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Development of Saccadic Suppression in Children

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[1]*Università degli Studi di Firenze, Florence, Italy;* [2]*University College London, Department of Psychology, London, United Kingdom;* [3]*Università Vita-Salute San Raffaele, Milan; Istituto di Bioimmagini e Fisiologia Molecolare, Consiglio Nazionale della Ricerca, Segrate Milan; Istituto Scientifico San Raffaele, Milan, Italy; and* [6]*Istituto di Neuroscienze, Consiglio Nazionale della Ricerca, Pisa, Italy*

Bruno, Aurelio, Simona Maria Brambati, Daniela Perani, and Maria Concetta Morrone. Development of saccadic suppression in children. *J Neurophysiol* 96: 1011–1017, 2006. First published January 11, 2006; doi:10.1152/jn.01179.2005. We measured saccadic suppression in adolescent children and young adults using spatially curtailed low spatial frequency stimuli. For both groups, sensitivity for color-modulated stimuli was unchanged during saccades. Sensitivity for luminance-modulated stimuli was greatly reduced during saccades in both groups but far more for adolescents than for young adults. Adults' suppression was on average a factor of about 3, whereas that for the adolescent group was closer to a factor of 10. The specificity of the suppression to luminance-modulated stimuli excludes generic explanations such as task difficulty and attention. We suggest that the enhanced suppression in adolescents results from the immaturity of the ocular-motor system at that age.

INTRODUCTION

We explore the visual world through very fast eye movements called "saccades," which cause frequent and rapid motion of the retinal image. However, the external world does not seem to move with each eye movement. Although it has been suggested that the spurious retinal motion is too fast to be perceived, and possibly masked by the fixations before and after the eye movement (Campbell and Wurtz 1978), it is now clear that very fast motion of low spatial frequencies (>500°/s), presented during fixation, can be perceived by our visual system. Best visual sensitivity at saccadic velocities is not impaired but simply shifted toward low spatial frequencies (Burr and Ross 1982).

Measurements of contrast sensitivity to gratings flashed briefly during saccades show a loss of sensitivity selective for low spatial frequency stimuli modulated only in luminance (Burr et al. 1982; Volkmann et al. 1978) but not color (Burr et al. 1994; for review, see Ross et al. 2001).

Sensitivity is very similar during fixation and saccades at the higher spatial frequencies, but at low spatial frequencies, sensitivity during saccades reduces sharply, reaching a 10-fold reduction of sensitivity at 0.02 cycles/°; these are the very frequencies that would otherwise be visible and highly conspicuous during saccades. The selectivity for spatial frequency might explain some of the conflicting data from earlier studies. Loss of sensitivity should depend on the spatial frequency content of the stimuli, typically high (e.g., small spots of light) in the luminance threshold studies (Krauskopf et al. 1966; Latour 1962; Zuber and Stark 1966) but low (large targets) in displacement studies (Bridgeman et al. 1975).

The selectivity of saccadic suppression led Burr et al. (1994) to propose that the suppression is specific for the magnocellular pathway, leaving the parvocellular pathway unimpaired. Additional support for this idea came from (Uchikawa and Sato 1995), who observed "Sloan's notch" (a dip in sensitivity at ∼570 nm) in the wavelength sensitivity function during saccades that closely resembles the wavelength sensitivity of P-ganglion cells in monkey (Ross et al. 1996).

The origin of the signal mediating saccadic suppression is still under debate. The retinal image motion produced by the saccadic eye movement may act as a mask for briefly flashed stimuli (Derrington 1984; Mackay 1970). An alternative explanation is that a top-down signal, a corollary discharge (Sperry 1950) or efference copy (Von Holst and Mittelstädt 1954) dampens the image motion signals. To disentangle the effects of the image motion from those of a saccade-related signal, Diamond et al. (2000) compared contrast sensitivity functions during real and simulated saccades (simulated by a mirror deflecting at saccade-like speed, amplitude, and acceleration). Real saccades produce a greater reduction of contrast sensitivity to low spatial frequency stimuli than simulated saccades, showing that an extra-retinal signal is necessary to suppress visual motion sensitivity during fast eye movement. The time course of suppression induced by real but not simulated saccades is very sharply defined, starting and ending ∼70 ms from saccade onset with maximum suppression at saccadic onset (Burr et al. 1994).

Another interesting question is how and where the magnocellular pathway is inhibited during saccades. One intriguing proposal is that suppression could act through contrast gain-control mechanisms (Burr and Morrone 1996). As gain-control mechanisms are present virtually everywhere in the visual processing pathways, the sites of saccadic suppression may be expected to be located at different levels. Psychophysical, neurophysiological, and neuroimaging results seem to support this expectation. Thilo et al. (2004) used transcranial magnetic stimulation (TMS) to show that suppression probably occurs early, before V2 or V1, supporting suggestions made earlier by psychophysical studies (Burr et al. 1994). Reppas et al. (2002) showed that saccadic suppression can be detected in LGN cells in monkeys. Thiele et al. (2002) found cells in MT in monkeys with suppressed activity or reversed directional selectivity during saccades, and Kleiser et al. (2004), using functional magnetic resonance imaging (fMRI), observed suppression-related activity in V5, V7, and V4 (see also Burr 2004). All

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these results suggest that suppression occurs at multiple levels of visual processing.

No studies to date have investigated the development of saccadic suppression. It is widely accepted that major development of visual sensitivity occurs during the first few years of life (for a review, see Fiorentini 1992; Teller 1997), including visual masking that reach adult-like strength in the first 2 yr of life (Morrone and Burr 1986; Skoczenski and Norcia 1998). Also many parameters of visually driven saccades, like accuracy, peak velocity, and latency, are mature by the end of the first decade of life (Fioravanti et al. 1995; Yang and Kapoula 2003). However, some aspects of saccadic motor control, particularly for anti-saccades and express saccades, seem to require longer to reach maturation, being still immature at the end of adolescence (Fischer et al. 1997; Fukushima et al. 2000; Munoz et al. 1998).

The aim of the present study is to measure saccadic suppression in children at a time when vision is mature but the saccadic motor system is not. If suppression is a consequence of visual masking, then it should be adult-like. But if, as we believe, suppression is mediated by an internal signal relative to eye position ("corollary discharge"), it may be different given the immaturity of the motor control. An uncertain and noisy eye-position signal may induce a stronger suppression to preserve perceptual stability during fast eye movements (Niemeier et al. 2003).

METHODS

Visual stimuli

Stimuli were generated at 200 Hz by a visual stimulus generator (Cambridge Research System VSG 2/4F) driven by Matlab and displayed on a Barco Calibrator monitor (medium decay time), which subtended $35 \times 24.5°$ at the viewing distance of 60 cm.

The stimulus was a horizontally oriented Gabor stimulus (35° wide and 24.5° high, space constant: 7.25 cycles/°) of a very low spatial frequency (0.15 cycles/°), which was displayed on an unstructured background (to avoid strong retinal motion signals that have been proved by Diamond et al. (2000) to reduce saccadic suppression). The stimuli were modulated either in luminance or in chromaticity: the luminance stimuli were made by summing the red and green sinusoidal gratings in the same phase, and the chromatic stimuli by summing them in counter-phase (subtracting them). For the equiluminant stimuli, the maximum stimulation along the L-M axis went from red (CIE, Commission Internationale de l'Èclairage, coordinates: $x = 0.489$, $y = 0.441$) to green ($x = 0.285$, $y = 0.581$) and produced a root-mean-square-cone contrast of 26%. Average luminance was 28 cd/m^2. Equiluminance was established by flicker photometry, adjusting the ratio of the red-to-green luminance modulation to produce minimal flicker of the stimulus when modulated at 20 Hz.

The luminance gabor stimulus was displayed for one frame of 5 ms; the chromatic modulated stimulus for five frames, 25 ms (persistence of the phosphors around 3 ms).

Eye movements

Eye movements were recorded by means of an infrared limbus eye-tracker (HVS SP150), with horizontal resolution of 0.01° and accuracy 0.1°. The infrared sensor was mounted below the right eye on the frame of lensless plastic goggles through which observers could see the monitor binocularly. The sensor was peripheral enough to be unnoticeable to the subjects. At the beginning of each session, subjects were asked to saccade to three dots arranged horizontally to calibrate the gain and the linearity of the eye-tracker.

For each trial, we stored the eye trace recorded by the eye-tracker, the presentation times for all the stimuli and saccadic latency together with the subject's response. Saccadic onset and termination were estimated by convolving the eye trace with the second derivative of a Gaussian function of 5-ms time constant and locating the local maxima and minima. The procedure evaluates the points of cusp, corresponding to local maxima of the eye acceleration. A later off-line analysis allowed us to check more accurately the quality of saccade and computer's estimate of saccade onset and the latency of the stimulus presentation from the saccadic onset. If the primary saccade was followed by a corrective saccade larger then ~2°, the trial was eliminated.

Subjects

Subjects were divided into four groups on the basis of their age at the moment of the experiment. *1*) Ten adolescents between the age of 12 and 14 yr [mean age = 12.9 ± 0.94 (SD) yr], five of them were female and five male; *2*) ten adult subjects between the age of 21 and 31 yr (mean age = 24.6 ± 2.6), seven of them were female and three male; *3*) three older adolescents between the age of 15 and 18 yr, two of them were female and one male; and *4*) three children between the age of 8 and 11 yr, all female.

Visual acuity was normal or corrected to normal in all subjects, and all but one had good color vision (AB is a red-green dichromat). Contrast sensitivity to luminance-modulated stimuli for both fixation and saccadic trials was determined for all the subjects, while contrast sensitivity to chromaticity-modulated stimuli was calculated for some subjects belonging to the first two groups (9 adults and 6 adolescents).

Procedure

Subjects sat in a dimly illuminated room facing the monitor onto which the stimuli were displayed. The head of the subject was constrained by a neck-rest (mounted on the back of a comfortable chair) to minimize head movements and to keep the distance between the eye and the monitor constant at 60 cm.

All the subjects ran two experimental conditions in separate sessions.

FIXATION TRIALS. Subjects maintained fixation on a black dot (0.5° diam), which was continuously displayed at the center of the monitor (0°). The gabor stimulus was randomly displayed above or below the horizontal midline, and subjects reported its position verbally. The same procedure was adopted for both luminance-modulated or for chromaticity-modulated stimuli.

SACCADIC TRIALS. At the beginning of each trial, subjects fixated a black spot (0.5° diam) displayed 8° left of screen center. The spot disappeared and an identical spot (saccadic target) appeared 8° right of monitor center, to which subjects saccaded. The stimulus presentation was triggered by setting a threshold on the eye-position deflection and usually occurred before 20 ms after saccade onset, the interval of maximum suppression in adults (Burr et al. 1994; Diamond et al. 2000). An off-line analysis rejected all trials with stimulus latency >20 ms to have maximal suppression. About half of the trials met the criterion and were included in the analysis for both groups (adolescent valid trials, 50%; adult valid trials: 52%). After the saccade, subjects reported verbally the position of the gabor stimulus as before.

To verify that the 20-ms delay from saccadic onset produced maximum suppression in adolescents as it does in adults, we measured performance to a stimulus of constant contrast (the value of which corresponded to the fixation threshold) presented randomly in the range –200 to +200 ms from saccade onset, for two subjects (1 from the 12- to 14-yr group, the other from the adult group; see RESULTS and Fig. 5).

Thresholds were measured by two-alternative forced choice. Subjects reported verbally whether the gabor stimulus was perceived above or below the horizontal midline of the monitor. The responses were recorded by one of the experimenters by means of a response box and stored in digital form for later analysis. An acoustic signal followed incorrect responses.

Contrast varied from trial to trial to home in on threshold (which was defined as the contrast level which elicited 75% of correct responses), using the adaptive QUEST procedure (Watson and Pelli 1983). The final estimate of threshold was obtained by fitting the frequency-of-seeing functions (percent correct vs. contrast) of all trials of a given condition with a cumulative Gaussian function. The two free parameters, contrast threshold and SD, were determined by minimizing the residual mean square error between data and prediction, using the simplex algorithm (Nelder and Mead 1964).

RESULTS

Sample psychometric functions for luminance-modulated stimuli are shown in Fig. 1 for a 13-year-old child (*right*), and a young adult (*left*). Sensitivity to stimuli triggered by the saccade (●) and during fixation (●) were shown. While the fixation thresholds were very similar for the two subjects, the saccadic threshold of the 14-year-old is about three times that of the adult. Note, however, that the psychometric functions are ordered and steep for both observers, suggesting that the young observer had no particular difficulty in performing the task under these conditions.

Figure 2*A* shows the psychometric functions during fixation and saccades for another 3 of the 10 children. All curves are orderly and smooth and have similar steepness. However, in all cases, the functions obtained during saccade are shifted by more than a factor of 10 toward higher contrast, indicating strong suppression.

Figure 3*A* shows a summary of all threshold results for these two groups of subjects for the luminance-modulated gabor stimuli. Contrast sensitivity measured during saccades is plotted as a function of contrast sensitivity measured during fixation. All the data points lay below the equality line (- - -) indicating that saccadic suppression is significantly present for both groups. It is also evident that the fixation sensitivities (abscissae) of the two groups are very similar (verified by Mann-Whitney U test: $z = -0.983$, $P = 0.353$). On the contrary, the contrast sensitivities of two groups during sac-

FIG. 2. Example of psychometric function of contrast detection to luminance modulate (*left*) and chromatic modulated (*right*) stimuli presented during fixation (■, ●) of just after the initiation of a saccade (■, ●) in 3 children. The contrast is express in RMS cone contrast; the luminance stimulus had a duration of 1 frame (5 ms), whereas the chromatic modulated stimulus of 5 frames (25 ms) to balance for visibility during fixation. Each point is the average of ≥6 trials.

cade (ordinate values) are clearly separate, indeed nonoverlapping. Contrast sensitivity of the adolescents is significantly lower than that observed in the adult group (Mann-Whitney U test: $z = -6.975$, $P < 0.0001$).

To exclude the possibility that the stronger suppression of adolescents could be limited to very brief stimulus exposures, we measured contrast sensitivity to luminance modulated ga-
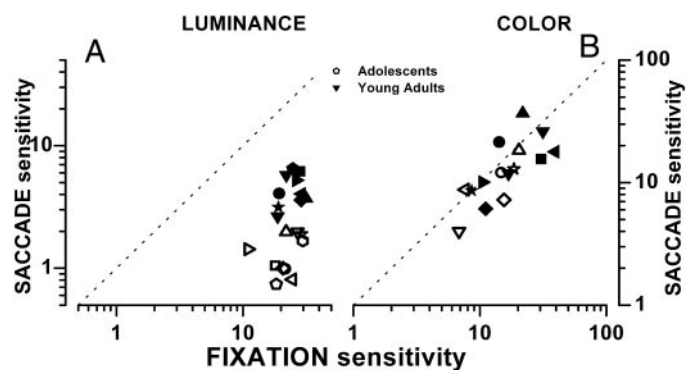
FIG. 3. *A*: contrast sensitivity during saccades plotted against the contrast sensitivity during fixation for luminance-modulated stimuli. The empty symbols refer to adolescents, filled symbols to adults; different symbol shapes correspond to different subjects. The dotted diagonal line shows equality in sensitivity. Fixation sensitivity: adolescents = 22.22 ± 1.8 (SE); adults = 24.85 ± 1.5; Mann-Whitney U test: $z = -0.983$, $P = 0.35$). Saccade sensitivity: adolescents = 1.35 ± 0,15; adults = 4.49 ± 0.4; Mann-Whitney U test: $z = -6.975$, $P < 0.0001$. *B*: RMS-cone-contrast sensitivity measurements for an equiluminant chromaticity-modulated gabor stimulus for adolescent and adult subjects. Fixation sensitivity: adolescents = 13.42 ± 2.19; adults 19.72 ± 3.51; Mann-Whitney U test: $z = -1.061$, $P = 0.328$. Saccade sensitivity: adolescents = 10.16 ± 1.97; adults = 16.48 ± 3.12; Mann-Whitney U test: $z = -1.179$, $P = 0.272$.
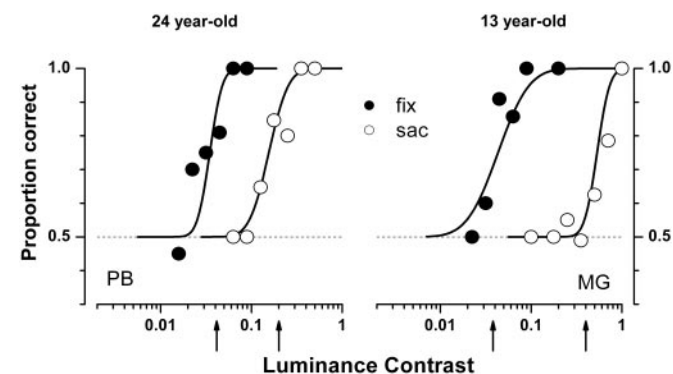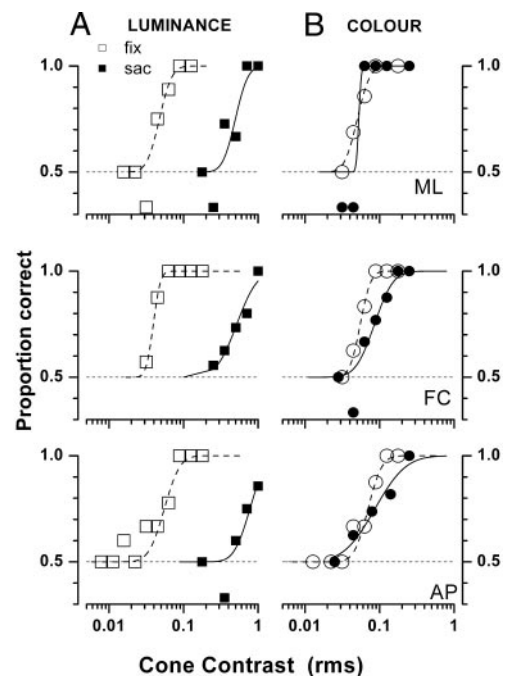
FIG. 1. Contrast sensitivity of a young adult (*left*) and adolescent (*right*) to a brief flash (1 frame = 5 ms) presented during fixation (●) of just after the initiation of a saccade (○). ↑, thresholds. Twenty-four-year-old subject: fixation threshold: 0.035; saccade threshold: 0.154; 13-yr-old subject: fixation threshold: 0.043; saccade threshold: 0.536. Each point is the average of ≥6 trials.

bors displayed for 25 ms for a few subjects. The poorer performance of adolescents during saccades did not vary with stimulus duration: the ratio in contrast sensitivity between fixation and saccade remains constant, presenting the luminance-modulated gabor for five frames to the same subjects (data not shown).

The difference did not result from a delayed presentation of the stimulus in the children. The average stimulus trigger delay was not significantly different between the data recorded in the adolescents and in the young adults, (adolescents: mean delay = 19.1 ± 0.69 (SE); adults: mean delay = 17.0 ± 0.81, Mann-Whitney U test: $z = -1.89$, $P = 0.063$). Although not statistically significant, the delay was slightly longer in children; this should induce a decrease in suppression with respect to the young adults. In addition, no statistically significant difference was observed between the mean saccadic latencies of the two groups (adolescents: 253 ± 16 ms; adults: 233 ± 17 ms. Mann-Whitney U test: $z = -0.68$, $P = 0.53$), indicating that the execution of the saccade is mature at this age, in agreement with previous reports (Fioravanti et al. 1995; Fischer et al. 1997; Munoz et al. 1998; Yang and Kapoula 2003).

Figure 2*B* shows example data obtained for the equiluminant chromatic stimuli for the same children illustrated for the luminance modulated stimuli (Fig. 2A). For these stimuli, subjects performed reliably, producing orderly and steep psychometric functions. However, for these stimuli the curves measured in fixation and in saccades overlap, indicating an absence of saccadic suppression, like in adults (Burr et al. 1994). Figure 3*B* shows the summary data of children and young adults, plotting contrast sensitivity (expressed as the inverse of cone contrast) measured during saccadic trials against contrast sensitivity measured during fixation. All the data points are scattered around the equality line indicating, as previously reported (Burr et al. 1994), that no saccadic suppression is detectable for color-modulated stimuli, neither for adults nor for adolescents. Comparing mean contrast sensitivity of the 12- to 14-yr-old group with that obtained in the 21- to 31-yr-old group, we found no statistically significant difference, neither during fixation (Mann-Whitney U test: z = −1.061, $P = 0.328$) nor during saccades (Mann-Whitney U test: $z = -1.179$, $P = 0.272$). On average, the point of equiluminance was not significantly different between adolescent and adult subjects.

To estimate the magnitude of saccadic suppression, we calculated the ratio of fixation to saccadic contrast sensitivity for each subject: the greater the index, the stronger the suppression. In the adolescents, suppression for luminance-modulated stimuli is much stronger: the average ratio of fixation to saccadic sensitivity is equal to 18.0 ± 2.1, more than three times as large as that observable in the adults (5.9 ± 0.5) and the difference is statistically significant (Mann-Whitney U test: $z = -3.268$, $P < 0.0001$). For the chromaticity-modulated stimuli, no difference is present between the two groups (adolescents: 1.4 ± 0.2; adults: 1.3 ± 0.2. Mann-Whitney U test: $z = -0.471$, $P = 0.69$), and none of the ratios is significantly different from unity, confirming that no suppression is present.

We also controlled for the importance of gender in determining the difference in the magnitude of suppression between adolescent and adult subjects. Both in adolescents and in adults, the amount of suppression for male subjects did not differ significantly from that for females (Mann-Whitney U test: adults: $z = -0.52$, $P = 0.69$; adolescents: $z = -0.77$, $P = 0.55$).

To examine maturation of saccadic suppression with age, we measured contrast sensitivity to luminance-modulated gabor stimuli during fixation and during saccades in three additional subjects between the age of 8 and 11 yr and in three subjects between the age of 16 and 17 yr. Figure 4 plots the index of suppression as a function of the age of all subjects ($n = 26$). There is a significant negative correlation ($r = -0.66$; $F = 18.9$; $P < 0.001$) between age and suppression index: the estimated linear trend having a slope of 0.76 ± 0.17 log-units/yr.

A possible explanation for the difference in the amount of saccadic suppression between young adults and children could be that the temporal dynamics of suppression differ between the two groups: children may have suppression that reaches a maximum after saccadic onset. To check this possibility, we measured the time course of saccadic suppression for two different subjects from the main experimental groups (FB = 14 yr old; SMB = 26 yr old*)*. To measure contrast threshold at fine delays before saccadic onset in young children would require many days of observation given the eye-triggering technique cannot be used and the saccadic latency of the children is highly variable. A more feasible technique is to measure performance for luminance stimuli at a fixed contrast (equal to fixation threshold) at various delay with respect to saccadic onset. The curves showing percent correct responses against stimulus trigger delay (Fig. 5) are not significantly different between the two subjects (Mann-Whitney U test: $z = -0.23$, $P = 0.817$). Suppression was maximal during the same range for both observers, 0–20 ms after saccade onset. However, the suppression was more enhanced for the 14-yr-old, who performed at chance over a wider range of delays compared with the adult (from 20 ms before to 20 ms after saccade onset). For both subjects, there was a similar facilitation effect for stimuli displayed well before saccade onset: the percentage of correct responses is slightly higher in the range −160 to −240 ms compared with that obtained in the range 160–240 ms. The facilitation effect, already described both for luminance and equiluminant grating (Deubel and Schneider 1996; Diamond et al. 2000), may reflect an increase of the attentional level of the
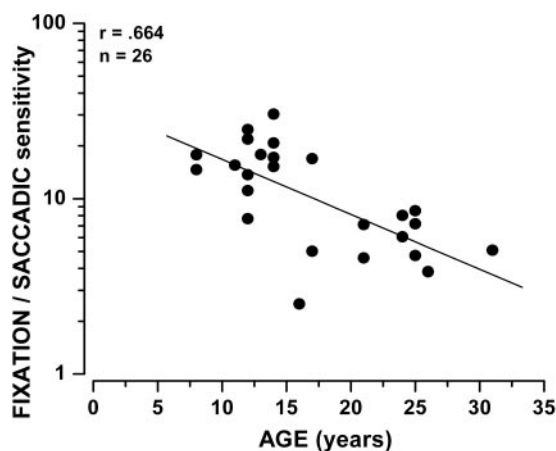

FIG. 4. The ratio of fixation to saccadic sensitivity is plotted as a function of age. The linear fit of the data (corresponding to the equation $Y = 25.291 - 0.762*X$) is also shown (—).
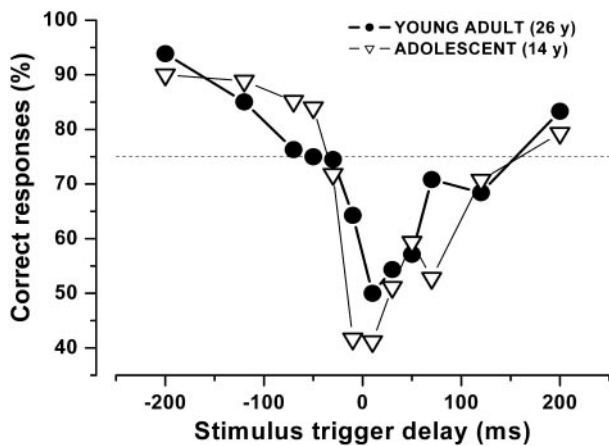
FIG. 5. Percentage of correct responses for detecting the position of a horizontally oriented gabor stimulus modulated in luminance briefly displayed at various times relative to the onset (0) of a horizontal saccade for a 14-yr-old subject (▽) and for a 26-yr-old subject (●). The contrast value of the gabor stimulus was constant across the trials, and it corresponded to the threshold value observed during fixation (defined as the contrast at which subjects are 75% correct, as indicated by - - -). A presaccadic performance at about 100% indicate the presence of presaccadic facilitation. Each symbol is the average of ≥40 trials.

subject before the execution of the saccade. These results show that saccadic suppression follows similar dynamics in both subjects.

DISCUSSION

The general aim of the present study was to investigate the maturation of saccadic suppression. The major results for luminance-modulated stimuli show that sensitivity attenuation during saccades is much stronger for younger subjects: the magnitude of the suppressive effect is more than three times as large as that observed in adults. No difference in the sensitivity to equiluminant chromatic stimuli was detected.

The difference in the amount of saccadic suppression between the two groups can be entirely ascribed to a difference in the sensitivity during saccades: thresholds during fixation was the same in adolescents as in adults (Fig. 3), in agreement with a large body of literature (for review, see Atkinson 2002; Fiorentini 1992; Teller 1997). Sensitivity to visual motion also seems to develop quite early (for a review, see Atkinson 2002; Wattam-Bell 1991) and is certainly adult-like in 10-yr-old children (Ellemberg et al. 2003). Our gabor stimuli did not move, but it is well known that brief exposure of stationary stimuli, particularly stimuli of low spatial frequency, excite motion detectors (Burr et al. 1982).

One may speculate that the increased suppression reflects an immaturity of visual masking mechanisms that could contribute in children, but not in young adults, to the overall suppression. However, two facts argue against such an explanation. First, the detrimental effects of visual masking are adult-like by ~2 yr of age (Candy et al. 2001; Macchi et al. 2003; Morrone and Burr 1986; Skoczenski and Norcia 1998). It would be strange if it were to increase above the normal adult level in adolescence with obvious implications to other aspects of visual function. In similar experimental conditions to those employed here, Diamond et al. (2000) showed that the contribution of visual masking to saccadic suppression is less than a

factor of 1.5. To explain the present data, we should hypothesize that visual masking in children should be stronger by a factor of ~3, which would impair many other visual functions.

Second, it would be strange if visual masking, equally strong for luminance and chromatic stimuli in adults, were selectively augmented in children only for luminance. The lack of suppression for equiluminant gratings is not due to the reduced cone contrast of the stimuli. To balance the reduced visibility of the chromatic grating, we need to increase the duration of the stimulus. However, we also checked that stimuli of 25-ms duration could be efficiently suppressed in agreement with previous evidence (Burr et al. 1994), indicating that, at difference with the luminance sensitivity, suppression for equiluminant gratings do not show a maturation trend.

Another possible explanation, similar to masking, is that the enhanced saccadic suppression reflects immature gain-control visual mechanisms in adolescence. On the basis of measurements of the impulse response function, Burr and Morrone (1996) suggested that saccadic suppression may operate through contrast gain mechanisms, an idea supported by measurements with real and simulated saccades (Diamond et al. 2000). Interestingly, M cells but not P cells show contrast gain control (Benardete et al. 1992), and the evidence suggests that M cells are suppressed during saccades (Burr et al. 1994). An immature contrast gain mechanism would reach maximum setting with a small input signal, producing a stronger suppression. There is no direct evidence that the contrast gain control mechanisms are immature in adolescents, but it would seem worthwhile to investigate this possibility. Some circumstantial evidence in patients affected by photosensitive epilepsy points to this possibility (Porciatti et al. 2000).

One possibility is that saccadic suppression is modulated by attention, and regulation of attentional resources is still immature in adolescents. Several studies using dual task paradigms indicate that top-down control over attention is still maturing during childhood and adolescence, even though the process of allocating attention is adult-like quite early in childhood (Atkinson et al. 1992; Karatekin 2004; Sireteanu and Rettenbach 1996). If the saccadic task requires more attention than the fixation task, the possible limitations of attentional resources in children could cause the greater suppression in adolescents. However, our results also show that in an equally demanding saccadic task with equiluminant chromatic stimuli, no suppression occurs either in adults or adolescents. Furthermore, the presaccadic facilitation effect, thought to be related to the attentional shift at saccadic target (Deubel and Schneider 1996), was equally present in both groups (see Fig. 5). Thus it seems unlikely that the results can be ascribed to deficits in attentional control in adolescents.

While we cannot completely rule out an immaturity of visual mechanisms, the present data would support the idea of an immaturity of the circuitry that controls or mediates an internal extra-retinal signal. Most of the classical and recent literature on saccadic suppression point to the existence of a corollary signal that mediates suppression (Bridgeman et al. 1975; Krauskopf et al. 1966; Latour 1962; Zuber and Stark 1966). Saccadic suppression presumably serves to preserve the stability of the visual world from the perturbations induced by the retinal image motion at saccadic velocities. Given that the motor system in children is still immature, it would be advantageous for the brain to allow a greater plasticity and a longer

stabilization period for the corollary discharge. For example in young children, eye-movement conjugation is different from in adults (Fioravanti et al. 1995), given the closed distance between the eyes and more probable asymmetry between the mechanical properties between the two eye (given that the head is still growing). It would be a greater advantage to the system to have a stronger suppression to eliminate the possibility of additional mismatch between the two eyes. Niemeier et al. (2003) simulated a visuomotor system that optimally integrated three imperfect signals in a saccadic task: a signal relative to the position of the stimulus on the retina, a motion signal, and an eye-position signal. They observed that this optimal integration unavoidably leads to suppression of displacement of an object during a saccade. The model accurately predicted the fact that the amount of suppression in human subjects increases linearly with the uncertainty of the eye-position signal (estimated by the SD of the postsaccadic position of the eye). In children the precision of landing accuracy, peak velocity and duration of saccades develop and mature very early, reaching adult levels by 5 yr (Fioravanti et al. 1995; Munoz et al. 1998; Yang and Kapoula 2003). However, other less automatic mechanisms tend to improve over a longer period of time (Fischer et al. 1997; Fukushima et al. 2000; Munoz et al. 1998). For example, latencies and frequency of express saccades, number of directional errors in the anti-saccade task and the coordination between the two eyes (Fioravanti et al. 1995; Yang and Kapoula 2003) continue to improve during adolescence and reach adult-like values around the age of 20 yr (Fischer et al. 1997). These immaturities could increase the uncertainty of the eye-position signal and induce a stronger suppression (Niemeier et al. 2003). All these studies also indicate that the voluntary component of saccadic control is still underdeveloped in adolescence. This would agree with the general belief that subcortical circuits develop before cortical circuits, particularly those mediating high cognitive function. Brain stem burst generators (probably controlling the more automatic saccadic parameters) is supposed to be mature very early (Brody and Vijayashankar 1977), frontal and prefrontal cortices, which would provide a top-down modulation of the saccadic system, are probably still developing during adolescence (Sowell et al. 1999). Interestingly, a recent fMRI study, (Luna et al. 2001) showed that children and adolescents performing an anti-saccadic task activate less than adults the superior frontal eye fields, intraparietal sulcus, thalamus, cerebellum, and superior colliculus, all structures implicated in programming and control of saccadic eye movements. All these findings suggest that the larger suppression we observed in adolescent subjects may be due, at least partially, to an underdevelopment of the motor ability and to a more noisy corollary discharge signal operating in the adolescence.

It is interesting that we found no statistical significant difference between male and female adolescents, given that males and females go through a different hormonal development that may result in sexually dimorphic cerebral structure and function (Killgore et al. 2001; Pilgrim and Hutchison 1994).

Whatever the underlying neuronal mechanisms, the present data show that adolescent children show far greater saccadic suppression than do adults, demonstrating that the visual system is still developing at that stage and is presumably very plastic.

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