Fundamentals of the objectiveFIELD® Analyzer (OFA®)

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Why do we need a new perimetric device?

The answer is partly that while Standard Automated Perimetry (**SAP**) remains the cornerstone of visual field testing, it has limitations that are perhaps not widely appreciated. Additionally, a different type of perimeter could provide extra statistically independent types of information that when combined could improve diagnostic and prognostic power. A more user-friendly device would also be welcome.

SAP limitations

SAP is a behavioural button-press test in which its stimuli are presented one-at-a-time to find a proxy for true sensitivity, a light threshold, at each location. Because the process is laborious and uncomfortable, participants often find it unpleasant,[1] and *it poses particular challenges for younger, or older persons, or physically frail individuals*.

Perhaps the biggest problem with SAP is that it suffers from poor reproducibility. This originates in part from the under-sampling of abrupt changes in sensitivity associated with visual field defects by SAP's typical Goldmann Size-3 stimuli, which are only 0.43 deg across. Even the relatively dense SAP 10-2 array of stimuli (**Fig. 1A**) leaves 96.4% of the macula untested, while the more commonly used and spatially broader SAP 24-2 pattern *misses 99.6%*. We have shown that SAP's poor sampling of the retina contributes greatly to its poor reproducibility[2, 3] Dysfunctional retinal neurons also contribute, but their contribution is about 12-times smaller.[4] Poor reproducibility impairs our ability to detect clinical changes over time, i.e. it affects *prognostic* power. Even a rapidly declining glaucoma patient needs to have 6 SAP tests over a 2-year period to have even an 80% chance of that decline being detected.[5] *In a busy clinical setting, this is rarely feasible.* By contrast, the *objectiveFIELD Analyser* (**OFA, Fig. 2A**), has better reproducibility, partly due to its larger stimuli (*cf.* the white dots in Fig. 1A with the yellow regions in **Fig. 1B**), and is faster than SAP.[6] The macular OFA stimulus ensemble of Fig. 1B makes comparisons with OCT retinal thickness data easier. OFA also has widefield tests. OFA measures physiological responses directly from the test subject through their pupils: users need only fixate their vision on a centrally located cross. So, it uses no button presses.

Another weakness of SAP is that the sensitivity losses it reports follow early retinal cell losses poorly. That is, changes in SAP sensitivity are a nonlinear function of ganglion cell loss.[7] As the eye damage progresses from minimal to quite severe cell loss, the slope of the function changes very little – the functional changes reflected by SAP are not truly representative of the severity of cell loss. Only after substantial damage has occurred do increases in damage

reported by SAP then change rapidly. By contrast, the sensitivity changes measured by OFA follow the decline in cell numbers closely (i.e. linearly), in principle allowing OFA to report damage earlier.[8] Plotting SAP mean defects (MDs) on OFA MDs in the same subjects shows this nonlinearity convincingly.[6]

A further issue with SAP is that the small size of Goldmann Size-3 stimuli means that the retinal contrast gain control system of magnocellular retinal ganglion cells (M-cells) that operates for natural stimuli is not engaged. When operating normally that system can change the responsiveness (gain) of M-cells by an order of magnitude in tens of milliseconds. To control that accurately, it uses a rapidly computed spatial average of contrasts over an area substantially larger than the M-cell inhibitory surround.[9] Macaque studies show that retinal gain control does not therefore operate for stimuli ≤ 0.5 deg across,[10] i.e. the diameter of Goldmann Size-3 stimuli. The larger, high contrast stimuli of OFA (e.g. Fig. 1B) thus test the visual system as it was designed to operate: in a world of dynamically changing spatially extensive stimuli. A corollary of that is that new information about the effects of glaucoma and other diseases upon that dynamic system may be revealed.

OFA Basics

OFA uses multifocal stimuli where the measured physiological response is the *relative* change in pupil diameter. Using relative change in pupil size is very commonly done,[11] partially because the pupil response dynamics are independent of size.[12] This also means that, in most cases, drugs that mildly alter pupil size, and at least one pupil is not stationary or quite irregular, are not an issue. Local iris defects caused by things like cataract surgery are generally not a problem. Age effects are also reduced. Ptosis presents a possible issue for OFA, but it doesn't track superior pupil size in larger pupils. During testing, independent stimuli are presented to the two eyes concurrently (halving test duration) and both pupils are recorded. The resulting direct and consensual responses are combined according to the measured signal to noise ratios, thus one poorly performing pupil is not a large issue. Pupil responses are sometimes thought to be sub-cortical, but this is only true for the extremely slow responses of the melanopsin containing retinal ganglion cells, [13] which regulate pupil size in bright light. Input to the pupil system from many cortical areas is well established.[14] We have provided evidence that the transient onset stimuli of OFA drive a cortical pathway,[15] probably through V2.[16] This appears to be an offshoot from the neural wiring supporting the accommodative triad. That system needs a rapid estimate of range to objects as is provided by stereopsis, which is computed in the cortex.[17]

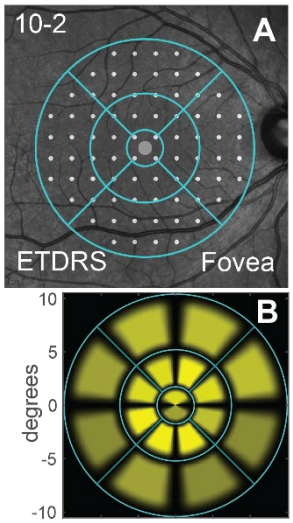

Fig. 1 A) The macula as delineated by the cyan **ETDRS*** grid. The central grey dot is the foveola. The small white dots are the tiny 10-2 SAP stimuli, which fail to test 96.4% of the macula. **B**) The OFA macular 10-EDTRS stimulus ensemble shown relative to the ETDRS grid. OFA also has wide-field stimuli testing ±30 degrees of the field. **Early Treatment Diabetic Retinopathy Study*.

OFA methods have been improving over time. Three innovations have been new stimulus methods each of which improved signal-to-noise ratios by 40%. These included: luminance balancing,[18] and clustered volleys.[19] Most recently, methods for fitting pupillary gain dynamics have done things like permit widefield[20] and macular methods[21] that test both eyes in under 90 seconds. With a few exceptions, OFA papers published since 2020 use the latest test methods (N=13). OFA has been demonstrated to be safe for individuals with migraine[22] and epilepsy.[23]

Dual-axis *versus* Half-axis testing

Another issue with SAP is that it is a *Half-axis* device because it only reports negative changes in sensitivity (**Fig. 2B**). It does not capture *increases* in sensitivity that may be indicative of phenomena such as glutamate excitotoxicity.[24] By contrast OFA is a *Dual-axis* device reporting increases and decreases in both sensitivity and response delays at every tested region. As will be described below, response delays often include information about damage that is independent of sensitivity, i.e. which may report on independent aspects of disease.

Hypersensitivity is an interesting example. We have shown that higher than normal sensitivities (hypersensitivity) are associated with early-stage diabetic retinal disease (DRD), e.g.[25, 26], providing better diagnostic power than SAP.[26] Hypersensitivities can predict which AMD eyes commencing anti-VEGF therapy will respond well, both initially[27] and over 15 months.[28] SAP testing using larger stimuli, that would engage retinal gain control, have been reported to show hypersensitive regions in glaucoma. Casson and Johnson referred to these areas as "high-sensitivity defects",[29] a nice succinct term. These could decay to regions of decreased sensitivity ("low-sensitivity defects") over 2 to 3 years.[29] Notice that for SAP to quantify hypersensitivity requires people to detect miniscule changes in contrast while on OFA hypersensitivity is an easy-to-measure larger than normal response.

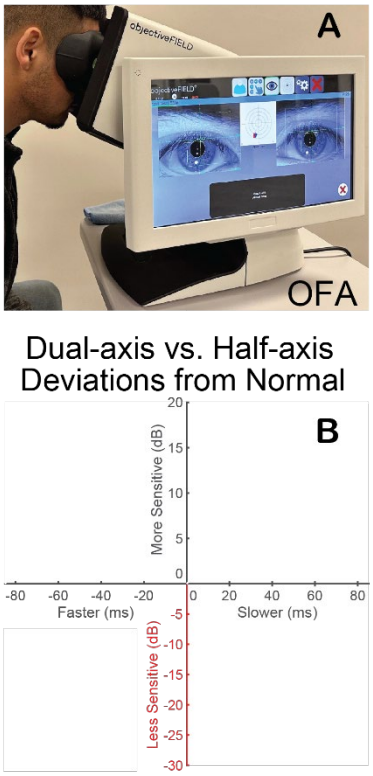

Fig. 2 A) An OFA in the clinic showing a test subject and the operator view of the videoed pupils. **B**) The difference between a Half-axis measuring device like SAP (red axis), and a Dual-axis device like OFA providing a broader data set.

Longer response delays are characteristic of later-stage DRD, and oedema related-changes in retinal thickness over 2 years, while SAP reports no change in the same subjects.[30] Our 5-year study of glaucoma showed that some eyes progress on sensitivity but not delay, or *vice versa*.[31] That phenomenon has been partially confirmed by others using an independent method: saccadic perimetry.[32] As in OFA, regions showing large response delays can occur where (nonlinear) SAP reports normal sensitivity. Others have reported increased saccadic delay and fewer express saccades in glaucoma.[33] Delays and sensitivities are differently correlated with AMD severity and Macular Pigment Optical Density,[34] indicating independent measures of the disease. OFA delays differentiate focal and generalised epilepsy,[23] provide high diagnostic power in multiple

sclerosis (MS),[20] and predict which MS subjects will change to progressive disease over 10 years.[35] Delays and sensitivities are differently affected at different field locations following mild concussion.[36] Overall, we have shown that measuring both sensitivity and delays can add significantly to diagnostic and prognostic power.

For example, we compared the diagnostic power of 44 different functional and structural tests from 23 studies attempting to discriminate eyes of normal controls from diabetic persons with no retinopathy (i.e. very early-stage DRD). The result was that OFA has by far the best performance.[37]

As OFA uses pupil responses, the issue of iris neuropathy arises. Iris-specific issues will affect all regions equally, either sensitivities or delays. Therefore, like the effects of cataract in SAP, the OFA pattern deviations will be unaffected by such global biases. Thus, pattern deviations will resolve true regional delay defects. We have quantified localised delay defects in early-stage DR independent of other factors.[26]

Long regional delays masquerading as SAP sensitivity loss?

The response delays observed with OFA can be upwards of 600 ms. This is comparable to the delays reported for saccadic perimetry in glaucoma.[32] Two studies have examined response delays in glaucoma patients when they were given more time to respond in SAP-like testing.[38, 39] In one study 25% of the glaucoma subjects had a mean delay of > 1 second.[38] Such delays could mean that in normal SAP testing that stimuli are missed, which are then interpreted as sensitivity loss. Another study was interested in performance at various limens of the contrast-response function for SAP-like stimuli.[39] Damaged areas of the field had mean delays of 996.3 ms for the 0 dB stimulus. In damaged areas six out of ten persons with glaucoma registered no response at all within the 2 s of allowed time. These studies suggest it is possible that long response delays could masquerade as SAP sensitivity loss. We followed glaucoma subjects over 5 years and demonstrated that sensitivity and delay losses can progress quite independently over time and across the fields.[31] Thus, a false SAP defect, due to a long response delay, could easily occur at a different location to true sensitivity loss. Independent evolution of OFA sensitivity and delay defects is in line with results from saccadic perimetry, and we have reported such results for OFA in MS,[35] DR,[26, 30, 40] and AMD.[28]

Returning to delayed responses using SAP-like stimuli, an interesting aspect of one of those studies was that persons with more acute diseases, AION and Stroke, had shorter response delays than the glaucoma patients.[38] That might suggest that some regional response delays might be due to subsequent degeneration of the cortex and optic radiations that has been reported in glaucoma.[41, 42] That appears to mirror demonstrations of trans-neuronal retrograde degeneration of macular retinal ganglion cells (RGCs) following striate cortical ablation in macaque monkeys.[43] In such studies RGC loss increases over periods between 1 and 9 years.[44] Both visual search[45] and reading speeds[46] are slower in persons with glaucoma. Comparing those data with OFA sensitivity and delay data would be interesting. Repeating those comparisons in persons with recent acute glaucoma (e.g. uveitic) and chronic glaucoma over several years could also be informative.

Summary

The 39 OFA publications to date include studies of 8 ophthalmic and brain diseases. The dual-axis OFA results appear to provide new, and statistically independent, information, improving diagnosis and prognosis. Its two high-resolution tests provide four 30-2 like reports in 8 minutes.[6] It's three lower-resolution 90-second tests are particularly useful for children[47] and infirm persons.[48] Both types of tests come in macular and wide-field versions. The stimuli of the fast macular test[21] match the test regions of the ETDRS grid used by OCTs (Fig. 1B) to report retinal thickness data, simplifying structure/function comparisons.

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