La Lettre de la SFEROV

Bulletin de liaison n°24 . novembre 2006

Chronique du Président

Les flons-flons se sont tus, les absents ont eu tort..
Les IXemes journées d'actualité de la SFEROV se sont terminées avec pour tous, acteurs et spectateurs, un sentiment légitime de satisfaction. Chacun a apporté sa contribution pour faire de ce congrès l'espace d'échange intellectuel cher à l'esprit SFEROV. En de nombreuses occasions, les discussions d'après conférences ont été d'un niveau supérieur aux conférences elles mêmes ce qui est l'expression d'interventions réussies faisant oublier l'intervenant au profit du sujet. L'implication amicale du service d'ophtalmologie de l'ENVT a été, comme lors de chaque session, un élément déterminant de la réussite de ces journées. Nos partenaires commerciaux ont assuré un soutien logistique et financier qu'il convient de saluer encore.
L'édition 2008 est déjà proche ; la SFEROV sera co-organisateur du congrès européen qui doit se tenir à Versailles. En attendant, et pour rester dans une dynamique de communication, le Bureau a décidé de la tenue de manifestations décentralisées à organisation allégée ; la première devrait se tenir à l'automne 2007 en région PACA. D'ici là, le Bureau a décidé de s'impliquer mieux pour rester proche des adhérents au travers d'une communication régulière sur les actualités de notre spécialité, par la voie du bulletin de liaison envoyé par courriel à fréquence bimestrielle. Par ailleurs, le site internet de la SFEROV : www.sferov.com , est en cours de refonte et proposera d'ici quelques semaines une meilleur interactivité, avec notamment un forum public doublé d'un forum à accès réservé aux membres de l'association.

Ateliers de Formation 2006

Le secrétariat des ateliers est désormais assuré par Frank Famose.

- Implantation : le 12 janvier 2007, à l'ENV Toulouse. (Moniteurs : P. Lazard - PF Isard) Atelier Complet
- Techniques de Microchirurgie et Phacoémulsification : les 5 et 6 juillet 2007 à Rueil-Malmaison. (Moniteurs : AS Augsburger – V. Meunier – B. Cantaloube. PF Isard – P. Lazard)
- Electrophysiologie visuelle fonctionnelle, en décembre 2007 à l'ENV Lyon. (Moniteurs : P. Lazard- P.E. Lallement- P.F. Isard)

Renseignements et Inscriptions : Frank Famose. Tel : 05 61 71 24 02 Fax : 05 61 71 65 52 @ : frank.famose@wanadoo.fr

Commissions

Plusieurs commissions ont été formalisées, et sont à même de répondre à vos questions dans des domaines en développement :

- génétique : responsable Philippe PILORGE
- ophtalmologie équine : responsable Marc VERNEUIL
- ophtalmologie des NAC : responsable Franck RIVAL

Prix SFEROV

Créé en 2004 , le Prix SFEROV, est attribué par un jury composé des membres du Conseil d'Administration pour récompenser un travail original récent (publication, mémoire, thèse..) en ophtalmologie vétérinaire. Les candidats à ce prix (chèque d'encouragement de 1000 €) peuvent soumettre leur travail au secrétaire de la SFEROV jusqu'au 31 décembre 2006.

Cotisation 2007

Grâce à une gestion rigoureuse et des dépenses transparentes, la situation financière de la SFEROV est saine, au point que le Bureau a décidé une fois encore de ne pas augmenter la cotisation annuelle. Elle reste fixée à 55 € ; vous recevrez prochainement l'appel de cotisation 2007.

ESVO

La SFEROV a resserré les liens avec la Société Européenne d'Ophtalmologie Vétérinaire (ESVO). Marc Verneuil est membre du bureau de l'ESVO depuis juin 2005. Le congrès annuel ESVO se tiendra à Gênes, en Italie, du 30 mai au 3 juin 2007. www.esvo.org

Bibliographie

Le bulletin de liaison vous proposera désormais une rubrique bibliographie. Sans souci d'exhaustivité, cette rubrique animée notamment par le Professeur REGNIER, permettra de connaître des travaux dignes d'intérêt publiés hors des circuits habituels.

Assessment of the dark-adaptation time required for recovery of electroretinographic responses in dogs after fundus photography and indirect ophthalmoscopy

Nalinee Tuntivanich, DVM; A. Lexi Mentzer; Danielle M. Eifler, DVM; Fabiano Montiani-Ferreira, DVM, PhD;
Janice Q. Forcier; Cheri A. Johnson, DVM, MS; Simon M. Petersen-Jones, DVetMed, PhD

Objective—To investigate the duration of dark-adaptation time required for recovery of electroretinographic responses after fundus photography or indirect ophthalmoscopy in dogs.
Animals—6 dogs.
Procedure—Initially, scotopic-intensity series of electroretinograms (ERGs) were recorded after 20 minutes of dark adaptation. The fundus of the left eye of each dog was photographed (n = 10) or examined via indirect ophthalmoscopy for 5 minutes with moderate- (117 candela [cd]/m2) or bright-intensity (1,693 cd/m2) light; ERGs were repeated after a further 20 or 60 minutes of dark adaptation (6 procedures/dog).
Results—Following 20 minutes of dark adaptation after fundus photography, the b- and a-wave amplitudes were reduced in response to brighter stimuli, compared with pretest ERGs; after 60 minutes of dark adaptation, ERG amplitudes had recovered. Following 20 minutes of dark adaptation after indirect ophthalmoscopy (moderate-intensity light), significantly lower b-wave amplitudes were recorded in
response to 2 of the brighter flash stimuli, compared with pretest ERGs; after 60 minutes of dark adaptation, ERG amplitudes had recovered. Following 20 minutes of dark adaptation after indirect ophthalmoscopy (bright-intensity light), all ERG amplitudes were significantly decreased and implicit times were significantly decreased at several flash intensities, compared with pretest ERGs; after 60 minutes of dark adaptation, ERG amplitudes and implicit times had returned to initial values, except for b-wave amplitudes recorded in response to dimmer stimuli.
Conclusions and Clinical Relevance—Results suggest that at least 60 minutes of dark adaptation should be allowed before ERGs are performed in dogs after fundus photography or indirect ophthalmoscopy.
(Am J Vet Res2005;66:1798–1804)

Why cornea is transparent and free of blood vessels, allowing vision

Scientists at the Harvard Department of Ophthalmology's Schepens Eye Research Institute and Massachusetts Eye Ear Infirmary (MEEI) are the first to learn why the cornea, the clear window of the eye, is free of blood vessels--a uni phenomenon that makes vision possible. The key, say the researchers, is the unexpected presence of large amount the protein VEGFR-3 (vascular endothelial growth factor receptor-3) on the top epithelial layer of normal healthy corn Diabetes can cause blood vessels to collapse, creating a hypoxic environment that generates vascular endothelial g factor (VEGF) and triggers angiogenesis. Pericytes (diamonds) detach, destabilizing vessels. Kazlauskas and Im hypothesize that endothelial cells (rectangles) receive specific instructions during this unstable state that dictate whe the vessels should grow or regress. In the case of diabetic retinopathy, too many vessels grow, eventually obscuring vision. According to their findings, VEGFR-3 halts angiogenesis (blood vessel growth) by acting as a "sink" to bind o neutralize the growth factors sent by the body to stimulate the growth of blood vessels. The cornea has long been kn to have the remarkable and unusual property of not having blood vessels, but the exact reasons for this had remaine unknown.
These results, published in the July 25, 2006 issue of the Proceedings of the National Academy of Sciences and in t July 17 online edition, not only solve a profound scientific mystery, but also hold great promise for preventing and cu blinding eye disease and illnesses such as cancer, in which blood vessels grow abnormally and uncontrollably, since phenomenon, present in the cornea normally, can be used therapeutically in other tissues.
"This is a very significant discovery," says Dr. Reza Dana, Senior Scientist at the Schepens Eye Research Institute, head of the Cornea Servic the Massachusetts Eye and Ear Infirmary, and an associate professor at Harvard Medical School, and the senior aut and principal investigator of the study. "A clear cornea is essential for vision. Without the ability to maintain a blood-v free cornea, our vision would be significantly impaired," he says, adding that clear, vessel-free corneas are vital to an animal that needs a high level of visual acuity to survive.
The cornea, one of only a few tissues in the body that actively keep themselves vessel-free (the other is cartilage), is thin transparent tissue that covers the front of the eye. It is the clarity of the cornea that allows light to pass onto the and from there to the brain for interpretation. When the cornea is clouded by injury, infection or abnormal blood vess growth, vision is severely impaired, if not destroyed. Scientists have been wrestling with the "clarity" puzzle for many decades. And, while some previous studies have revealed small clues, none have pointed to one major mechanism, this study. In most other tissues of the body, blood vessel growth or angiogenesis occurs in response to a need for increased b flow to heal an injured or infected area. The immune

system sends in growth factors such as vascular endothelial grow factor (VEGF) to bind with a protein receptor called VEGFR-2 on blood vessels to trigger vessel growth. Three forms VEGF--A, C, and D--bind with this receptor. Two of them, C and D also bind with VEGFR-3, which is usually found o lining lymphatic vessels, to stimulate the growth of lymphatic vessels. Dana's team began to suspect the involvement VEGFR-3 in stopping blood growth in corneas when they noticed unexpectedly that large amounts of the protein see to exist naturally on healthy corneal epithelium, a previously unknown location for the receptor. Dana and his team already aware from clinical experience that the epithelium most likely played a role in suppressing blood vessel grows the cornea, having witnessed blood vessels develop on corneas stripped of their epithelial layers. They began to theorize that the large amounts of VEGFR-3, in this new, non-vascular location, might be attracting an sucking up all the C and D VEGF growth factors, thereby blocking them from binding with VEGFR-2. And, because binding took place in a non-vascular setting, the growth factors were neutralized. To test their theory, the team condu a series of experiments. They conducted chemical analyses that demonstrated that VEFGR-3 and the gene that expressed it were indeed present on the corneal epithelium. Next, in two separate experiments, they compared corn with and without epithelial layers that were injured. They found that only the corneas without epithelial layers develop blood vessels, implicating the role of the epithelium in suppressing blood vessel growth. To further prove their theory added a VEGFR-3 substitute to corneas stripped of their epithelial layers and found that vessel growth continued to suppressed, replacing the normal anti-angiogenic role of the epithelium. Finally they exposed intact corneas to an ag that blocked VEGFR-3 and found that blood vessels began to grow, formally demonstrating that the corneal epithelium key to suppression of blood vessels and that the key mechanism is expression of VEGFR-3. "The results from this series of tests, confirmed our belief that the presence of VEGFR-3 is the major factor in prevent blood vessel formation in the cornea," says Dana, who says that the discovery will have a far reaching impact on the development of new therapies for eye and other diseases. "Drugs designed to manipulate the levels of this protein heal corneas that have undergone severe trauma or help shrink tumors fed by rapidly growing abnormal blood vessel he says. "In fact, the next step in our work is exactly this."
Science Daily
August 1, 2006

Contacts :

Le Bureau de la SFEROV est toujours à votre disposition. Merci de privilégier les contacts e-mail ou fax.

Président : P.F. ISARD
Centre d'Ophtalmologie Vétérinaire. 4550 Les Grands Vignobles. 74320 Sevrier. T :0450190101. F : 0450190985. @ : contact@ceovet.com

Vice Président (relation avec les instances nationales) P. LAZARD.
Clinique Vétérinaire. 80 rue Pereire. 78100 Saint Germain en Laye.T : 0134513133. F : 0134515516. @ : ladelou@wanadoo.fr

Vice Présidente (congrès européen 2008) V. MEUNIER. 30 rue Faÿs. 94300. Vincennes. T:0143982107.@v.meunier@free.fr

Vice Président (relations internationales) M. VERNEUIL. Clinique Vétérinaire. Bd de la Libération. Chemin du Micocoulier. 83490 Le Muy T : 0494458787. F: 0494458611. @ : marc.verneuil@cegetel.net

Secrétaire : Ph. PILORGE. Clinique Vétérinaire.
68-70 rue Papu. 35000 Rennes. T : 0299590122. F : 029959615
@ : p.pilorge@wanadoo.fr

Secrétaire adjoint (ateliers et manifestations) F. FAMOSE Clinique Vétérinaire. 42 rue Lucien Servanty. 31700 Blagnac. T: 0561712402 F: 0561716552. @ : frank.famose@wanadoo.fr

Trésorier : P.E. LALLEMENT .Clinique Vétérinaire. 20 avenue des Tignes. 06400 Cannes. T: 0493390727. F: 0493683558.@:pelallement@aol.com.

Membres :

B. CANTALOUBE (relations partenaires commerciaux).Clinique Vétérinaire. 61 route de Sète. 34300 Agde. T+F: 0467773064. @ : b.cantaloube@wanadoo.fr

Ph. DURIEUX. Clinique Vétérinaire. 35 rue des Cordeliers. 77100 Meaux. T: 0164341155. F: 0164333235. phil.durieux2@wanadoo.fr

F. GOULLE. Clinique Vétérinaire. 1 rue du Dr Nancel Penard. 33600 Pessac T : 0556450475. F : 0556150079. @ : frederic.goulle@wanadoo.fr

A. REGNIER (validation scientifique) ENVT. 23 chemin des Capelles. 31076 Toulouse Cédex 3. T : 0561193832. F : 0561193203. @ : a.regnier@envt.fr

F. RIVAL. Clinique Vétérinaire.192 avenue de Romans. 26000 Valence . T : 0475433391. F : 0475431460. @ : franck.rival@wanadoo.fr