Eyelid closure causes myopia in humans

D.J. O'Leary and M. Millodot[1]

Laboratory of Experimental Optometry, University of Wales Institute of Science and Technology, Cardiff (CF1 3NU, Great Britain), 29 January 1979

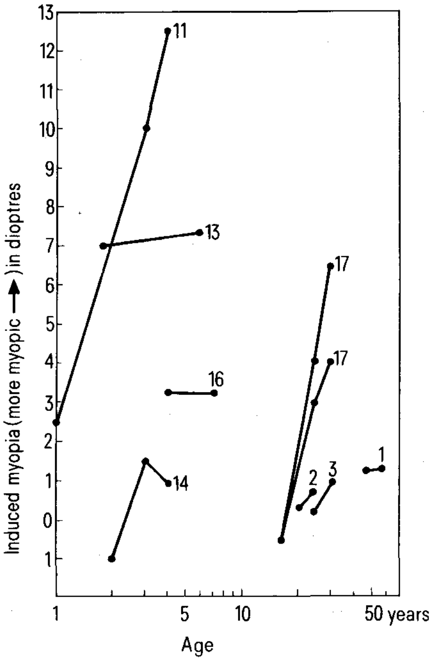
Summary. Humans with unilateral ptosis are more myopic in the closed eye than in the normal eye. The effect seems unrelated to visual loss. In pairs of humans ranked for sleep and myopia, the more myopic tends to sleep the more. We conclude that eyelid closure causes myopia.

It has recently been established in several studies that neonatal eyelid closure induces myopia in different animals: in monkeys[2], tree shrews[3], cats[4], and most recently in chicks[5]. The amount of induced myopia varies greatly amongst species[6], and even between individual members of the same species[7]. Some of these variations may be artifacts of the surgical techniques used to effect closure[7], or can be explained by the visual environments of the animals[5,8]. We report here the effects of nonsurgical lid closure on the refractive error of humans.

Firstly, we examined the refractive state of 18 cases of ptosis; our results are summarized in the table. It can be seen that, in 12 out of the 13 cases where the closure is unilateral, the eye affected by ptosis is more myopic than the normal eye ($p < 0.005$). The mean refractive errors (SD) are: affected eyes -1.69 dptr (5.8), normal eyes $+1.13$ dptr (4.6). Taking the normal eyes as controls, eye closure has induced, on average about 2.75 dptr myopia, an amount comparable with that found in monkeys[2,7]. Since the closure in our subjects was always incomplete, we do not think that this result reflects the maximum extent of the effect in man. In bilateral cases where 1 eye was affected more than the other (Nos 9 and 17) the more affected eye was also more myopic (or less hyperopic).

For some subjects we were able to obtain data from previous refractions; these cases are summarized in figure 1. The tendency for the progression of myopia to be greater in younger subjects is quite evident, and in this respect also, man seems similar to monkey[2]. In 3 of the 4 children, the myopia has very nearly stabilized, although at greatly different levels. This may mean that there are constitutional limits to the effect for each individual and again this is in agreement with the variations found in monkey[2,7].

We could find little support for the hypothesis that deprivation of form vision (as a result of eyelid closure) is responsible for inducing myopia in man[8]. Although the visual acuities of our subjects with unilateral ptosis are, on average, lower in the affected eye (0.60) than in the normal eye (0.75), the theory leaves several cases (Nos 1, 6, 8, 10 and 14 in the table) unexplained. If we assume that the visual loss is secondary to the anisometropia, then we find only 1 case not explained (No. 3). This does not rule out the possibility that more subtle visual effects may be important, as has been shown in various experiments on chicks[5]; it does



The progress of myopia in ptosis for 7 unilateral cases (refraction of affected eye minus normal eye) and 1 bilateral case (No. 17). The number beside each case refers to the subject's case number in the table.

Ptosis and refractive error in humans

No.	Age of onset (years old)	Duration (years)	Affected eye(s)	Equivalent refraction*	Visual acuity
1	0	54	R	R+ 4.75 L+ 6.00	1.00 1.00
2	0	24	R	R− 3.25 L− 2.25	0.4 1.0
3	14	34	L	R+ 2.25 L+ 1.25	1.0 0.9
4	0	18	L	R− 5.00 L− 6.00	1.0 0.6
5	0	66	R	R− 7.00 L− 3.00	0.1 1.0
6	0	66	L	R+ 5.75 L 0.00	0.2 1.0
7	12	30	Both (R = L)	R+ 3.00 L+ 2.50	0.6 0.6
8	57	12	L	R+ 4.25 L+ 3.00	0.5 1.0
9	0	3	Both (R > L)	R+ 3.00 L+ 5.50	– –
10	0	41	Both (R = L)	R+ 1.00 L+ 2.25	0.25 0.17
11	0	4	L	R 0.00 L − 12.50	0.5 0.1
12	0	12	Both (R = L)	R 0.00 L 0.00	1.0 1.0
13	0	2	R	R− 6.50 L+ 1.00	– –
14	0	4	R	R+ 8.00 L+ 9.00	0.5 0.5
15	0	3	L	R+ 3.50 L+ 4.00	1.0 0.7
16	0	7	R	R− 3.25 L 0.00	0.17 0.33
17	16	13	Both (L > R)	R− 4.75 L− 6.00	0.25 0.25
18	0.5	21	L	R− 2.75 L− 4.50	1.0 0.7

* Sphere + ½ cyl.

suggest that deprivation of form vision alone is not sufficient to explain the induction of myopia in man.

On the assumption that it was eye closure per se (rather than visual interference) which was the triggering factor in this type of myopia, we then compared the relative duration of sleep in siblings and cohabitants with their relative refractive errors; 133 such pairs of people were evaluated. In 85 out of 133 pairs the person who slept the more (an easy observation in people living together) exhibited the greater degree of myopia ($p < 0.005$). This sample was not taken from a wide section of the population: We selected cases from the families of our students and associates. To ensure that the result was not unduly biased by its narrow population base, we interviewed myopes with non-academic backgrounds, and obtained a further 57 pairs. In this group the more myopic slept the more in 39 cases ($p < 0.01$), confirming our first observations.

Further evidence of the effect of partial or total eyelid closure is provided by a) cases of infantile hemangiomas[9] and b) cases of external ophthalmoplegia[10]. In both conditions it was found that the affected eye was associated with marked myopia relative to the normal eye.

Taken in conjunction with our own observations this seems to indicate that there is a very basic relationship between ocular coverage and myopia in humans, as well as in other species[2-5].

We do not suggest that the enormously complex aetiology of myopia can be reduced to an equation involving only the palpebral aperture. It is noteworthy, however, that in the normal reading posture, the aperture is reduced; we also note that many races prone to myopia, (e.g. Chinese[11]) have a narrow palpebral aperture.

1 We are grateful to Mr J.C. Dean Hart, Bristol Eye Hospital, for providing us with some case histories.
2 T.N. Wiesel and E. Raviola, Nature *266*, 66 (1977).
3 S.M. Sherman, T.T. Norton and V.A. Casagrande, Brain Res. *124*, 154 (1977).
4 J.R. Wilson and S.M. Sherman, J. Neurophysiol. *40*, 891 (1977).
5 J. Wallman, J. Turkel and J. Trachtman, Science *201*, 1249 (1978).
6 F. Giffin and D.E. Mitchell, J. Physiol. *274*, 511 (1978).
7 G.K. von Noorden and M.L.J. Crawford, Nature *272*, 53 (1978).
8 E. Raviola and T.N. Wiesel, Invest. Ophthal. *17*, 485 (1978).
9 R.M. Robb, Am. J. Ophthal. *83*, 52 (1977).
10 A. Salleras and J.C.O. de Zarate, Br. J. Ophthal. *34*, 662 (1950).
11 I. Borish, Clinical refraction, 3rd edn. The Professional Press Inc., Chicago 1970.

Photoperiodic regulation of winter diapause in the grass spider

K. Kurihara[1]

Division of Entomology, National Institute of Agricultural Sciences, Nishigahara, Kita-ku, Tokyo 114 (Japan), 4 December 1978

Summary. Winter diapause of the grass spider was experimentally proved to be photoperiodically regulated. Critical daylength for the termination of diapause; being shortened during winter, was suggested to ensure successfull hibernation of this species.

Diapause as an important means of seasonal adaptation has been extensively explored only in particular groups of arthropods, insects and mites[2]. Although a few reports have described the presence of diapause in Araneae[3-7], the significance of photoperiod in regulating the diapause of spiders has not generally been recognized. The author elucidated the occurrence of a winter diapause in the grass spider, *Agelena limbata* Thorell (Agelenidae), and demonstrated experimentally the important role of photoperiod in the maintenance of this diapause.

The grass spider is a most common species of univoltine life cycle in Japan[8]. The overwintered 2nd-instar spiderlings emerge from the egg-sacks in late March to early April. They construct platform webs on weeds or shrubs and develop into adults in late July to early August presumably after 4–5 moults. The adults copulate in mid to late August and each female lays about 70 eggs in a egg-sack in late September to early October. The eggs hatch 1–2 weeks later, and the spiderlings moult into the 2nd instar in about 15 days. The 2nd-instar spiderlings then enter diapause and

Photoperiodic responses of the grass spider during the overwintering period

Time of transfer			Photoperiod (light: dark) h						
Day after the 1st moult	Date		10:14	12:12	13:11	13.5:10.5	14:10	14.5:9.5	16:8
0	Nov. 5	%*	7	31	5	98	98	100	95
	(12)**	day*	165	140	145	26	39	29	28
40	Dec. 15	%	0	18	82	100	95	99	88
	(12)	day	0	125	48	27	28	28	25
91	Feb. 4	%	97	34	100	100	99	98	–
	(8)	day	50	55	19	18	18	16	–
131	Mar. 16	%	–	–	100	92	–	–	54
	(3)	day	–	–	13	16	–	–	13

* '%' indicates the percentage of emergence of 2nd instar spiderlings, and 'day' indicates average duration in days from the transfer of egg-sacks to the emergence. ** Values in parentheses are the number of egg-sacks used.