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Psykinematix: A New Psychophysical Tool for Investigating Visual Impairment due to Neural Dysfunctions

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In this paper, we first review the most common neurodegenerative diseases and mental disorders, and their effects on visual functions. Secondly, we present Psykinematix, a new Mac OS X software package dedicated to visual psychophysics. Because it does not require any programming skill, and provides an intuitive GUI that abstracts most of the inherent difficulties, this new tool provides effortless means to create and run the complex psychophysical paradigms developed by vision scientists. Finally, we discuss how Psykinematix could help clinician researchers replicate fundamental studies, and better investigate visual functions that are impaired by aging and neural dysfunctions, such as shape and motion processing.

1. Introduction

The purpose of this paper is threefold: first we provide a "clinical" review of the visual impairments found in the most common neurodegenerative diseases and mental disorders. The aim of this review is to stress the importance of perceptual dysfunctions, the nature of perceptual abnormalities in these diseases and disorders, and the critical need to develop perceptual tests appropriate for future clinical trials. Secondly we present Psykinematix, a new tool dedicated to visual psychophysics we developed to ease the elaboration of standard protocols, by providing a simple experimental paradigm (Method/Procedure/Stimulus) and an intuitive graphic user interface (GUI) that abstracts most of the psychophysical concepts. Third, we emphasize some features in Psykinematix that address requirements that are typical of clinical applications.

2. Visual impairments due to neural dysfunctions

Below we review the pathologies and known visual symptoms for the most common neurodegenerative diseases and mental disorders: Alzheimer's (AD) and Parkinson's (PD) for the neurodegenerative diseases, autism and schizophrenia for the mental disorders. Neurodegenerative diseases such as Alzheimer's or Parkinson's disease pose some of the greatest challenges in an aging society (about 21% of population is 65 years and over in Japan in 2007, up from 12% in 1989), while prevalence in autism has dramatically increased since the 1980s in children and schizophrenia in young adults is ranked the third-most-disabling condition.

2.1 Alzheimer's disease (AD)

AD is the most common form of neurodegenerative disorder in the elderly, and is characterized pathologically by synaptic dysfunction, and clinically by a progressive

decline in memory and cognition and a progression in dementia. Currently, AD has no cure or preventive solution, and understanding all behavioral, anatomical, and physiological aspects of this disease is obviously of utmost importance worldwide. Diagnosis is usually confirmed with behavioral assessments and cognitive tests, often followed by a brain scan if available. While agreeing that the memory deficit is usually the initial sign of AD, researchers have long known that AD is characterized by impairments in several additional domains, including visual functions and attention. Indeed, visual dysfunctions are prevalent in AD as more than 60 percent of the people with Alzheimer's have a decline in one or more visual capacities. The neuropathology of this disorder affects several brain areas that are devoted to processing of low-level visual functions as well as higher-order visual cognition and attention.

2.1.1 Visual impairments in AD

Vision disorders in AD span from structure (retinal and cortical) to function (cortical activation) to behavior (basic vision, hallucinations, perception, cognition, attention, and everyday activities). Visual impairments in AD most commonly occur in four basic areas: motion, depth, color and contrast. Some people with AD are unable to correctly sense movement[1)], and their perception of structure from motion (SFM) may be also impaired[2)]. The visuo-spatial disorientation in AD is thought to arise from deficits in the perception of self-motion via optic flow, which contains information about heading direction and the three dimensional structure of the visual environment[1)]. Persons with Alzheimer's also may lack the ability to recognize depth, and three-dimensional objects may appear flat. Color perception diminishes with age, and the ability to see contrast is also reduced in people with Alzheimer's.

The magnocellular hypothesis of visual dysfunction in AD proposes exceptional vulnerability of functions associated with the magnocellular pathway and by extension with the dorsal processing stream. As AD patients may also suffer from a perceptive and associative agnosia, problems may occur also in the ventral pathway, which is specialized for object recognition and at the fundamental basis of perceptual organization. Moreover it has been reported that AD patients who perform poorly on face discrimination, also show impairments on all tests relying on perceptual organization (Gestalt principles) such as figure-ground segregation, proximity, and continuation. Moreover, visual dysfunctions in color discrimination, stereo-acuity, contrast sensitivity, and backward pattern masking were shown to be a significant predictor of cognitive dysfunction (such as object recognition) in AD, suggesting that visual deficits in AD may have a strong functional impact on performance in specific cognitive domains[3)].

2.2 Parkinson's disease (PD)

Parkinson's disease is another neurodegenerative disorder that often impairs the sufferer's motor skills, speech, and other functions. Because there is no definitive test for the diagnosis of PD, the disease must be diagnosed based on clinical criteria. It is characterized by muscle rigidity, tremor, a slowing or, in extreme cases, a loss of physical movement. The primary symptoms are the results of decreased stimulation of the motor cortex by the basal ganglia, normally caused by the insufficient formation and action of dopamine, which is produced in the dopaminergic neurons of the brain. Non-motor symptoms include autonomic dysfunction, cognitive/neurobehavioral abnormalities, sleep

disorders and sensory abnormalities such as lack of olfaction, paresthesias and pain. These problems are all caused by the degeneration in the brainstem of people with PD. This degeneration results in low levels of dopamine, an important chemical messenger in the brain. At present, there is no cure for PD, but medications based on L-dopa or surgery can provide relief from the symptoms.

2.2.1 Visual impairments in PD

In addition of stiffness and tremor in Parkinson's disease, people with Parkinson's usually develop some manifest eye problems, such as stare because they don't blink as frequently as they used to and involuntary closing of the eyelids is also a frequent occurrence. Eye movement disorders are apparent on examination, although they do not always cause problems from a functional standpoint. The eyes may have difficulty fixating on objects and following them as they move. Occasionally, because of a lack of eye coordination, people experience double vision, which may be present only when looking in certain directions.

There is also good evidence for impairments of early visual processing in Parkinson's disease. Symptoms caused by these abnormalities include reduced vision, poorer color vision, and difficulty appreciating the correct location or orientation of an object, though these visual abnormalities are unlikely to be uncovered during routine neurological examination. Using more sophisticated testing techniques, both psychophysical and electrophysiological (such as the visual-evoked potential VEP and the pattern electro-retinogram ERG) studies have shown abnormalities of motion, spatial and color perception[4]. Impairments in color discrimination (CD) and contrast sensitivity (CS) are established signs of Parkinson's disease[5], and are known to deteriorate over time[6]. Both deficits correlated with age, and the chromatic deficit additionally correlated with higher impairment of motor function. Treatment with L-Dopa is also known to improve color vision in AD.

All these visual deficits may influence overall motor function and lead to enhanced motor impairment. However it is not clear whether all of these problems are the result of the degeneration in the brain or whether some may be due to lowered dopamine levels in the retina itself. Since dopaminergic amacrine cells in the retina are known to be involved in the control of the overall visual spatiotemporal sensitivity[7], visual deficits in PD have been interpreted as the consequence of progressive, select pathology of dopaminergic neuronal processing in the retina, leading to loss of spatiotemporal tuning and distorted retinal input to higher visual centers. All evidence has pointed out the retina for the likely site of origin of these impairments[4].

Despite its dopaminergic deficiency, the retina may not be the only site of visual pathology in PD, as more complex visuo-cognitive difficulties, e.g., impairment of consciously controlled visual information processing, have also been identified in PD: the visuo-spatial sketchpad, a component of the working memory system, shows a specific selective impairment in PD, and visual categorization deficits have also been found suggesting involvement of posterior parietal cortex. Concurrent electrophysiological recordings of primary and visuo-cognitive responses have revealed that the impairment of higher order visual processing in PD is not simply a consequence of retinal dopaminergic deficiency. Electrophysiological, neuropsychological, and functional neuroimaging data imply that both frontal and

posterior cortico-subcortical circuits may be involved[8].

2.3 Autism

Autism (more generally autism spectrum disorders, ASD) is a brain development disorder that first appears during infancy or childhood, and generally follows a steady course without remission. Impairments result from maturation-related changes in various systems of the brain. Autism is one of the five pervasive developmental disorders (PDD), which are characterized by widespread abnormalities of social interactions and communication, and severely restricted interests and highly repetitive behavior. Individuals with autism have also difficulty with processing and responding to sensory information.

2.3.1 Visual impairments in ASD

Vision problems are very common in autistic people. They often use visual information inefficiently, and have difficulty maintaining visual attention. They are unable to visually "hold still", and frequently rely on a constant scanning of visual information in order to gain meaning. This symptom reflects their inability to integrate their central and peripheral vision, and as a result of this poor integration their difficulty to normally process visual information. For example, once central focus is gained, they can ignore side vision and remain fixated on a task for excessive periods of time, and often seem to be looking off to the side of an object. Eye movement disorders and strabismus are also common. Since the visual system relates to motor, cognitive, speech, and perceptual abilities, these areas may also be negatively affected by their visual impairments.

The most prominent visual symptom in autism is the aberrant local and global processing characterized by a superior perception of fine details, and a failure to integrate local information into a global percept (also referred as 'weak central coherence'[9,10]). Another important symptom in autism is the impaired motion perception that may be also linked to the abnormal perceptual integration: Children with autistic disorder show increased motion coherence thresholds for random-dot kinematogram stimuli (RDK) as compared to controls[11,12], and also show reduced sensitivity to biological motion, but it is unclear whether these deficits result from a general developmental delay rather than being specifically related to autism[11]. Nevertheless, the intact or enhanced performance on static spatial tasks and inferior performance on dynamic tasks have suggested a reduced sensitivity of the visual magnocellular pathway or a deficit of the cortical dorsal stream. Young adults with autism also show a significantly lower sensitivity to second-order (texture-defined) motion stimuli compared to their normal sensitivity to first-order (luminance-defined) stimuli[13] suggesting a reduced neuronal integration rather than impairment in motion perception per se. Autistic people also perform worse on static second-order stimuli, but also surprisingly better on static first-order stimuli compared to normal in terms of orientation identification, while having normal flicker contrast sensitivity, indicating normal functions along the sub-cortical magnocellular and parvocellular pathways, and some atypical neural connectivity at an early cortical level such as enhanced lateral inhibition[14].

However when using plaid motion stimuli instead of RDK stimuli, which can be perceived either as a coherently moving pattern or as two transparent gratings sliding over each other, no evidence of impaired global motion perception was reported in autistic people, a result that can be accounted by spatial frequency processing in

autism[15]. Moreover another recent EEG study in autism has attributed the specific impairment in object boundary detection to a dysfunction of horizontal connections within early visual areas, and suggested that atypical horizontal interactions might reflect a more general neural abnormality in autism disorder[16]. Evidence of early perceptual integration deficits is however still controversial as some studies reported comparable performances between normal subjects and an accurately diagnosed sample of autistic children for both contour integration and global motion perception[17,18]. Some other studies have claimed magnocellular or/and dorsal deficits do not appear to be sufficient to explain impairment of motion perception in autism, and have suggested that abnormalities at a higher cortical level, in the superior temporal sulcus (STS), may provide a neural basis for the range of motion-processing deficits observed in autism, including biological motion perception[19].

2.4 Schizophrenia

Schizophrenia is another severe and disabling brain disorder characterized by abnormalities in the perception or expression of reality. It most commonly manifests as auditory hallucinations, paranoid or bizarre delusions, or disorganized speech and thinking with significant social or occupational dysfunction. Onset of symptoms typically occurs in young adulthood. Diagnosis is based on the patient's self-reported experiences and observed behavior. No laboratory test for schizophrenia currently exists. Although no common cause of schizophrenia has been identified in all individuals diagnosed with the condition, currently most researchers and clinicians believe it results from a combination of both brain vulnerabilities (either inherited or acquired) and life events. Much work in the cognitive neuroscience of schizophrenia has focused on attention, memory, and executive functioning. To date, less work has focused on perceptual processing. However, perceptual functions are frequently disrupted in schizophrenia, and consistent deficits in visual processing are observed in schizophrenia.

2.4.1 Visual impairments in schizophrenia

Schizophrenia is generally associated with deficits in higher-order processing of visual information at a cognitive level, but several studies have nevertheless evaluated the integrity of early visual processing in terms of magnocellular and parvocellular visual pathway activity in order to evaluate the overall pattern of visual dysfunction in schizophrenia. Using VEP, it has been reported that a dysfunction of lower-level visual pathways was more prominent for magnocellular than parvocellular biased stimuli. Since the magnocellular pathway helps in orienting toward salient stimuli, a global magnocellular dysfunction could contribute to higher-level visual cognitive deficits in schizophrenia[20,21]. However the claim that magnocellular deficits are linked to schizophrenia has been recently undermined by several studies[22–24], suggesting either the absence of an association between magnocellular deficits and schizophrenia, or the presence of such deficits at more integrated levels at which parvocellular and magnocellular paths interact. Deficits in contrast sensitivity for moving and static gratings, form discrimination in noise and dot motion discrimination have also been reported in patients with schizophrenia[25].

Poor contextual processing is another hypothesis to account for diverse cognitive deficits associated with schizophrenia. People with schizophrenia fail to use contextual information to disambiguate visual information because of hypo-activity in NMDA glutamate receptor channels. Surprisingly, it has been

reported that weak contextual suppression can make people with schizophrenia more accurate at contrast discrimination[26], suggesting that weak cortical suppression from context may be a general feature of schizophrenia. This contextual processing may result from deficits in two other important mechanisms: gain control and visual integration. Gain control studies in schizophrenia clearly show that patients have difficulty modulating neuronal responses to take advantage of the surrounding context, and that gain control deficits may account for the reported deficits in contrast detection and magnocellular pathway. There are also numerous examples of poor form processing in schizophrenia (object recognition, grouping, perceptual closure, contour integration, face processing, and reading) that would seem to directly implicate integration deficits. This is thought to partially result from decreased NMDA-modulated lateral excitation in the visual cortex and the consequent reduction in synchronization of this neural activity. Reductions in gain control and integration can account for findings from a number of experimental paradigms (including contrast detection, gestalt processing, motion perception, and eye-movement control). The neurophysiology of both processes is likely to involve effects of glutamatergic activity at NMDA receptors and interactions between parvocellular and magnocellular pathways[27].

2.5 Summary

In all the neural dysfunctions we reviewed, the cognitive capabilities are primarily affected. However vision is also always impaired to some degree, although this feature is not widely recognized by most clinicians. The prevalence of basic visual deficits raises naturally the question of their impact on cognitive functions, and suggests that some cognitive impairments may simply result directly or indirectly from deficiencies at a perceptive level rather than from a core cognitive problem. Moreover, because cognitive and vision impairments are not widely recognized as closely linked, vision testing and cognitive testing are not conducted at the same visit or by the same provider. When realizing the links between the vision and cognitive impairments, one can realize the importance of testing for visual risk factors in pre-symptomatic and early phases of the disease or disorder, and that such testing may help with secondary and tertiary prevention. Treatments and outcomes could also benefit from a better understanding of the perceptual deficiencies.

The nature and importance of perceptual abnormalities found in these neural dysfunctions stress the need to develop appropriate perceptual tests able to quantify the affected visual functions. A psychophysical approach can provide the most objective and quantitative assessment of the impact of the neural diseases (as well as their progressions and outcome of clinical treatments) on the visual functions. While brain imaging techniques are powerful tools when investigating cerebral functions and neuro-chemical changes, they could be of little use for quantifying deficits in visual functions and could be extremely burdensome when used to regularly monitor the progress or remission of neurodegenerative diseases and mental disorders. Other tools, like behavioral assessments and cognitive tests, are cost-effective but only adequate for obtaining a qualitative assessment of the visual deficits. It is clear from our review that the psychophysical testing of visual functions should be an important asset amongst the diagnostic and research tools available to clinicians.

A tool delivering adequate psychophysical testing should be versatile enough to investigate

the various visual deficits, in particular those that are common to the neural dysfunctions we previously reviewed, that is in terms of contrast, color and motion (both simple and complex) perception, spatial and temporal integration, Gestalt principles, and object perception. In the next section, we present a new software tool, Psykinematix, which provides advanced features that address the requirements for these clinical applications.

3. Psykinematix

Psykinematix is a new low-cost software package dedicated to visual psychophysics that takes advantage of the most advanced computer technologies (OpenGL & Mac OS X). This tool was developed with the goal of simplifying the creation of complex experimental paradigms in the context of psychophysical and electrophysiological studies of spatial, temporal and color aspects of human vision.

The Psykinematix software eases the elaboration of standard protocols, by providing a simple experimental paradigm (Method/Procedure/Stimulus) and an intuitive graphic user interface (GUI) that abstracts most of the psychophysical concepts. It was created with the following requirements in mind:

– Complete streamline workflow (experimental design, display calibration, data collection and analysis) in a standalone software package to minimize the need for 3rd party softwares,

– Intuitive graphic user interface (GUI) to easily navigate through the many available options,

– No programming or script required to give the most users access to all features,

– Intuitive way to create visual stimuli using a "What You See Is What You Get" approach (WYSIWYG),

– An affordable solution (including a free demo for educational purpose).

3.1 Overview

Psykinematix has the ability to calibrate the experimental display, create and run standard psychophysical protocols, generate and present complex dynamic visual stimuli, collect the subject's responses, and analyze results on the fly. To do so, Psykinematix consists in an integrated solution composed of 5 clearly defined components:

– A design tool that allows the user to create the experimental protocol in term of an events hierarchy. A wizard is also available to create "canned" experiments.

– A calibration tool to measure the display geometry, perform the Gamma correction and specify the color properties of the display in term of colorimetric or radiometric measurements.

– A subject tool to manage lists of the subjects and groups as well as the experimental sessions run by each subject.

– A plotter tool to visualize and analyze the session results. Data can be fitted with various psychometric functions. Graphs and data tables can be printed or exported to Excel files.

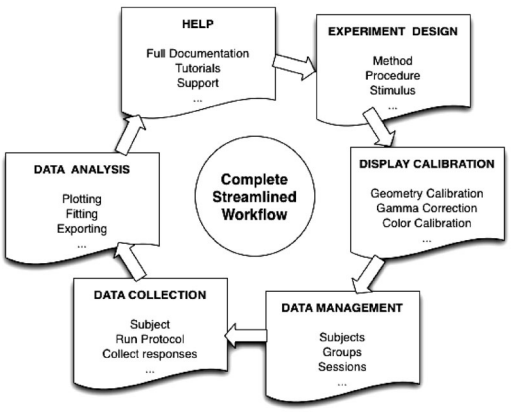

Fig. 1. Psykinematix worflow

– A built-in help tool that provides the full documentation, and access to tutorials for each component.

These 5 components constitute a complete streamlined workflow for creating and running a new experiment from scratch as illustrated in **Figure 1**. These components are available at all time in the top toolbar of the GUI (see right part in Figure 3).

3.2 Experimental paradigm

As illustrated in **Figure 2**, Psykinematix uses a simple hierarchical description of the experimental protocol, composed of the following events:

– Experiment: the root event that specifies the authorship information, display settings, independent variables, and input/output devices to be used during the experiment;

– Method: this event specifies how the dependent variables change with each trial: method of constant stimuli, staircase, bayesian estimation, and block design are all supported;

– Procedure: this event specifies the subject's task (forced-choice, yes/no, discrimination, adjustment), how the subject provides the responses (i.e. which input for a given choice) and some trial properties;

– Stimuli: these events specify the stimulation applied to the subject's visual system. Stimuli can be simple or complex, static or dynamic, built-in or custom, or even imported from a file.

This simple hierarchy describes events that are functionally linked during the execution of the protocol: first the experiment event configures the session, and then executes the method event. The method event runs the experimental loop, and executes a procedure event for each trial. The procedure event executes the stimulus events and waits for the subject's response. The subject's response is sent back to the method event in case this information is required to set up the next trial. The stimulus events generate all stimuli on the fly and render them with the specified values from independent and dependent variables.

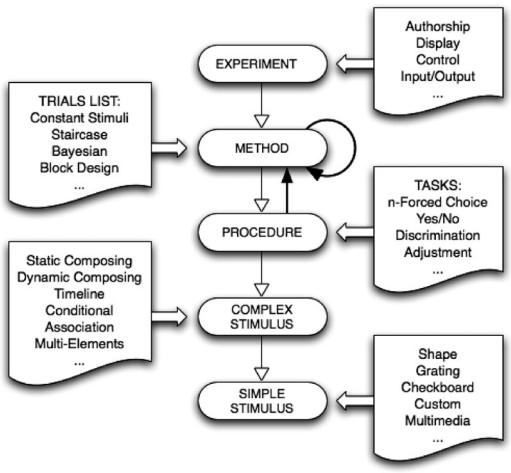

Fig. 2. Psykinematix experimental paradigm: Method/Procedure/Stimulus

3.3 Graphic User Interface (GUI)

The GUI is an important aspect of every high-level application. In Psykinematix, the GUI allows the navigation across the various components, the creation of the experimental protocol, and the customization of every property for all events that compose the protocol.

For example, as illustrated in **Figure 3**, the events hierarchy that describes an experiment can be created manually or automatically using the Wizard tool.

Figure 4 illustrates the GUI window to create a drifting Gabor stimulus by customizing a carrier*envelope kind of stimuli. Note that: 1) the stimulus is automatically previewed in the inset, 2) the Gabor contrast is set through a dependent/independent variable, 3) the drifting is simply produced by specifying a time-varying spatial phase.

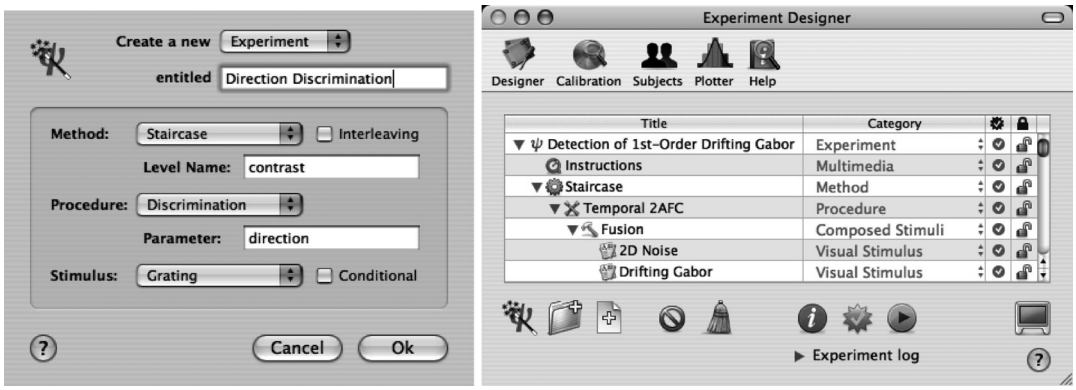

Fig. 3. Example of experimental protocol created using the Wizard tool (left). Once validated, the experiment hierarchy appears in the Designer component (right).

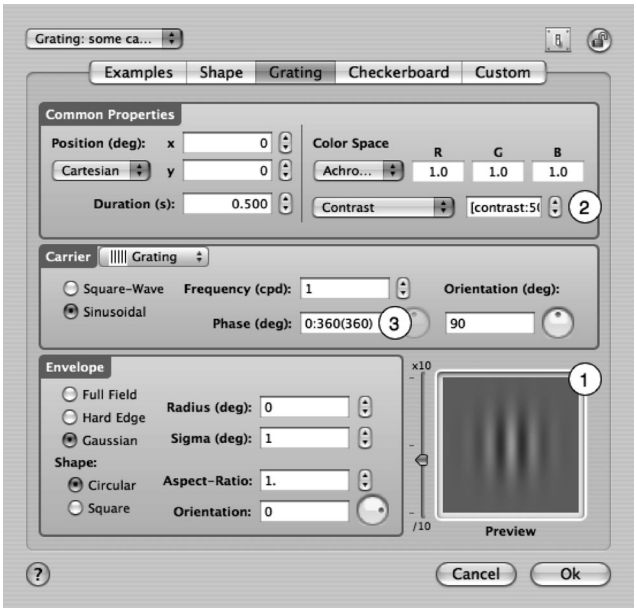

Fig. 4. Drifting Gabor stimulus.

4. Clinical applications

The neural dysfunctions we reviewed in section 2 show some clear deficits in motion perception, spatial integration, and contextual modulation. Integration and contextual modulation are two fundamental processes performed by the visual cortex, which are thought to be impaired in several neural dysfunctions that involve long-range (intra- or inter-cortical) interactions. Long-range interactions play indeed a critical role in visual perception, not only because they are involved in both integration and contextual modulation, but also because they are the keystone in perceptual learning, the experience-dependent cortical plasticity seen throughout life. It is also thought that the same mechanisms used in perceptual learning are recruited for functional recovery after central nervous system lesions

and neurodegenerative diseases.

Use of relatively complex stimuli is required when investigating visual integration or contextual modulation. We present here 3 examples of complex stimulus that demonstrate the advantages of using Psykinematix to create and run psychophysical experiments to investigate these visual processes known to be impaired by neural dysfunctions: RDK for motion integration, multi-elements configurations to explore deficits in Gestalt principles, center-surround stimuli for contextual modulation. Any clinical application should also take advantage of Psykinematix experimental paradigm "Method/Procedure/Stimulus" by associating either an adaptive method for fast measurements or a method of constant stimuli for more precise measurements, to a nAFC/2IFC forced-choice or discrimination procedure, with dependent variables controlling any aspect of the complex stimulus. Whatever the selected experimental design is, Psykinematix can quantify the task performance in terms of thresholds and sensitivity by fitting a psychometric function to the experimental data, and provide quantitative measurements that can be finally compared between normal and clinical groups.

4.1 Motion integration

Motion integration is impaired in all four neural dysfunctions we reviewed: random-dot kinematogram stimuli (RDK) are often used to demonstrate an integration deficit in motion perception[1,2,17]. RDK are composed of individual micro-elements ("dots") that have either a coherent motion (ie. those dots with the same motion on average) or a non-coherent motion (ie. random motion on average). Because a single dot does not provide enough information about the global motion, this task requires the integration of local motion to produce a perception of global motion. Motion coherence thresholds for direction discrimination are generally measured, that is the proportion of dots with coherent motion necessary to achieve a given performance criterion. RDK can then be used to explore either simple (uniform) motion or complex motion (optic flow).

Figure 5 shows an example of stimulus created by Psykinematix using its "RDK" feature: one simply specifies the kind of micro-elements to be used, how they should be moved (speed, life-time, coherence, direction, etc.), and at which location. Note that with Psykinematix, it is very easy to control for important visual parameters: one can specify the spatial frequency content of each micro-element, rather than using simple dots (or small disks) with unspecified spatial properties as it has been usually done in past studies.

Using a staircase method and a direction discrimination procedure, Psykinematix is able to measure motion coherence thresholds for a variety of motion types (uniform, radial, angular). Such an experimental protocol can be

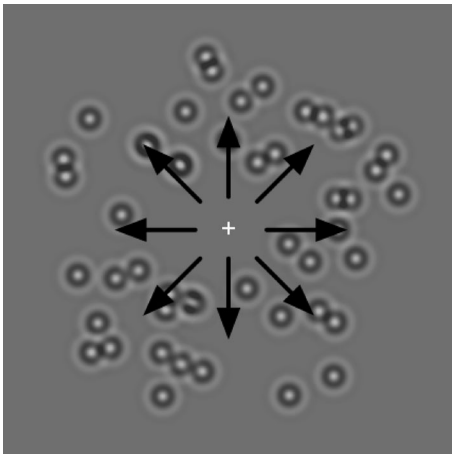

Fig. 5. Example of RDK stimulus with radial motion ('optic flow') with isotropic Gabor micro-elements.

readily applied to quantify the effects of the different neural dysfunctions on motion integration.

4.2 Spatial integration

Spatial integration is impaired in some of the neural dysfunctions we reviewed. However, spatial integration can take several forms that may not involve the same neural mechanisms depending on the kind of stimulus. For example, lower sensitivity to static and dynamic second-order (texture-defined) stimuli has been reported in autism[13,14], and some deficit has been reported for contour integration, another form of spatial integration, in schizophrenia and with aging[27,28] but not in autism[17].

Figure 6 shows another example of stimulus created by Psykinematix using its "Multi-Elements Field" feature: one simply specifies the kind of micro-elements to be used, then how they should be pasted in a grid. Note that positional and orientation jitters can also be specified to assess the robustness of the integrative process.

Such stimulus can be used to investigate the global integration of local orientation, somewhat analogous to the use of RDK in motion perception. This kind of stimulus is also at the basis of the contour integration paradigm that involves the selective integration of micro-elements oriented along a 'path' embedded in a noise field (micro-elements with random orientation). Contour integration has become a popular paradigm to investigate the Gestalt principles that form the basis of shape perception (proximity, continuation, closure)[29]. Sensitivity to contour is generally obtained by measuring detection thresholds using a method of constant stimuli with a nAFC/2IFC forced-choice procedure as function of various conditions (curvature, spacing, closure).

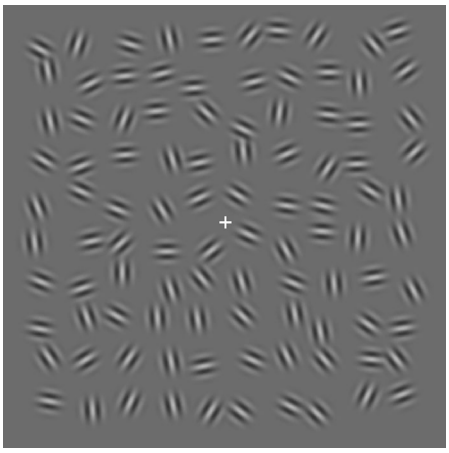

Fig. 6. Field of Gabor patches.

4.3 Contextual modulation and figure-ground segregation

Contextual visual processing is fundamentally impaired in schizophrenia[26], as well as figure-ground segregation in Alzheimer's disease. Figure-ground segregation is another form of contextual modulation thought to result from the competitive interactions between integration and segmentation of visual information[30]. A common form of contextual interaction in visual cortex is center-surround inhibition that serves as a form of local contrast-gain control.

Figure 7 shows an example of center-surround stimulus created by Psykinematix using its "Custom" feature that describes the stimulus with a set of analytical expressions (note that x and r and built-in 2D variables, z is output image):

```
sigma=0.1; sf=5; radius=2.5          # Parameters
n=unoise(r,1,1)                      # 2D Noise
gs=exp(−(r^2)/(2*sigma^2));
gb=cos(2*pi*x*sf)* gs                # Filters
in=norm(conv(gs,n),0,1);
on=norm(conv(gb,n),0,1)              # Textures
s=r<radius;
c=exp(−((rect(r−radius/2))^2)/(2*sigma^2))
                                     # Envelopes
z=in*s*(1−c)+on*c                    # Center-
                                       Surround
```

There are many other experiments that

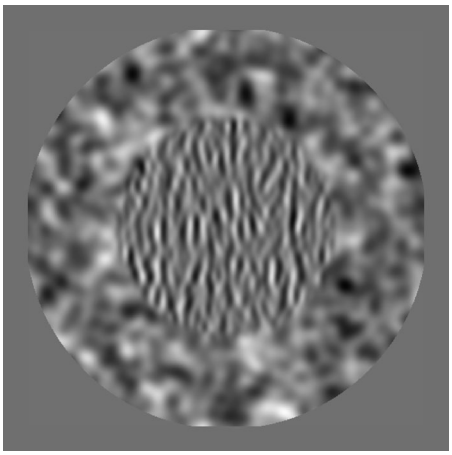

Fig. 7. Example of 2D noise stimulus with oriented fine center and non-oriented coarse surround.

involve contextual modulation that are straightforward to implement using Psykinematix, such as Polat-Sagi flanking experiments that measure contrast facilitation through lateral interactions, or orientation discrimination in presence of visual context[31]. Using a staircase method and a nAFC/2IFC forced-choice procedure, Psykinematix is able to measure detection thresholds for the target as function of the properties of the contextual surround.

Conclusion

The most common neurodegenerative diseases and mental disorders show a range of visual deficits that affect various levels of the visual system. The experimental paradigms used to investigate these visual deficits are generally based on published psychophysical studies that can be challenging for clinicians to replicate without the help of vision scientists, thus limiting their applications in clinical research. As we have seen in the clinical review, studying the visual impairments due to neural dysfunctions requires indeed elaborate experiments (e.g. threshold and sensitivity measurements through various methods), complex visual stimuli (e.g. made of multiple static or dynamic visual elements that may overlap in a linear or nonlinear way), and possibly interfacing with electrophysiological or brain imaging equipment (e.g. EEG, fMRI).

Psykinematix offers many capabilities that address these experimental requirements: it has the ability to calibrate the experimental display, run standard psychophysical protocols, generate and present complex dynamic visual stimuli, collect the subject's responses, and analyze results on the fly. Psykinematix is also versatile enough for the investigation of visual functions at various levels, from contrast sensitivity to object recognition. Finally, it can also interface with external devices through serial, USB or network connections. Psykinematix also addresses requirements that are welcome for clinical applications: a complete streamline workflow, a simple experimental paradigm, a clean interface, no need for programming, a WYSIWYG approach to stimulus creation, and finally affordability.

In conclusion, Psykinematix could become a valuable tool for clinician researchers to help them replicate fundamental studies, and better investigate visual functions that are impaired by aging and neural dysfunctions, such as shape and motion processing. Ultimately Psykinematix may also constitute an experimental platform through which vision scientists and clinicians could talk the same "language" and exchange ideas and expertise.

Disclaimer: Psykinematix is a commercial product (www.psykinematix.com)

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