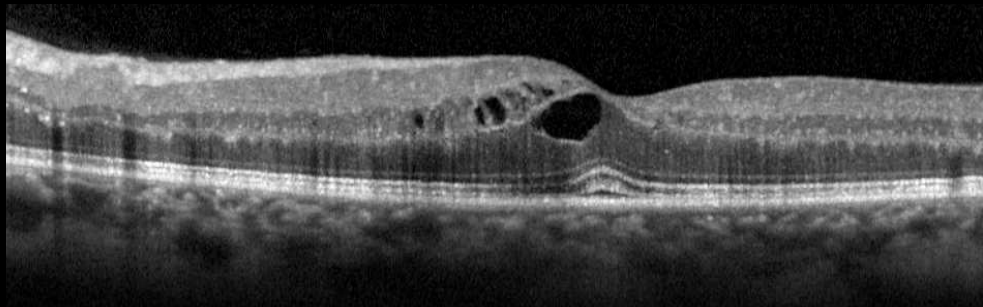


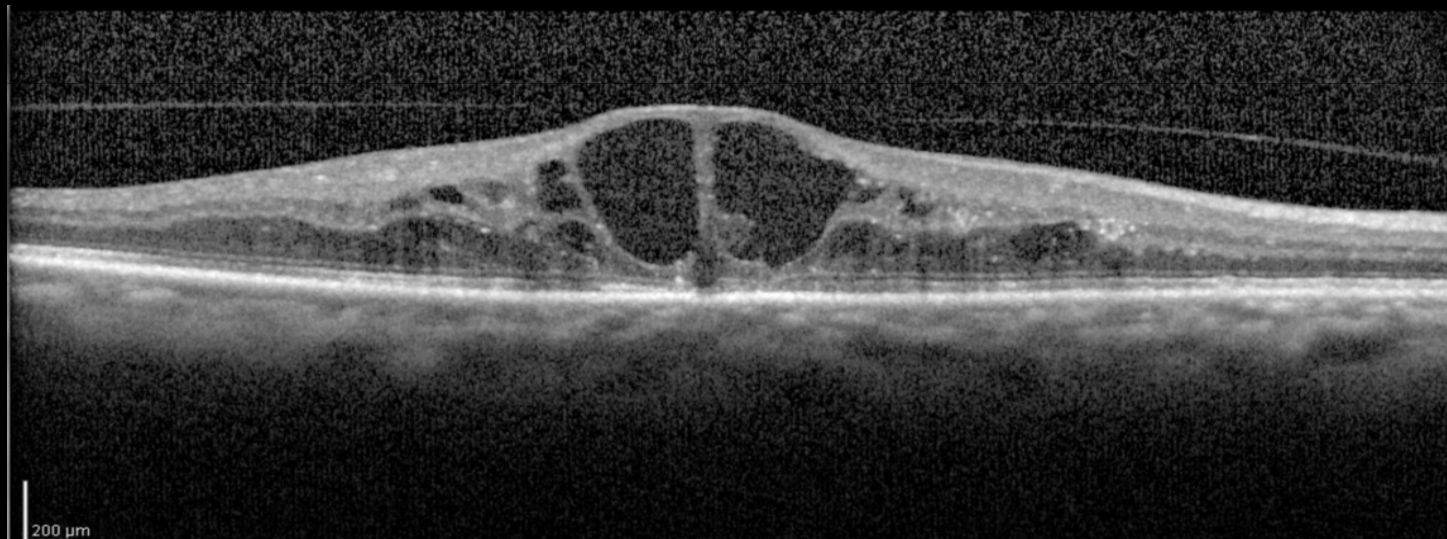
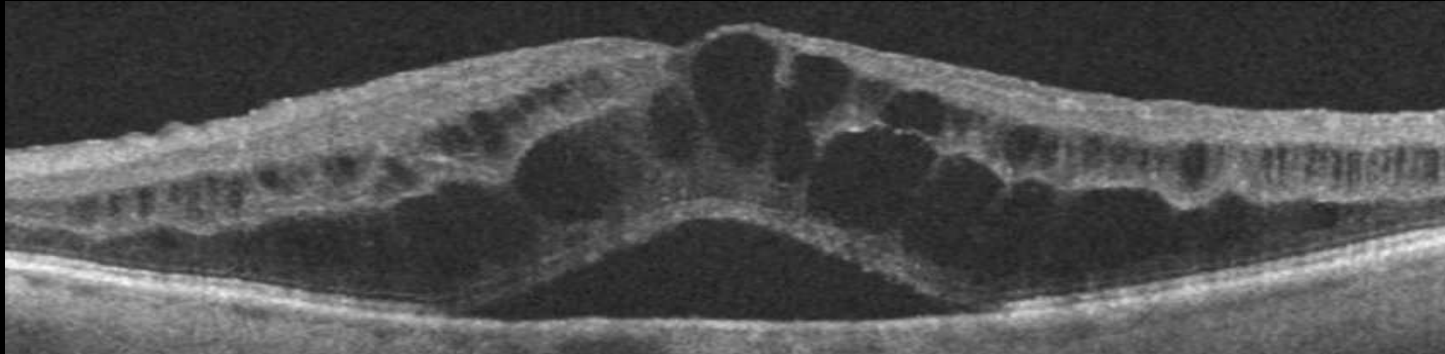




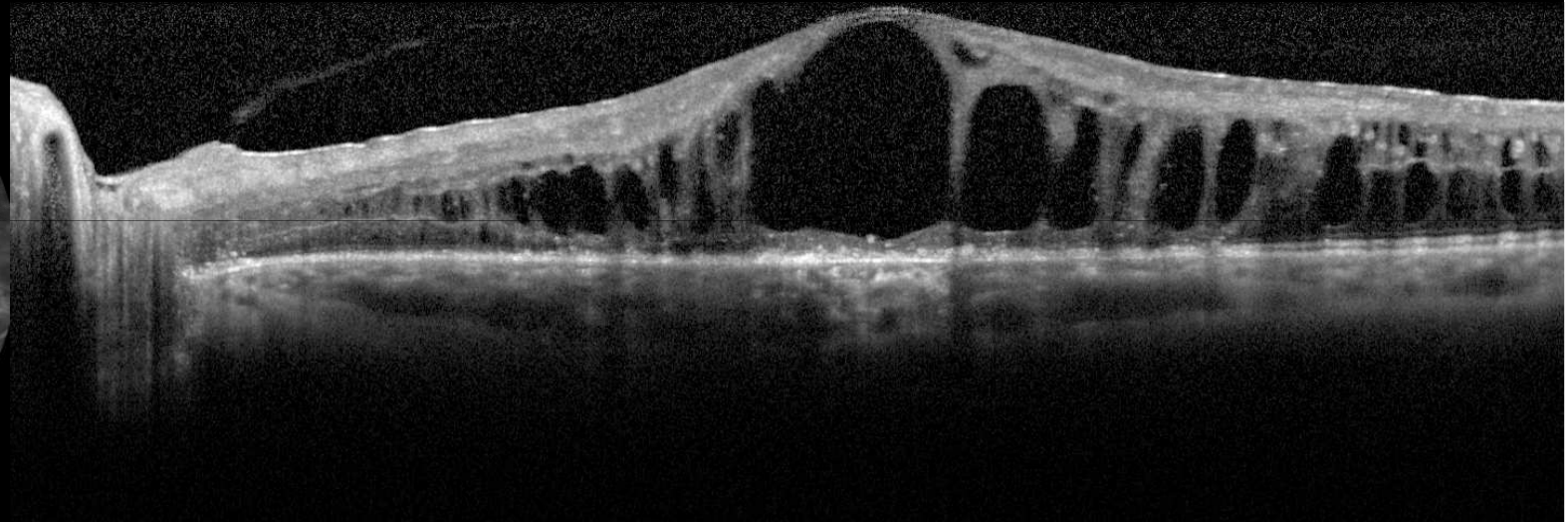
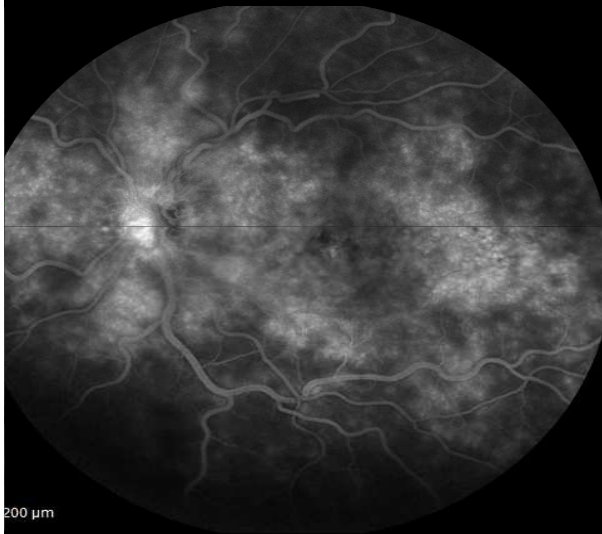


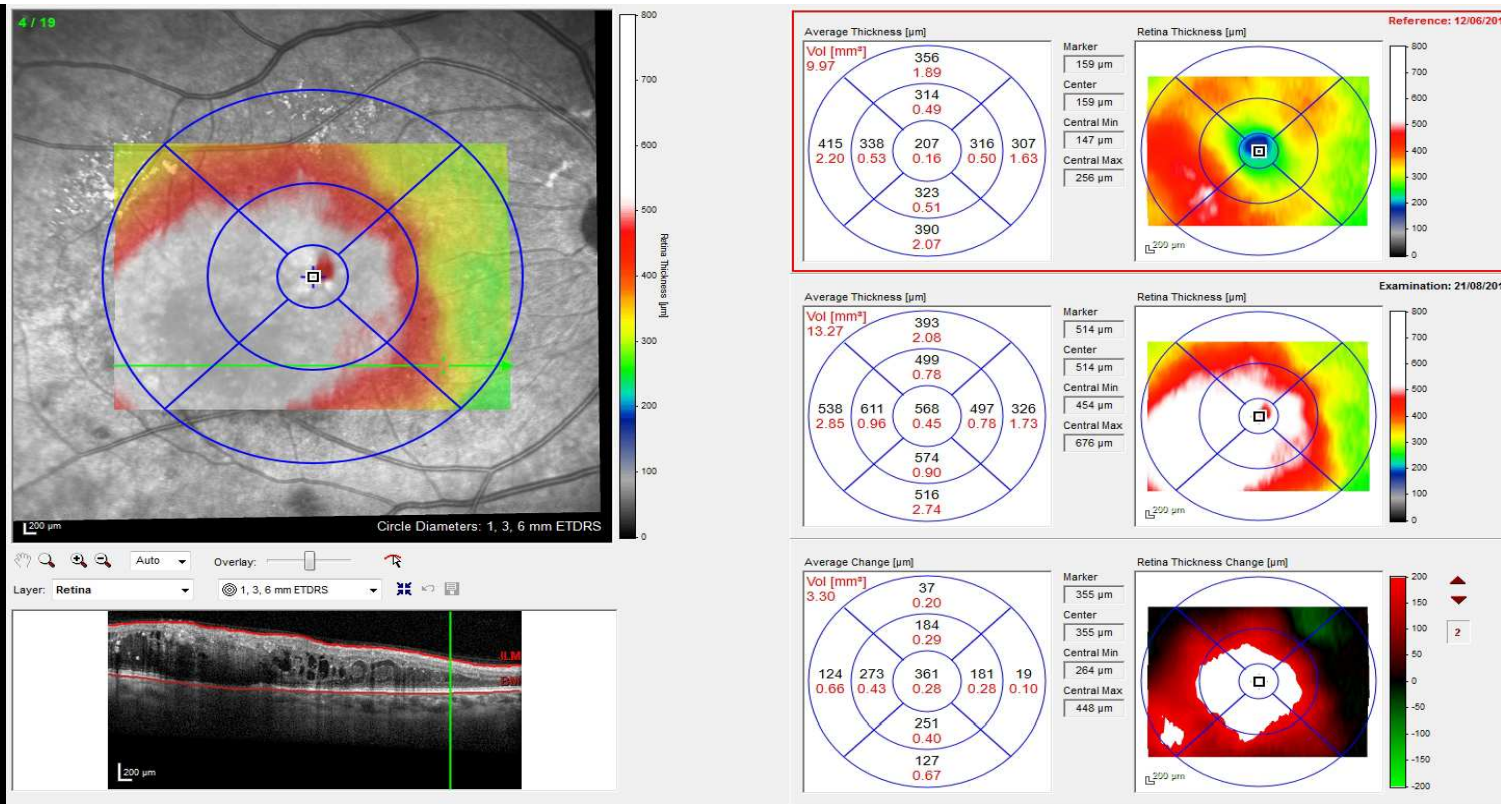
- The concept of CME was invented by FA
- It is uncertain whether or not non cystoid and cystoid ME are different entities
- The macula is particularly susceptible to fluid accumulation
- The fovea is particularly prone
 - To detach
 - To develop a cyst





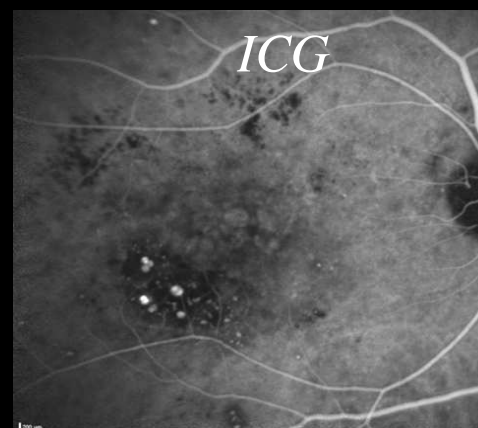
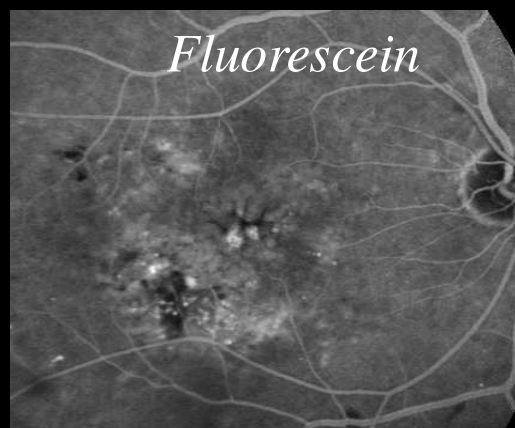
ASPECT DE SCHISIS DE CERTAINS OM CHRONIQUES





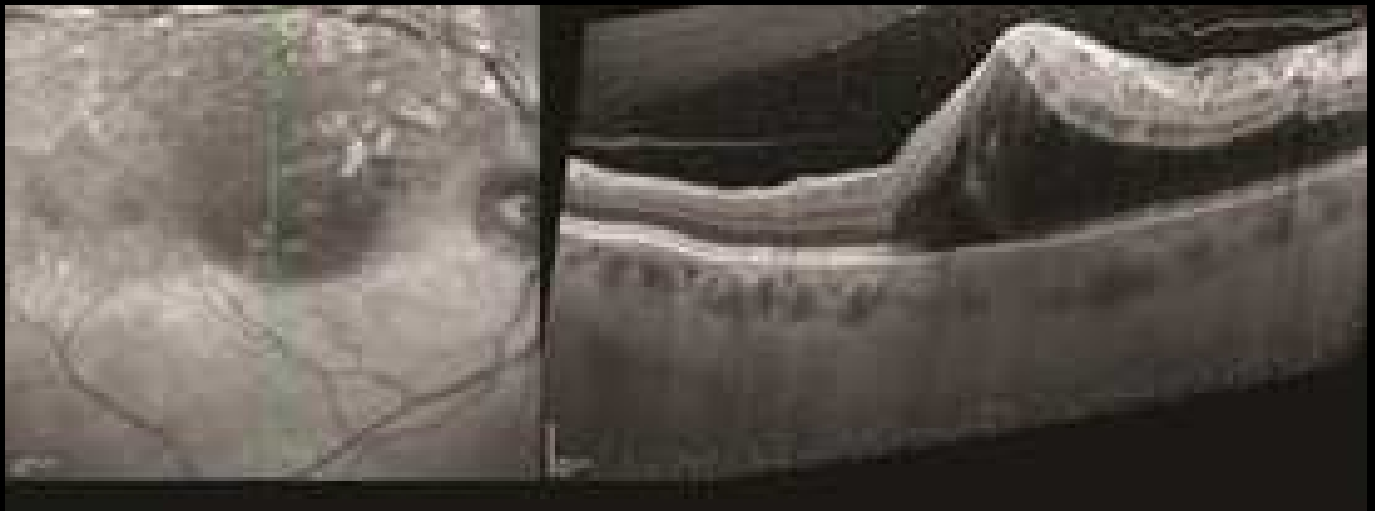
21/08/2018, OD

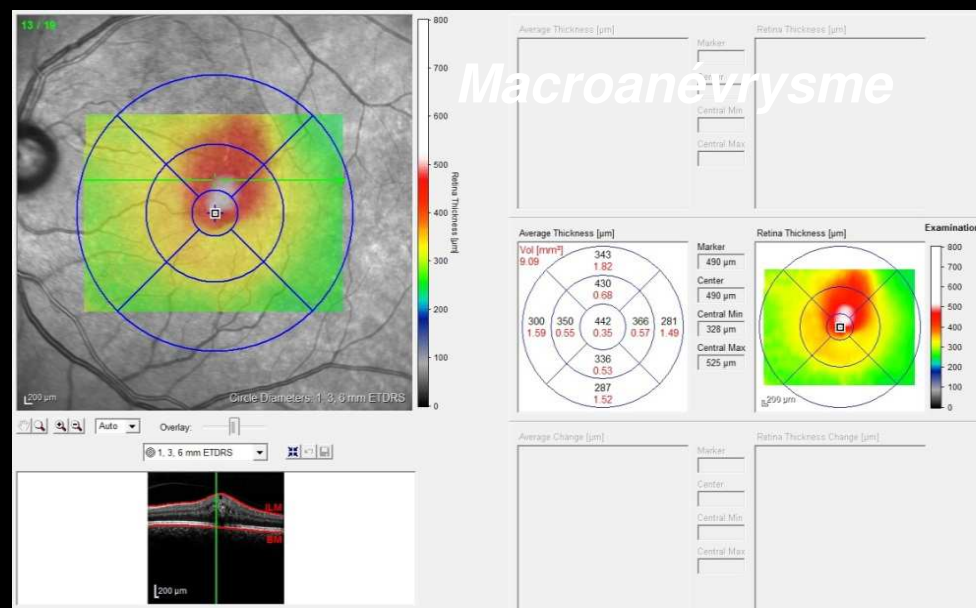
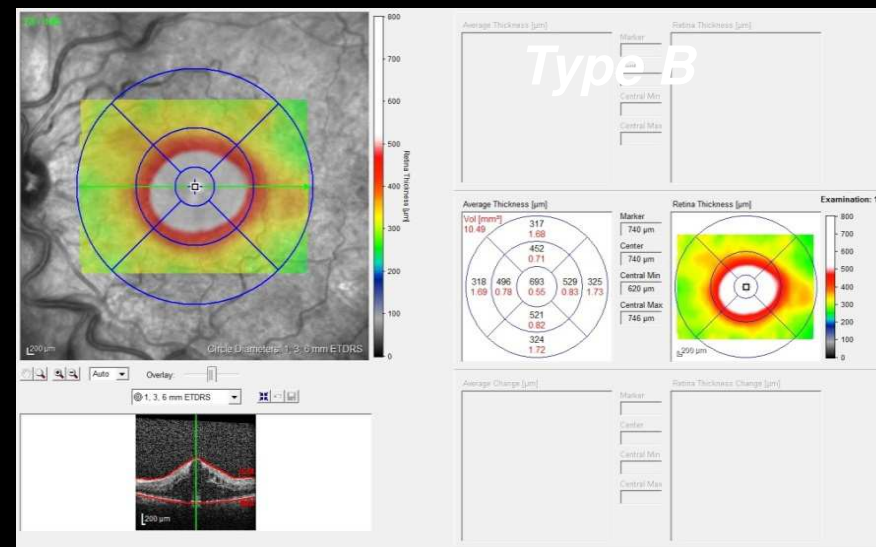
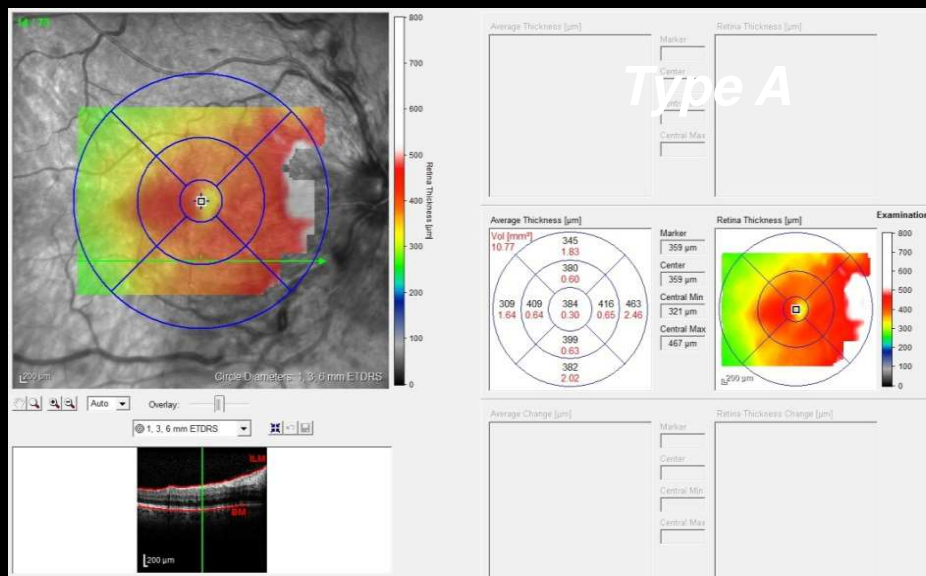
HEIDELBERG
ENGINEERING

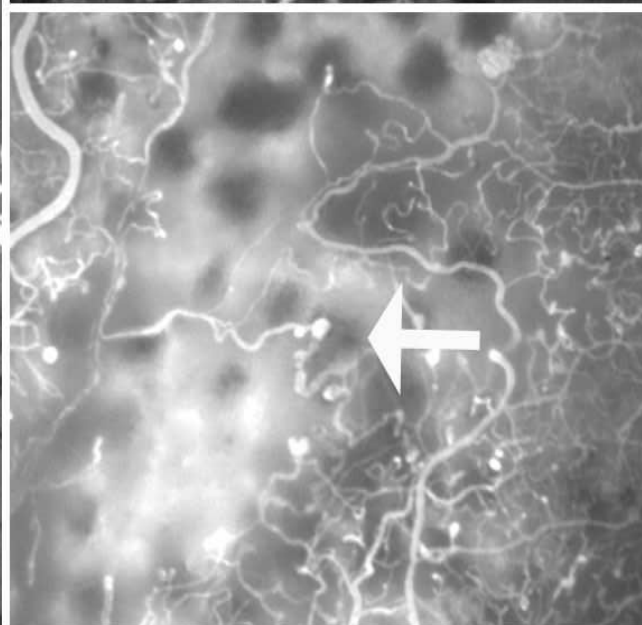
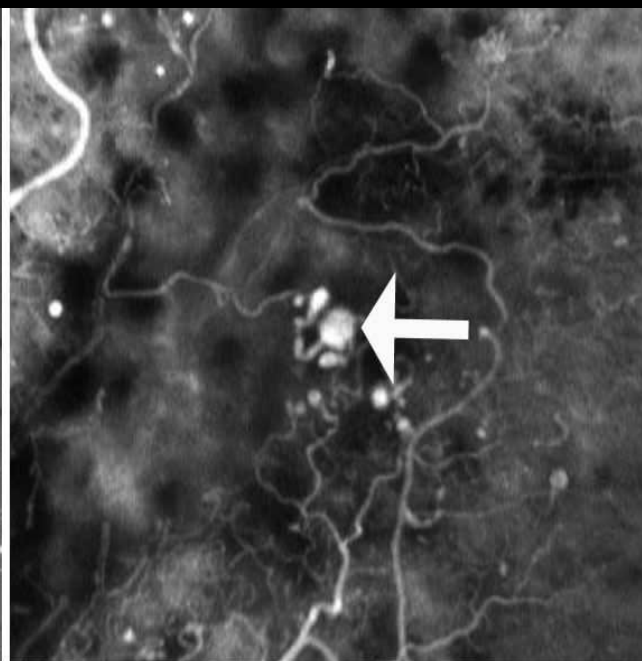
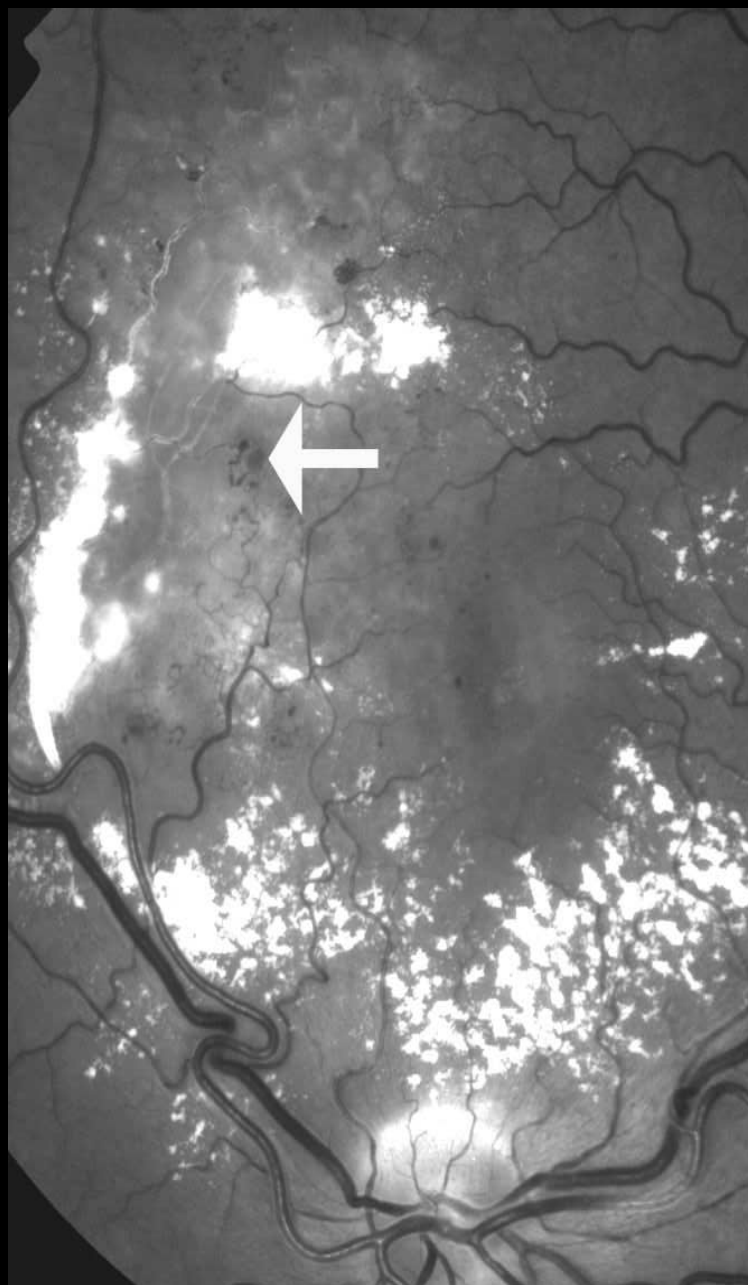


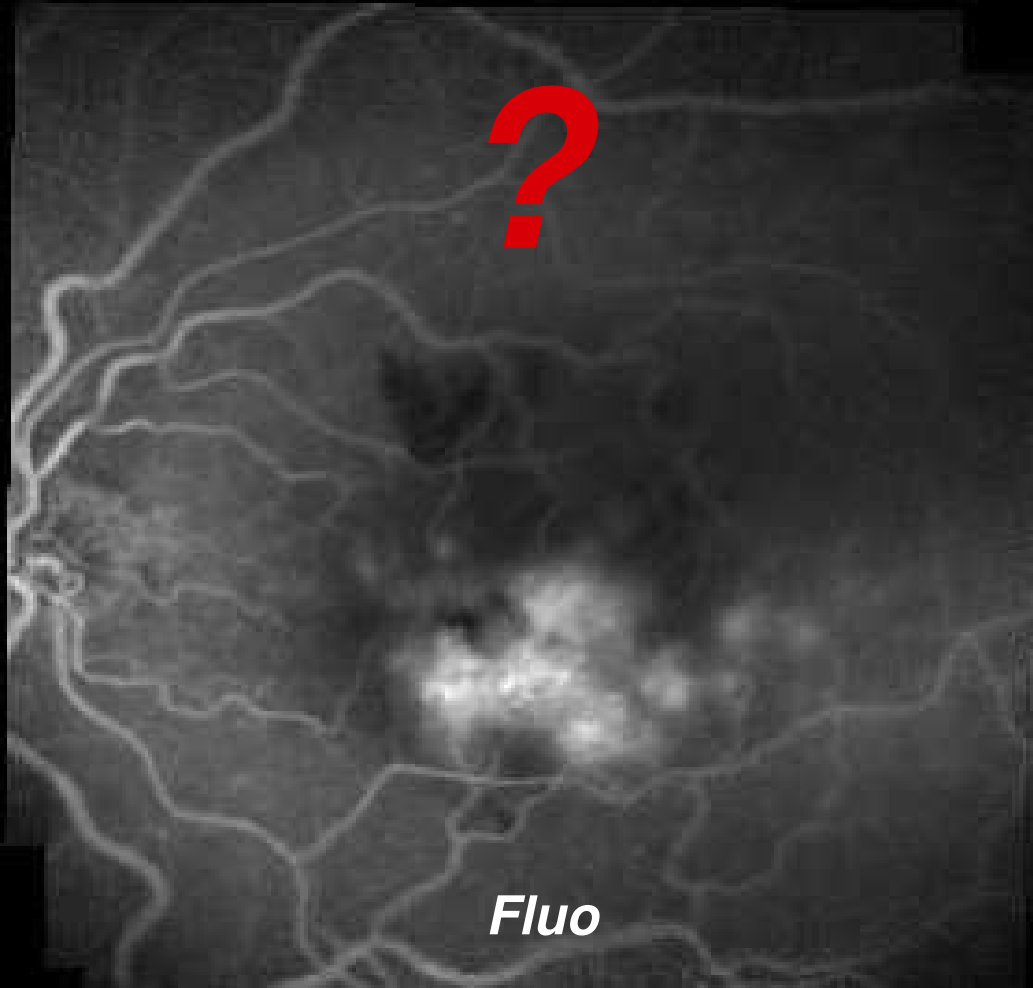
HTA AIGUE ET ŒDÈME MACULAIRE

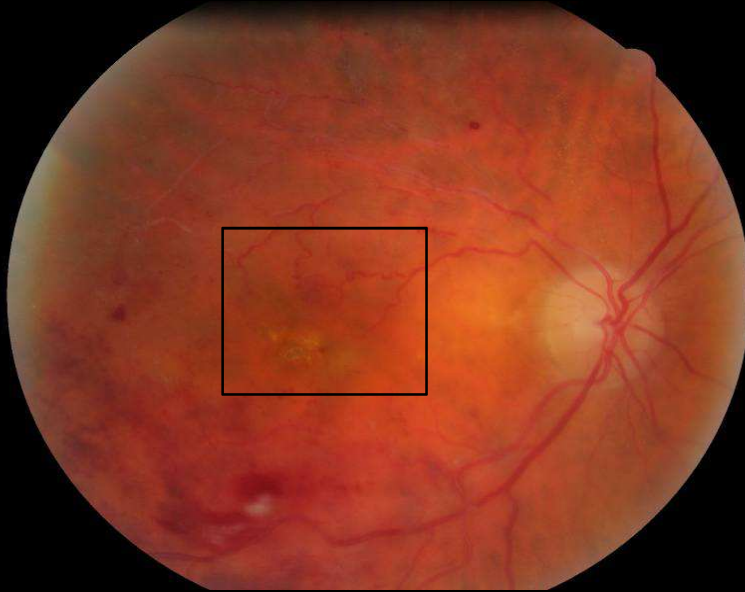
- L'HTA aigue est une cause de déclenchement d'œdème maculaire (bien qu'aucune étude ne le mentionne...)
- Il semble logique d'attendre l'effet d'un traitement antihypertenseur avant d'injecter
- Mais protéger la rétine en attendant l'effet du traitement antihypertenseur peut être prudent
- Un nombre réduit d'injection peut être justifié







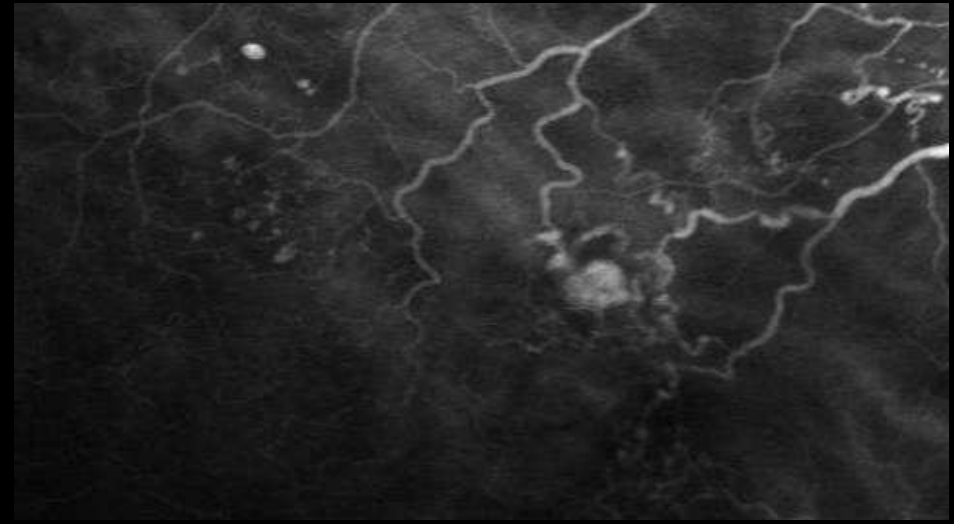
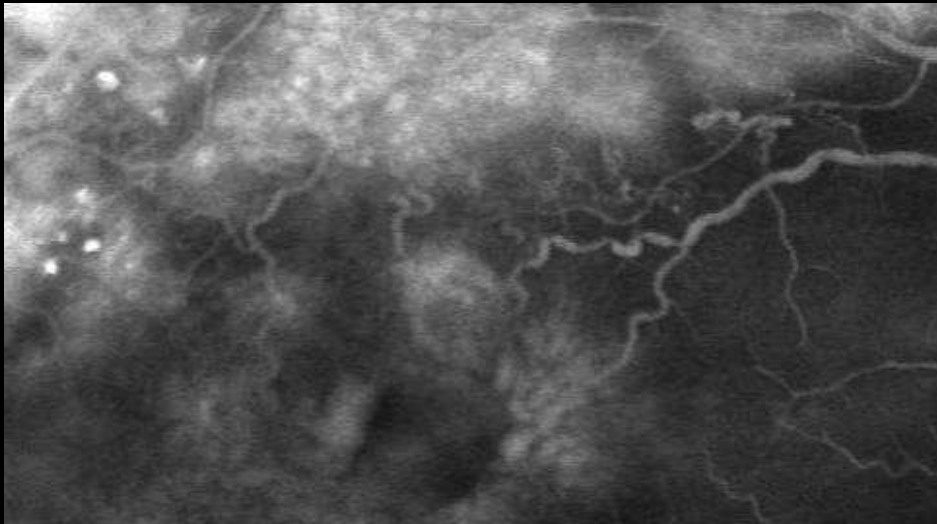




Fluorescein



Indocyanine green

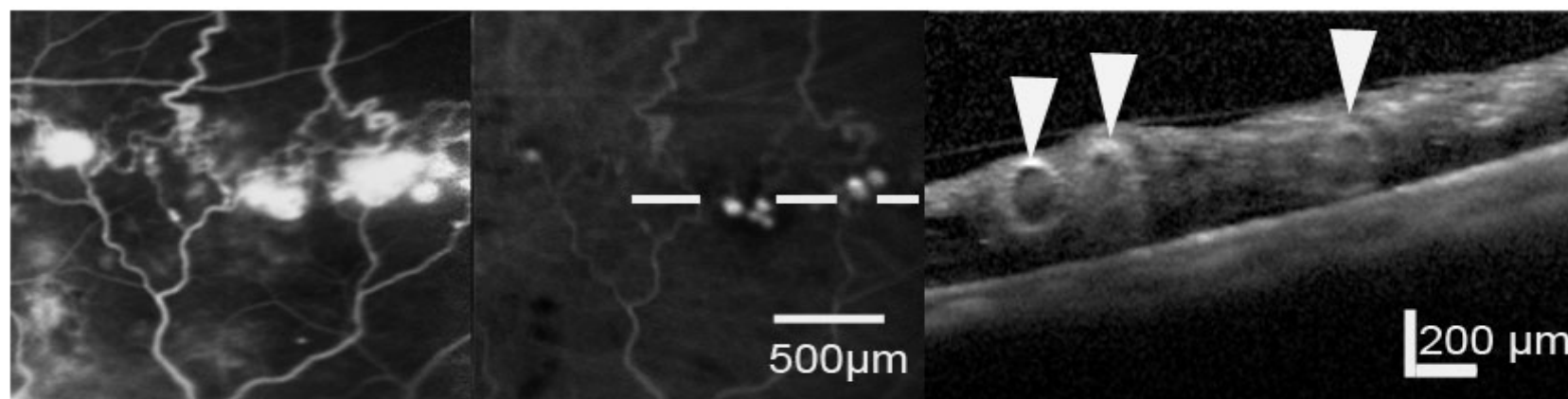
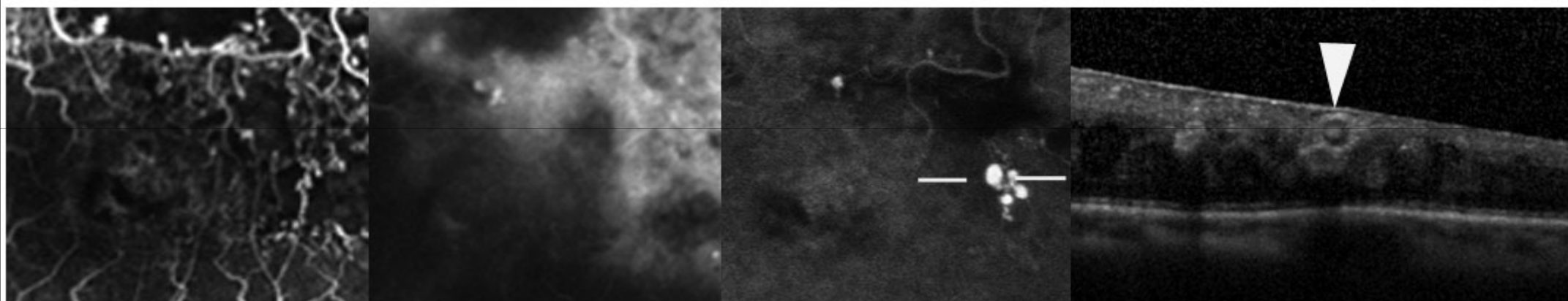
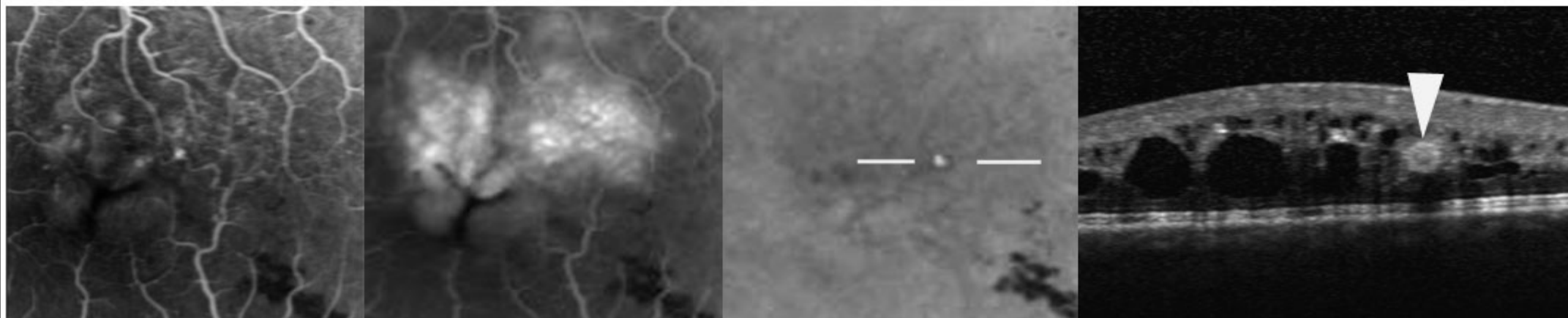


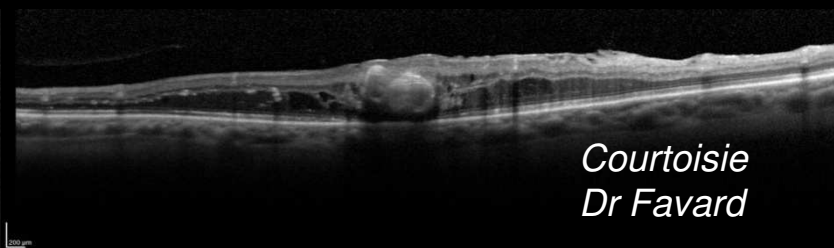
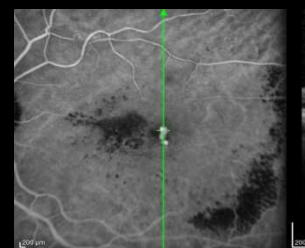
FA early

FA late

ICGA late

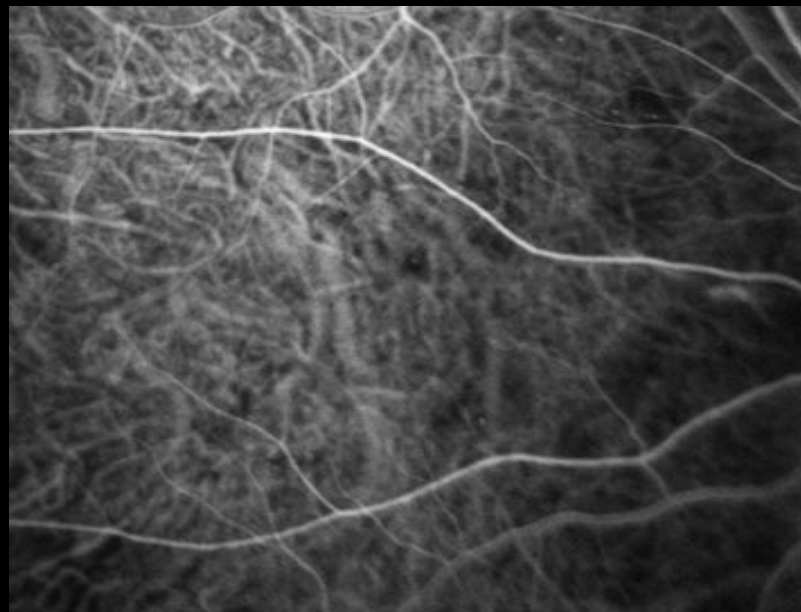
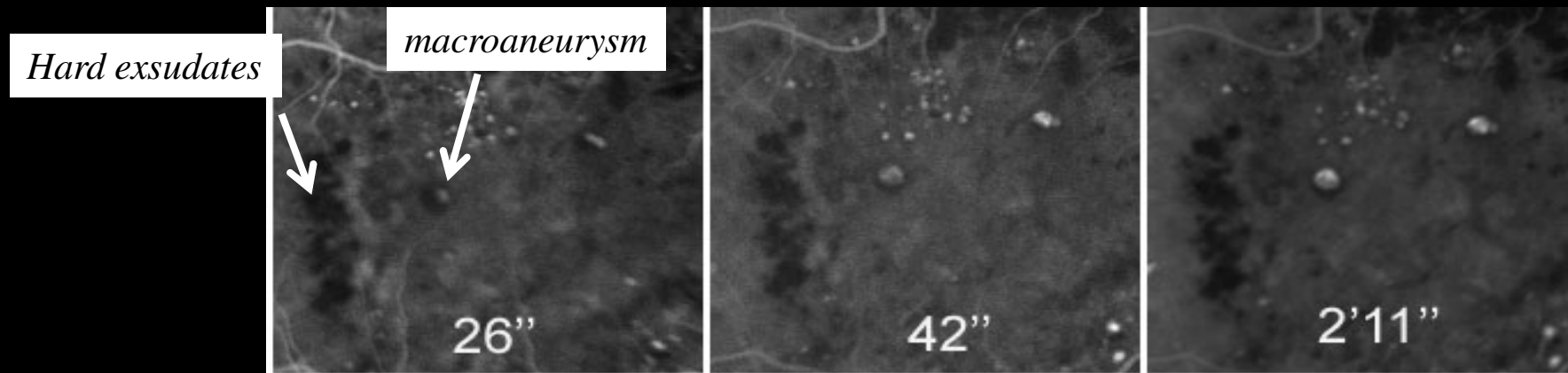
OCT

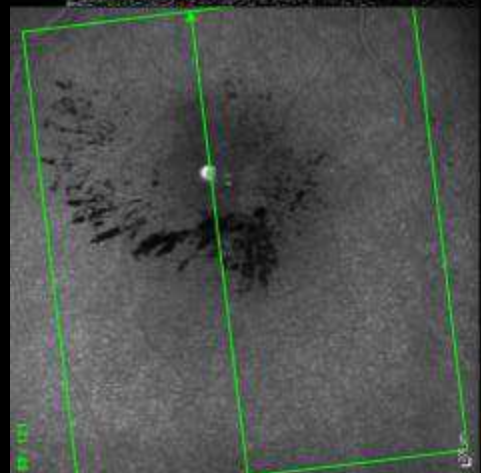
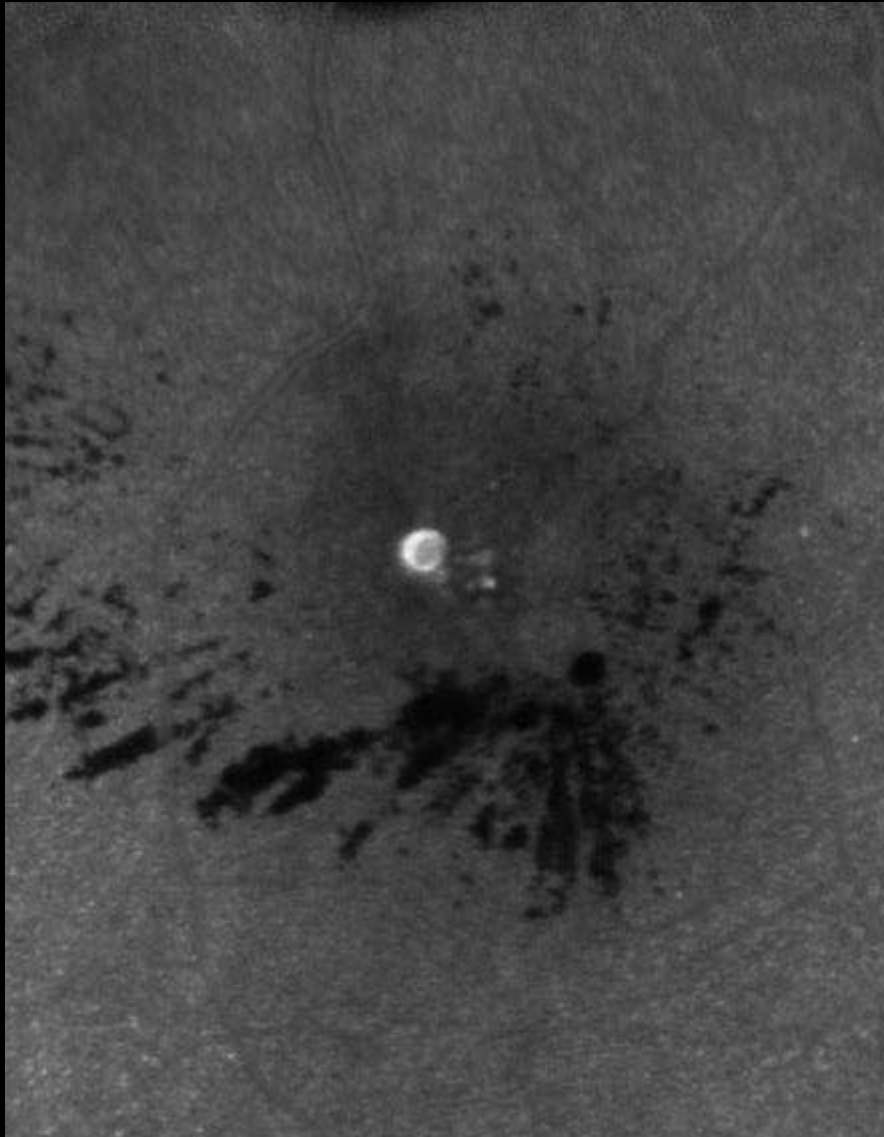
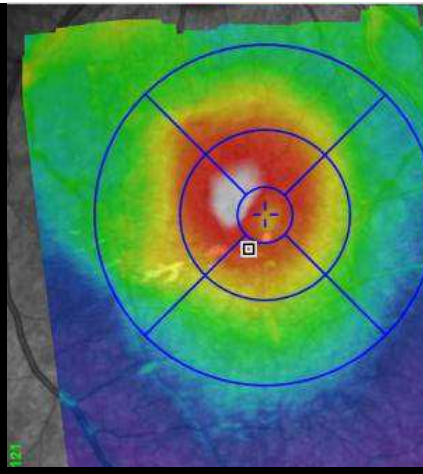


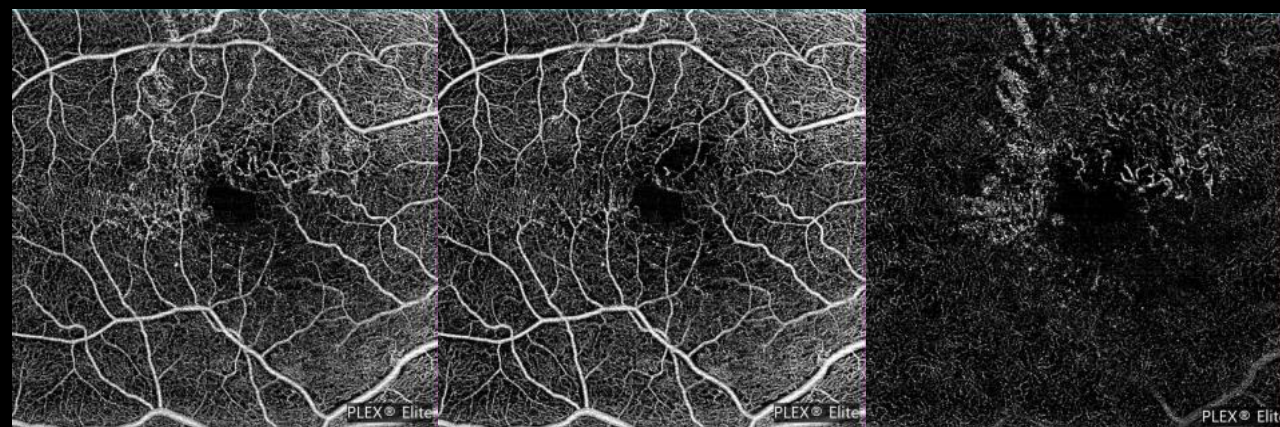
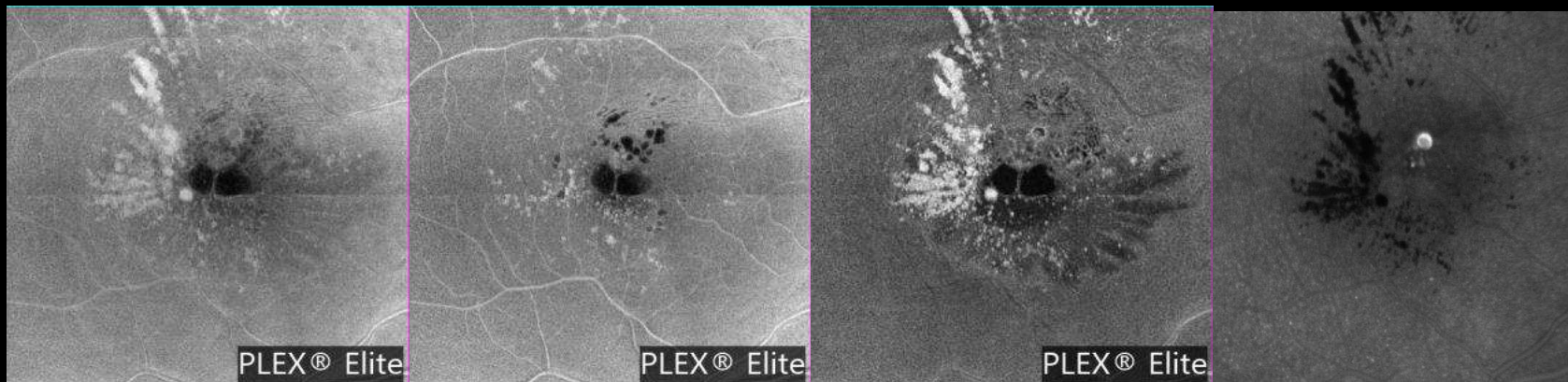


*Courtoisie
Dr Favard*

THE AMPHIPHILIC NATURE OF INDOCYANINE GREEN PROBABLY EXPLAIN THE PROGRESSIVE IMPREGNATION OF FIBRIN AND/OR LIPIDS WITHIN THE LUMEN



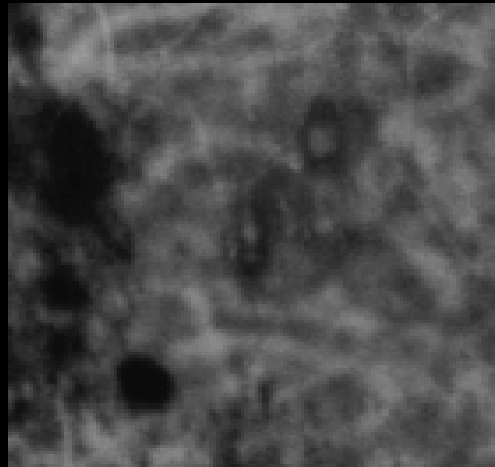




Retina

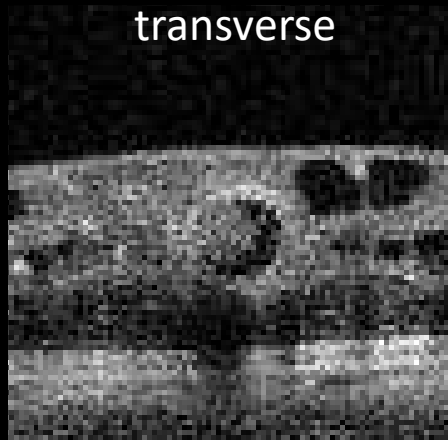
Superficial

Deep

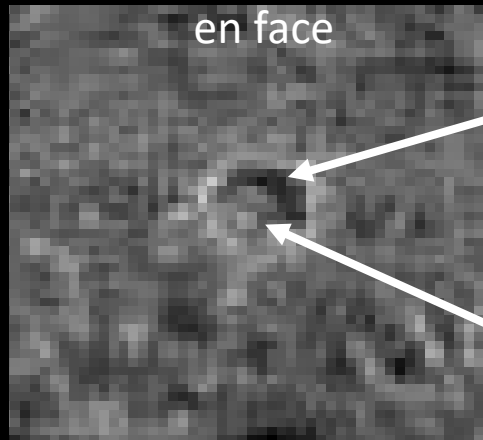


Discrete
diffusion

Hypofluo relative



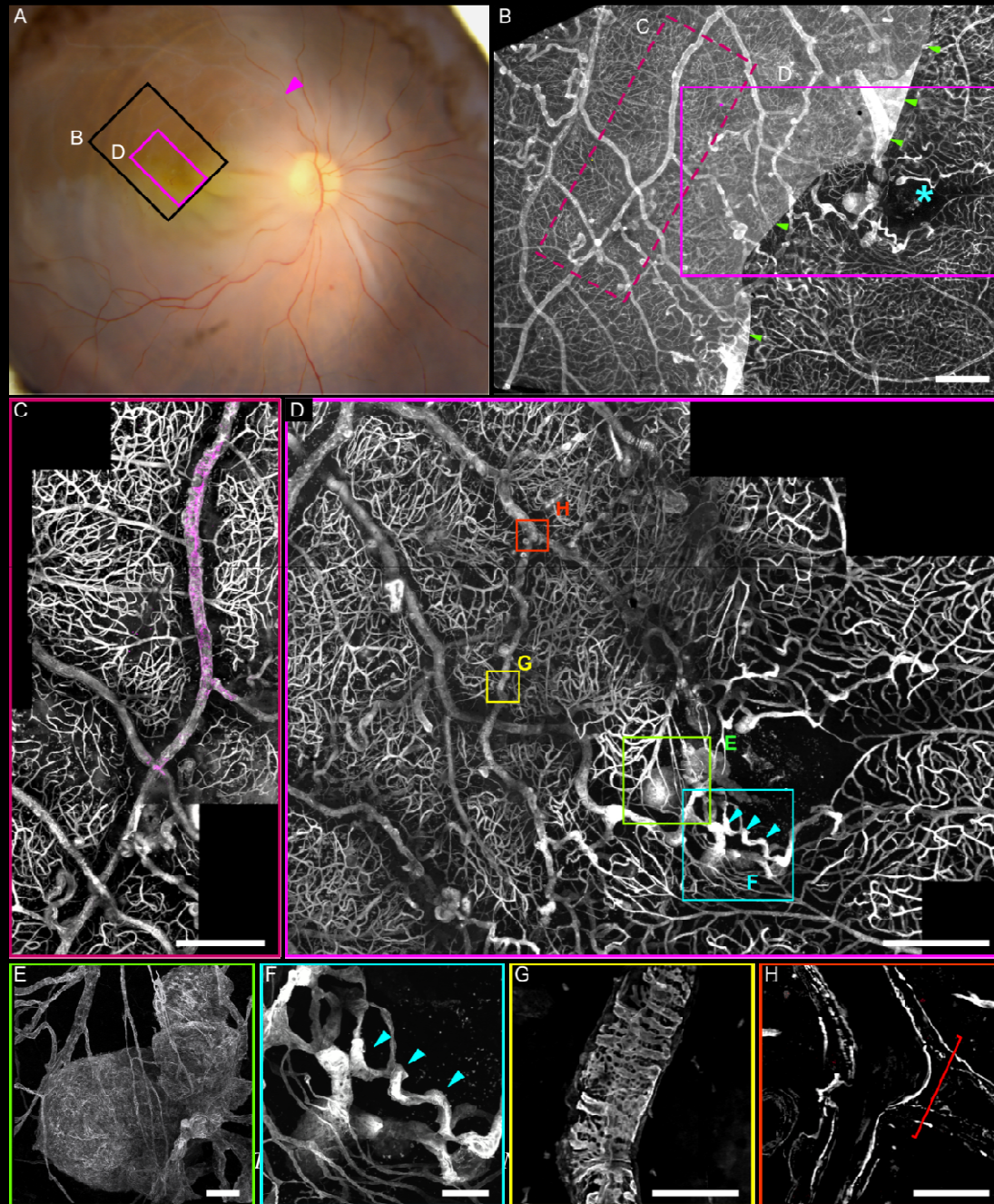
transverse

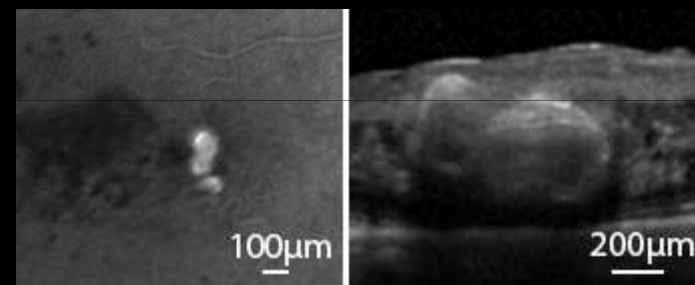
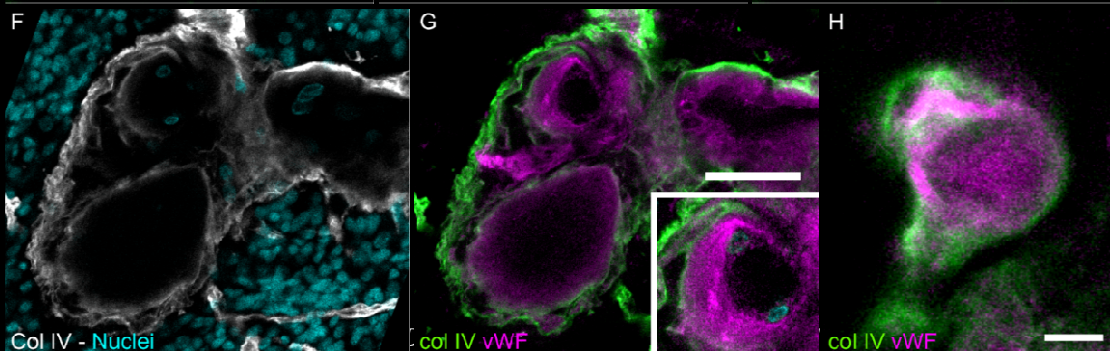
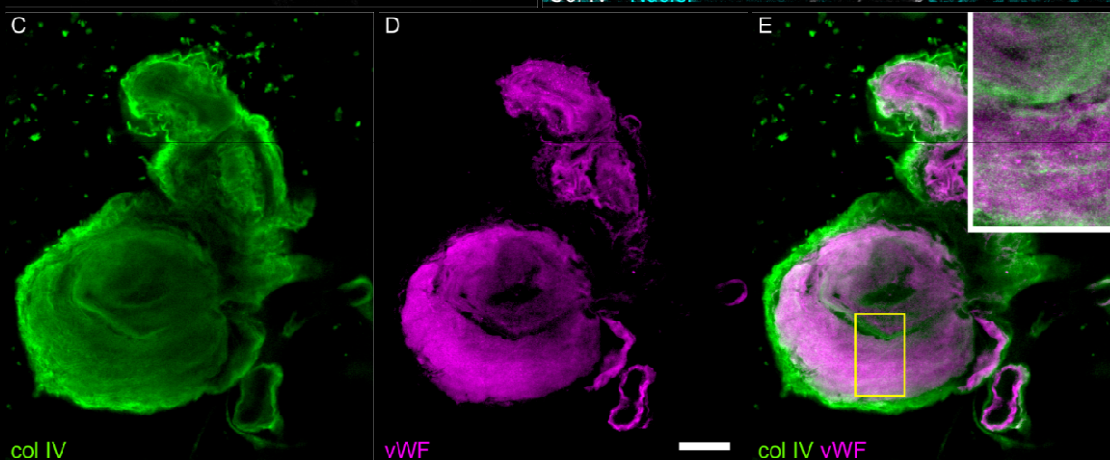
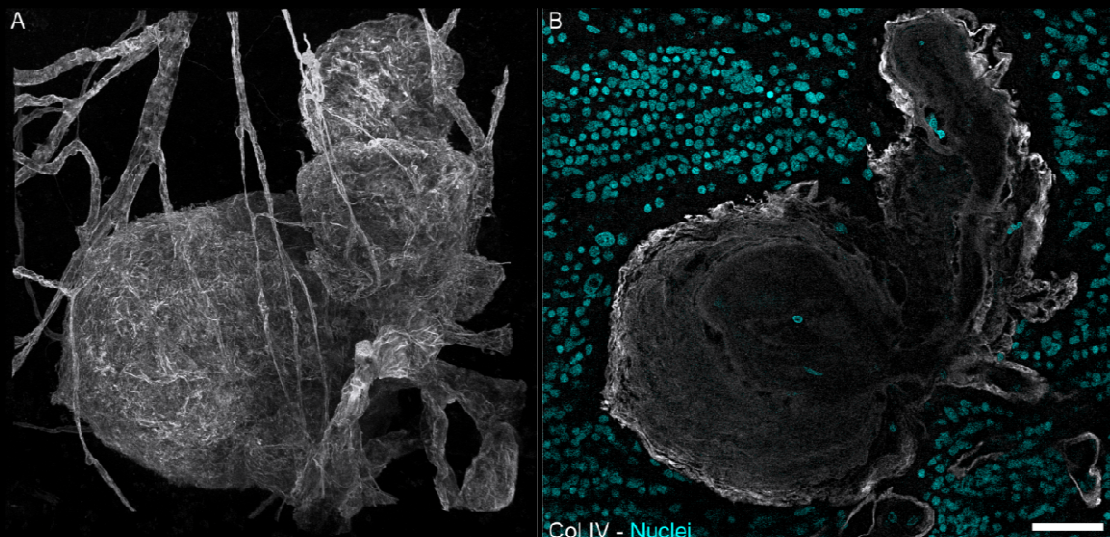


en face

Partie nonréflective:
dépôts lipidoprotéiques

Partie
isoréflective:
Circulation des GR

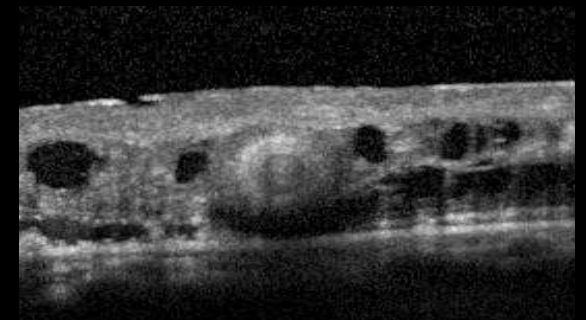


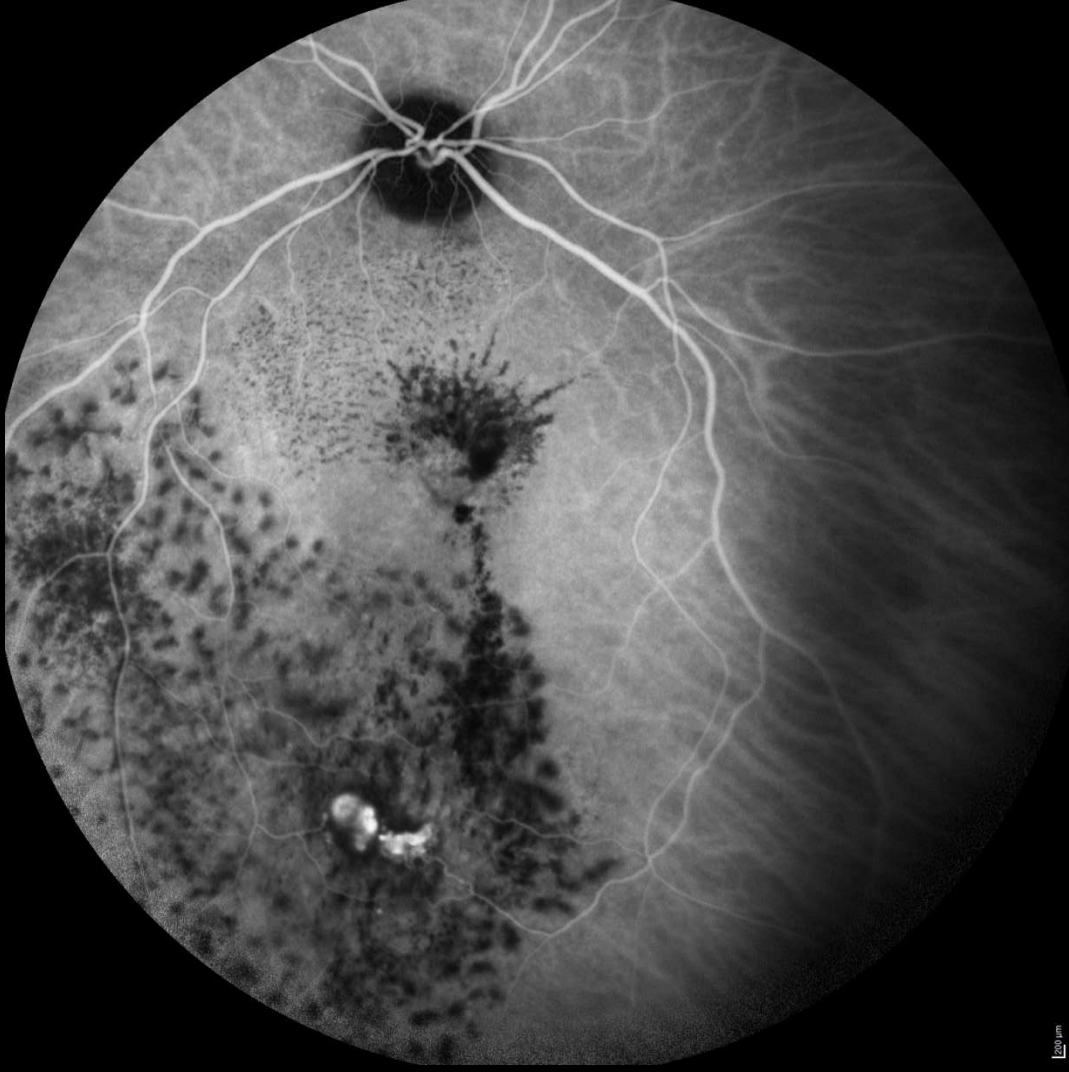
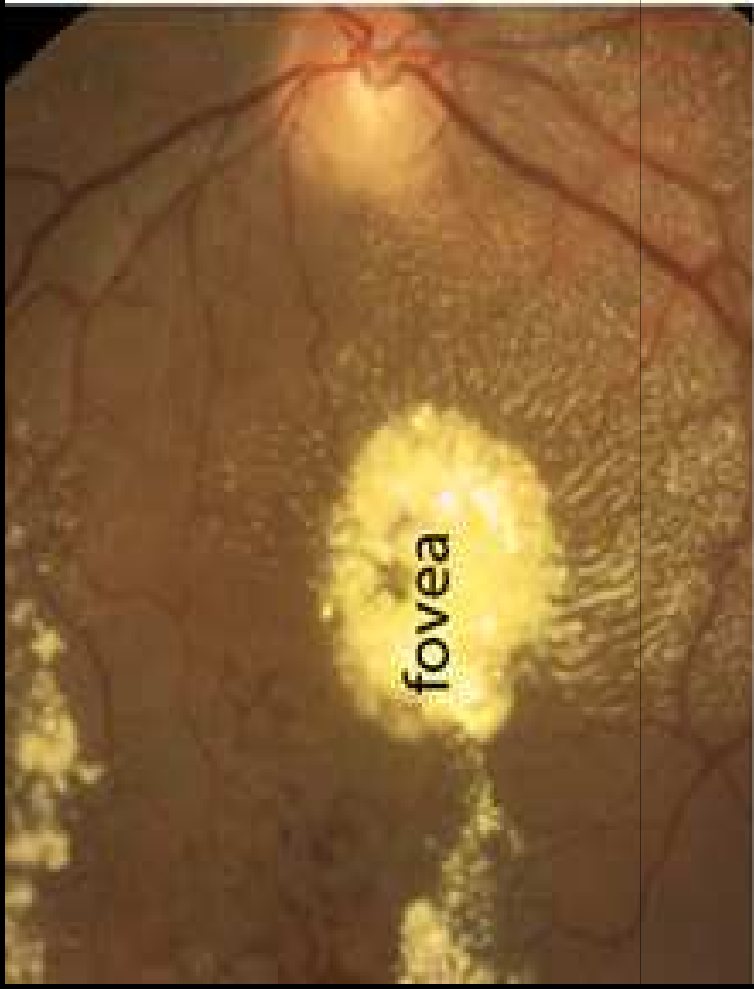


Darche et al, submitted

HISTOLOGY

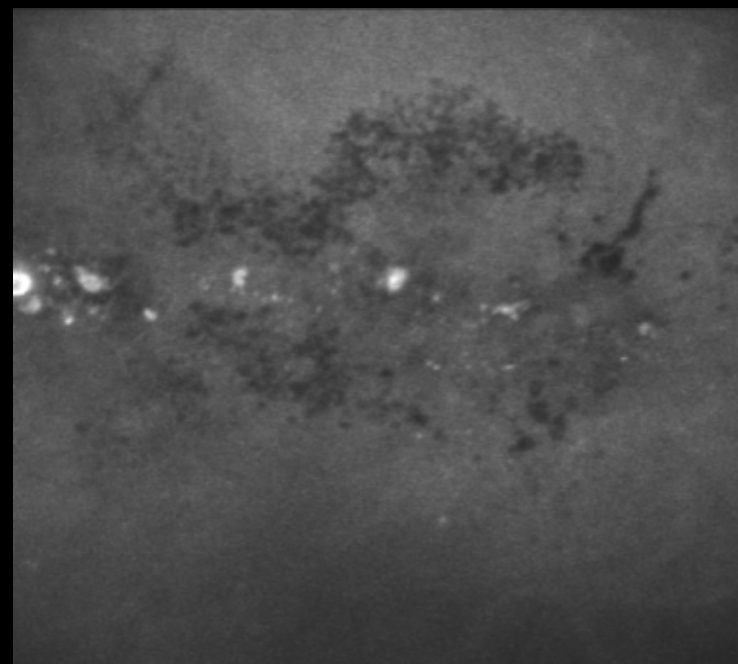
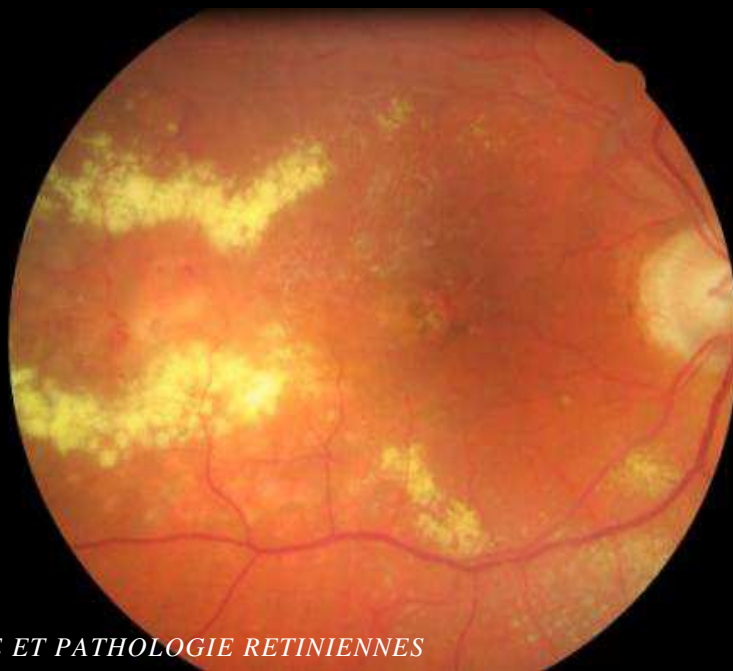
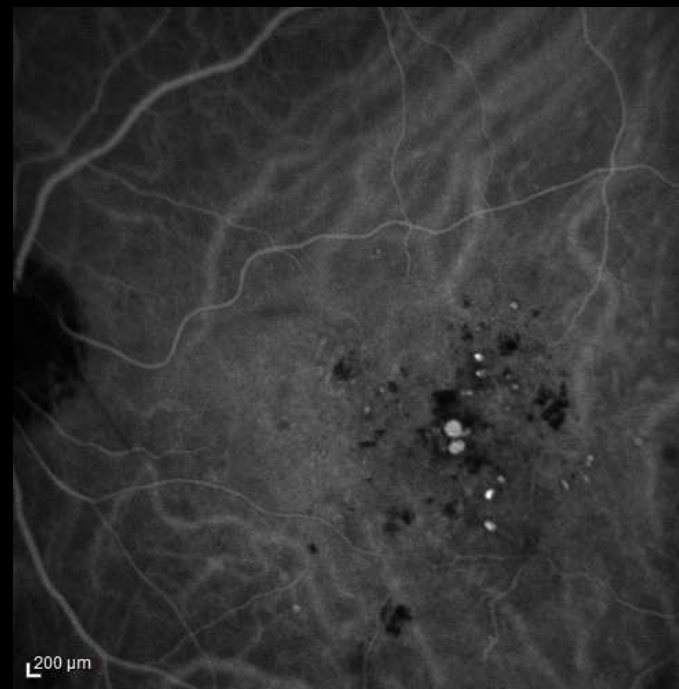
- All macroaneurysms show parietal thickening (which may be even thicker than the lumen itself)
 - Histology suggests that thickening occurs through the deposition of plasma material
 - Asymptomatic macroaneurysms show limited wall thickening
 - Hence, parietal thickening may be directly correlated to the damage to the blood-retinal barrier, and may help to identify photocoagulation targets. This may also explain why hard exsudates are strong indicators of the presence of macroaneurysms.
- Most macroaneurysms show intraluminal material
- → most of the volume of the macroaneurysm is occupied by noncirculating elements, hence flow is reduced, hence its poor visibility by OCTA



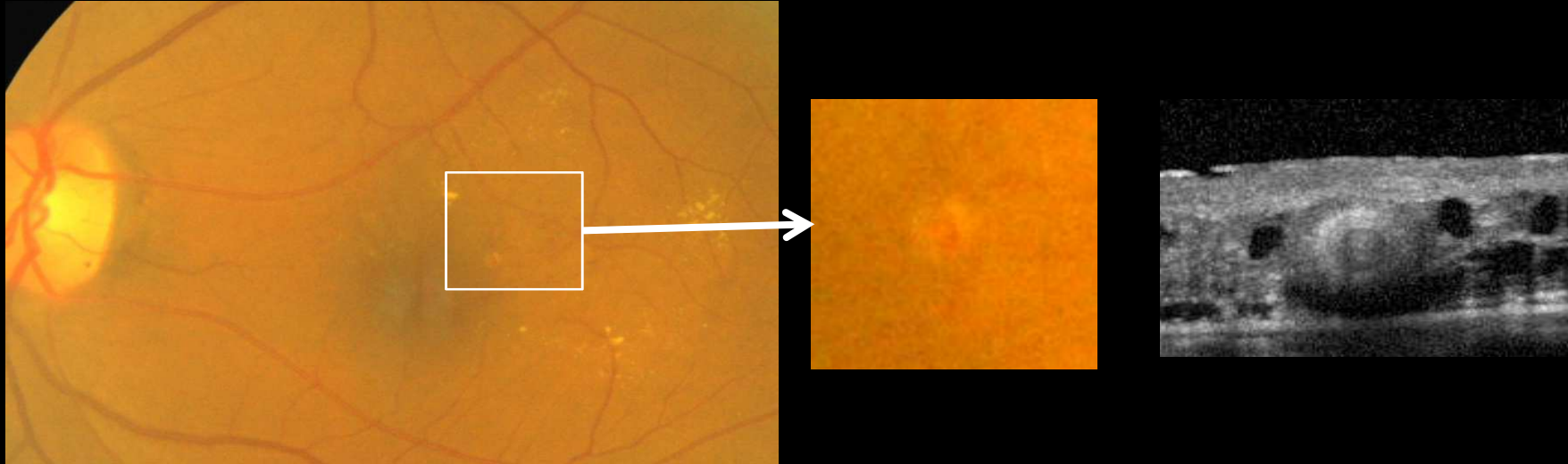


100 μm

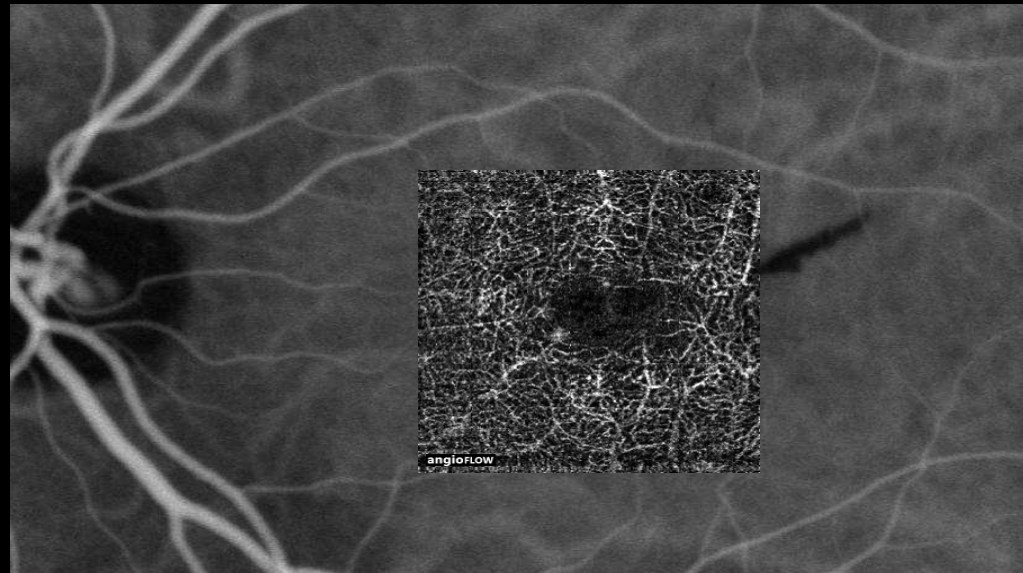
09/10/2013, OD
ICGA 10:59:88-55° ART(34) [HR]



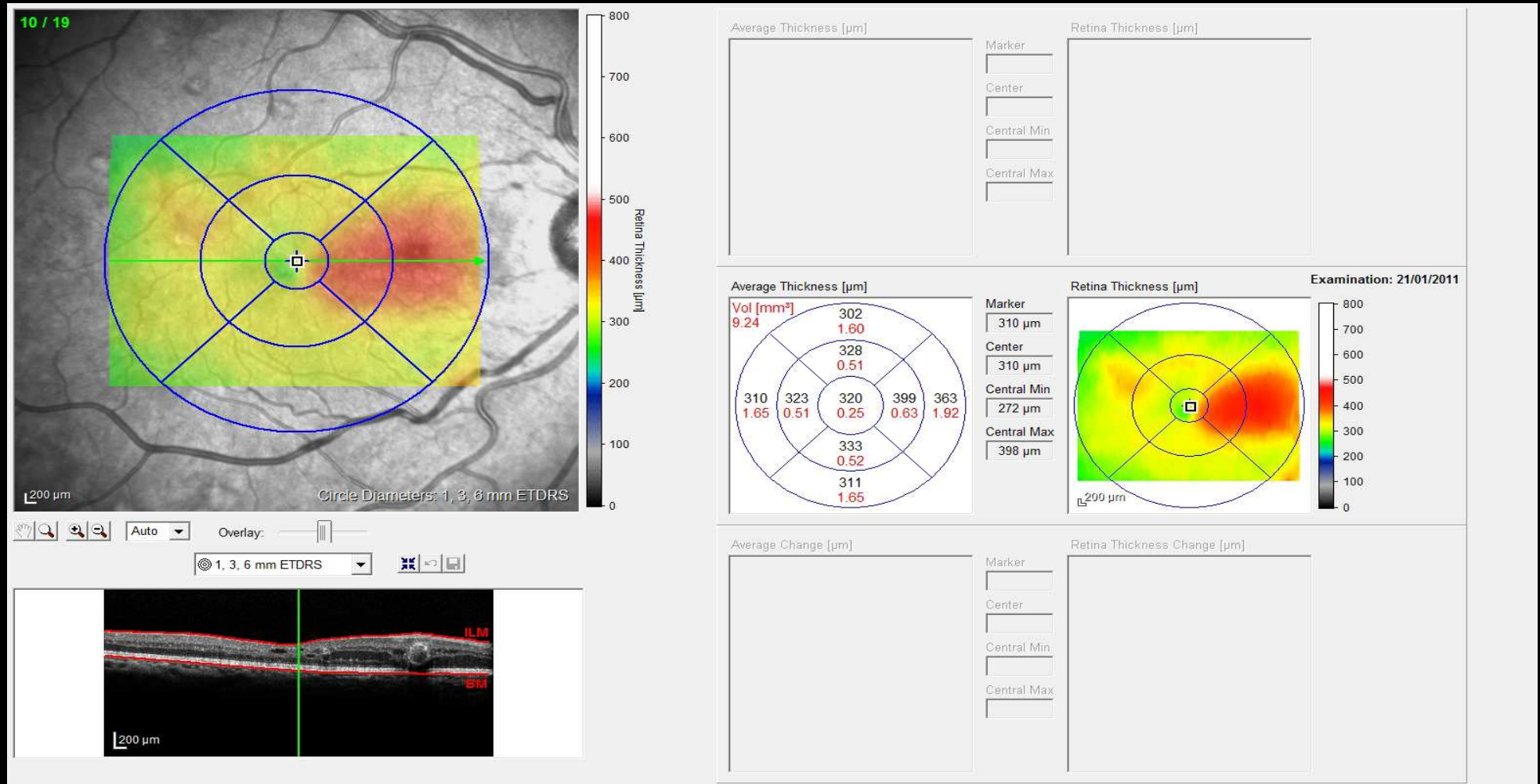
OCT SHOWS PARIETAL THICKENING AND INTRALUMINAL MATERIAL



OCTA is poorly contributive because there is a very slow blood flow



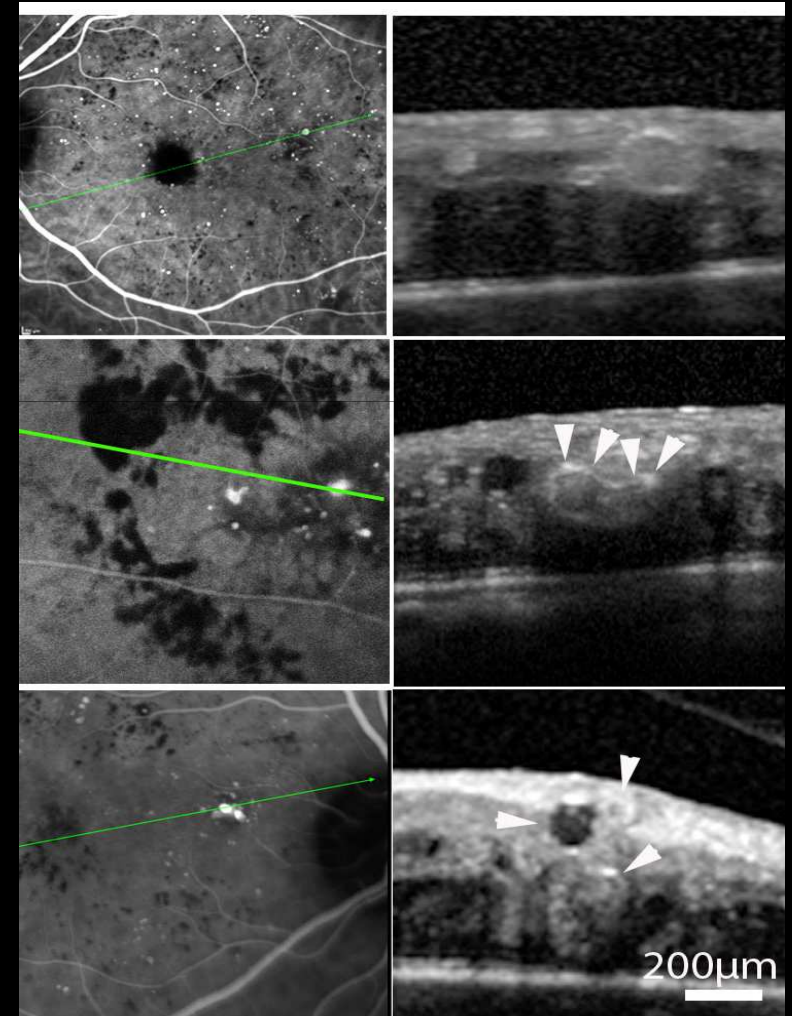
THE MAPPING IS OF INTEREST TO IDENTIFY A TELCAP

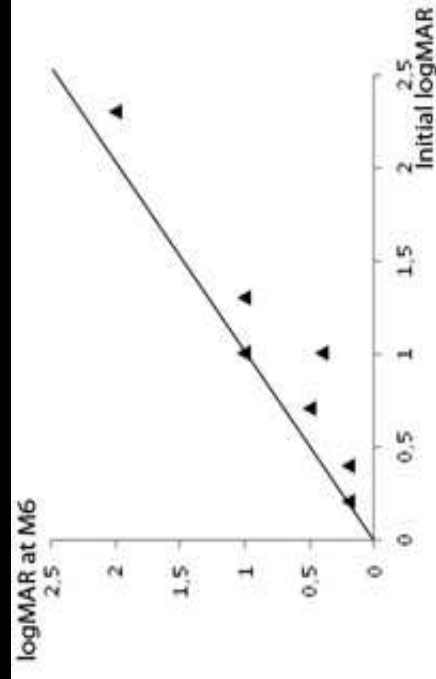
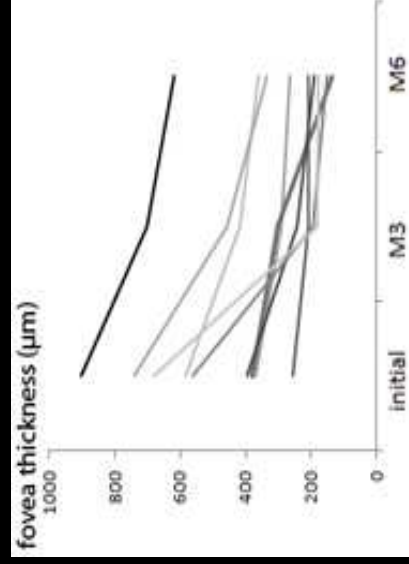
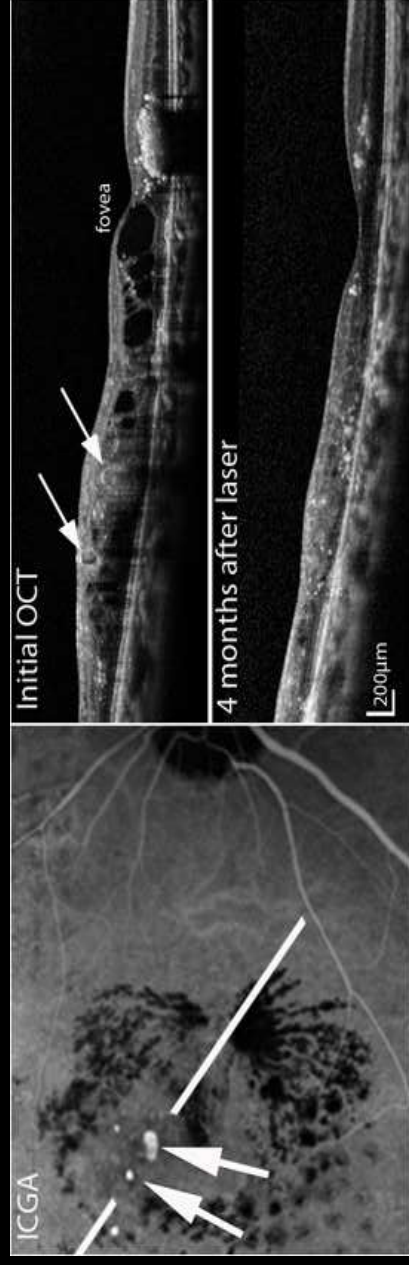
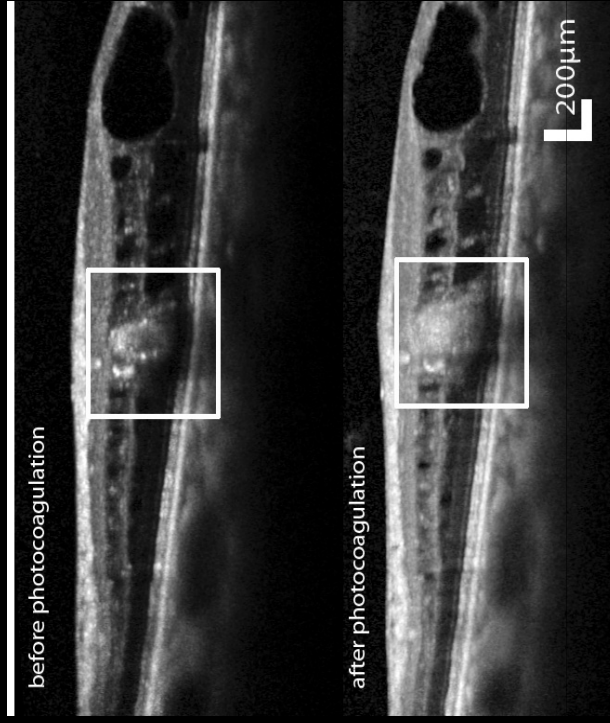


- The incidence of macroaneurysms increases with both the duration and severity of macular edema
 - In RVO of >12 months duration,
 - BRVO: 37% (n=27)
 - CRVO: 24% (n=18)
- > 90% of eyes with hard exsudates*

COMMENT NOMMER CES LÉSIONS?

- Le terme « macroanévrisme » prête à confusion avec les macroanévrismes artériels
- Ces lésions peuvent se présenter sous la forme d'agrégats coloré par l'ICG
- Il s'agit histologiquement de télangiectasies
- Le terme « *telangiectatic capillaries* » (TelCaps) paraît plus adapté





PLACE DU LASER GUIDÉ (NAVILAS®) ?

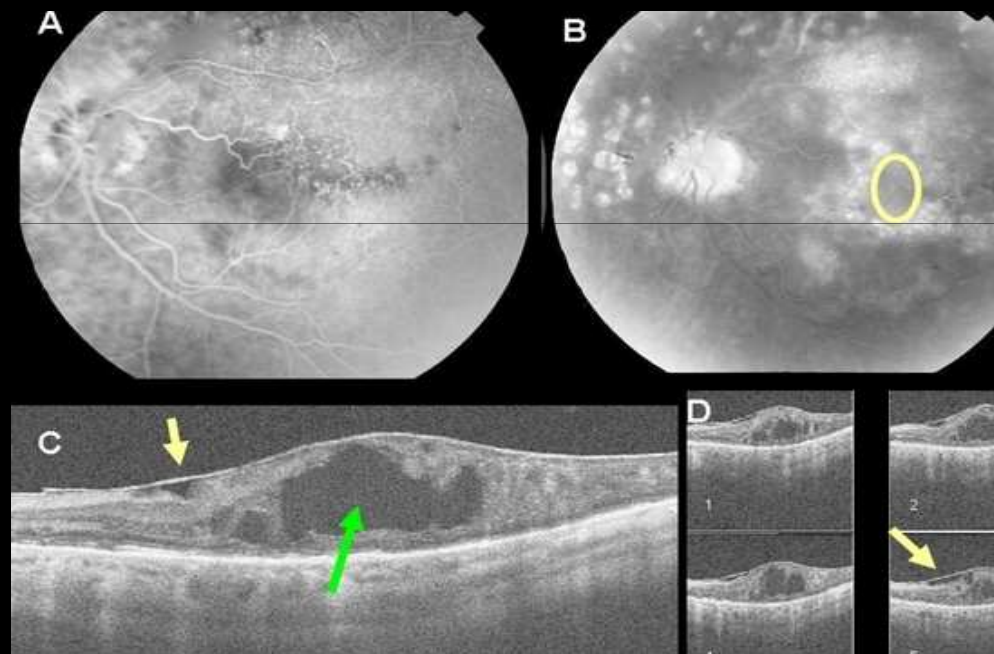
- Localisation plus facile (superposition ICG), traitement plus exhaustif



- Mais contrôle de la focalisation ?
 - Plutôt en l'absence d'OM contrairement au traitement « manuel » ?

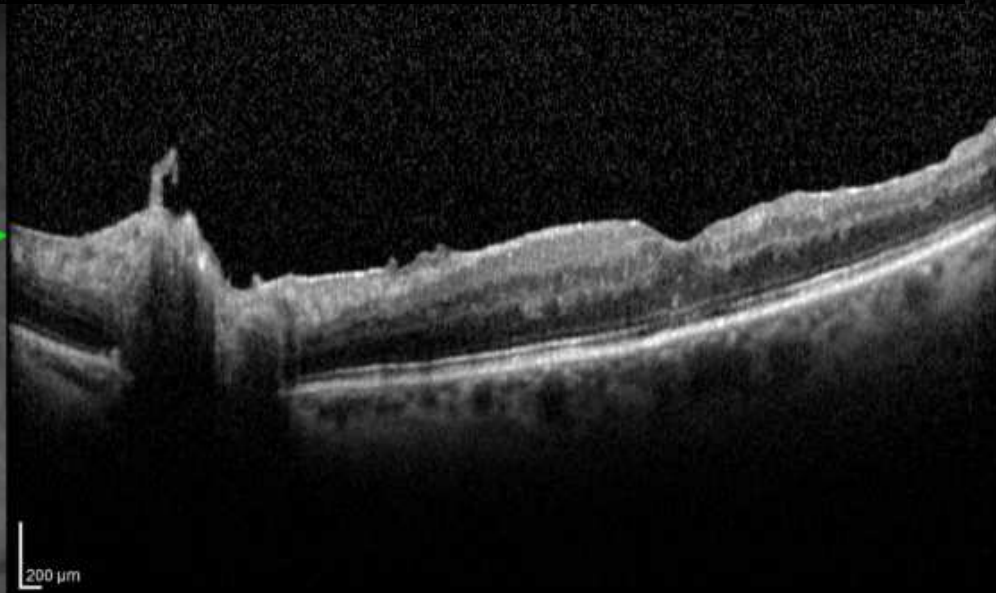
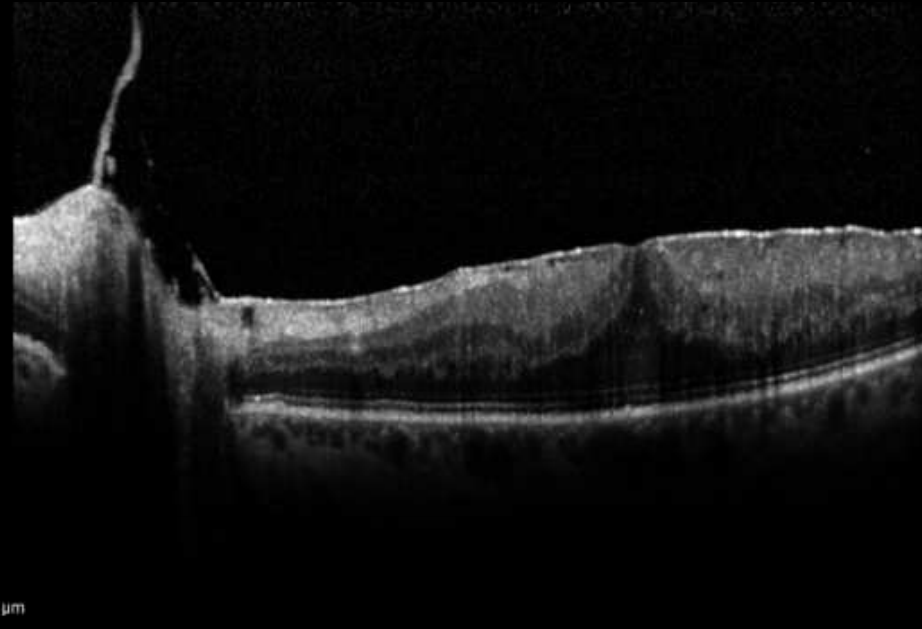
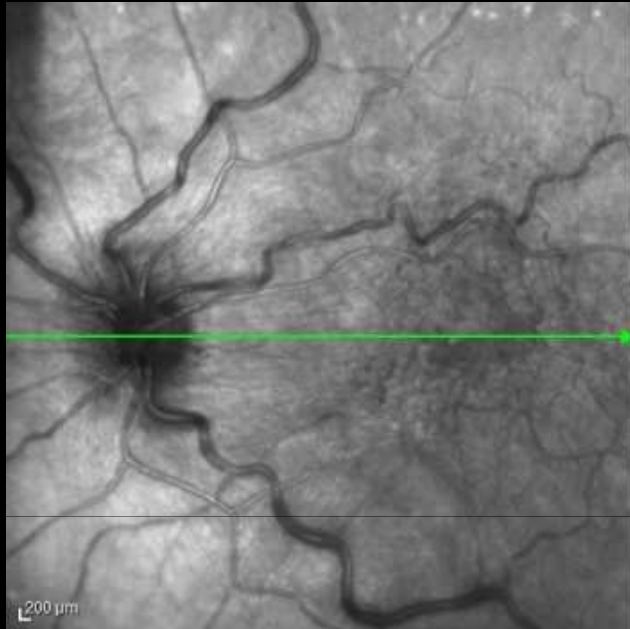
• MEMBRANE ÉPIRÉTINIENNE ET OVR

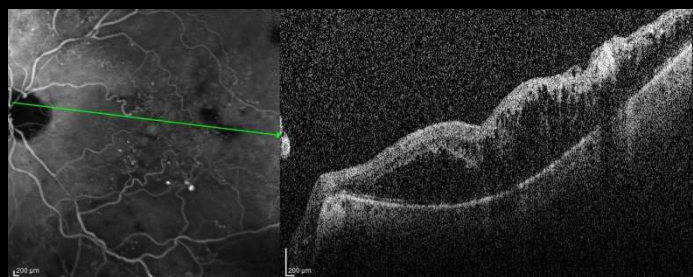
- Facteur de récurrence après IVT
- Parfois difficile à diagnostiquer en raison des autres remaniements rétiens – intérêt de l'OCT



- Plus fréquente avec les anti-VEGF? Avec le laser?

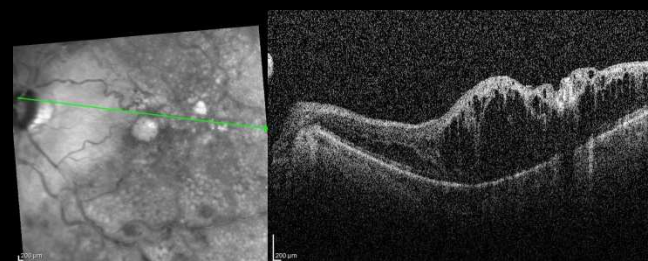
Autres complications: Développement d'une membrane épirétinienne





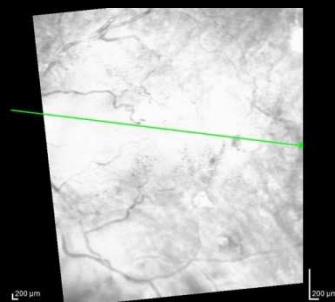
08/02/2019, OS
ICGA/OCT 12-10 14 30° ART [HS] Q. 28

HEIDELBERG
engineering



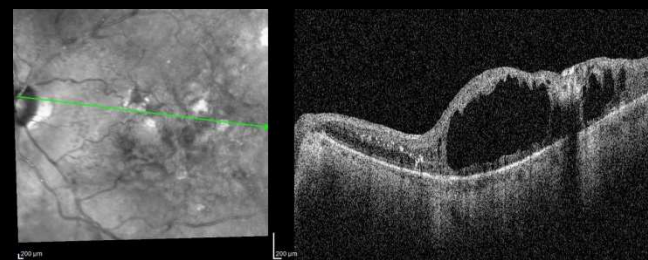
14/06/2019, OS
IR&OCT 30° ART [HS] Q. 24

HEIDELBERG
engineering



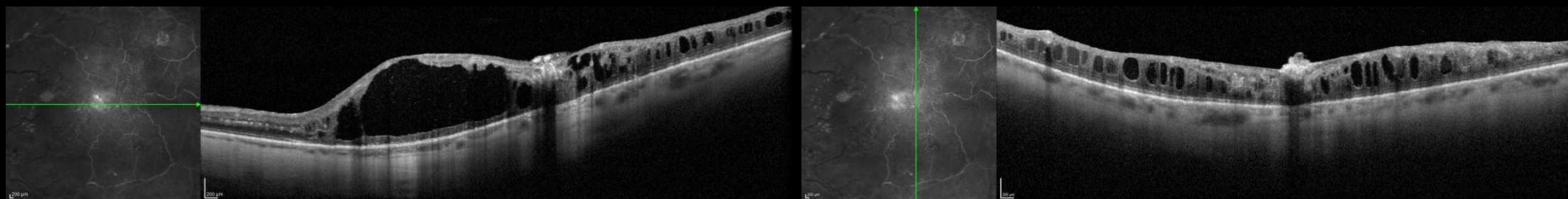
13/09/2019, OS
IR&OCT 30° ART [HS] Q. 28

HEIDELBERG
engineering



10/12/2021, OS
IR&OCT 30° ART [HS] Q. 28

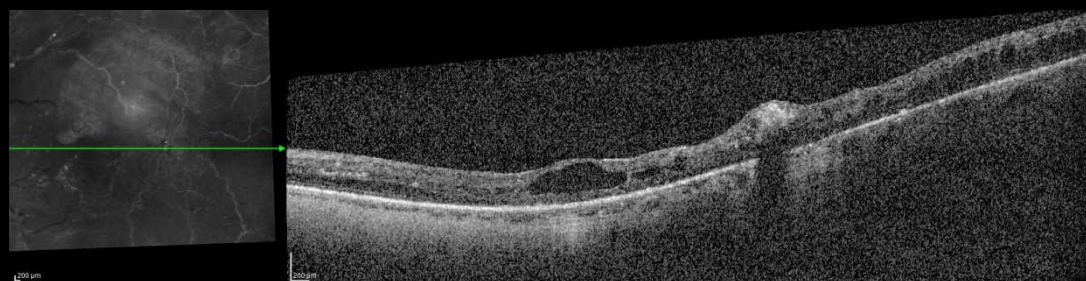
HEIDELBERG
engineering



10/12/2021, OS
BR&OCT 30° ART [HR] ART(47) Q: 36

10/12/2021, OS
HEIDELBERG
BR&OCT 30° ART [HR] ART(16) Q: 35

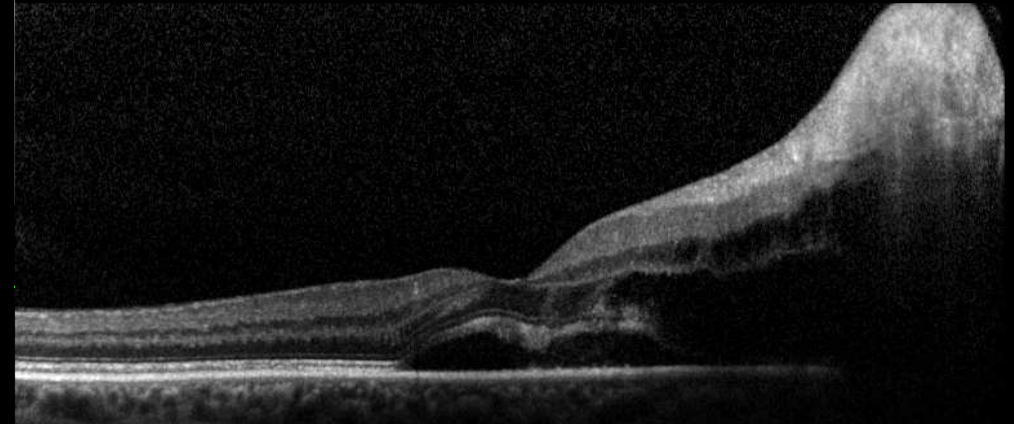
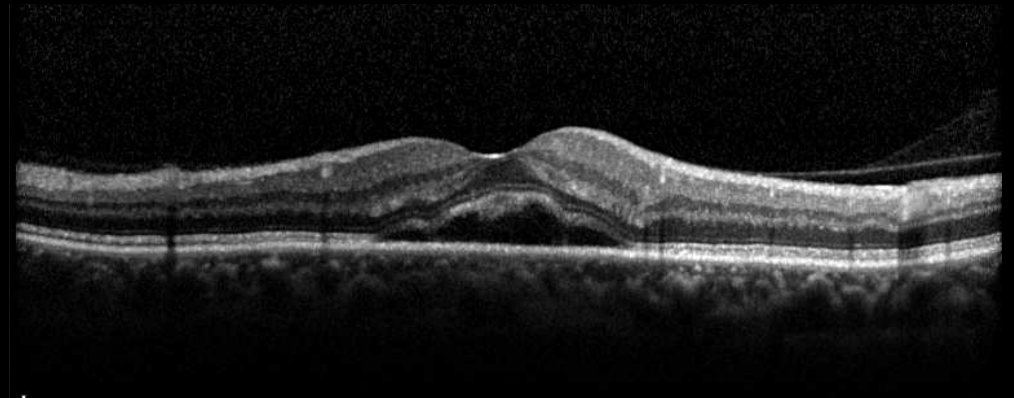
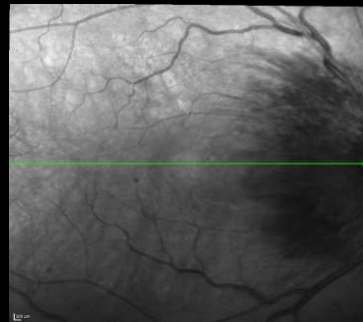
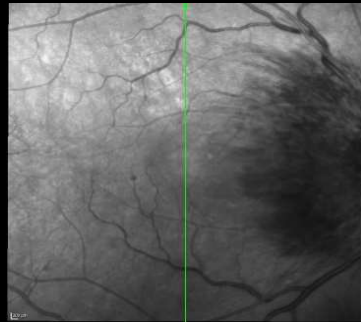
HEIDELBERG
engineering



11/02/2022, OS
BR&OCT 30° ART [HR] ART(3) Q: 21

HEIDELBERG
engineering

DIFFUSION LATÉRALE D'UN ŒDÈME PAPILLAIRE



Cas clinique du Dr Vignal

FACTEURS AGGRAVANTS DE L'OM



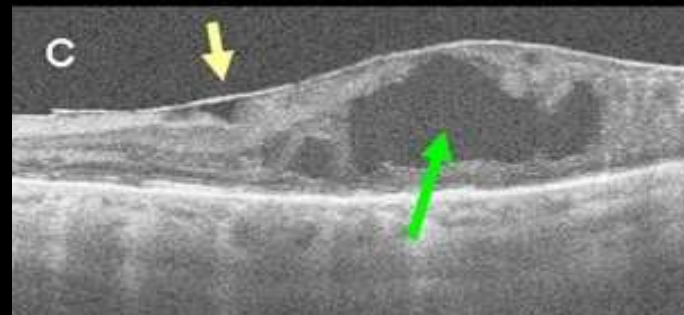
prostaglandines



HTA



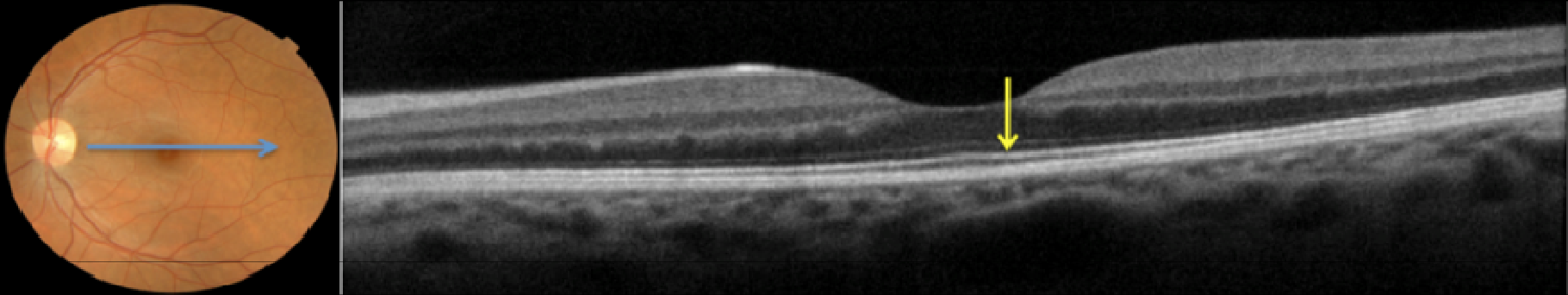
macroanévrysme



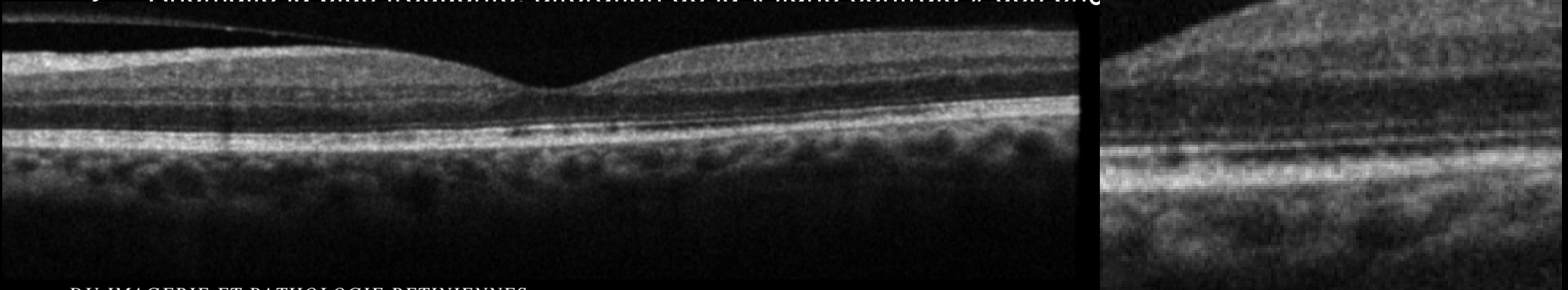
MEM

RÉSULTATS MORPHOLOGIQUES (2): ANALYSE QUALITATIVE

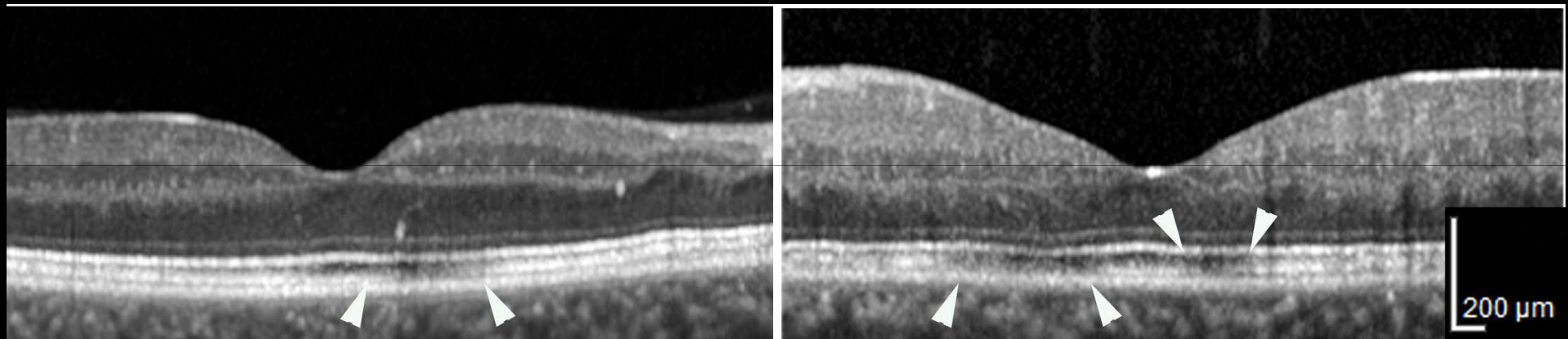
- Aspect normal: 27 % (tous AV $\geq 10/10$)



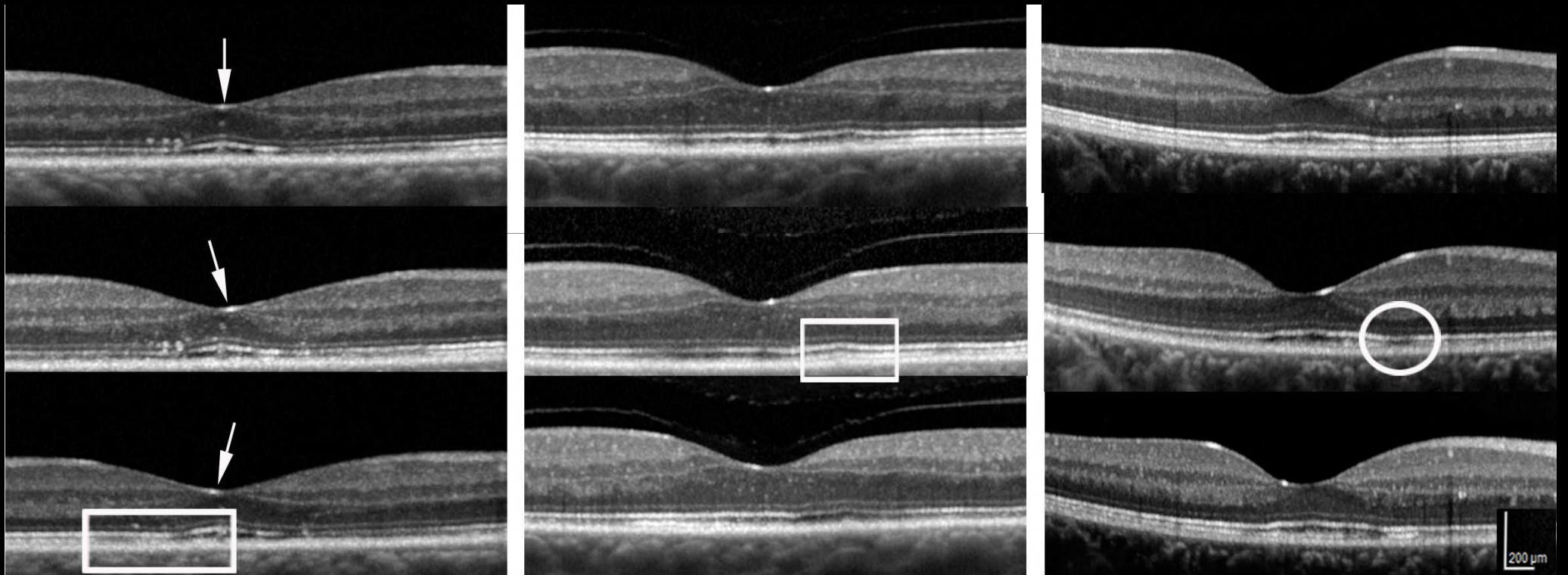
- Anomalie la plus fréquente: altération de la « ligne centrale » des pnc

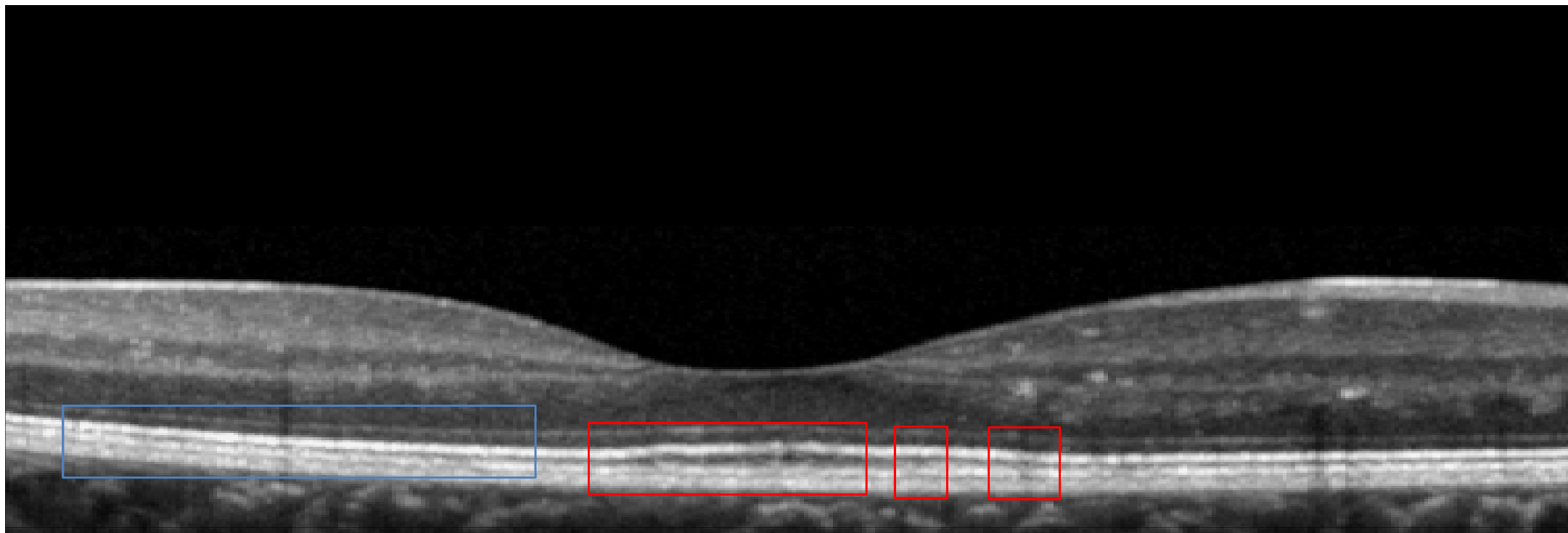


Séquelle d'OM: les segments externes sont très souvent désalignés

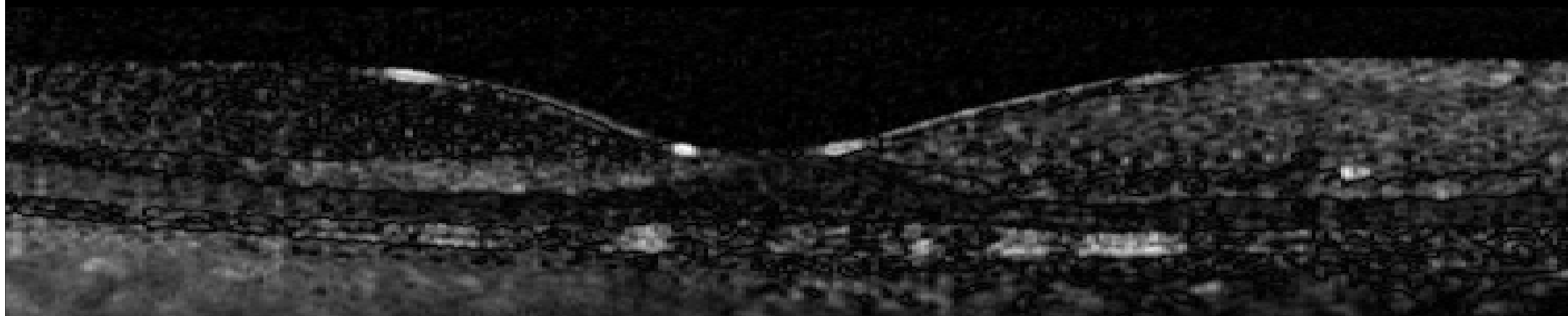


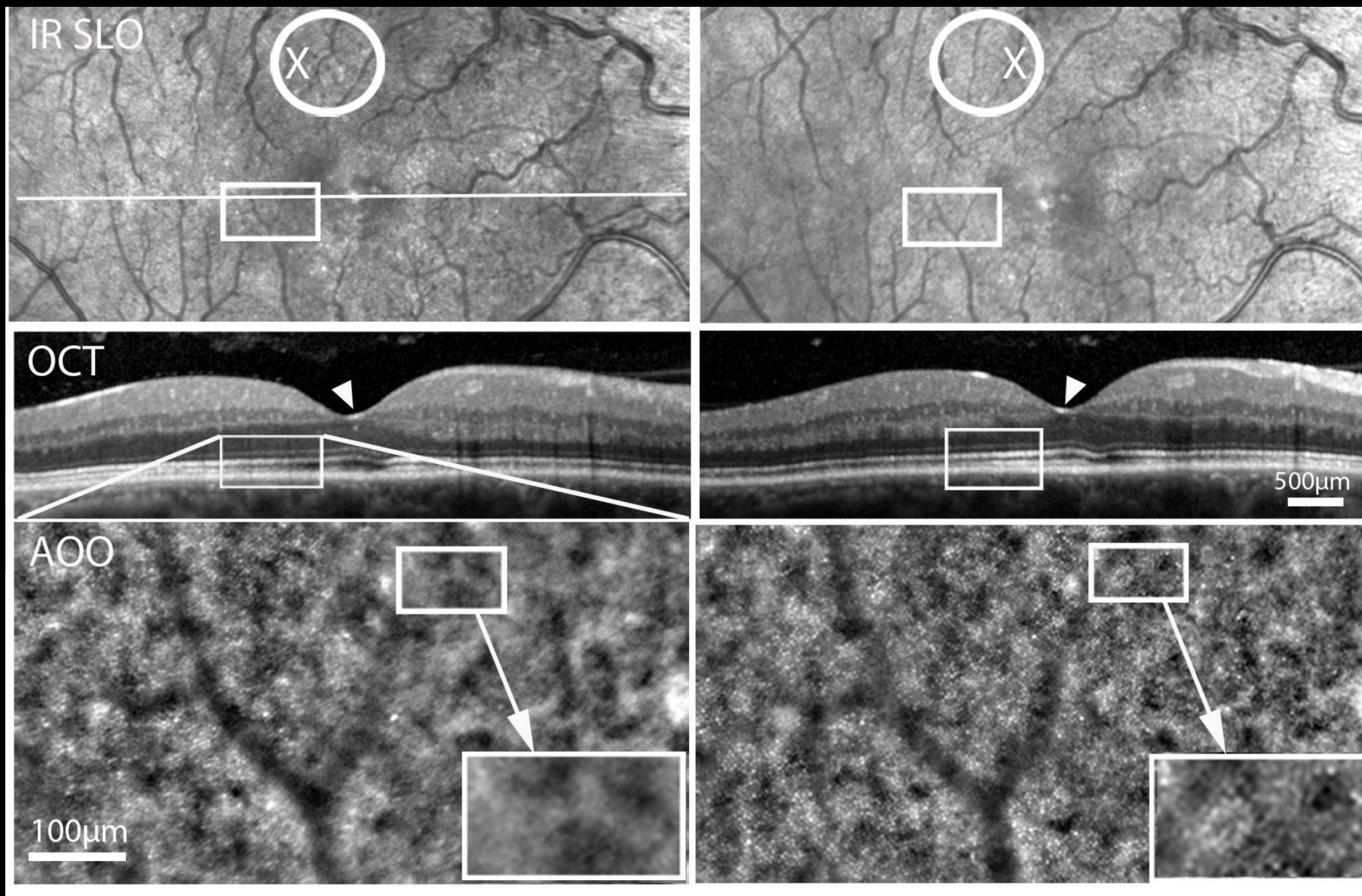
OCT multiangle après œdème maculaire





STD 2-3



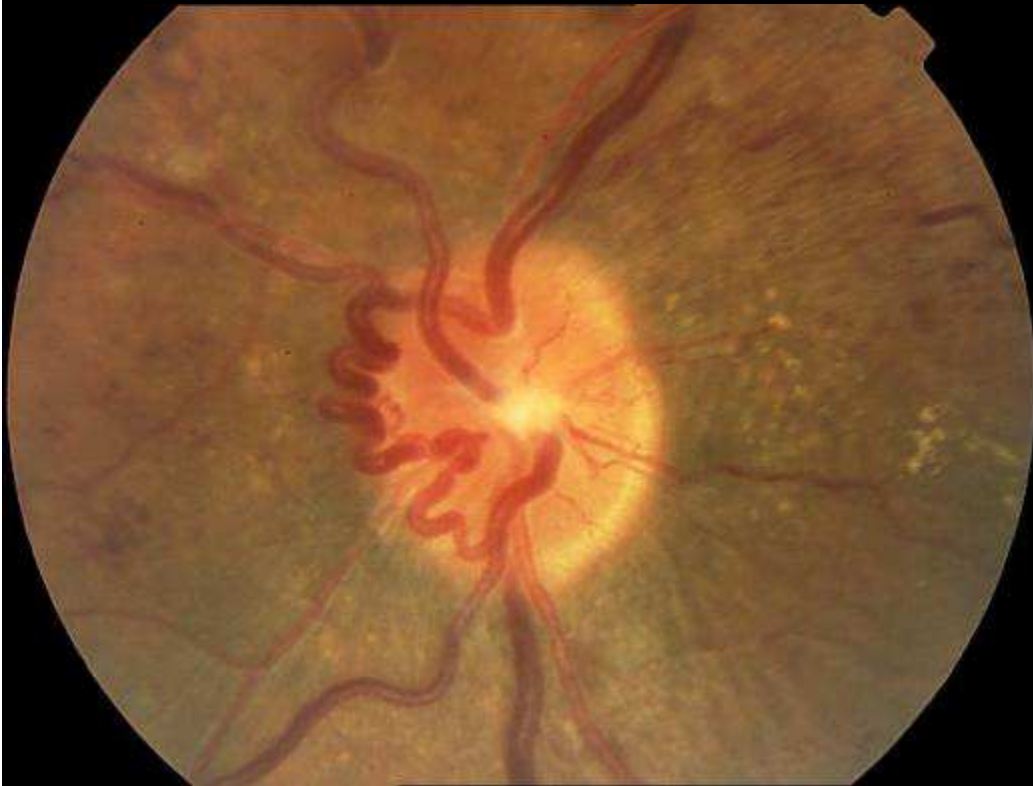


GUÉRISON

➤ Reperfusion de la VCR

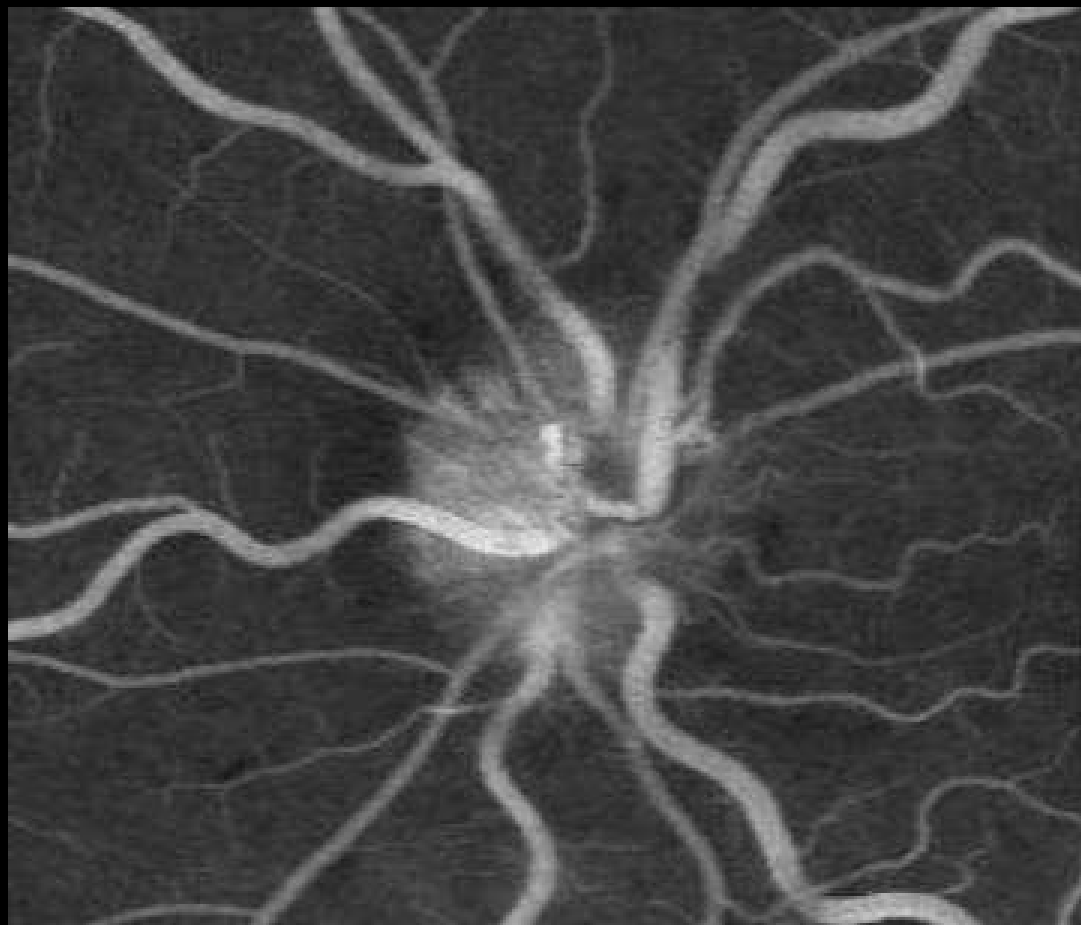
Et/ou

➤ Circulation collatérale

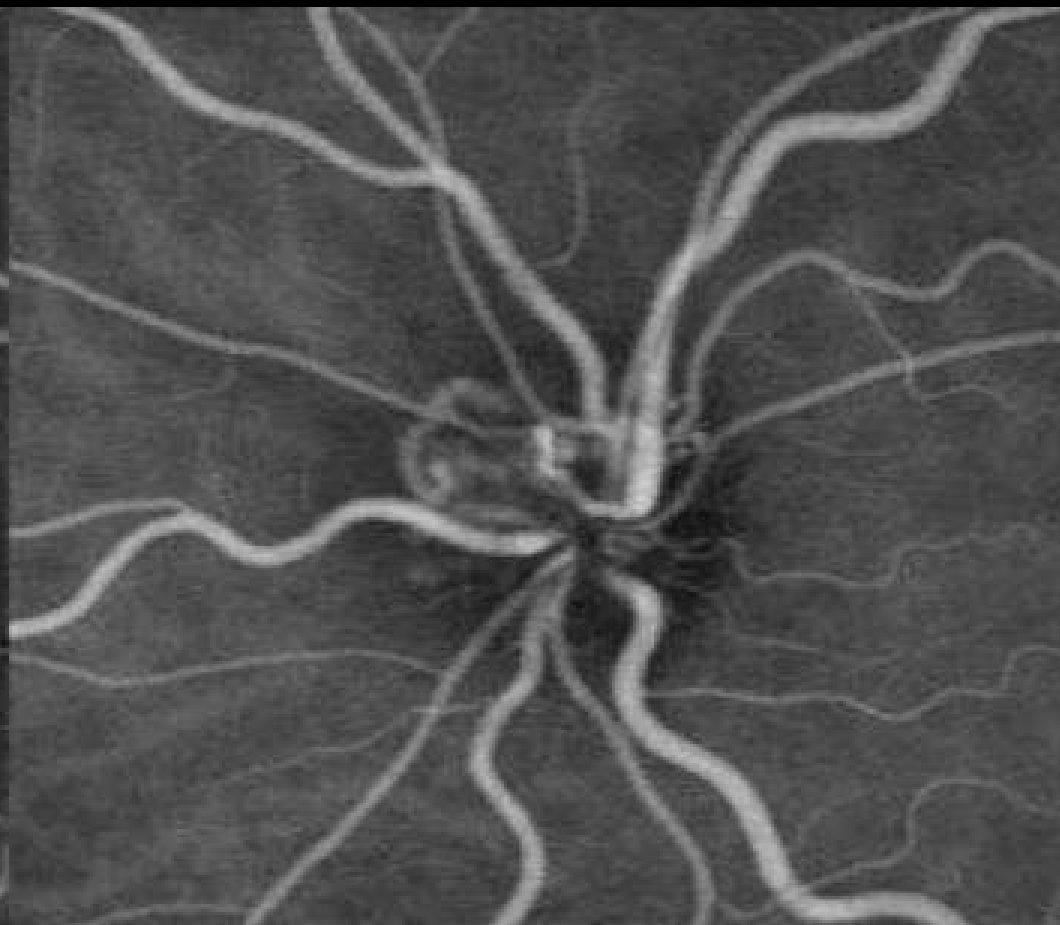




fluor



ICG





200 μ m

04/12/2012, OD

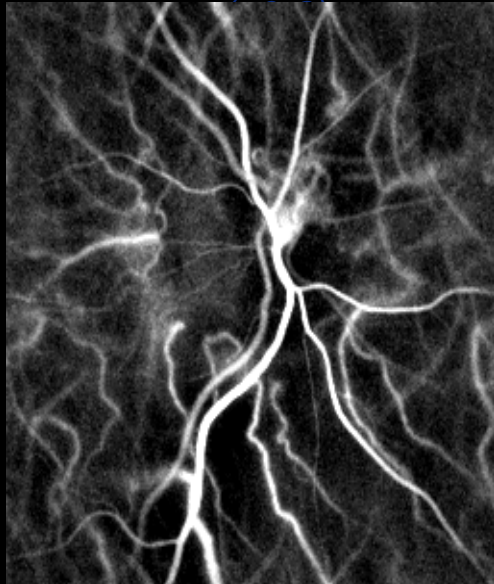
IRAF 55° ART(10) 91732.1 [HR]

HEIDELBERG
ENGINEERING

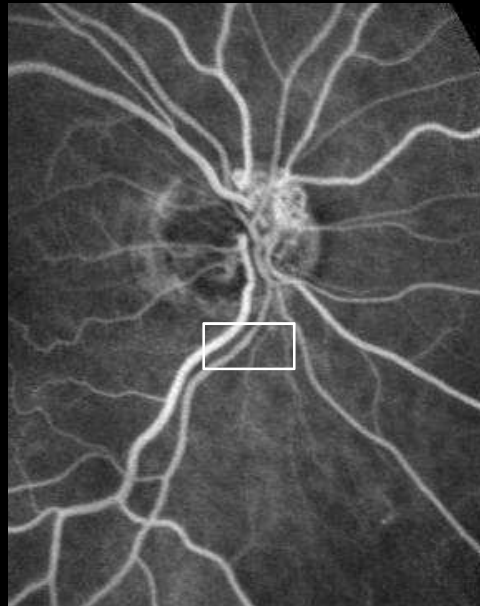
191108
TAM0140
OD

- $IR = 0.96$
- $IP = 2.17$
 $IR = -1.11$
- $IP = -0.54$

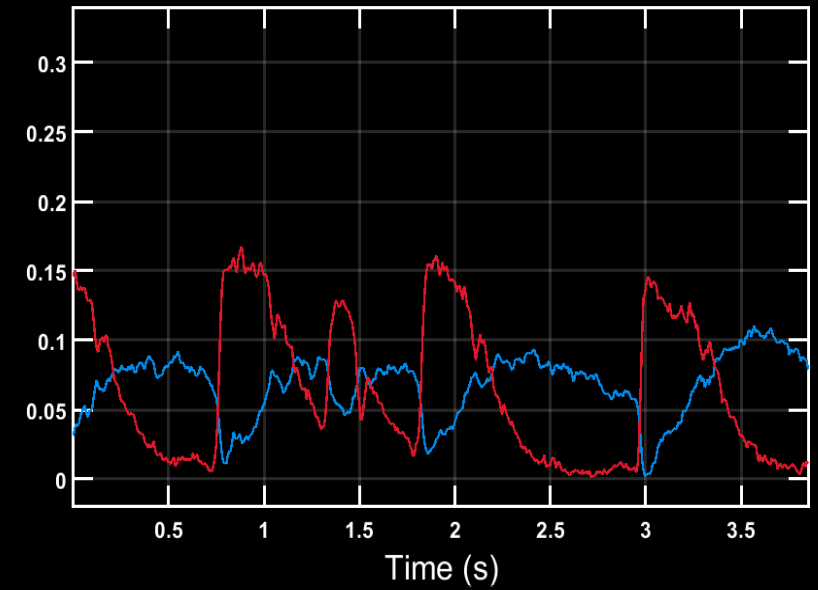
Power Doppler movie



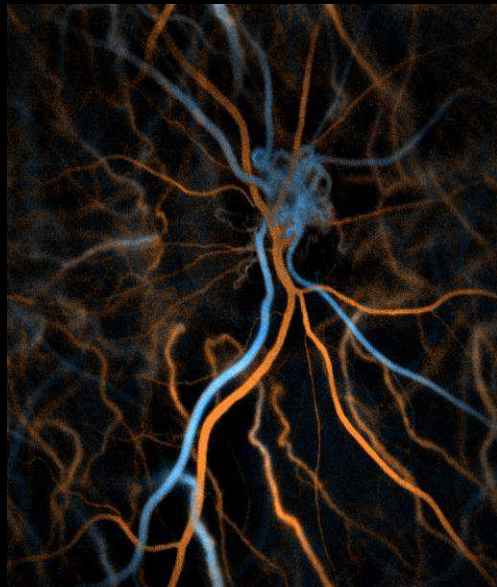
Collatérale
Power Doppler, 2-37 kHz



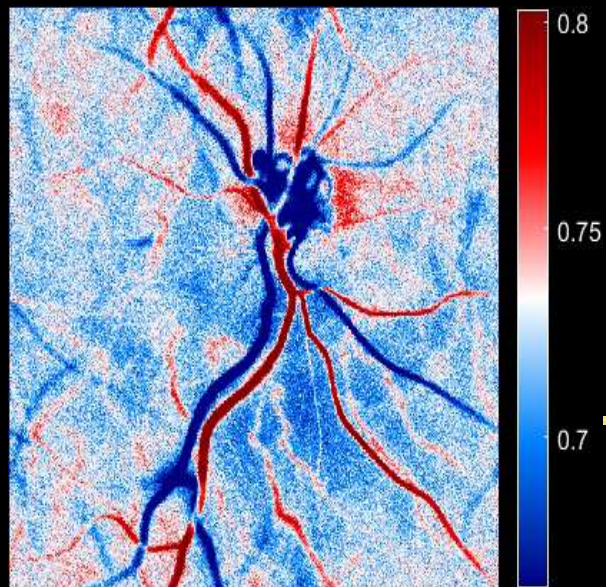
Power Doppler variations



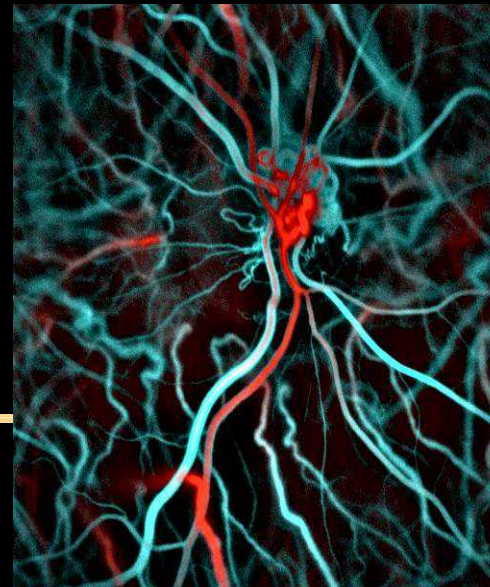
Systole / Diastole



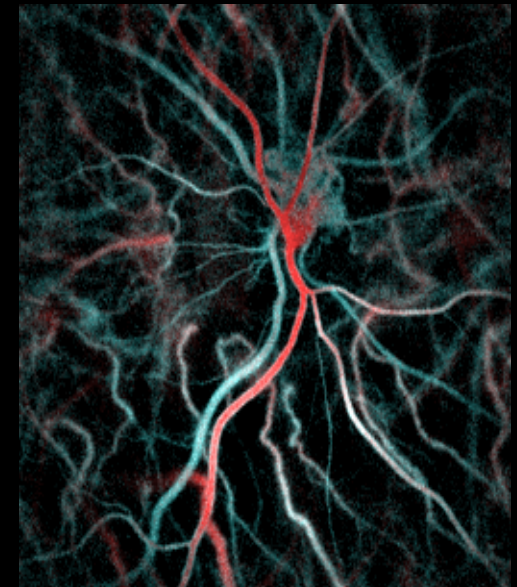
Resistivity map



Low flow / high flow



Composite movie



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

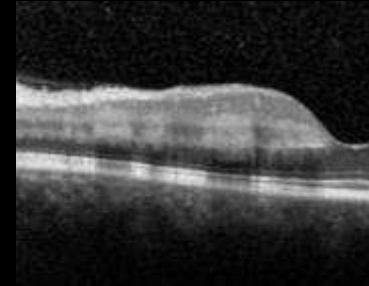
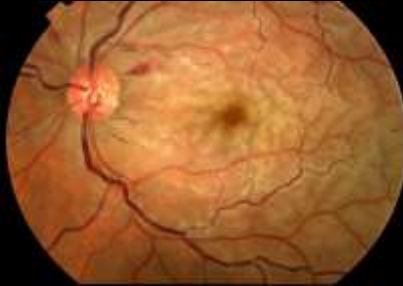
- Type A: Occlusion brutale révélée par bas débit aigu, suivi d'un oedème papillaire
 - Résolution habituelle en quelques semaines
 - Risque essentiel: atrophie de la rétine intermédiaire
 - Possibilité de formes graves si bas débit sévère

- 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

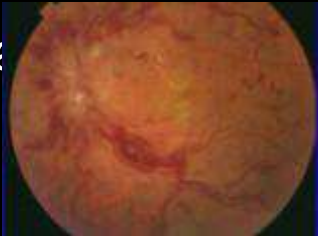
- Remodelage micro vasculaire proportionnel à la durée de l'OVR
 - Collatéralisation
 - Non perfusion ($\pm$ Angiogenèse)
 - Macro anévrysmes

OVR?

➤ Type A précoce: ischémie aiguë



➤ Type A tardif: capillaire



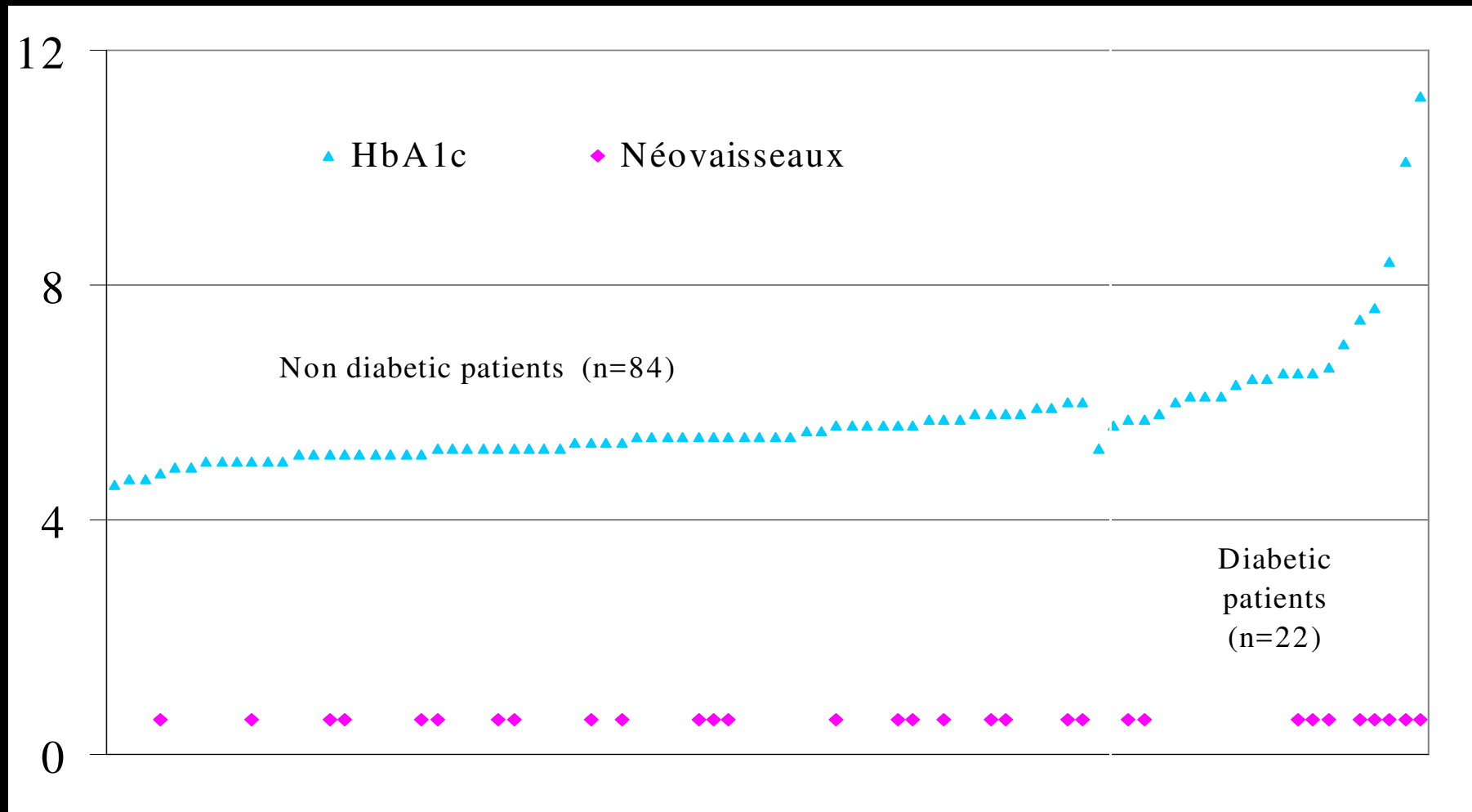
➤ Type B précoce: dilatation veineuse

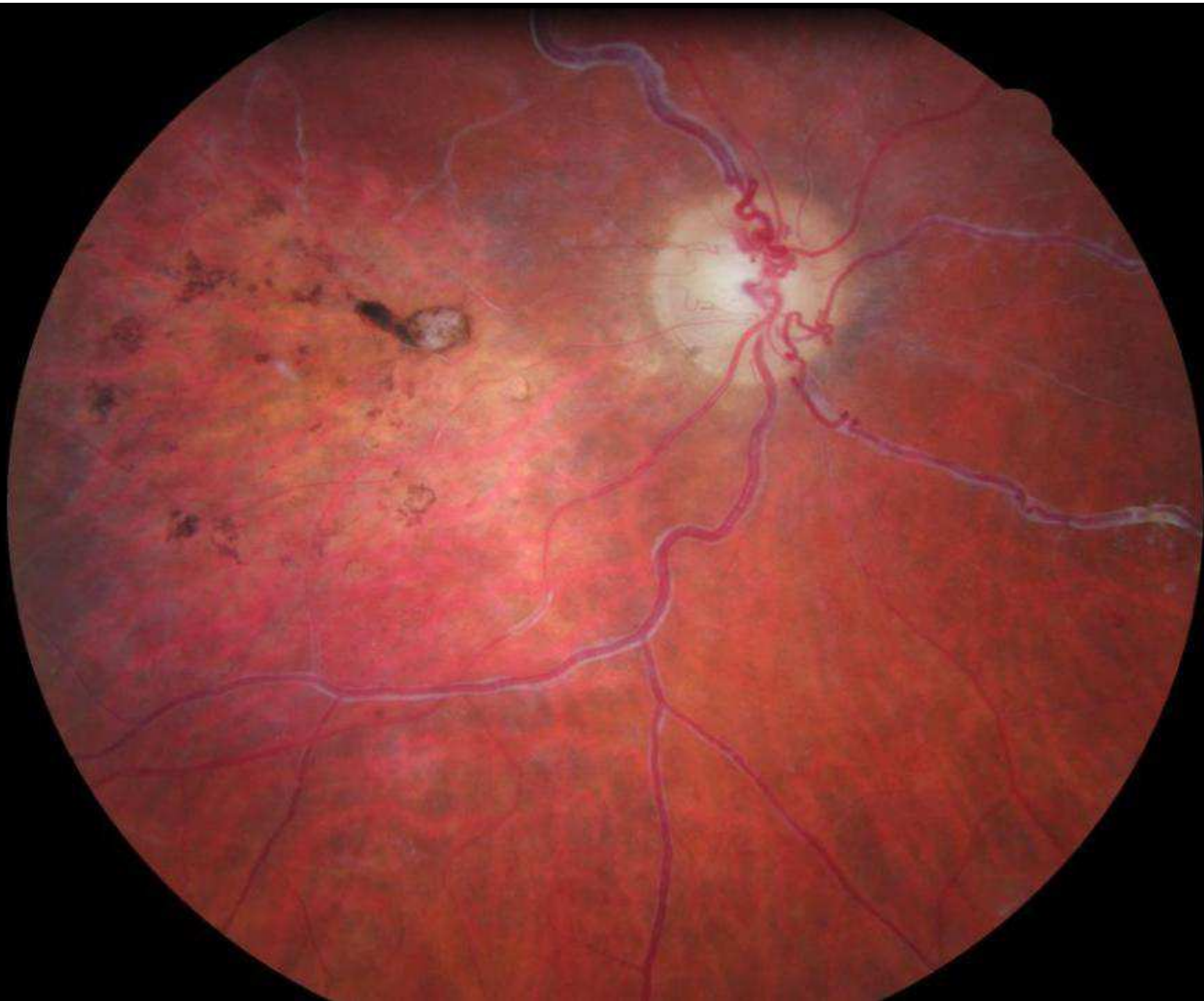


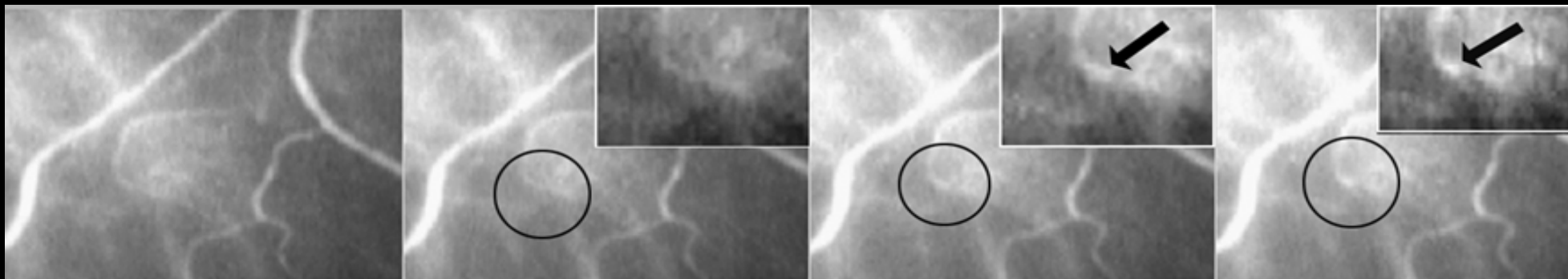
➤ Type B tardif: œdème



HbA1c AND NEW VESSELS







systole

diastole

