

D KÖMPF*

Klinik für Neurologie,*

Klinik für Innere Medizin†

Medizinische Universität zu Lübeck,

Ratzeburger Allee 160

D-2400 Lübeck 1

Federal Republic of Germany

References

- 1 Stenflo J. Structure and function of protein C. *Semin Thromb Hemost* 1984;10:109-21.
- 2 Broekmans AW, Bertina RM, Reinalda-Poot J, et al. Hereditary protein S deficiency and venous thromboembolism. *Thromb Haemost* 1985;54:273-7.
- 3 Egeberg O. Inherited antithrombin deficiency causing thrombophilia. *Thromb Haemost* 1975;13:516-30.
- 4 Esmon CT. Protein C: Biochemistry, physiology, and clinical implications. *Blood* 1983;62:1155-8.
- 5 Seligsohn U, Berger A, Abend M, Rubin L, Attias D, Zivelin A, Rapaport SI. Homozygous protein C deficiency manifested by massive venous thrombosis in the newborn. *N Engl J Med* 1984;310:559-62.
- 6 Tarras S, Gadia C, Meister L, Roldan E, Gregorios JB. Homozygous protein C deficiency in a newborn. Clinicopathologic correlation. *Arch Neurol* 1988;45:214-6.
- 7 Broekmans AW, van der Linden IK, Veltkamp JJ, Bertina RM. Prevalence of isolated Protein C deficiency in patients with venous thrombotic disease and in the population with anticoagulant therapy. *Thromb Haemost* 1983;50:350 (Abstr).
- 8 Broekmans AW, Veltkamp JJ, Bertina RM. Congenital protein C deficiency and venous thromboembolism. *N Engl J Med* 1983;309:340-4.
- 9 Mannucci PM, Vigano S. Deficiencies of protein C, an inhibitor of blood coagulation. *Lancet* 1982;ii:463-7.
- 10 Wagner T, Schwieder G. Wertigkeit verschiedener Protein-C-Bestimmungsmethoden bei kongenitalem Protein-C-Mangel und unter Phenprocoumontherapie. *Ärztl Lab* 1987;33:136-40.
- 11 Wintzen AR, Broekmans AW, Bertina RM, et al. Cerebral haemorrhagic infarction in young patients with hereditary protein C deficiency: evidence for "spontaneous" cerebral venous thrombosis. *Br Med J* 1985;290:350-2.
- 12 Vogt T, Besser R, Thoenke F, Hopf HC. Therapie der Sinusthrombose bei angeborenem Protein-C-Mangel. *Med Klin* 1987;82:801-3.
- 13 Barbui T, Finazzi G, Mussoni L, et al. Hereditary dysfunctional protein C (protein C Bergamo) and thrombosis. *Lancet* 1984;ii:819.
- 14 Schwieder G, Vieregge P, Wiedemann G, Wagner T. Kongenitaler Protein-C-Mangel und thromboembolische Erkrankungen. *Dtsch Med Wochenschr* 1987;112:425-8.
- 15 Griffin JH, Evatt B, Zimmerman TS, Kleiss AJ. Deficiency of protein C in congenital thrombotic disease. *J Clin Invest* 1981;68:1370-3.
- 16 Horellou MH, Conard J, Bertina RM, Samama M. Congenital protein C deficiency and thrombotic disease in nine French families. *Br Med J* 1984;289:1285-8.
- 17 D'Angelo A, Landi G, Vigano d'Angelo S, et al. Protein C in acute stroke. *Stroke* 1988;19:579-83.
- 18 Krayenbühl HA. Cerebral venous and sinus thrombosis. *Clin Neurosurg* 1966;14:1-24.
- 19 Barnett HJM, Hyland HH. Non-infective intracranial venous thrombosis. *Brain* 1953;76:36-49.
- 20 DiRocco C, Iannelli A, Leone G, Moschini M, Valori VM. Heparin-Urokinase treatment in aseptic dural sinus thrombosis. *Arch Neurol* 1981;38:431-5.
- 21 Druschky KF, Neundörfer B, Erbguth F, Kilian KD, Kömpf D. Heparinbehandlung bei Sinusvenenthrombosen mit intrazerebralen Blutungen. *Fortschr Neurol Psychiatr*.
- 22 Pabinger-Fasching I, Bertina RM, Lechner K, Niessner H, Korninger C. Protein C deficiency in two Austrian families. *Thromb Haemost* 1983;50:810-3.
- 23 Clouse LH, Comp PC. The regulation of hemostasis: the protein C system. *N Engl J Med* 1986;314:1298-304.

Accepted 6 September 1988

Geometrical optics of the retinal image stabilisation device

Sir: An optical device giving partial or almost-complete stabilisation of the retinal image has been described¹ and tested^{2,3} to treat patients with continuous oscillopsia caused by nystagmus, usually due to acquired neurological disease such as multiple sclerosis. It can relieve such oscillopsia, and consists of a high-plus spectacle lens used together with a high-minus contact lens.

In many of these patients the amplitude of oscillopsia is less than would be predicted from the amplitude of their nystagmus, so that the image only needs to be partially stabilised. A means is then needed for predicting how much retinal image stabilisation ("RIS") will be needed in each case, and for calculating the correct lens power required to achieve it.

An index of oscillopsia has been developed, defined as the ratio of angular amplitude of oscillopsia ("O") and angular amplitude of nystagmus ("N"). O/N can vary from 0 (no oscillopsia, in spite of nystagmus) to 1 (oscillopsia of the same amplitude as nystagmus). The index of

retinal image stabilisation required is also equal to O/N, since the proportion of the nystagmus that is perceived as oscillopsia is also the proportion of the nystagmus that must be cancelled by the device.

Partially stabilising the retinal image is equivalent to increasing the amount of rotation of the eye required to move the image through a given angle on the retina (rotational magnification). Retinal image stabilisation therefore bears a simple relation with rotational magnification, which is a well-studied property of plus spectacles.

Rotational magnification of the image on the retina (RM_r) is here defined as the ratio of the angular motion of the eye scanning a scene (N) and the resulting angle of retinal image-slip (P). RM_r differs from the amount of rotational magnification in terms of distance across the scene (RM_s) when, as here, the device causing the rotational magnification also causes angular magnification of the retinal image. What we need is to apply sufficient RM_r to reduce the retinal image-slip (originally equal to N) by the amount of the oscillopsia (O). The remaining image-slip P does not cause oscillopsia since it is compensated by a central mechanism ("C"), whose site and mode of action is not known.

Then if:

N = amplitude of nystagmus

O = amplitude of oscillopsia

C = amplitude of central compensating signal

P = amplitude of residual retinal image-slip

N = C + O (1)

RM_r = N/P (2)

From (1), O/N = 1 - (C/N)

From (2), (RM_r - 1)/RM_r = 1 - (P/N)

For optical neutralisation of oscillopsia, we have said that P = C

So O/N = (RM_r - 1)/RM_r = RIS (3)

The RM_r is generated by the combination of spectacle and contact lens, which if the image is in focus on the retina form a galilean pair. A galilean pair is afocal, so where:

f_s = focal length of spectacle (in metres)P_s = power of spectacle (in dioptres)f_c = focal length of contact lens (in metres)P_c = power of contact lens (in dioptres)

d = separation between principal planes of spectacle and contact lens (in metres)

f_s = -f_c + dOr, 1/P_s = d - (1/P_c) (4)

The galilean pair also causes angular magnification of the image (AM), which is given by:

AM = -P_c/P_s (5) (see ref 4, p205).

Now we need to find how the amount of RIS obtained varies with the lens power.

The rotational magnification generated in terms of movement of the gaze across the scene (RMs) by a plus spectacle lens can be shown to be given by:

$$RMs = \frac{1}{1 - Ps(d + r)} \quad (6) \text{ (see ref 4, p248)}$$

where: r = radius of the eye, from its centre of rotation to the principal plane of the contact lens

Ps = power of spectacle lens (in dioptries)

But what we need is the rotational magnification in terms of retinal image-slip (RM_r), not in terms of movement of the gaze across the scene (RMs), because the scene is magnified by the optical device.

So, $RM_r = RMs/AM$.

Substituting into (6) to obtain RM_r :

$$RM_r = \frac{1}{[1 - Ps(d + r)]AM} \quad (7)$$

Put (7) into (3):

$$RIS = \frac{1}{[1 - (d + r)Ps]AM} - 1$$

$$= \frac{1}{[1 - (d + r)Ps]AM} - 1$$

$$= 1 - AM[1 - (d + r)Ps] \quad (8)$$

From (5), substitute into (8) for AM:

$$RIS = 1 + Pc/Ps - Pc(d + r)$$

From (4), substitute for $1/Ps$:

$$RIS = 1 + Pc(d - 1/Pc) - Pc(d + r)$$

$$= 1 + d.Pc - 1 - d.Pc - Pc.r$$

$$= -Pc.r$$

So the amount of RIS obtained is directly proportional to the contact lens power and the radius of the eye. For each combination of Pc and d , Ps is fixed by the need to form an afocal galilean pair. If d is varied, Ps and AM will vary as a consequence, but RIS will be unchanged. It follows that if r is taken to be a constant, a nomogram can be calculated giving all combinations of RIS , Ps , Pc and d . An example of a nomogram for $r = 1.25$ cm has been constructed.² The results are only

approximate, because the equations used throughout are those of thin-lens geometrical optics, while to obtain clinically appropriate amounts of RIS , Ps and Pc are both high.

I am grateful to PEK Donaldson and N Rushton for help with the algebra.

DN RUSHTON
MRC Neurological Prosthesis Unit
Institute of Psychiatry,
De Crespigny Park,
London SE5 8AF, UK

References

- 1 Rushton DN, Rushton RH. An optical method for approximate stabilisation of vision of the real world. *J Physiol (Lond)* 1984;**357**:3P.
- 2 Rushton DN, Cox ND. A new optical treatment for oscillopsia. *J Neurol Neurosurg Psychiatry* 1987;**50**:411-5.
- 3 Leigh RJ, Rushton DN, Thurston SE, Hertz RW, Yaniglos SS. Effects of retinal image stabilization in acquired nystagmus due to neurologic disease. *Neurology* 1988;**58**:122-7.
- 4 Rubin M. *Optics for Clinicians*. Gainsville Triad Scientific Publishers, 1974.

Accepted 26 August 1988

J Neurolog Neurosurg Psychiatry. First published as 10.1136/jnnp.1989.52.1.137 on 1 January 1989. Downloaded from http://jnnp.bmj.com/ on March 28, 2023 by guest. Protected by copyright.