

Viewpoint • I See Models Everywhere

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The title of the present paper is what the first author's PhD advisor, and later long-time colleague, Professor Larry Stark (Figure 1), a neurologist and bio-engineer, frequently stated. He "saw" and conceptualized all things in the world in the context of a "model". That is, there was input (I) to a "black box", and its resultant output (O), under feedback control. See Figure 2 for a simplified model of the accommodative and vergence systems. Of course, the ultimate goal was to conceptualize, develop, and mathematically describe the inner workings of the individual components and their interactions, such that their output and overall responsivity reflected the real world (i.e., it was

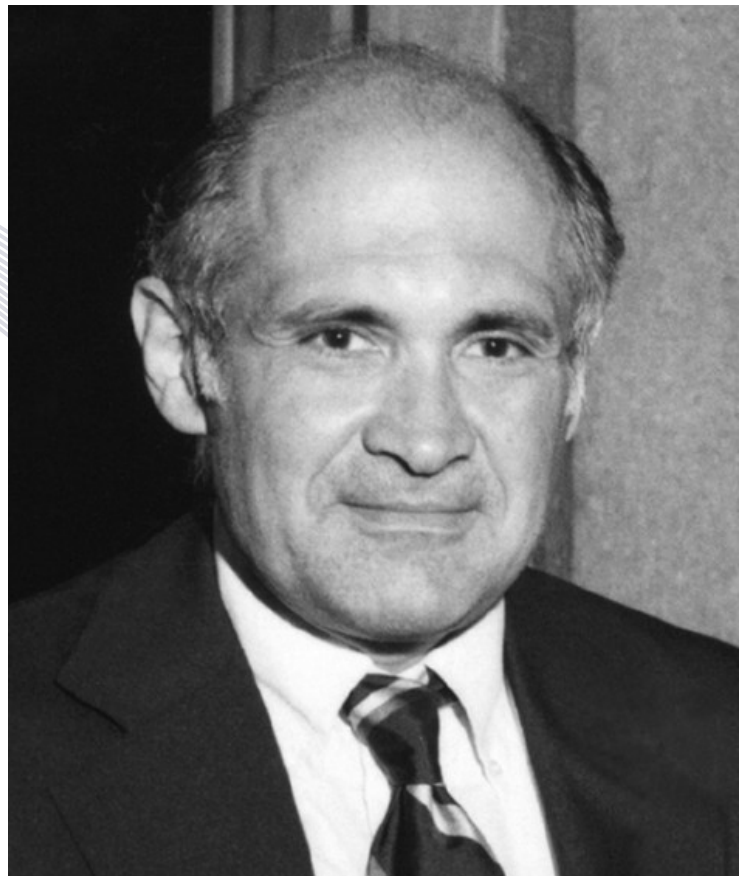


Figure 1. Lawrence W. Stark, MD Accessed 5/14/2025 <https://optometry.berkeley.edu/alumni/hall-of-fame/lawrence-stark/>

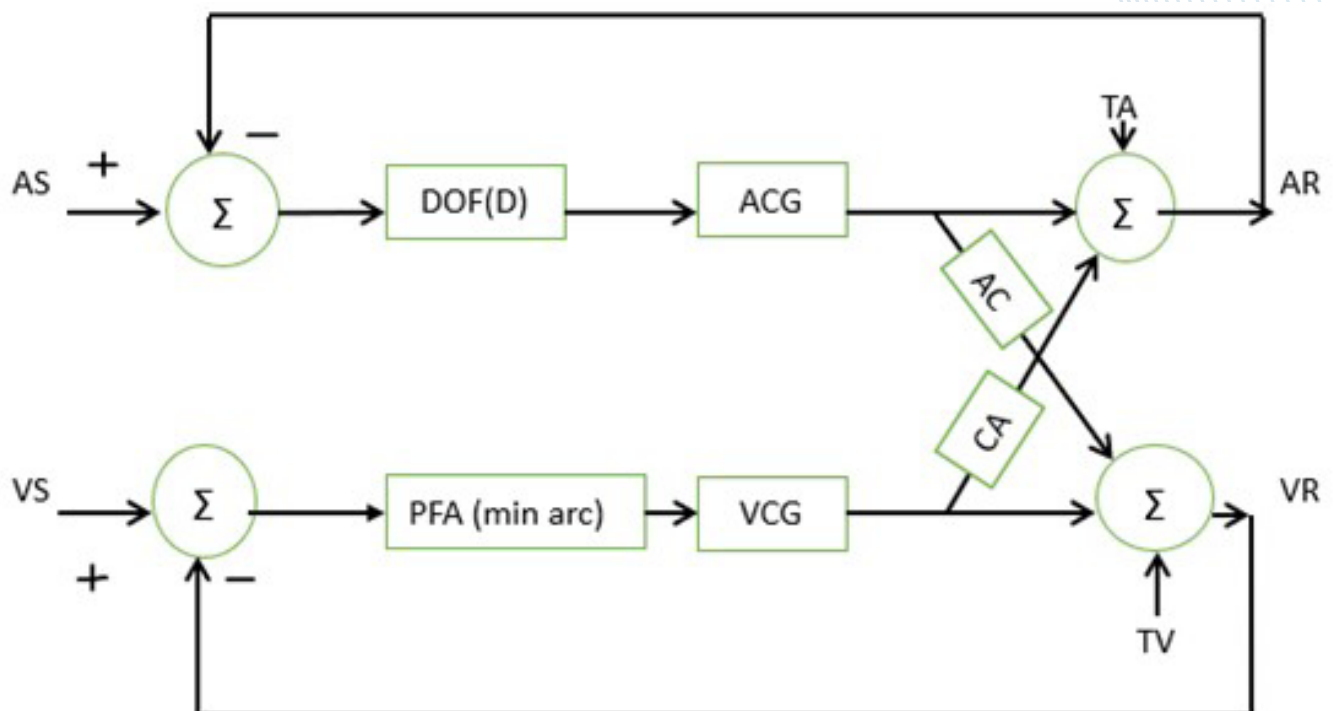


Figure 2. Symbols: DOF = depth-of-field/focus, PFA = Panum's fusional areas, ACG = accommodative controller gain, VCG = vergence controller gain, TA = tonic accommodation, TV = tonic vergence, AC = accommodation crosslink gain (~AC/A ratio), CA = vergence crosslink gain (~CA/C ratio), AS = accommodative stimulus, AR = accommodative response, VS = vergence stimulus, and VR = vergence response

homeomorphic). This input/output, bioengineering approach could be used to describe any physiological system (e.g., heart regulation, leg movement), as well as weather patterns and economic trends. For him, it was usually applied to the visual system, especially its oculomotor control (e.g., accommodation, vergence).¹⁻³

Why are such models of importance to optometry? That is a fair question to ask. They provide a testable, overall/global framework for understanding the neurophysiological control of the "closed-loop" (i.e., under feedback control) response: for example, vergence dynamics in normals versus those with strabismus.⁴ By doing so, one is essentially performing a "dry dissection" of the system in question. However, one must first create a model of the normal human oculomotor system and then demonstrate its accuracy by comparing the model's output to actual, normal physiological responses. Once proven to be accurate, one can then proceed to explore the abnormal system, with a goal of assessing alterations/changes in the values for the various components in a systematic manner, and furthermore to assess how these components might be involved in producing some oculomotor dysfunction, such as strabismus, ill-sustained and slowed accommodation, or convergence insufficiency.³

One way to do so is to perform a "sensitivity analysis" of the system in question.⁵ In such an analysis, each individual component's nominal/normal value is purposely changed one at a time—for example, the depth-of-focus (DOF) element for accommodation by +50% and then by -50%—and determine its effect on the output value. If the change is small, then that particular element/model parameter is considered not to be "sensitive", and thus is unlikely to be critical in causing some abnormal oculomotor condition. In contrast, if the output is changed significantly, it is thus deemed to be "sensitive", and the parameter is likely to play a major role in the etiology of the visual oculomotor abnormality. For example, with respect to the steady-state, static vergence system, the crosslink controller gains, namely accommodative convergence (AC) and convergence accommodation (CA) (i.e., effectively the AC/A and CA/C ratios, respectively), which function as multipliers of the input, have been demonstrated to be particularly sensitive to any change in value. Thus, they are likely to contribute to some oculomotor dysfunction, such as accommodative esotropia. In contrast, the model parameter tonic vergence (TV) was found to be insensitive to changes in its nominal value and therefore unlikely to contribute to some vergence dysfunction.

In addition, these models allow one to assess the

effect of some visual intervention, such as near add spectacles for myopia control and related defocus on the retina,⁶ as well as oculomotor-based vision therapy.⁷ In the latter case, by assessing either subjectively in the clinic (e.g., vergence flipper rate) or in the laboratory (e.g., convergence peak velocity), any improvement in dynamic responsivity can be attributed to that intervention and then the specific contributory model parameters identified, such as vergence latency and vergence controller gain (VCG). These parameters can be targeted in future interventions (e.g., vision therapy). In contrast, if there is little or no response change, then that specific intervention/technique would not be used in future cases.

Excellent models have been developed for a wide range of visual conditions/dysfunctions.³ Some of these include strabismus, refractive development (e.g., myopia), nearwork-induced transient myopia, nystagmus, visual attention, and more. In the future, it is hoped that these computer-based models will become more user-friendly and thus become part of the contemporary optometrist's clinical armamentarium in providing a better understanding of the etiology and preventative aspects of a patient's care, as well as guide potential therapeutic interventions.

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