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ENLARGING VISUAL FIELD AND REDUCING BLIND SPOTS, Continuation

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"e. SUMMARY OF MECHANISM OR PERIVASCULAR SPACE PRESSURE VARIATIONS.

". . . . . It would seem fair to expect variations in pressure within the perivascular space under two forms:

I. Increased pressure, and
II. Decreased pressure.

"I. Increased space pressure may arise from:

1. Increased quality of tissue fluid, actively with
   a. increased retinal metabolism as after stimulation and with inflammatory processes; or
   b. passively when vitreous aqueous enters retinal tissue and escapes through the spaces.
2. Decreased rate of escape of tissue fluid, as with
   a. obstruction of space by post inflammatory debris, by
   b. mechanical obstruction of outflow channels by intraocular or retro-ocular masses.
   c. hydrostatic back pressure as by increased intracranial pressure and
   d. vasodilation- - particularly venous stasis - - arterial dilation implies rapid blood flow and increased fluid dilation implies rapid blood flow and increased fluid pickup so that space pressure is rapidly lowered.
3. Increased osmotic pressure above that of blood or aqueous vitreous.

"II. Decreased space pressure may arise through

1. Decreased production as with
   a. Absence of stimulation; or
   b. lowered level of response, as by uremia[7], morphinism –
2. Increased outflow as with high rate of blood flow;
3. Increased cross area of space as with:
   a. contraction of astrocytes, or
   b. decrease in vessel caliber.
4. Relatively low osmotic pressure in space as compared to blood or vitreous aqueous."

Evans then continues with this:[20] "The neurogenic hypothesis hinges on our knowledge of the arrangement of the fiber-bundles in the optic pathway and retina; and although there is no doubt but that many of our present ideas will have to be modified, they are at present in a much more satisfactory state than ever before."

He concludes with the following table:

"LOCALIZATION OF LEASIONS ACCORDING TO THE TWO HYPOTHESIS

1. Neurogenic hypothesis - - fiber – bundle - - emphasizes maximum sensitivity of the fiber - - vascular relations secondary and indirect.
   A. The lesion may be located along the
      I. extraocular fibers, in the
      optic pathway - - nerve, chiasma, tract, radiations, cortex: and may originate through
      a. mechanical pressure,
      b. inflammatory processes,
      c. degenerative processes,
      d. secondary to various vascular disturbances as
         1. mechanical pressure
         2. Unreadable

Note: It is obvious that the actual histological structure first addicted is often in dispute – quine for instance,

      e. metabolic disturbances as
         1. in the nerve head, or among the
         2. retinal fiber – bundle, and may originate from
            a. mechanical - -
               1. pressure of tumors
               2. traumatic division of fibers,
               3. soar-tissue contraction -?-
               4. compression at scleral ring - - glaucoma;
            b. inflammatory foci - - theoretic specific toxin;
            c. degenerative processes as:
               1. post-inflammatory neuritis or retinitis, primary atrophies -Lieber's disease-
               2. hereditary - - tapetoretinal degenerations;
            d. elective toxins - -
               1. tobacco - - alcohol - - papilllomacular – location of action disputed-
               2. quinine -?- peripheral

"II. May act from regions which are
   A. Extraocular, as with
      1. mechanical factors, as
         a. increased intracranial pressure,
         b. direct pressure or traction on vessel by tumor, et
         c. orbital affections disturbing lymph and blood flow
      2. inflammatory and degenerative
         a. intracranial vascular disease---- with stasis;
         b. orbital affections disturbing lymph and blood flow

3. systemic disturbances acting indirectly through
   a. general circulatory insufficiency,
   b. toxic disturbances - - metabolic, inflammatory, etc.,
   c. reflex disturbances - - psychic, etc.

B. Intraocular, as with
1. mechanical pressure
   a. on vessels by tumors, etc.,
   b. increased intraocular pressure,
   c. local vascular obstruction - - thrombi.
2. inflammatory lesions - - associated edema;
3. post-inflammatory repair - - debris in perivascular space;
4. locally acting specific toxic bodies - - quine and histamine;
5. local degenerative processes – arteriosclerosis, perivascularitis, etc.,
6. systemic disorders with specific ocular lesions, as blood dyscrasia.
7. systematic disorders with specific toxins acting on the sympathetic hormonal vascular control mechanism – on Rouget cells, etc.-"

Evans also has this to say:[22] "One may thus speculate as to a system of interpretation based entirely on this "fiber-bundle-perceptive-organ-mechanism" -neuroscotometry-,, as differentiated from that of the "vascular-system=perivascular-space-mechanism" -angio-scotometry-.

"The assumption would appear to the writer to be contrary to the generally accepted theories - in general physiology- which hold that the synapse is the most sensitive element of the neuron chain. To be sure, the nerve c fibers in the retinal area are non-medullated and hence more exposed than the same fibers before they traverse the fenestrations of the lamina cribrosa. Moreover, these fibers may be peculiarly sensitive to pressure.

We do not find this sensitiveness to hold true for the intracranial portions of the pathway, or even for the intra-orbital portions of the optic nerve; for in both these structures we have seen release from pressure -removal of compressing growths- result in full recovery of a previously restricted visual field. This may occur after months of continuous and severe pressure. Pressure seems to be accepted as the prime offender by the originators of the neurogenic theory.

The angiogenic theory assumes that the most sensitive element of the neuron chain is the synapse, though it is possible that under some conditions the cell bodies, or even the nerve fibers, may be the most sensitive to a specific or elective to support this idea … Perhaps he weakest point of the neurogenic theory is ha tit has been impossible to correlate a specific field defect with corresponding fiber-bundle damage; and the most obvious deficiency of the angiogenic theory is the impossibility of correlating a particular field defect with direct evidence of synapse damage."

The foregoing data should help in our understanding of the means whereby syntonics does work, and as further help in understanding this I append the following cases:

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TECHNIC USED IN CHARTING FIELDS STEREOCAMPIMETER

ILLMINATION: Special stereocampimeter lamp.

LENSES: Collimating lens in instrument.

FIXATION: Small cross before one eye; before the other eye which fuse to

FUSION : Adjusted on Wells chart first.

N. B.: Eventually, I put all of the prism on one eye – the eye not under test – decreasing the amount until I had the proper alignment. After the one eye was tested, the prism was shifted to that eye and the other eye was examined. Right eye examined first. The reason for this shift of the prism and was to tend to lessen the amount of chromatic aberration and thereby keep the color of the stimulus purer.

STIMULI: Blind spot; 1^{o} white and colors; motion wire enameled white and moved up, down, and inwards toward fixation device. Order; Red, Blue, White, Green, Motion. Right Eye first.

RECORDING: At saturation for white and colors; at perception for motion.

N.B. At a lecture given in Kansas /city by Brombach, his instructions in taking the motion field is this: The standard motion stimulus, which is black wire with a vey small white tip. Is dipped into a can of enamel so the around 2" or 2 1/2" of wire is covered with this paint and then it is left to dry. This is jiggled in a movement perpendicular to the axis in simultaneously from the area of no perception to the area where motion - not white- is first perceived and this is noted.

CAMPIMETER – ATTACHMENT TO BROMBACK PERIMETER

ILLUMINATION: Same as above

LENSES: NONE if myopic up to several diopters, 43.50 as a collimating lens if not very myopic or hyperopic; +3.50 added to hyperopic. Rx if presbyopic.

FIXATION: White mark at center of fixation.

STIMULI: Blind spot by 1^{o} white - - that is then put-a-way; then tested by 10/2 white, blue, red, green, in that order. If the blind spot for that color extends completely around the central area, we continue with that color from the blind spot until we have completed that area, then go on to the next color. Having completed the blind spot, we then bring in the 10/2 stimulus from the periphery until seen. Order: green, red, blue, white.

CHECKING: After having done all of this, we then check for the Seidel sign for color: order Green, red, blue white. Even if the blind spot -scotoma- for color, extends for a considerable distance, we make the perimacular area, there is, of course no use to make this test for the particular color. The direction of checking for this attest is always at right angles to the direction of the blood vessels.

RECORDING: Always at saturation. If the color fades at any time, we must always move the stimulus in more yet.

The attached field charts accompanying these case reports are actual tracings of the original charts and are exactly the same size as the originals. The method for making them is this: The semi-transparent paper is laid over the original chart and tracings made. For comparison with the actual charts I am enclosing the following charts:

1. Bausch and Lomb Stereocampimeter chart
1. American Optical Brombach Perimetric chart
1. American Optical Brombach Campimetric chart

CASE REPORT 1

B. D. male age 19, high school senior, myope. Progressive. Notice size of blind spots and field involvement. Technic used: υω (Upsilon Omega) daily; prism base is placed over each eye, a pair at a time in 1 Δ steps – total of 2 Δ each time an increase was made - "starting with 1, going up to 5, returning in steps of 1 again, increasing the prism as the phoria thru them returned to orthophoric; at infinity, decreasing the same way. Final lens Rx substantially the same as the old Rx. We are having some impacted third molars looked after now and think that he is going to be a pretty normal young man. One morning when he came in for his field chart he was so mad that he could kill, inasmuch as he had thought that someone had stolen something from him; he searched all over the neighborhood for the miscreant. Undoubtably he had the urge and the motive for murder, and his blood chemistry would have shown it - - I did not need a blood chemistry test to know that - - his green field was larger than his red. I asked him to rest for 15 minutes, by that time he quieted down, his blood sugar undoubtedly went down, and his field became normal.

* *

CASE REPORT II

R. G. male, clerk. Intelligent and had noticed when he saw both a vertical and horizontal line, they would not be seen with the same intensity. In other words, he had some of astigmia. His old lenses were -2.50 spheres. He definitely showed cylinders needed, but it was a shifting type, and we could not determine the axes. Notice the green interlacing with red. Compare with the later PM charts, and notice that they are normal. Note progressive reduction of the size of the blind spots. Notice too, the Seidal sign for red in the right eye on April 16 he had a good deal to drink the day before, and it certainly affected him. Among other things that helped produce his excellent condition the end was the fact that during the first week I sopped all nicotine. We also took him off of coffee, and cholate, etc. He is very asthenic, ad the use of
Μυ (Mu Upsilon), υω, αω (Alpha Omega) made his indigestion much better, too all indicating of course, that his biotype was he thing at fault. He finally quietened down so we could give the small cylinder needed, and there was no fluctuation in the axes.