Steiger on refraction: a reappraisal

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SUMMARY Seventy years ago Steiger, a Swiss ophthalmologist, found the distribution of corneal powers to follow a normal (binomial) curve. He noted a wide range of values among emmetropes, and he also knew that their axial lengths varied significantly. He expected that normal distributions would be found for other components of refraction and also for refraction as a whole, and in seeking a controlling mechanism he recalled the multifactorial pattern of inheritance of such characteristics as stature. The present study employs modern mathematical techniques to test the validity of 2 related hypotheses: that the components of refraction are correlated and that a polygenic mode of inheritance is responsible for determining the refractive power of the eye. In the light of this study and of other modern knowledge about refraction Steiger's work is reassessed. Most of his views are vindicated, although his assumption of a normal distribution for refraction as a whole could not be justified. His contribution to the understanding of refraction establishes him among the great names in ophthalmology.

For his bounty
There was no winter in't, an autumn 'twas
That grew the more by reaping . . .
(*Anthony and Cleopatra*, V, ii)

Invented by Helmholtz in 1856, the keratometer as perfected by Javal and Schiötz in 1881 found a ready application clinically for assessing any existing astigmatism. From observations on the measurement of the radii of curvature of the cornea, Bourgeois and Tscherning in 1886¹ suggested that the corneal power was distributed in a normal (binomial) manner—a view firmly established by Steiger from a study of the corneal power in 5000 eyes in boys aged 6 to 7 years. Fig. 1 taken from his crucial monograph² published in 1913 is a historical landmark. The material for his curve of distribution had come from some 20 schools at Zurich at which he served as oculist, interim data being recorded from 1895 onwards.

On the strength of his curve of distribution of the powers of the cornea Steiger made 2 inspired and far-reaching guesses. He held that the other components of refraction and also refraction as a whole followed a normal distribution; and that this normal distribution recalled the multifactorial pattern seen in such a hereditarily determined character as stature. Both assumptions, though fundamentally valid, were not entirely correct.

Fig. 1 The normal (binomial) distribution of power of cornea in 5000 eyes in boys.² Unbroken line: measured values. Dashed line: normal (binomial) distribution.

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Fig. 2 Curves of distribution of refraction and its components in 194 eyes: (a) power of cornea (D); (b) depth of anterior chamber (mm); (c) power of lens (D); and (d) axial length (mm).³

Refraction curves

Steiger's assumption as to the normal distribution of the components of refraction has been fully borne out by the measurements made with procedures that have become available since his time: Fig. 2 is illustrative.³

On his further assumption that the refraction of the eye as a whole also follows a normal curve he concluded that emmetropia and the refractive errors are merely points on such a curve, and that all refractions

result from the free association of normally distributed individual components. In this scheme of things, presented graphically in Fig. 3 taken from his monograph, he employed the 2 components available to him: his own corneal measurements, which showed a range of 12 D in emmetropia, and the axial length in emmetropia which Mauthner in 1876 had found in post-mortem material to extend between 22.25 mm to 26.24 mm.

Steiger did not plot the distribution of refraction in

Fig. 3 Theoretical curve of range and distribution of ocular refraction on the assumption of free association of cornea and axial lengths, both normally distributed.²

the general population, but the relevant data now available do not support his assumption that the distribution would be normal in character. Fig. 4 is illustrative; it is seen that in the general population there is a marked excess of refractions around emmetropia, a consequent deficiency of moderate refractive errors, and some excess of the extreme refractive errors. The concentration of refractions around emmetropia suggests that the refraction of the eyes as a whole results from correlation of the components of refraction—and not from their free association—a suggestion that can be tested mathematically.

Statistical assessment of the relationship of the components of refraction

Correlation of multiple individual components is not easy to express mathematically, as the function to be expressed is rather complicated, involving laborious effort. The electronic computer was only just becoming available when the data on refraction and its components in 194 eyes in adults aged 20 to 50 years were published in 1957.³ This material is now used here for the method of simulation made possible by the computer to assess statistically the relationship of the components in refraction.

The 3 major variables which determine the ocular refraction (K) are (i) the overall axial length of the eye; (ii) the power of the cornea, considered as a single refracting surface; (iii) the equivalent power of the crystalline lens. To these may be added one more of lesser importance: (iv) the depth of the anterior chamber, which, for this purpose, includes the corneal thickness.

Unfortunately a knowledge of these 4 dimensions is not sufficient for accurate calculation. The thickness and individual surface powers of the crystalline lens are also required. In the experimental data utilised for this study these additional dimensions had been found by phakometry. Nevertheless, to introduce them as additional variables would have made it impossible to arrive at an algebraic expression suitable for statistical analysis.

A sufficiently close approximation is made possible by the fact that the 2 principal points of the human crystalline are very close together, being usually

Fig. 4 Curves of distribution of refraction in the general population: (a) in 1033 National Service recruits in England⁵; (b) in 194 eyes of adults in London.³

Fig. 5 The simplified schematic eye used as the model for statistical analysis.

separated by less than 0.2 mm. Without introducing serious errors the real crystalline of known equivalent power can hence be replaced by a hypothetical lens of the same power but of zero thickness, situated at the mean position of the 2 principal points. This, in effect, was the artifice used by Gullstrand in the simplified version of his schematic eye. It provides an ideal basis for the purpose of this study.

The scheme is shown in Fig. 5, in which all the distances are to be expressed in metres:

A₁=vertex of the cornea
A₂=anterior vertex of the real crystalline lens
M'=fovea
M_R=eye's far point of refraction, conjugate with M'
d=depth of anterior chamber
w=(d+0.0024)=distance from corneal vertex to position of hypothetical crystalline lens
z=overall axial length of eye
k=A₁M_R whence K=1/k

Additionally,
x=equivalent power of crystalline lens
y=corneal power.

Table 1 Simulation of combination of optical components in 1000 random eyes. Normally distributed components uncorrelated.

Run	1	2	3	4	5	6	7	8	9	10
Mean K	0.22	0.24	0.03	-0.04	0.06	0.33	0.29	-0.01	0.22	0.21
SD K	3.03	2.99	2.91	2.87	2.93	3.04	3.00	2.94	3.15	2.94
√β ₁	0.05	0.03	0.03	0.10	0.20	0.10	0.11	0.09	0.10	0.14
β ₂	0.11	-0.17	-0.23	10.23	0.29	0.12	0.02	0.11	0.09	0.04
Frequency histogram										
Less than -10.0	0	0	0	0	1	1	0	0	0	0
-10.0 to -9.0	0	0	0	0	1	1	1	0	1	0
-9.0 to -8.0	4	1	2	0	0	2	1	3	2	2
-8.0 to -7.0	2	5	3	3	0	2	5	6	4	3
-7.0 to -6.0	17	9	13	11	13	8	8	7	15	8
-6.0 to -5.0	22	30	31	28	18	20	16	31	28	19
-5.0 to -4.0	36	41	45	45	43	39	36	43	42	44
-4.0 to -3.0	62	68	59	69	61	61	69	67	61	61
-3.0 to -2.5	24	32	35	42	57	40	39	41	51	26
-2.5 to -2.0	45	47	60	54	49	42	55	43	44	45
-2.0 to -1.5	57	50	59	61	71	49	53	53	54	78
-1.5 to -1.0	68	47	53	66	56	77	71	67	60	63
-1.0 to -0.5	66	51	66	68	71	60	57	64	45	63
-0.5 to 0.0	73	63	64	75	68	71	56	67	63	71
0.0 to 0.5	67	70	66	68	60	59	66	73	59	68
0.5 to 1.0	71	84	62	52	59	67	65	89	66	73
1.0 to 1.5	65	66	76	57	62	52	63	59	60	54
1.5 to 2.0	52	55	56	48	63	61	60	57	71	51
2.0 to 2.5	59	66	58	51	52	48	44	53	48	53
2.5 to 3.0	36	43	33	48	38	50	51	23	34	48
3.0 to 4.0	65	69	78	78	72	83	73	62	75	69
4.0 to 5.0	49	45	33	34	41	44	55	42	44	49
5.0 to 6.0	30	34	29	23	19	27	26	27	41	23
6.0 to 7.0	12	16	12	12	6	17	14	13	13	10
7.0 to 8.0	10	2	6	3	11	10	7	5	10	14
8.0 to 9.0	6	4	1	4	4	4	7	3	5	2
9.0 to 10.0	2	2	0	0	4	5	2	2	4	3
More than 10.0	2	2	0	0	4	5	2	2	4	3

Total refraction of eye K derived from formula:

$$K = \frac{1.336 - x(z - w)}{z - 0.7485wx(z - w)} - y$$

where x = lens power (D), y = corneal power (D), z = axial length (m), w = depth of anterior chamber in metres (+0.0024).

[illegible]

Fig. 6 Distribution of computerised ocular refraction when the components are (a) uncorrelated and (b) correlated.

lation. Both distributions are shown in Fig. 6 (one run of each).

There is therefore clear confirmation of a correlating mechanism, a mechanism which the simulation model faithfully reproduces. The model itself is also validated. It will be noted that the mean value of K in most runs is small and positive, as one would expect.

Heredity in refraction

The teaching on refraction that prevailed during the second half of last century appears to date back to Donders, who presented his records of refraction in a graphical form. This was a rudimentary curve of distribution in which emmetropia dominated and on which he remarked: 'From this figure it is very clear that the emmetropic is the normal eye.'⁶ By implication hypermetropia (which he had distinguished from presbyopia) and myopia were abnormalities: one was an underdeveloped and the other an overdeveloped eye.

The immense literature on myopia and its causation that these views stimulated was largely speculative. Various environmental causes were incriminated to explain the development of myopia in childhood. These normally centred around schooling, where poor illumination, faulty posture, and difficult script (such as the German and Hebrew) were involved. As distinct from these, physiological factors were also blamed: the accommodation which was involved in learning; the convergence in all such tasks with resultant compression and distortion of the globe by the extraocular muscles; and traction by the superior

oblique muscle on the posterior pole of the eye—all these found their proponents. To explain why, under similar conditions, these environmental and physiological causes were effective in only a minority of children the hypothesis of a predisposing cause such as a postulated low orbit (leading to deformation of the globe) and a short optic nerve (limiting ocular movements) had to be introduced. In this welter of postulates and assumptions some hard observations were not lacking, the most significant being the recognition that there was a familial background in many cases of myopia.

Steiger emphasised the familial factor, and in the opening years of the century, when Mendelian inheritance was rediscovered, he broadened the discussion by insisting that not only myopia but all refraction was genetically determined. He stressed that the components of refraction—each with its own mode of inheritance—had to be studied to disentangle the complexities of inheritance. His view that multifactorial inheritance was involved was an early application of the ratios Mendel had established for the presence of more than one factor. But the early stages of human genetics had to overcome the difficulty that, while monofactorial inheritance in man was known in disease, multifactorial inheritance was not very definitely established. The present-day concept of polygenic inheritance in man was emerging but slowly.

Steiger's achievement

Breaking with constants and categories, Steiger freed the teaching on refraction from the environmental and mechanistic strangleholds that had pervaded in

the nineteenth century. But his biomathematical approach, coming as it did before genetics had revealed the immensities of variation, showed 3 substantial deficiencies. In the first place, in stressing that refraction as a whole follows the normal distribution pattern followed by its components, he overlooked the fact that in biology the whole is more than the sum of its parts, and that integration into an organ is a different process from that which shapes the individual components. Profuse as nature is with its raw material, it is selective in the end results.

Secondly, Steiger barely touched the extreme degrees of refractive errors. It is now clear that refractions over $-4.0D$ and over $+6.0D$ are determined by anomalous lengths that fall outside the range seen in emmetropia.⁷ These clinically important refractive errors are not accommodated in Steiger's scheme. In fact they do not fit into his scheme, and it is only recently that they have been recognised as a distinct group not only optically uncorrelated but also genetically specific.^{8,9}

The third deficiency in Steiger's work stems from his difficulty with the higher refractive errors. He was right in his assumption that all refractions are genetically determined, but critical evidence has come only recently from findings on refraction and its components in twins⁸ and in family studies.⁹ And it is clear now that all refractions other than those of extreme degrees are indeed determined polygenically, but the extreme refractions with anomalous axial length are inherited in a strictly monogenic manner.⁹ It is no mean achievement that Steiger should have sensed an almost complete solution to a difficult genetic problem.

The deficiencies in Steiger's work are mere blemishes in a monumental achievement that carries

the mark of genius. He died in 1920 at the age of 58. Siegrist, in an obituary notice in the *Neue Züricher Zeitung* on the humble local school oculist, considered Steiger's monograph with its wealth of corneal measurements as ranking with Javal's *Mémoires d'Ophthalmométrie*. Apparently none of Steiger's contemporaries found this valedictory notice incongruous. Forty-four years later there was a sensitive appreciation of Steiger's life and work by a Swiss critic.¹⁰ Today, 60 years after his death, Steiger's place in ophthalmology is secure; it is with Helmholtz, von Graefe, Donders, Bowman, and Nettleship.

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