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ENLARGING VISUAL FIELDS AND REDUCING BLIND SPOTS

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I have been a user of syntonics of several years now and have found it extremely valuable in helping me in my practice of optometry. As a testimonial to this fact I shall review several cases- -which many of us find are run-of-the-mill cases. Of course, it is most interesting in any of these eye problems to have been of assistance to the patient; but, of even greater interest is the knowledge of why the precise procedure used was of the benefit that it was, and the manner in which this improvement in the condition was brought about. We have all heard from Spitler his theory of how the action of the photons discharge from the one neuron to the other, and I think it would be very interesting to review what other authorities have to say about the physiochemical imbalance which results in a disturbed relationship in the neurological set up. I have chosen as my criterion the normal and abnormal physiolchemical relationships as revealed by the perimetric and campimetric examinations. I believe that the work that Evans has done in his research of angioscometry has fitted in admirably with the work that Spitler has done, and with your permission I shall quote to a great extent from the work and the references that Evans has compiled.

Evans[1] says, "We must recall that edema in the pathological sense of the word is the product of a very complex series of events[2]. The main features in its mechanism, according to McLeod[3]are: 1. Diffusion pressure. 2. Capillary blood pressure. 3. Osmotic pressure of blood proteins. 4. Differences of electrical potential. 5. Variations in the permeability of the membranes concerned." Please note feature No. 4 again. I will repeat it. Differences of electrical potential. Does this work in with syntonics or not?

Wegner[4], using Starling's heart-lung preparation and revival of the visual purple as the index, has shown that after being deprived of blood – as in death – for 22 minutes, the eye regains its function, but not after 45 minutes.

Evans further states -vide supra-[5]: "It is obvious that we cannot relate scotometry to these various entities, nor would it be desirable to do so, for the clinical aspects of the present studies would become obscured by the effort. It is significant to recall a statement from the classical studies of Barlow[6] who says: 'Edema is mainly due to two factors: 1. The starvation of the tissues by loss of the nutrient blood supply, and 2. The accumulation of waste products of their own metabolism…. The amount of lymph which escapes from the vessels is determined by the needs of the tissues rather than the condition of the blood vessels.' When looked upon in this way the increase of tissue fluid in pathologic conditions is simply an exaggeration of the physiologic. The main factor emphasized in the production of the defects just reviewed – the widening phenomena of angioscotoma – is venous stasis but it equally obvious that a great many less conspicuous influence should be considered in the final analysis. Nevertheless, venous stasis is common to all, the cell of the tissue for an excess of fluid follows venous stasis very promptly in the retina. This is necessarily so when we recall the high rate of retinal metabolism.[7]

To be sure, the nerve fibers in general, and even the cell body, can be deprived of oxygen for quite a long time and still recover – of Reference to Wegener supra – but the synapses are much more sensitive.[8] We know from, a vast amount of experimental work that the conductivity of the synapse can be altered by many agents and conditions.[9] The most classical of these are strychnine, nicotine, lack of oxygen, and particularly fatigue. In fact, the susceptibility of the synapse to fatigue apparently plays a major role in safeguarding the visual apparatus by allowing an interval for the removal of waste. Moderate degrees of circulatory insufficiency.[10] - Fischer maintains that the reduction in oxygenation acts chiefly by the increased production of acids which greatly increase the affinity of the tissue colloids for water and at the same time alter the colloidal state of the capillary endothelium, and thus increase capillary permeability - and this insufficiency – would present an easy step towards angiopathic retinal disease and produce - a scotoma very similar to those already shown. It is an exaggeration of the physiologic type. In more advanced cases, such a defect would practically obliterate the central field for minute object would practically obliterate the central field for minute objects, but larger objects would still demonstrate the angioscotoma until such time as the edema became quite massive and easily discoverable by the ophthalmoscope."

Evans further states[11]: "The central field thus furnishes a means of demonstrating the effect of minor degrees of uncomplicated cardia embarrassment which," according to Yater and Wagener, "provides no evidence through ophthalmoscopic examination." "The call for excess of tissue fluid," he adds, according to H. G. Wells[12], "to combat and repair is a common finding when inflammatory processes are going on. The development of a tubercule – of a scotoma- immediately adjacent to the nerve head may present the appearance of swelling of the optic disc. The scotoma in such a case will be helpful in making an early diagnosis of retinitis juxtapapillaris though the classical sector defect of fiber damage may not develop until a later date when atrophy replaces the early functional depression associated with edema." Shade, quoted by Wells, points out that in inflamed areas the H-ion concentration may be increased to fifty times the normal. This is most significant when we recall that the active retina is already acid in reaction."

Evans also states:[13] "Dilation of the perivascular system in an effort to withdraw waste and supply nutriment is apparently related to the wedge-shaped defects and widened angioscotoma. The fanning out toward the periphery is suggestive of a peripherally located – inflammatory process …. a more restricted wedge-shaped defect may persist in the region after the atrophic stage is reached…. The angioscotoma nearest the lesion shows a widening and fanning out so that a characteristic wedge-shaped defect results with its apex toward the blind spot and connected to it by a widened angioscotoma. This finding is most typical in the purely mechanical forms of edema, whereas the classical arrangement is departed from more and more as edema is less and less the dominating factor. Wedge-shaped defects are of course not entirely limited to lesions that are angiopathic in origin. The classical quadrant and hemi-field defects of pathway lesions are really wedge-shaped defects…. It has been pointed out defects with the apex pointing towards the blind-spot are ocular and angiopathic in origin, but where the apex is toward the macula the lesion is primarily in the pathway. The picture is rendered atypical, however, where increased intracranial pressure is superimposed."

Much though I have quoted from Evans, I feel that I must continue to quote him for some little time, as this man has devoted an immense amount of work on this subject and has complied a monumental amount of evidence in support of his theory was those of you men who have read his book

have found out. His entire theory works out so beautifully with the theory of spectrum therapy, the different types of ionization created by the different bands, the trouble at the synapse, and the fact that although he does not go into treatment of any of his cases, merely presenting them as evidence yet by taking advantage of the facts as he presents them, and applying syntonics as Riley taught us, we find that many many patients are grateful for the benefit which they have derived from there syntonic applications, and the evidence is in the field charts - - because we find that if the patient has receiving the right kind of therapy, whether it is by lenses, as the case of B. D. will show; by medical attention, as we do not show in this series of cases as we are not discussing that at this time, although it must, of course be kept in mind; by remedial reading treatments, as the case of B.M. will show, on which syntonics was also used, but only to a slight extent; or wholly by syntonic applications, as the majority of these attached cases will show; I repeat, if the patient has received the right kind of care, then the results will be conclusively shown, and shown as dramatically as we will shortly show by means of reproductions of the field charts.

I have used the word "angioscotoma" and "angioscotometry." Evans has coined them, and their meanings are as follows: A scotoma in which the retinal vessels seem to project. More closely defined[14], it is a system of field study in which the defect is angiogenetic in origin. It is further explained in the hypothesis that he offers. Furthermore, he uses minute white objects to map out the involved areas. As will be shown, he maps out the area affected by each minute blood vessel which will not permit the objects he uses minute blood vessel which will not permit the objects he uses – graduated from 0.25 mm up, and accurate to within 0.01 mm either way-to be seen. I have not used that small a stimulus, as I believe that I have found the $1/2^{o}$ white and color stimuli accurate enough for my purposes. I do not mean to decry his method, but, inasmuch, as mine works so satisfactorily for me, I think I shall continue to use it for clinical patients in my own office practice, and possibly combine the use of his precise method in research work. Also, he does not mention Brombach, and the important work that Brombach has done in helping differentiate between different types of toxemias by means of the chromographic studies which undoubtedly, we have all found so useful. Brombach's theory does not coincide at all with Evans' theory as to the enlargement of the blind spot. In fact, in his booklet "Blind Spot Measurements and Remedial Reading Problems" -1937- he states this: "The following standard cases appear to demonstrate the importance of enlargements of the blind spot not associated with disease, but resulting from transitory visual aphasiam, the apparent reason appearing to be a desire to eliminate double vision or abnormalities in binocular perception. There is no doubt in my mind that many cases of young people present reading problems as a result of the reduced usable area between the blind spots and the fixation point. This will naturally reduce speed and efficiency of reading, due to improper perception of groups of letters and words and may produce symptoms which resemble word blindness … Enlarged blind spots reducing the area of visual perception will not permit satisfactory results in intelligent reading unless their causes are analyzed and eliminated. A very simple procedure, occlusion of one eye for a few days, will establish the status of enlarged blind spots.

1. If they are physiologic in character and voluntarily produced by the higher centers of the brain to permit comfortable vision, they will reassume normal shaped when the control tests are being made.
2. If they are the results of pathology, no change in relative size will be observed.

The question arises in my mind as to the main factor which has caused this phenomenon to take place. In a letter to me, Brombach states, "I firmly believe that they are a part of the defense mechanism put into practice whenever binocular instability interferences occur with visual comfort. How this is accomplished is unknown to me, and I believe that laboratory investigations have not as yet given a define clue…Enlargements of the blind spots due to functional activity recover after the occlusion of one eye, while those due to pathological activity…. respond only to therapeutics."

Could it not be that there is some phenomenon, physiological and not psychological in character which takes place – and results e.g. in a slight papilledema and thereby interferes in the transference of the slight impulse between neurons – whether located in eye, per se, or brain- i.e., at the synapse, and by means of our syntonic applications we build a better pathway for the discharge of this neural energy; then if we have made no other changes in the environment of this individual, and he continues to read and does not manifest this enlargement phenomenon, we will be able to maintain that the entire phenomenon has taken place because of the physiological involvement. Basing our hypothesis on this assumption, is it not probable that the occlusion of the eye gives the body a change to clear up the physiological cause of the interference at the synapse, since ordinarily, if the act is repeated the same phenomenon occurs; whereas, after the correct syntonization the enlargement of the blind spot phenomenon is not repeated. I do not particularly care whether Brombach's theory works or Evans'; different. I do know that several cases will show marked changes in color fields due to exogenous toxemia, such as Mrs. J. L., with the half cup of coffee, and also as B. H. with the same change; endogenous toxemias such as menstrual flows in B. W. and Mrs. J.V.L.; changes in the blood chemistry in anger in B. D. Also, inasmuch as no "authority" outside of Wold of Marshallton, Iowa, even mentions the use of syntonics, although he credits it in aiding a blind man to see again, and then he does not think that it helped cataracts very much - - again, I repeat, I shall continue to use that which I can have shown to me is correct, whether authorities praise it or condemn it. I know that you, too, have felt the same way about it, or you, too, would not be using syntonics and get the results that you do get.

Inasmuch as Evans has shown us much research on angioscotometry and has tabulated his findings so nicely I shall continue to quote him. In regard to his hypothesis of his theory of angioscotometry he tabulates it in this manner:

"a. MINUTE ANTAOMY"

One must begin the summary by recalling the neuron chain of retinal structure.

The first, a rod-cone link, forms a synapse with the outer filament of the bipolar cell, the inner filament of which forms a synapse with the ganglion cell. The axons of the ganglion cells build up primary centers in the brain. It will be remembered that the synapse is that "organ: in the neuron chain which regulates the force of the transmitted nerve impulse".

> It has been seen, as in the brain, that the cells of the retina and their processes - with evidence lacking on the rods and cones – are surrounded by a fluid space; these are built up into a perineural space as the fibers of the cells form fiber-bundles. The perineural spaces empty for the most part into the perivascular spaces which pass with the vessels through the optic nerve head into the body of the nerve. It is not clear whether or not the fluid from the perivascular space empties into the vaginal space, about the perivascular space probably follows the vessel through the membranes of the nerve so that the fluid is discharged into orbital channels. The significant features in this course are that the perivascular space is exposed so the inelastic

resistance of the lamia cribrosa and to the pressure variations in the vaginal space – of the spinal fluid- and orbit.

"b. FUNCTIONAL RELATIONS"

We will begin by assuming a relatively static condition of all the mechanism concerned. Let us say that we are to study a healthy young adult physically and mentally at rest, and that the eye is in a state of moderate light adaptation … In view of evidence presented by physiologists we accept the vitreous as a storehouse of nutritive material - - particularly sugar, which is used by the inner layers of the retina - - and a rapidly acting reservoir of fluid. This fluid may pass into the retina as far as the synapse between the outer ends of the filaments of the bipolar cell layer and the ends of the filaments from the rod and cone layer. This fluid does not supply the rod and cone layer which, because of its extremely great metabolic rate, needs the entire choriocapillaris blood-bed to meet its demands. The vitreous fluid, sometimes called vitreous aqueous, upon entering the retina becomes the so-called tissue fluid and comes to occupy the pericellular spaces, the perifibrillar spaces – about the cell fibrils-, the perineural spaces -about the fiber-bundlers-, and the perivascular spaces through which it drains through the optic nerve head and nerve to the vaginal space of the nerve into which it flows to mingle with its homologue, the cerebro-spinal fluid. Or, as previously suggested, it may empty into the tissue spaces of the orbit…. This flow…. Is probably a sort of tide-like ebb and flow, the rate and volume of movement depending on variations of retinal demand. *Samojloff referred to the idea that the accumulated fluid of the perivascular system may press on adjoining tissue in glaucoma. He presented no evidence, however.[17]- That this posterior drainage route conveys a relatively small proportion of the fluid content of the eye is obvious. Priestly-Smith suggested about one-fiftieth of the entire amount; but if one stops to realize that this system needs only to meet the nutritive demands of the less active layers - - conductive - -of the retina, and that it may accommodate much larger amounts of fluids in disease conditions, one will see that its importance is nonetheless vital. How, then are we to explain the extreme sensitivity of the retina when this system is interfered? In the chapter on retinal edema we pointed out that the synapse is the most easily interrupted point in the neuron chain and that it is designed so to act as a sort of fuse to protect the much more refined rod and cone layer. It is not more sensitive to response, for it is not able to respond to light at all, but it is more sensitive to deleterious agents, for which purpose it is especially adapted. Now does the aqueous element of the vitreous enter the retina-tissue spaces?

We can postulate pores or ostis in the internal limiting membrane or we can assume that osmosis is capable of performing the task. The process is not necessarily reversible. The retinal fluid does not normally return to the vitreous. It is not even necessary to postulate a vitreous source for the fluid as it can, and probably does, in part originate from the blood of the retinal-vessel system. We do have evidence that the vitreous is a contributor[18] however, and Magitot[19] has reiterated his opinion that the choroid, retain, and iris are directly responsible for the production of this fluid.

Pressure upon the globe forces more fluid into the perivascular space system through simple increase of the hydrostatic pressure. In this connection one must not fail to keep in mind not only osmotic pressure but even gaseous interchange." Contraction of the fibers of the astrocytes - - which send footplates to form the wall of the perivascular space - - also increases the amount of fluid within

the space by virtue of the greater cross-section thus within the space by virtue of the greater cross-section thus created. Should the blood vessel contract, this again would result in an increased area of the space. On the contrary, should the vessel dilate, fluid in the space would be forced back into the retinal space or out into the posterior channels of escape. Since the escape of fluid is relatively slow as compared to the rapid dilation of the vessel, there would be at least a temporary damming up of the fluid with a consequent edema of the synapse. This interruption of function would result in loss of response of the rod or cone of the corresponding chains.

"c. SPECIFIC APPLICTIONS OF THE HYPOTHESIS"
This we shall delete here.

"d. ORIGIN OF THE PATTERN OF THE SCOTOMA"

Should local perivascular-space drainage be retarded, the perivascular system of that area would alone show defective function through a characteristic wedge-shaped defect. The blending of a number of these would give rise to a sector or even quadrant defect, showing base toward the field periphery and apex toward the blind-spot.

When there is obstruction at the exits through the nerve head, the amount of disturbance of the perivascular spaces about the hook-like vessels from the Circle of Zinn will be proportionate to the rapidity of onset and the degree of the obstruction, the amount of vascular distention, and the rapidity with which the more peripheral perivascular spaces distend by the action by the action of the astrocytes and transmitted pressure. The result will be an enlarged blind-spot through blending of the hook-like angioscotoma…..Had the angioscotoma no other value in a diagnostic sense, its study would still be justified, because it provides a topographic outline which not only helps in the localization of various processes, but also supplies a guide to show whether this or that area has been covered in a particular study." *N. B. Also, whether you are doing the right thing to your patient, syntonically or otherwise. -

CONTINUED IN NEXT ISSUE