

I'VE GOT A PROBLEM WITH "CONVERGENCE INSUFFICIENCY" AND OTHER "DIAGNOSIS"

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Journal of Behavioral Optometry Volume 11/ 2000/ Number 4

"In optometry, as in other health disciplines, good patient care starts with the right diagnosis. Equally important to assuring a good outcome is prescribing the proper treatment." - AOA News 11/1/99

The term "diagnosis: conjures up an image that all the relevant components have been identified—that their relationships have been recognized and, where existing knowledge permits, the etiology has been understood.

Differential diagnosis somehow suggests a "magna cum laude" in the realm of diagnoses—that out of a conglomerate of overlapping signs and symptoms an extremely specific judgment has been made. One has gotten "to the heart of the matter."

Diagnoses are obviously quite important because treatment protocols are almost always yoked to them. Diagnoses tell you "what's wrong."

But, what if there is a disagreement concerning the relevance of certain factors? What if some clinicians believe that a patient's symptoms are somehow related to his environment or nutrition and others do not? Could that not reflect itself in a difference in diagnoses? So what is a diagnosis?

Diagnosis is defined as the "identification of a disease or condition by a *scientific evaluation* of physical signs, symptoms, history, laboratory tests and procedures." (1)

Webster's Dictionary further adds that it is the "investigation or analysis of the cause or nature of a condition, situation, or problem;" also, that it is "a statement or conclusion concerning the *nature or cause* of some phenomenon." (2)

The Dictionary of Visual Science (3) considers it to be "...the determination of the refractive, muscular, or functional *origin* of the sources of visual discomfort or difficult."

Since diagnoses are dependent upon symptoms, signs, procedures, and history, it seems apparent that they are also dependent upon the operational model of the practitioner and his academic training and experience. A case in point is *convergence insufficiency*. As a behavioral optometrist, I've had considerable difficulty accepting what is fondly known as "CI" as a diagnosis—as an entity to be treated in itself—as *the* problem. I've also felt confused by other so-called diagnoses such as heterophoria, reduced stereopsis, suppression, and so on. It seems to me that these are descriptions, observations, and findings of some problem—in effect, aspects of more intrinsic or global conditions.

In addition, I believe that a diagnosis requires a "collective" agreement concerning its characteristics. Dyslexia is still controversial, in part, because no universally recognized set of characteristics identify it. So—let's discuss CI.

- I. London defines CI as: “the condition in which the exo deviation, either phoria or tropia, is greater for near vision than distance vision.” (4) He further states: “No commonly accepted cut-off criteria for the difference between distance and near deviations are available. A reasonable starting point is 10 of difference between the two measurements...”
- II. In *The Oculorotary Muscles* by Scobee, (5) we are told: “There is no single set of definitions which might be considered classical. One set is as follows:
- “Convergence Insufficiency: if there is either esophoria or esotropia and it is greater at far than at near, there is convergence insufficiency.
- “Divergence Insufficiency: if there is exophoria or exotropia, and it is greater at near than at far, then it is divergence insufficiency.”
- III. In the series, *The Optometric Clinical Practice Guidelines*, produced by the American Optometric Association (AOA), the monograph titled *Accommodative Vergence Dysfunction* (6) provides us with the following:
- “Classic convergence insufficiency consists of a receded NPC [how much?], reduced PFC [how much?], exophoria at near [how much?], and deficiencies in NRA [how much?]. However, not all patients with CI have all of these clinical findings.”
- IV. *Ocular Accommodation, Convergence and Fixation Disparity...A Manual of Clinical Analysis* by David Goss, O.D., Ph.D. (7) indicates the following:
- “In 1897, Duane described four types of binocular vision syndromes:
The four are:
1. Convergence Insufficiency
 2. Convergence Excess
 3. Divergence Insufficiency
 4. Divergence Excess
- Some authors have used variations of Duane’s terms, but we will stay with the original ones which are still useful.
CI: Distance Phoria: Approximately ortho
Near Phoria: High Exophoria [how much?]
- V. Le Chac (8) in the OEP monograph, *Nonstrabismic Vergence Problems*, defines CI as follows:
- “CI is a condition where the exophoria is greater at near than at far.” Diagnostic Findings:
Low AC/A
Positive fusional vergence is low
Reduced NPC
Poor sensory fusion and suppression.
Again, I ask, “how much?”
- VI. Duke Elder, (9) on the other hand, states that “the diagnosis is based on the presence of orthophoria for distance, the periodic increase of relative divergence as the nearpoint is approached, the remoteness of the nearpoint (beyond 9.5cm.), the low prism convergence (below 15), and normal prism divergence.

VII. Cox (10) states that “CI is a *syndrome* of binocular vision dysfunction.” He further states that “clinical findings include an exo deviation at near, very little or no deviation with distance fixation (generally a small exophoria) and a deficit in positive relative convergence. Other etiologic features involve deficient fusional convergence, inadequate accommodative responses or a breakdown of the interaction between accommodation and convergence. However, the diagnosis of CI is complicated by compounding sources of eyestrain such as stress, fatigue, refractive status, etc.”

VIII. The Dictionary of Visual Science (3) (p. 151) contributes the following:

- “1. Exotropia or high exophoria in near vision in association with a relatively orthophoric condition in distance vision, a relatively low increase in convergence being associated with an increase in accommodation.
2. The condition of exophoria or exotropia in near vision, exophoria or exotropia in distance vision being considered *divergence excess*.
3. A condition in which esophoria or esotropia is greater at far than at near.
4. Inability to converge the eyes to the average or normal near point of convergence.”

Unless I am overlooking an important aspect of the term diagnosis, I cannot understand how one can make a *scientific evaluation* of a condition that is so loosely defined, which has no concrete landmarks, and which, clinically has no universal protocol for obtaining the data.

As I initially indicated, treatment regimens are presumably based upon appropriate and accurate diagnoses. It is my belief that too often syndromes, (mal)adaptations, and peoples’ responses to a variety of stressors are being “diagnosed” as the condition(s) to be treated. Where this occurs, the clinical thrust becomes one of eliminating signs and symptoms and identifying it as the “cure or correction.”

Syndrome, by the way, is defined as “a complex of signs and symptoms resulting from a common cause or appearing in combination.” (1) (p. 1047).

Allopathy is defined as a “system of medical therapy in which a disease or an abnormal condition is treated by creating an environment that is antagonistic to the disease or condition, as an antibiotic toxic to a pathogenic organism...or an iron supplement...in iron deficiency anemia.” (1) (p. 40). A case in point is an observation made by Mary A. Lynch, M.S., M.D. 11. (Please see Addendum.)

In like manner, if we are able to move the NPC in or reduce the exo, enhance the PFC, etc., the patient is cured. Give minus to a myope, he is cured. After all, the myopia is a refractive “error,” and are not the glasses, therefore, “corrective” lenses?

Behavioral optometry truly moved away from allopathic constructs—it considered itself “holistic” (12) and searched for “core” etiologies. Nearpoint stress pattern became a diagnosis and myopia a symptom. Twenty to twenty-five percent of all patients referred to me by optometrists have been diagnosed as having CI as the problem. As one attacks a bacteria, I am asked (in a sense) to attack the CI.

If we can establish that CI and a number of other labels are not diagnoses but rather aspects of more global conditions, it may free clinicians to delve into more “bottom line” dynamics and findings in order

to make a diagnosis—to understand what is truly occurring. An understanding of focal-ambient integration and the impact of disruptions in their relationships created exciting clinical protocols and new “tools” as, for example, the use of yoked prisms.

Morse and Jiang, (13) in a paper relating to virtual reality, state: “we speculate that the most likely cause of the reduced accommodation was sympathetic nervous system arousal...Sympathetic overstimulation...could lessen the accommodative response and reduce the gradient AC/A while increasing exophoria at near. We were not able to test this hypothesis in this experiment, but we do have data that support our contention that the increased exophoria at near and the reduced AC/A is due to the reduced accommodation among the symptomatic group.”

It seems to me that the clinical implication of that study is that, were we to deal with this group of patients therapeutically, quieting the “sympathetic arousal” rather than “correcting” the AC/A relationship or exo would be indicated. Sympathetic arousal would, indeed, be our diagnosis—the condition that requires our attention.

Imagine, if you will, a universal understanding of the pervasive impact of vision upon our nervous system and the symptoms/syndromes such stress responses can create. Imagine clinicians probing to understand the trigger mechanisms and predisposing states of specific patients—of truly diagnosing the “dis-ease.”

Imagine a clinical postulate in which elimination of symptoms or syndromes is no longer accepted as a “correction.”

Acknowledgements

My thanks to Dr. John Streff for his assistance and to Drs. John Pulaski and Raymond Gottlieb for reviewing this paper.

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Addendum

“Considering the frequency of asthma, hypertension, hypotension, glaucoma, ulcer disease, and abnormalities of sweating, temperature, cardiac rhythm, respiration, sexual, bowel and bladder function, *it is amazing that the autonomic system gets essentially no direct treatment.* Rather, those symptoms produced by lack of homeostasis of this system have been attacked with a vengeance but with no correction of the problematic system.

“Diabetes mellitus, brainstem multiple sclerosis, Gullain-Barre’s syndrome and infarction are often associated with disorders of the autonomic function. And, our medical response has been reduced to a barrage of pharmacological antidotes: antihypertensives, psychotropic drugs, atropinics, alpha-and beta-adrenergic stimulating and inhibiting agents but in no single case is this treatment directed at the problem, only the symptom. None of this central or peripheral clinical pharmacology addresses the system directly.” (11)