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THE OCULAR MANIFESTATIONS OF RIBOFLAVIN DEFICIENCY*

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Ten years ago, Bessey and Wolbach[1] described and explained the corneal vascularization which occurs in rats maintained on a riboflavin-free diet. Shortly afterward Sydenstricker, Kruse, and their associates[2] published their observation of the same phenomenon in man. Unfortunately there is still some argument about its clinical significance. This is mainly caused by a faulty discrimination between normal and pathologic aspects of the vascular system of the limbus and the failure to recognize different types of true corneal vascularity. It is the purpose of this paper to review critically the different opinions and to compare them with my own experiences. Another and more important purpose is to present an explanation for the apparent incompatibility of findings regarding the ocular manifestations of riboflavin deficiency.

WHAT IS CORNEAL VASCULARITY?

Unmistakable corneal vascularity has been described by Bessey and Wolbach,[1] Sydenstricker and associates[2] and Kruse and associates.[3] Much trouble could have been avoided if Sydenstricker, Kruse, and their group had not included "conjunctivitis," "engorgement of the limbic plexus," and "circumcorneal injection" as early signs of aribo-flavinosis, and if some of the subsequent workers had first made themselves familiar with the variations of the limbus as visualized by the slitlamp.

Corneal vascularity is a condition in which newly formed blood vessels leave the limbic plexus and centripetally enter the subepithelial space of the true cornea. This state, and nothing less, is acceptable as corneal vascularity.

The limbus is not a line but a band about one-mm. wide, consisting of the wedge-

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shaped edge of the sclera which overlaps the corneal tissue, the cornea being inserted into the sclera like a watch glass. The sclera bevels off from full thickness at the equatorial border to extreme thinness at the corneal edge. This area is plentifully provided with blood vessels which comprise the limbic plexus.[4]

The limbic plexus is not always visible in the living eye as the vessels frequently are empty, in which state they do not show. The plexus responds readily to mechanical or chemical irritation, which causes a conjunctival hyperemia and opens the vessels of the limbus. The state of filling of the limbic vessels may change from hour to hour, depending on light, wind, dust, accommodative strain, and so forth. They can be demonstrated clearly, according to their state of filling, with the aid of the slitlamp. The true cornea is usually sharply demarcated from the limbus, particularly when the light reflected from the iris is used, and normally is devoid of blood vessels.

Hyperemia of the limbic plexus, circumcorneal injection, and engorgement of the limbus have been regarded as early signs of ariboflavinosis. While this certainly may be the case, these signs are entirely nonspecific. The sprouting out of new vessels from the limbic plexus is preceded by engorgement and increased activity of the plexus itself. This condition will or will not respond to riboflavin administration, according to its etiology. For practical clinical purposes it must be insisted upon that new vessels must appear in the cornea before one can speak of corneal vascularity. The importance of a correct anatomic diagnosis has been emphasized before by Gregory,[5] Ferguson,[6] Mann,[7] Stern,[8] and others. It is stressed here again because much of the confusion regarding the causal relationship between riboflavin deficiency and corneal vascularity has been due to the various interpretations of the extent of "corneal invasion."

Let is be stated then once more that circumcorneal injection—that is, hyperemia of the blood vessels of the limbus—although it may represent the first stage of corneal vascularization, is a completely nonspecific response of the eye to any kind of irritation, or to an intraocular upset like iritis or glaucoma. It is entirely useless for the evaluation of an individual's state of nutrition with riboflavin.

Corneal vascularity itself is not always easy to detect. It requires considerable experience with the slitlamp to recognize the sometimes very fine and short capillaries which often can be seen only by indirect illumination in the light reflected from the iris.

The corneal vascularization of well-developed riboflavin deficiency always occurs in the entire circumference of the cornea. This fact is important for correct diagnosis. In view of the fact that riboflavin deficiency is nutritional and present throughout the organism, it is to be expected that both eyes show equally the resulting vascularity. This is the case in the great majority of patients. But there are exceptions to this rule. It will be shown later that disease or trauma may condition vascularization in one eye when the degree of riboflavin deficiency is insufficient to produce signs in the other eye.

Differential diagnosis of corneal vascularization

Invasion of the cornea by blood vessels occurs in several pathologic conditions of the eye. In rosacea keratitis it is usually accentuated in the upper quadrants of the cornea, but may also extend all around the limbus. This disease, however, is characterized by subepithelial infiltrates which increase in size and progress toward the center and the deeper layers of the stroma. Eventually the epithelium over these areas becomes eroded and the ensuing ulcers attract blood vessels which form a vascularization of the fascicular type.[9] This clinical picture is different from that of the ariboflavinotic type of corneal vascularity, and reports by Johnson and others[10] that rosacea keratitis responds to riboflavin therapy have been vigorously con-

tradicted by Fish,[11] Wise,[12] and others. It may be that a coincidental riboflavin deficiency can aggravate a rosacea keratitis, in which case some benefit may be derived from riboflavin administration.

Vascularization of the cornea also occurs in phlyctenular (eczematous) keratitis. In this condition the arrangement of the vessels is similar to that described in riboflavin deficiency. However, this fact presents no difficulties in the differential diagnosis. It has been shown by Landau and me[13] that phlyctenular keratitis is really a riboflavin deficiency conditioned by an allergic reaction. These cases respond so well to riboflavin medication that this alone should be proof enough for this contention. They display, in addition, low riboflavin excretion levels in the urine, and it can be categorically stated that phlyctenular or eczematous keratitis is a clinical variation of riboflavin deficiency in an allergic subject.

Another condition characterized by invasion of the cornea by blood vessels is trachoma. The trachomatous pannus is, of course, of an entirely different type, occupying only the upper half or third of the cornea. It cannot, therefore, be confounded with the ariboflavinotic vascularization. Landau and I[14] have shown that certain cases of clinically healed trachoma display a degree of activity of the pannus which is out of proportion to the conjunctival lesions. These cases show an additional corneal vascularity in the remainder of the circumference, and this condition responds readily to riboflavin administration. We take this as proof that this type of pannus is an exacerbation due to a riboflavin deficiency; the preformed channels of the originally collapsed, inactive pannus vessels are opened again when riboflavin deficiency causes the limbic plexus to throw out new vessels in the previously unaffected parts of the cornea.

These are the only known clinical conditions in which corneal vascularity more or less resembling that of riboflavin deficiency can be observed. As has been shown, the resemblance is only superficial in the case of rosacea keratitis, except possibly in some instances where the vascularity may be due to a primary riboflavin deficiency which prepared the cornea for an invasion by blood vessels in the presence of a conditioning factor.

Corneal vascularity of the same type as in riboflavin deficiency has been found to occur in rats on a diet deficient in one or more of the essential amino acids (tryptophane, leucine, phenylalanine, and so forth). Nothing much is known as yet of the pathogenic mechanism of this vascularization; it is probably due to some metabolic upset in the corneal cells caused by the absence of these substances. A similar type of vascularization occurs in zinc deficiency,[15] and sodium deficiency.[16] Although this vascularity is of the same type as that in ariboflavinosis, clinically it usually need not be taken into consideration in the differential diagnosis because it seems to occur only in experimental conditions which, it is believed, are unlikely to be encountered in clinical practice.

Specificity of corneal vascularization

It is necessary to discuss here critically the statement of a number of workers that corneal vascularity is not an unequivocal sign of riboflavin deficiency. Stannus,[17] in expressing this opinion, quotes several authors to whom he attributes statements to this effect. In checking the original papers one finds, in some instances, Stannus's interpretation of the authors quoted open to criticism; in other cases it seems likely that Stannus read into these papers a meaning which the authors never wished to express.

Goldsmith[18] looked, in a nutritional survey of patients in a charity hospital, for "vascularization of the cornea (grossly visible)" and "conjunctival congestion." She writes: "In a number of our patients injection of the conjunctival vessels appeared as a band extending across the bulbar conjunctiva from the xanthus to the limbus. . . ." This description and the expression "grossly visi-

ble" indicate that this author did not use a slitlamp, and what she describes is, of course, not corneal vascularity.

Kodicek and Yudkin[19] have made a survey of school children and considered in it encroachment of vessels upon the clear cornea as a sign of riboflavin deficiency; they write: ". . . Invasion of the cornea itself by capillaries from the limbus is more likely to be of definite dietary origin, and by considering only children showing this we think we are dealing with cases of real deficiency . . . the degree of vascularization which we have taken as abnormal is almost certainly due to riboflavin deficiency. . . ."

Keath-Lyle and others[20] say: "Vascularization of the cornea is not necessarily evidence of deficiency in the diet. . . . Riboflavin is not the only nutrient concerned in the prevention of corneal vascularization." However, these authors included hyperemia of the limbic plexus as corneal vascularity, and examined in their survey only "the nasal, inferior and temporal quadrant of both eyes . . ."—that is, they were not concerned with vascularization in the whole circumference. Moreover, they arrived at a score of vascularization by adding the number of corneal capillaries in each of the three quadrants observed, thus accepting cases of localized vascularity into their total number. It has already been pointed out that this is inadmissible.

Machella and McDonald's paper[21] concerns nine patients who failed to improve with riboflavin although they "showed the accepted picture of riboflavin deficiency." Two of their cases had chronic conjunctivitis without corneal vascularity, two others "vascularizing keratitis" which is not further described and may be anything, and five cases suffered from rosacea keratitis which does not respond to riboflavin.

Pett[22] failed to cure 43 percent of persons with corneal vascularity with riboflavin. His method, however, is objectionable; in his investigations "a record was made of the appearance of the limbus between 5 and 7 o'clock of the circle" and the rest of the cornea was disregarded.

Another authoritative paper quoted by Stannus is that by Tisdall, McCreary, and Pearce[23] who found a high prevalence of corneal vascularization in a group of healthy pilots of the Canadian Air Force; they reproduce excellent photographs of the cornea which, however, leave some doubt whether the actual cornea was invaded by blood vessels in all their cases, and it is not unlikely that they included some cases of limbic congestion. All the same they arrive at the following conclusions: "(1) The incidence of vascularization of the cornea among healthy young adults in Canada is surprisingly high and seems to vary with the riboflavin-containing foods in the diet. (2) Riboflavin in large dosage for a period of 2 months decreased the vascularity of the cornea in a large percentage of cases. . . ."

Their actual figures were: progressive decrease in the vascularity in 70 percent and clearing or improvement of symptoms in 95 percent. The administration of placebos in a control group caused no change in the vascularity. They found furthermore that the subjects under investigation were on an average daily intake of riboflavin of only 1.6 mg. It is not clear why Stannus quotes this excellent paper in support of his thesis that corneal vascularity is not always a sign of ariboflavinosis.

Finally, there is a paper by Youmans and others,[24] the result of a mass observation trying to define whether corneal vascularity may not be due to destruction of riboflavin by excessive light. The very object of this investigation indicates that the authors do not question the specificity of corneal vascularity, and in fact they say nowhere that it is not due always to riboflavin deficiency—be it local or systemic.

Scarborough[25] found circumcorneal injection in 34 percent of 204 cases, and in 68 percent of subjects over 50 years of age. He observes correctly that this condition is not caused by riboflavin deficiency, as ad-

ministration of riboflavin by mouth or parenterally did not influence it, and that a therapeutic test is the best criterion in establishing a diagnosis.

Scott[26] includes "circumcorneal vessels" and "invasion of the clear cornea with not more than one arcade" in his cases and it seems doubtful whether even the latter were true corneal vascularity. Wiehl and Kruse[27] investigated the problem within the framework of their work on nutrition and stated: ". . . but those with capillaries noted as having extended into the cornea in one or both eyes have been counted as cases of aribroflavinosis. . . ." It is obvious that they accept this as an unequivocal sign of ariboflavinosis and do not doubt its significance.

Sandstead[28] found 80 to 93 percent of vascularization of the cornea of children and young adults, but here again it is open to doubt whether he distinguished between true vascularity and circumcorneal congestion.

Stannus also mentions a paper by Wilson which I have been unable to find.

Those then are the workers on whose papers Stannus and others depend when they argue that corneal vascularity is not an unequivocal sign of ariboflavinosis. Who are the other workers taking the same attitude?

Anderson and Milam[29] doubt that milder degrees of corneal vascularity are due to riboflavin deficiency. The cases of corneal vascularity on which they base their opinion were, however, on an average daily intake of 0.5 to 1.3 mg. of riboflavin which is below the recommended daily allowance of 1.5 to 1.8 mg. It is doubtful, furthermore, whether these cases of "milder degrees" were not really only limbus congestion.

Boehrer and others[30] were unable to find corneal vascularity in six subjects who received 0.47 mg. of riboflavin daily for five weeks. This is too short a period of time to allow vascularization to make its appearance, and, in addition, the authors were not sure of the coöperation of their subjects.

McCreary, Nicholls, and Tisdall[31] make an important point which, however, does not disprove our thesis: they found that corneal vessels of ariboflavinosis never disappear entirely but become invisible when they are anemic and collapsed. They can refill at any time in response to a nonspecific irritation. This observation corresponds with that in trachomatous pannus, where the vessels persist as empty channels all through the life of a patient. This fact appears, therefore, to be a potential source of error. Even if it is agreed that corneal vascularity of the type discussed here is originally always due to riboflavin deficiency, a flare-up at a later stage may be caused by a nonspecific reaction to any kind of irritation. In this instance no response can be expected to riboflavin medication.

To summarize: there is no known clinical condition other than ariboflavinosis that produces the type of corneal vascularization seen in riboflavin deficiency. There are indications that the catalytic chain of respiratory processes in the cornea may occasionally break down at a different link—iron, amino acids, zinc—but it seems to happen most frequently at the riboflavin link.[32-36] The ariboflavinotic type of corneal vascularity is, therefore, not an absolutely pathognomonic sign of riboflavin deficiency, but unless a primary or secondary deficiency in these or related substances exists it is definitely indicative of a riboflavin deficiency and will respond to riboflavin. The therapeutic test provides the final answer.

It is necessary to mention here a paper which has caused me grave concern in the process of formulating the above conclusions. The findings of Borsook and others[37] in a survey of Californian aircraft workers are in direct opposition to the reasoning presented here. These workers found some degree of corneal vascularity in 100 percent of their subjects, and failed to observe any influence of riboflavin on this condition. In talking to one of the observers (E. A.), I was convinced that it was apparently true corneal vascularity which the authors had seen. This perturbing state of affairs is at

present entirely inexplicable to me. However, I am convinced that this unique paper must be based on some kind of misapprehension. No similar claim has been made by any of the other investigators on record, and to my mind it is inconceivable that every single individual of the 1,200 subjects of this survey should be an exception to the conclusions arrived at in the previous pages.

Does riboflavin deficiency always produce corneal vascularity?

The results of a survey in the Nutrition Clinic of the Health Department of New York City will be useful in the attempt to arrive at an answer to this question.

Two hundred and fifty new cases were seen in the course of nine months. These cases were predominantly in children and young adults from families in the lower-income classes, and the reason for their attendance was suspected malnutrition found by the school physician. It was to be expected, therefore, that nutritional deficiencies would be found in these persons.

Analysis of their diet on a sample day, usually that preceding the day of their attendance in the clinic, showed in fact that a great proportion of them had an intake of riboflavin of less than the recommended daily allowance of the National Research Council.[38] In this group, 72 children (29 percent) had a riboflavin intake of 80 percent or less. In spite of this, the incidence of corneal vascularity was very low.

Altogether 12 cases of corneal vascularity were seen. Only one of them had had a deficient riboflavin intake on the sample day, the others gave a satisfactory account of their diet, but it must be pointed out that no record could be obtained of their previous diet. What is more relevant is that six of them, who could be induced to attend the clinic regularly, were treated with riboflavin (15 mg. daily by mouth), and that all six responded to treatment. The vascularity became invisible within 2 to 4 weeks.

It is interesting to note, although no explanation can be found for it at present, that three of the cases of vascularization were obese young girls of 7, 12, and 17 years of age. They were put on a reducing diet of 1,200 calories a day which failed to influence the vascularity. When they were given 15 mg. riboflavin daily by mouth the vascularity disappeared within 3 to 4 weeks.

To return to the question: Does riboflavin deficiency always cause corneal vascularity? Of the 250 patients attending the clinic 29 percent had probably a riboflavin intake of 80 percent or less of the normal, and yet only about five percent showed corneal vascularity. This does not seem to provide an affirmative answer to the question.

An explanation may be found in a series of experimental observations. Williams and others[39] found no corneal vascularity in four subjects maintained for nine months on about 0.8 to 0.9 mg. of riboflavin per day. However, neither this amount nor about 1.1 mg. per day prevented a certain degree of depletion of tissue riboflavin as measured by load tests.

Keys and others[40] observed no corneal vascularity in their test subjects who for nearly six months were supplied with only about 0.9 mg. of riboflavin per day, and Davis and others[41] found that 0.6 to 0.7 mg. per day failed to cause corneal vascularization. About 1.3 mg. per day were required to prevent tissue depletion. Sebrell, Butler, and Wooley[42] found that 0.5 mg. per day was followed by corneal vascularization after 3 to 8 months.

It seems, therefore, that a low intake of riboflavin must continue for a prolonged period before anatomic signs of the deficiency and particularly corneal vascularity make their appearance. How low the tissue concentration of riboflavin has to fall in the cornea before corneal vascularization sets in has been shown experimentally by Bessey and Lowry.[43]

In rats on a riboflavin deficient diet the first signs of corneal vascularization ap-

peared only when the riboflavin concentration of the cornea fell to less than 50 percent of the normal. In rats on a completely riboflavin-free diet this stage was reached in three weeks, but in animals on a diet containing only slightly less than the optimum it needed a considerably longer time.

These studies and experiments may explain why our patients with an inadequate riboflavin intake failed to show corneal vascularity. Inadequate as it was compared with the recommended daily allowances, it was still sufficient to prevent a serious depletion of the cornea over the period of observation. The absence of corneal vascularity in patients on a diet moderately deficient in riboflavin is not a contradiction to the statement that riboflavin deficiency always causes corneal vascularization—if it lasts long enough to produce a low enough riboflavin concentration in the tissues.

Why is it, then, that patients with a low but not absolutely deficient riboflavin intake show vascularity of the cornea at all? In view of the foregoing it would require a very long period of time with a very low riboflavin intake, in fact an intake which is extremely unlikely to have existed for any very long period of time among the patients of our survey.

Observations on a group of seven patients (Table 1) not included in the Nutrition Clinic figures provide an answer to this question. These cases were selected in the Special Cornea Clinic of the Manhattan Eye, Ear, and Throat Hospital because of a pronounced degree of corneal vascularity. Some of them had undergone a corneal graft operation which was followed by vascularization of the graft, others had been attending the clinic prior to an intended corneal graft operation for corneal opacities acquired in the course of an earlier corneal disease.

The most interesting cases in this group are Cases 2, 4, and 5. These patients showed corneal vascularity after corneal graft operation while the second eye was normal. Their riboflavin intake was inadequate, but not grossly so, and the riboflavin level in the urine was low but not on a serious deficiency level. Each presented a heavily vascularized cornea which endangered the result of the operation. Such a relatively mild riboflavin deficiency would not be expected to cause corneal vascularization, and the unoperated eye failed to show it. Only the eye which had undergone surgical trauma displayed vascularity.

This vascularity responded most satisfactorily to riboflavin administration. Within 4 to 6 weeks all corneal vessels, even the deeper ones, became less engorged, the circulation became sluggish, "beading" made its appearance which indicates that blood corpuscles are captured in capillaries which close down, and the smaller vessels became

TABLE 1

OBSERVATIONS ON PATIENTS IN THE SPECIAL CORNEA CLINIC

No.	Daily Intake	Riboflavin Output	Operated on		Vascularity in		Result of Riboflavin Therapy
			R.E.	L.E.	R.E.	L.E.	
		(in gamma)					
1	61%	250	x	x	x	x	Excellent
2	39%	135		x		x	Excellent
3	70%	250	x	x	x	x	Good
4	39%	250		x		x	Excellent
5	54%	220	x		x		Good
6	90%	900	Mooren's ulcer both eyes with extensive vascularization				None
7	100%	440	Healed T.B. keratitis with vascularity after graft operation				None

to a large extent invisible. The general injection of the conjunctiva and the photophobia improved to the same degree. This response to riboflavin is highly significant.

Pirie[44] has pointed out that riboflavin fulfills a special function in the organism, and that its therapeutic use can only be expected to be effective when these specific functions are upset. The beneficial effect of riboflavin in corneal vascularity after corneal operations must therefore be due to a riboflavin deficiency which had not reached a degree sufficient to cause spontaneous vascularization; in fact, the unoperated eye failed to show it. It cannot be argued that any healing of tissue injury is accompanied by a growing-in of blood vessels into the traumatized area because this does not apply to the cornea. Vascularization after corneal transplantation is an abnormal and alarming sign which usually precedes an opacification of the graft.

Jolliffe[45] insists that biochemical changes and an impairment of function usually precede morphologic changes in deficiency conditions. Corneal vascularity is a relatively late result of these changes and the expression of a well-established deficiency. It represents the response of the organism to a degree of impairment of function which makes the continuation of normal metabolic processes impossible.

A deficiency disease may not be caused primarily by an inadequate diet but by interference with absorption or utilization of the essential nutrients, or by increased requirement, destruction, or excretion. Jolliffe has termed these states "conditioned" malnutrition.

In the case of corneal vascularization after surgical trauma to the cornea, the trauma can be regarded as the conditioning factor. The healing process after the operation causes increased activity of the corneal epithelium and stroma with a correspondingly higher oxygen requirement. In the presence of a nutritional deficiency of riboflavin, a point is reached sooner or later where the respiratory enzyme, of which riboflavin is a prosthetic group, is unable to keep up a sufficient oxygen supply.

An injured cornea thus requires more oxygen, consequently more respiratory enzyme, and therefore more riboflavin than a healthy one. A degree of riboflavin deficiency which remains subclinical under normal conditions will lead to corneal vascularization, which relieves the respiratory deficit, when pathologic conditions have caused the corneal metabolism to be on a higher level.

It has been shown by Lowry and Bessey[46] that a prolonged deprivation of riboflavin causes a definite handicap to the healing process of experimental corneal lesions.

It can be concluded that a relative riboflavin deficiency, which was too mild to cause either corneal vascularity in the healthy eye or other signs—glossitis, dyssebacia of the nasolabial fold, cheilosis—was the cause for the vascularization of the cornea in the eyes which had undergone an operation. The cases which had an adequate riboflavin intake and a normal riboflavin level in the urine were of a different etiology (Mooren's ulcer, tuberculous keratitis) and failed accordingly to respond to riboflavin medication.

The practical conclusions from these facts are obvious—every patient for corneal graft operation should be investigated as to his dietary habits and riboflavin should be administered pre- and postoperatively as a matter of routine. This routine has been followed with advantage by Franceschetti[47] and Rosso.[48]

The conditioning factor can assume different forms. While examining unselected patients in Palestine, I found a considerably higher prevalence of ariboflavinotic corneal vascularity among them than among the selected, malnourished patients of the Nutrition Clinic of New York City Health Department. I do not believe that this is due to a less satisfactory diet among the patients in Palestine; the very high incidence of seasonal Koch-Weeks conjunctivitis and trachoma in Palestine probably puts an extra strain on

the corneal metabolism and represents a conditioning or precipitating factor for the corneal vascularization.

The same applies to phlyctenular keratitis as shown by Landau and me; in this instance a subclinical riboflavin deficiency is conditioned by an allergic reaction and results in the so-called phlyctenular pannus which is nothing but the typical vascularization of ariboflavinosis. In a case report on a patient who had developed heavy vascularity of the cornea caused by a serious riboflavin deficiency conditioned by superficial corneal injuries I[49] arrived at the same conclusions. And finally, I observed a patient with maximal vascularization of the cornea in chronic riboflavin deficiency, conditioned by an acute sore throat with high fever which increased the riboflavin requirements of the organism.[50]

This, then, is the answer to the question why not every case of nutritional riboflavin deficiency displays signs of corneal vascularity. Unless the deficiency has reached considerable proportions, or *unless a conditioning factor comes into play,* corneal vascularity will fail to appear.

Summary

1. Corneal vascularity is a condition in which newly formed blood vessels enter the normally avascular corneal periphery; limbic congestion and circumcorneal injection may be early stages, but they are nonspecific and not pathognomonic for ariboflavinosis.

2. Corneal vascularity of riboflavin deficiency is of a type not observed in any other known clinical condition and is pathognomonic.

3. Riboflavin deficiency always causes corneal vascularity—if it lasts long enough to produce a low enough riboflavin concentration in the tissues.

4. The appearance of corneal vascularization may be precipitated by conditioning factors such as chemical or mechanical trauma to the cornea in the presence of a sub liminal riboflavin deficiency.

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Ophthalmic Miniature

To George Whately

Passy, 23 May, 1785

. . . By Mr. Dolland's saying that my double spectacles can only serve particular eyes, I doubt he has not been rightly informed of their construction. I imagine it will be found pretty generally true, that the same convexity of glass, through which a man sees clearest and best at the distance proper for reading, is not the best for greater distances. I therefore had formerly two pairs of spectacles, which I shifted occasionally, as in travelling I sometimes read, and often wanted to regard the prospects. Finding this change troublesome, and not always sufficiently ready, I had the glasses cut and half of each kind associated in the same circle. By this means, as I wear my spectacles constantly, I have only to move my eyes up or down, as I want to see distinctly far or near, the proper glasses being always ready. This I find more particularly convenient since my being in France, the glasses that serve me best at table to see what I eat not being the best to see the faces of those on the other side of the table who speak to me; and when one's ears are not well accustomed to the sounds of a language, a sight of the movements in the features of him that speaks helps to explain; so that I understand French better by the help of my spectacles. . . .

B. Franklin.