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REVIEW PAPER

A primer on motion visual evoked potentials

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Abstract Motion visual evoked potentials (motion VEPs) have been used since the late 1960s to investigate the properties of human visual motion processing, and continue to be a popular tool with a possible future in clinical diagnosis. This review first provides a synopsis of the characteristics of motion VEPs and then summarizes important methodological aspects. A subsequent overview illustrates how motion VEPs have been applied to study basic functions of human motion processing and shows perspectives for their use as a diagnostic tool.

Keywords Visual evoked potential · Electrophysiology · Magnetoencephalography · Motion perception · Clinical diagnosis · Area MT

Introduction

The perception of motion is one of the fundamental tasks of the visual system (see [1] for a concise review). Motion visual evoked potentials (motion VEPs) have been used for several decades to investigate motion processing in humans, both in basic and in clinical research. Yet, many of their characteristics have only been revealed relatively recently, in parallel with advances in the general knowledge about the human visual system and its motion-processing mechanisms.

The cortical circuits specialized in processing motion are considered to be part of the magnocellular system, or, more strictly speaking, part of the dorsal stream as the cortical analog to the subcortical magnocellular system [2–4] (see [3], though, for a review of the problems of this view).

Two properties of motion processing mechanisms are particularly imprtant for recording and understanding motion VEPs: First, motion-specific mechanisms are commonly defined to be direction-specific [5]. Second, adaptation plays an important role in motion processing. This can be demonstrated psychophysically via the motion aftereffect [6, 7]. Both properties are also found in motion VEPs.

The first measured electroencephalographic responses related to motion were possibly those evoked by the retinal image shift during eye movements, recorded by Gastaut and Régis [8] and Evans [9] around 1950. They are also known as 'lambda wave' [e.g., 10, 11]. Dating from the early 1950s, the first report on steady-state VEPs to a moving stimulus is almost as old: Marshall and Harden [12] used repeatedly expanding rings to demonstrate the capabilities of a new cathode ray tube (CRT) stimulator.

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Since procedures for recording motion VEPs are not standardized, this review first provides a general characterization and a methodological overview. Next, applications of motion VEPs in basic and clinical research as well as perspectives for their diagnostic use are summarized. In addition to VEP studies, recent findings using magnetoencephalography (MEG) are also taken into account. Rather than to discuss a specific application in great detail, the aim of this review is to provide a broad overview that will serve as a starting point for more in-depth reading.

General characteristics

Motion-onset VEPs

The majority of studies used motion-onset VEPs, where the stimulus consisted of a pattern that started moving after being stationary for a sufficient amount of time to allow the neural activity evoked by the on- and off-set of the preceding stimulus to cease.

MacKay and Rietveld [13] were the first to systematically assess various properties of motion-onset potentials, including effects of eccentricity, pattern, and direction. However, their potentials were atypical compared to many later studies, since they were characterized by a negativity around 60 ms, followed by a positive deflection.

In most studies, the motion-onset VEP is dominated by an occipital and occipito-temporal negativity that peaks between 150 and 200 ms after motion onset (Fig. 1). For this component, several designations have been used in the literature, such as C_{II}, N200, N1, or N2. In the present review, the latter, N2, will be used, taking into account that an earlier negativity has been reported and that it resembles the pattern-reversal N2 in timing and some other properties (see below).

The motion-onset N2 is often preceded by a positivity that is weak at occipito-temporal locations, but usually stronger at the occipital pole [14]. It seems to be largest for foveal stimulation [15]. In addition, there is a vertex positivity around 200 ms. No sizable variability of the shape

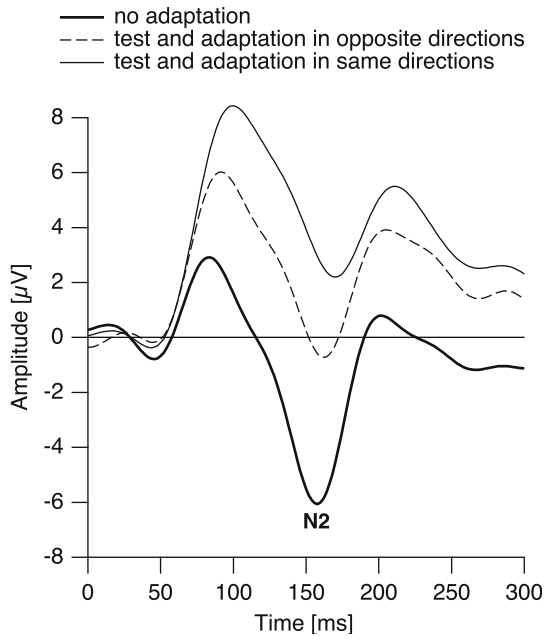

Fig. 1 Sample motion-onset VEPs recorded from an occipito-temporal electrode, dominated by the N2 negativity. The traces represent different adaptation conditions: no adaptation, adaptation in the opposite direction and adaptation in the same direction as the test motion. Adaptation is partly but not fully direction specific. Experimental details can be found in [17]

and size of the motion-onset VEP has been found for different times of the day [16].

Based on a variety of reasons, the occipito-temporal negativity around 150–200 ms is generally considered to reflect motion processing: First, relatively low stimulus contrasts are sufficient to elicit a large response [4, 18–22]. Second, it appears to have its origin in area MT (V5) [4, 23–26], possibly with contributions from V3/V3A or nearby [27, 28]. This corresponds well to the localization of motion-processing mechanisms found with other techniques such as functional magnetic resonance imaging [e.g., 29–31] or positron emission tomography [e.g., 32, 33], though the actual role of area MT in motion processing is not yet fully understood [34]. Third, it is highly susceptible to adaptation [35–39] and the amount of signal reduction depends on the relative directions of adaptation and test motions [25, 40–45], as illustrated in Fig. 1. Adaptation is not fully direction-selective. Therefore, the N2 component has been

interpreted as a compound of two types of responses, one of them reflecting veridical motion processing and the other originating from various neural circuits sensitive to temporal luminance modulations [46].

The N2 amplitude often differs between hemispheres [e.g.,15, 47–49]. In a sample of 80 subjects, Kubová et al. [14] found in 60% of the cases a higher amplitude in the left hemisphere. This was uncorrelated to handedness.

Motion-reversal VEPs

A few studies investigated motion-reversal stimuli, recording transient [50–54] or steady-state responses [42, 55–58]. The term 'steady-state' refers to the case that the time interval between stimuli is too short for the response to decay before the next stimulus is presented. Many studies used abruptly reversing stimuli, but sinusoidal motion has also been applied [59, 60].

Steady-state reversal VEPs are affected by unidirectional pre-adaptation [42, 56, 59]. This is most likely also true for transient reversal VEPs, but has not yet been verified. Steady-state reversal VEPs exhibit a considerable interindividual variability but prolonged stimulation does not result in sizable adaptation [42]. A high interindividual variability was also found for transient reversal VEPs [51, 52]. Generally, positive components around 100 ms are more pronounced than in motion-onset VEPs, but it is not clear to which degree this is due to differences in adaptation. Adaptation might also be the reason why Henning et al. [53] only obtained very weak responses to motion reversal stimuli.

It is likely, though unproven, that reversal stimuli generally underestimate the motion responses. If the response of the neural population stimulated by one direction was simply replaced by response of the population sensitive to the other direction, a zero net effect would be expected [42]. The fact that motion reversal VEPs can be recorded even if other stimulus discontinuities are accounted for [57] might be due to short-term adaptation processes [61], differences between onset and offset latencies [62, 63], and potentially other transient effects.

Motion-offset VEPs

Motion-offset stimuli have only been used in a few studies, e.g. to investigate the motion aftereffect [64] or certain attentional effects [65]. Generally, motion-offset potentials seem to have a negative deflection, but are smaller and less uniform than onset potentials [15, 39]. Their latency seems longer, at least when measured at Cz for slow speeds [66]. Dominant positive peaks have also been reported [67].

Other types of motion-related VEPs

In addition to other brain areas, pattern reversal stimuli also activate area MT [53, 68, 69]. The assumption that a pattern reversal stimulus is virtually identical to a motion stimulus [70] is probably too simplistic, but there is increasing evidence that at least some components of pattern-reversal VEPs are in part evoked by the apparent motion that occurs during the reversal. This has in particular been suggested for the N2 deflection, based on curve shape, stimulus contrast effects, and adaptability [19, 21, 71–73]. On the other hand, at least parts of the pattern-reversal P100 have recently been reported to have its origin in area V3 and MT [74], though the stimuli used in that study where not very typical and the results contradict with several earlier studies. Interestingly, the pattern reversal P100 is also drastically reduced with simulated nystagmus [75], which might hint towards a connection to motion processing. Clarke [76] found that simultaneous motion-onset does not change the response to pattern onset. In contrast, Mackie et al. [77] did find such changes. The authors suggest that simultaneous pattern and motion onset evokes responses similar to a superposition of separately-measured pattern-onset and motion-onset potentials, but the study does not actually verify this directly. Buchner et al. [78] report that pattern-reversal evokes an early negativity with an onset around 30 ms and a peak around 45 ms that originates from the vicinity of MT.

Most studies have focused on transient or steady-state motion VEPs. Patzwahl et al. [79] were an exception. They used DC amplifiers to record sustained neural activation to ongoing object motion.

It is beyond the scope of this review to address event-related potential components associated with cognitive processing, such as the P300 [e.g., 80–82] or changes in certain EEG frequency bands [e.g., 83, 84]. The fields of object motion [e.g., 85] and biological motion [e.g., 86] are also largely spared. Although this review focuses on perceptually continuous motion, studies on two-frame apparent motion are included where particularly relevant.

Recordings and analysis

Stimulus patterns

A wide range of patterns can be used for recording a motion-onset VEP. Random-dot patterns, sine wave gratings and checkerboard or dartboard patterns have been frequently used. From a theoretical viewpoint, the spatial frequency distribution of a pattern might matter. For instance, spatial and temporal parameters are linked by the speed of the stimulus. Furthermore, many models of motion perception, such as the Hassenstein-Reichard detector [87, 88], are sensitive to changes in temporal parameters. Practically, the effect of the stimulus pattern seems to be minor [17, 36, 89, 90]. For central presentation, the size of the stimulus field has only little effect [89].

The motion-processing mechanisms are relatively sensitive to low-contrast stimuli. Consequently, contrasts in the range of 2% are still sufficient to evoke a large VEP response [18–20] and many studies use low-contrast patterns in order to enhance the motion-specificity of the VEP responses. Very low-contrasts result in increased latencies [19]. A very low luminance of 0.003cd/m^2 is sufficient for evoking a motion response [91, 92].

Direction and speed

Generally, motion-onset VEPs can be evoked by any direction of movement. However, radial motion seems to yield higher amplitudes [93] and has been proposed to reduce optokinetic nystagmus [42, 93]. Expansion was found to elicit larger responses than other directions [94, 95]. Since radial motion may be perceived as a flow field similar to those associated with a forward or backward movement in space, additional cortical circuits might be stimulated, such as the vestibular cortex [96]. A higher amplitude for radial motion agrees well with the recent psychophysical findings that thresholds in a motion discrimination task are lowest for radial motion [97]. Tobimatsu et al. [98] found a larger N2 for horizontal motion, while their VEPs to radial motion are dominated by a positive deflection around 200 ms. Since they report only few stimulus details, it is difficult to estimate where the differences to the aforementioned studies originate from. For rotating patterns, N2 amplitudes and latencies do not depend on the direction of motion [91]. Using gratings that moved orthogonally to their orientation, Maffei and Campbell [99] showed that lower steady-state amplitudes are obtained for oblique motion directions than for cardinal directions, though several stimulus details of that study are unclear. They did not find such an effect in the electroretinogram. Delon-Martin et al. [95] report that the type of movement (translation, contraction, expansion, rotation) differentially affects the strength and temporal characteristics of extrastriate and parietal activations.

As a general rule, latency decreases and the amplitude increases with increasing speed [e.g., 21, 45, 67, 100–102]. In a study by Kuba and Kubová [15], however, the most negative N2 was found for speeds around 6°/s, with an increase of the P1 amplitude and a roughly constant P1–N2-difference for higher speeds. Typical speeds are in the range of 1–10°/s, but higher speeds have been used by some investigators. Varying the speed of a light spot in six steps from 0.4 to 500°/s in an MEG experiment, Kawakami et al. [103] have probably tested the widest speed range of any study.

While the angular speed (usually measured in degrees per second) is most commonly used to describe motion stimuli, occasionally the contrast (or temporal) frequency (in Hertz) is given instead, in particular for regular gratings. It represents the frequency of the flicker that occurs at any given spot within the stimulus field while the stimulus pattern is moving across. It has been suggested that a sufficiently low contrast frequency (< 6 Hz) is important for recording

motion-onset VEPs [15, 104]. It is not clear, however, to what degree this favors either the magnocellular or the parvocellular pathway, since their respective temporal frequency sensitivities are largely overlapping with a slightly higher sensitivity of the magnocellular pathway for high frequencies [3].

Most of the more recent studies used pixel-based computer monitors for stimulus display. Strictly speaking, the motion is therefore not continuous. Nevertheless, it is assumed that for most purposes the approximation is sufficiently good and studies on apparent motion (see further below) suggest that the same cortical mechanisms are involved in the processing of apparent and continuous motion.

Stimulation schemes

A series of studies have demonstrated that adaptation during repeated stimulation is a crucial problem in the experimental design that can be alleviated by inserting long stationary intervals between motion epochs [35–38]. Bach and Ullrich [39] have analyzed this quantitatively by varying the duty cycle (i.e. motion duration divided by total trial duration). In typical low-adaptation experiments, stimulation sequences for motion-onset VEPs often consist of brief motion epochs of 200–300 ms duration, separated by stationary epochs of 2–3 s, keeping the duty cycle in the range of 10–15%. One should be aware, though, that local luminance adaptation may occur during stationary epochs. This has been suggested by Heinrich et al. [17] and might also cause the 'sustained component' described by Markwardt et al. [90], which oscillates with a frequency corresponding to the temporal frequency of the stimulus.

In order to isolate 'veridical', i.e. direction-specific, motion responses, several approaches have been proposed for motion-onset VEPs. In one of them, the interval between two test motion epochs is filled with motion having a direction opposite to the test motion, leaving just a short stationary epoch before the onset of the test motion [e.g., 25, 41, 45]. This anti-directional motion ensures that non-direction-specific cortical circuits are adapted, leaving the response dominated by direction-specific mechanisms that are sensitive to the test direction. Another approach is the use of a flickering stimulus to adapt the non-specific circuits. This can be achieved by assigning a limited lifetime to each dot of a random dot pattern, as suggested by Maurer and Bach [46]. It has the additional advantage that the transition between a flickering stimulus and a motion stimulus should be largely invisible for non-directional mechanisms. Under the conditions tested by Maurer and Bach, though, the response did not become completely direction-selective. In many cases, adapting with anti-directional motion seems to be the more reliable approach since it ensures that the temporal characteristics of adaptation and test stimuli match. However, in cases where the experimental paradigm does not allow for anti-directional adaptation, the second approach will be valuable. A third way of isolating veridical motion responses is to measure responses to motion and to noise separately and then subtract the two resulting curves, ideally leaving only the direction-specific component [105].

For motion-reversal stimuli, a different method has been proposed. The issue here is that the position of a pattern would be exactly repeated before and after a reversal. Wattam-Bell [57] has solved this problem by replacing the random-dot stimulus by a new uncorrelated pattern both at the times of the reversals and midway between the reversals. The recording is then analyzed in the frequency domain, where the response at the reversal rate reflects directional processes, while responses associated with pattern changes are found at twice the reversal rate. However, this method does not take into account that the direction-selective response itself might contain harmonics, even in absence of the pattern replacement. These harmonics would be discarded together with the pattern-replacement response.

Recording locations

Which electrode locations should be chosen if only a small number of channels is available? For the N2 component Göpfert et al. [48] have suggested positions 5 cm to the left and right of Oz (see American Encephalographic Society

[106] for nomenclature and locations of EEG standard positions). This has been adopted by a series of studies [e.g., 17, 19, 107]. More recently, some studies used EEG standard positions such as O_1, O_2, PO_7, and PO_8 [42, 108]. Other positions used include Oz and Cz, usually in addition to the occipito-temporal electrodes.

Linked ears have frequently been used as a reference. Some studies compared how the Oz recording changes if the reference is changed from the ears to a frontal position, as recommended by the ISCEV standard for flash and pattern VEP measurements [109]. They found that the curve shape changes somewhat [e.g., 15], and that the N2 amplitude may be reduced [39]. Similar findings were reported by Kuba [16] for the occipito-temporal derivations. Multi-electrode topographic mapping studies [16, 28] may be used to estimate the effect of arbitrary reference locations.

As already mentioned, the N2 amplitude is often lateralized. Therefore, in some studies a 'virtual electrode' was introduced corresponding to the occipito-temporal electrode with the largest response as determined individually for each subject [e.g., 42, 107, 110].

Analysis

The motion-onset VEP consists of a superposition of several components. Assumptions regarding the exact nature of this superposition will influence the choice of analysis procedures. Those studies that employed only a small set of electrodes usually assessed the peak amplitude of the N2 negativity. It has been proposed that the preceding positivity is actually only the beginning of a longer-lasting component which is largely masked by the N2 deflection [17, 108], which is supported by topographic studies showing that with strong adaptation a stable occipital positivity persists until around 170–180 ms after motion onset [111]. This does not necessarily mean that measuring the difference between P1 and N2 peak values is the most appropriate measure for determining the size of the N2 peak. Rather, changes in the size of N2 might affect the size of P1 without indicating any change in the underlying positivity. One future possibility to disentangle superimposed components might be through modeling of the neural response and determining the parameters of the model. A first attempt to apply this approach to motion VEPs has been made by Kremláček et al. [112].

In order to quantify the veridical motion response contained in N2, some studies measured N2 amplitudes as the percentage of the unadapted baseline amplitude that remained after anti-directional adaptation [41, 45]. As the relative contributions of motion and flicker responses might vary between subjects, there is no guarantee that this approach will reduce inter-subject variability. Furthermore, the method is usually inadequate in those cases where the 'flicker response' varies with experimental conditions [17]. Problems also arise if the N2 peak does not reach below zero in strongly adapted states, as this would yield negative percentages. In many cases, taking absolute amplitude values obtained with anti-directional adaptation might therefore be the best solution. Many of the studies cited in the following sections did not attempt to isolate veridical motion responses, though this will not always be mentioned explicitly.

If the VEP was recorded with a dense array of electrodes covering the whole head, various further analysis techniques can be applied, such as principle or independent component analysis [111, 113, 114] and source localization [e.g., 23, 28, 78].

Steady-state VEPs can be analyzed in the frequency domain, where the response can be found at the frequency corresponding to the stimulation rate and/or its harmonics[1]. In addition to its elegance, this type of analysis also facilitates statistical assessment [115, 116]. However, steady-state potentials can be difficult to interpret since different components of the single-stimulus responses may be superimposed destructively. Furthermore the common practice of only evaluating a single harmonic might miss important features of the signal.

[1] Strictly speaking, the frequency of a motion-reversal stimulus is equal to half the reversal rate since one stimulus period encompasses both parts of the 'back and forth' movement.

Applications in basic research

Development and aging of motion-sensitive mechanisms

De Vries et al. [117] investigated the development of motion-sensitive systems using a complex definition of motion specificity and correcting for the individual development of contrast responses. Since their stimulation sequence most likely resulted in a considerable amount of adaptation, the results are difficult to interpret.

Norcia et al. [118] discovered that an asymmetry in monocularly recorded steady state VEPs recorded to apparent motion is present in infants of 6 months and younger. Wattam-Bell [57] recorded steady-state reversal VEPs in infants at two speeds. They found first significant direction-specific responses for speeds of 5°/s after approximately 10 weeks of age. At around 13 weeks of age, the response to 20°/s became significant with the responses to the slower speed still being larger. By the end of the study, the response amplitudes were equal. Braddick et al. [119] measured motion-reversal and orientation-change steady-state VEPs in infants in the age of 5–18 weeks and found that the first harmonic of the signal became significant at a later age for motion than for orientation stimuli, which may be interpreted as evidence for a separate development of motion and orientation selectivity. It should be noted, though, that looking at the first harmonic only might not provide a full account of all relevant age-related changes in the signal, and furthermore some of the orientation response might be caused by local luminance effects despite the precautions taken in the design of the experiment and motion adaptation might additionally affect the results. Several studies have used oscillatory displacement stimuli, where a pattern jumps between two position thereby producing two-frame apparent motion. Infants at 7 weeks of age need 10 times larger displacements than adults to evoke a VEP response. At 1 year of age, there is still a factor of five between infants and adults. Infants also exhibit a nasalward/temporalward asymmetry in their motion response [120]. This diminishes by 6–8 months and neonates also do not exhibit motion VEP asymmetry [121].

Hollants-Gilhuijs et al. [49] have shown that the hemispheric asymmetry also undergoes developmental changes. In children, the strongest VEP response for hemifield stimulation is found in the ipsilateral hemisphere. In adults, on the other hand, one hemisphere is dominant irrespective of the stimulated hemifield. With peripheral stimulation, however, Mitchell and Neville [122] found a stronger response in the contralateral hemisphere for adults but not for children. Comparing subjects in the age range of 21–72 years and stimulus contrasts of 0.04 and 93%, Korth et al. [123] found that latencies generally increase with age, while amplitude reductions only occurred for low-contrasts. A latency increase with age for adults above 18 years was also reported by Langrová et al. [124], who did not find a systematic amplitude effect using a stimulus contrast of 10%. For children and adolescents, Langrová et al. found a continuous reduction in latency by a total of about 100 ms in the tested age range of 5–18 years of age. This agrees with Mitchell and Neville [122], who furthermore note that children generally produce 'unorganized' motion VEPs. Processing of motion seems to mature at an even later age than processing of color [125].

Direction bandwidth

Using an adaptation paradigm with test directions varied in steps of 45° relative to the adaptation direction, Hoffmann et al. [41] estimated that the direction specificity of adaptation has a bandwidth in the order of 90° (FWHM). In a subsequent and more elaborate study, Maurer et al. [44] computed the direction bandwidth of neural motion detectors from a population model that used N2 amplitudes as input parameters. In that study, test directions were sampled in steps of 15° and the estimated detector bandwidth was 62° at occipito-temporal electrodes.

Spatial and temporal stimulus characteristics and speed

As mentioned earlier, N2 latency decreases and the amplitude increases with increasing speed. In steady-state reversal VEPs, however, Wattam-Bell [57] found a maximum amplitude at 20°/s.

The reason for the difference between N2 amplitudes and steady-state responses is unknown. Besides a real decline of the neural response for high speeds, there is also the possibility that changes in the shape of the response evoked by a single reversal might result in a smaller steady-state response even in cases where the single-response amplitude does not decrease. Also, the analysis might have interpreted the occurrence of a higher harmonic as a response decrease.

Several studies investigated the underlying cortical mechanisms in more detail. Particularly interesting is the question whether speed is coded additively (i.e. higher speed results in larger responses of the same neural population) or whether there are 'speed channels' (i.e. distinct neural populations process or code different, though possibly overlapping, speed ranges). Müller et al. [100] investigated test speeds up to approximately 6°/s and found evidence for additive coding within broad speed channels. In their experiment, as an overall trend, adaptation at a certain speed affected VEPs to all test speeds to a similar degree, and higher adaptation speeds resulted in more adaptation. Results for their highest adaptation speed (4°/s), however, did not fit into this scheme. In a later study, Müller et al. [126] report that adaptation speeds of 1 and 4°/s for adaptation produce the same amount of adaptation when a 2°/s test stimulus is used. The authors interpret this as evidence that there is more than one speed channel involved, based on the following reasoning: The motion response increases with speed. A higher speed should therefore result in more adaptation. If the range of speeds was covered by only one speed channel, then adapting with 4°/s should cause more reduction of the VEP than 1°/s. In a further study [127] the authors found some difference between the amounts of adaptation induced by slower and faster motion, which was explained by differences in adaptation time constants that might have had more effect in this latter study due to the timing of the stimuli. Müller et al. [43] provided further evidence for the existence of channels by combining psychophysical measurements with VEPs. Heinrich et al. [45] have shown that adaptation with very high stimulus speed (32°/s) does not result in a reduction of the direction-specific response to low speeds (3.5°/s) and vice versa, suggesting that the two speeds are processed by separate speed- and direction-selective channels.

A major question in motion processing is whether there are mechanisms that are speed-tuned irrespective of the spatial properties of the stimulus pattern. The literature is not consistent in this respect. According to Göpfert et al. [89], spatial frequency does not affect N2 amplitude. Markwardt et al. [90] report that stimulus speed is the determining factor for N2 amplitude and latency. Wang et al. [128], on the other hand, suggest that the MEG amplitude depends in a complex manner on temporal and spatial frequency, while it is only the latency that reflects stimulus speed. Heinrich et al. [17] have investigated the pattern-specificity of motion adaptation. Using sine wave gratings as well as one-dimensional and two-dimensional noise, they found that adaptation is largest if both adapting and test stimuli use the same pattern type. Even though some adaptation might be inherited from earlier areas, these results show that processing in MT is at least indirectly affected by the stimulus pattern rather than by stimulus speed alone.

Using a moving bar, Gallichio and Andreassi [129] report that both apparent motion and continuous motion (as approximated on a CRT screen) evoke similar responses. They found small differences, but these might originate from the inevitable physical differences between the stimuli rather than from fundamental difference between both stimulus types. This assumption is further supported by two MEG studies in which apparent motion activated area MT like continuous motion [130] and in which the amplitude was closely correlated to subjective estimates of motion smoothness [131].

Adaptation and its temporal dynamics

As mentioned earlier, adaptation is a crucial factor in the design of motion VEP experiments and can be used as a tool to investigate functionally defined neural populations. A qualitative investigation by Wist et al. [40] suggests that the time constants of adaptation of the motion-onset VEP are in the range of several seconds. Hoffmann et al. [107] tracked the time course of adaptation assuming an

exponential characteristic. They report time constants of 2.5 and 10.2 s for adaptation and recovery, respectively, of the occipito-temporal N2 component. This study did not distinguish between direction-specific and non-specific responses since the composition of the N2 was not known at that time. Nevertheless, the time constants are of the same order of magnitude as those measured psychophysically, which are also in the range of several seconds, depending on stimulus speed [107, 132, 133]. Uusitalo et al. [134] presented motion epochs of 45 ms in sequences with various interstimulus intervals and computed a recovery time constant of 0.8 s. This is substantially shorter than psychophysical results, but similar time constants have been found for short-term adaptation in macaques [61]. Taken together, the studies by Hoffmann et al. [107] and by Uusitalo et al. [134] suggest that adaptation occurs on various temporal scales, probably with different underlying mechanisms. Interestingly, adaptation does not seem to affect N2 latency [40].

Prompted by animal studies and theoretical models, Heinrich et al. [108] assessed whether the continuousness of the adapting motion affects adaptation. They did not find a sizable effect when they varied the onset rates of intermittent motion between 1.4 and 5.6 onsets/s while keeping the duty cycle constant. Amano et al. [135] report that MEG responses measured to speed changes are larger during an adapted state, paralleling psychophysical findings. This supports the idea that adaptation might be a means of optimizing the dynamic range of perception [c.f. 136].

Kobayashi et al. [64] aimed at measuring a neural correlate of the perceptual motion aftereffect. The authors presented either 10.8 s of continuous unidirectional motion or, for the same duration, oscillatory motion reversing every 2.4 s. They compared the responses after motion offset and found an increased positivity around 160 ms. However, the study does not separate correlates of the perceptual motion aftereffect from differences in the motion-offset response due to adaptation-related changes in the activation level of motion-processing mechanisms.

Kremláček et al. [137] found that a further effect can affect the N2, which they interpreted as a mismatch negativity (MMN) and which might potentially be confounded with adaptation. Such a component is known primarily from auditory evoked potentials and is considered to be unrelated to adaptation [138]. It occurs with stimuli that are presented infrequently within a series of repeated stimuli. In the study by Kremláček et al., the effect manifests itself as a more negative potential for rare stimuli in the time range of 145–260 ms. Their stimulus sequence largely excludes adaptation (or the lack thereof) as a confounder.

Color

If isoluminant rather than luminance-defined patterns are used, motion-onset VEPs differ in a number of properties [22, 139–141]. At speeds of less than 2 cycles/s, amplitudes are reduced and latencies are longer. In an MEG study, Anderson et al. [4] found that area MT does not respond to isoluminant motion stimuli. Morand et al. [142] investigated motion processing in the koniocellular pathway by using 'tritan' (S cone isolating) stimuli, but without assessing the motion-specificity of the VEP responses. Tritan and luminance-defined motion produced similar responses, leading to the conclusion that koniocellular and magnocellular pathways share the same neural substrate for motion processing. In addition, early ($\approx$40 ms) activations were found for the tritan stimuli. It is not fully clear whether these are color-related rather than motion-related.

Second-order motion

Victor and Conte [143] found no difference in motion VEPs when comparing first- and second-order motion. Ellemberg et al. [144] raised the concern that the stimuli used by Victor and Conte did not represent pure second-order motion, due to the relatively large size of the checks from which the stimulus was constructed. Therefore, they performed a similar experiment with improved second-order stimuli and found that latencies are longer for second-order than first-order stimuli. Furthermore, higher contrasts are needed for second-order motion to evoke a sizable response. Chakor et al. [54] reinvestigated the issue with reversal VEPs and found that the latency is only increased at low contrasts. With a

second-order stimulus based on the local speed of a dot pattern, Sofue et al. [145] did not obtain any MEG response.

Eye movements

Both stimulus motion and eye motion can result in identical retinal motion. Nevertheless, the visual world seems stable during eye movements. Thier et al. [146] investigated this using the Filehne illusion. They found the first effect in the VEP around 300 ms and concluded that the distinction between both types of motion is achieved upstream from area MT from which the earlier N2 arises. Hoffmann and Bach [147] compared conditions where retinally identical motion is induced either through eye movements, through physical stimulus motion during fixation, or through stimulus motion during eye movements. They isolated a correlate of head-centric motion detection that peaked around the same time as the N2 and was localized occipitally. A second component occurred around 300 ms occipitotemporally. Tikhonov et al. [148] compared two conditions where the same stimuli and eye movements yielded different percepts due to changes of a reference stimulus and obtained an effect with the same latency as the N2 in Hoffmann and Bach's study, but with a different scalp distribution and a likely origin in the medial parietooccipital sulcus.

Armington and Bloom [149] measured the retinal and cortical potentials evoked by saccadic image shifts. For both, they found a high correlation between the response amplitude and the magnitude of the saccade. Since an occipital bipolar pair of electrodes was used, the responses cannot be compared directly to other studies. However, the timing suggests that they do not simply reflect the neural processes that initiate the saccades or electrooculographic crosstalk. Kleiser and Skrandies [150] used VEPs to investigate motion processing during saccadic eye movements. They found that global field power and latencies were unchanged by saccades, but the topographic distribution changed significantly. They concluded that extraretinal information is used to extract velocity information and that identical stimuli activate different neural populations during saccades than during fixation.

Attention, task effects and interactions with other sensory modalities

Torriente et al. [151] report a reduction of the N2 amplitude when they instructed the subjects to attend to static rather than moving line elements in a transparent motion display. PazoAlvares et al. [152] found an increase in N2 amplitude for a rare ('deviant') motion direction, compared to a frequent motion direction, even if subjects had to attend to unrelated stimuli presented at the location of the fixation mark. This effect declines with age [153].

Anllo-Vento and Hillyard [154] used apparent motion and color to assess location and feature based attention in the dorsal and ventral streams. They found effects of location based attention to include the time interval of the motion N2, while feature based attention effects were found at later times intervals. Both types of attention yielded more negative signals in the occipito-temporal region, that were interpreted as selection negativities. Martín-Loeches et al. [155] also used apparent object motion as a dorsal-stream stimulus. Subjects had to attend to one of the two possible motion directions. The study found a smaller N2 amplitude for attended motion directions that was interpreted as a selection positivity. It is not fully clear from these two studies whether the selection positivities and negativities reflect the attentional process itself or represent a modulation of the activity that is associated with stimulus processing. However, Anllo-Vento and Hillyard [154] report that attention to color and to motion direction exhibited different scalp distributions. This suggests that there are at least separate mechanisms for color and motion. Valdes-Sosa et al. [156] used transparent motion to investigate the effect of attention. When subjects attended to one of the moving planes, N2 amplitudes recorded to changes in direction of the other plane were drastically reduced.

Niedeggen et al. [157] investigated whether transient motion blindness in a dual task paradigm is occurring at the stage of sensory processing. The subjects had to detect brief periods of coherent

motion among periods of incoherent motion. The coherent motion was cued by a color stimulus, but detection of the coherent epoch was impaired when it occurred within 300 ms after the cue. The study showed that both the motion N2 and the cognitive P300 component were reduced, but only the P300 showed a correlation with the psychophysical performance in individual trials, suggesting that sensory processing does not account for the impairment in coherence detection. This was confirmed by a second study, where Niedeggen et al. [158] recorded VEPs to motion distractors to track sensory processing. In an experiment involving direction changes of two transparently moving surfaces, Rodríguez and Valdés-Sosa [159] investigated how the occurrence of one stimulus affects the N2 recorded to a second stimulus that was presented shortly after the first one. They found reduced amplitudes when subjects incorrectly identified the direction of the second stimulus, and a marked reduction if the two stimuli occurred on different rather than the same motion surfaces, consistent with psychophysical performance.

Using a shape-deforming motion stimulus, Fort et al. [160] demonstrated that N2 amplitude and latency are affected by the type of the subject's task. In an identification task, the amplitude was larger, while onset, peak and offset latencies of the N2 were longer than in a detection task.

Effects of cross-modal attention were analyzed by Beer and Röder [161]. They provided the subjects with auditory and visual motion stimuli and required them to perform either an auditory or a visual task. All effects in the motion VEP occurred later than the N2 component.

Armstrong et al. [162] found that the processing of visual motion, but not color, is affected in congenitally deaf subjects. In particular, the N2 was larger and more anteriorly distributed than in hearing subjects. Chlubnová et al. [104], on the other hand, report smaller amplitudes for deaf subjects. The authors explained this lack of agreement with differences in the stimulus selectivity for the magnocellular system.

Probst and Wist [163] report that all components of the motion VEP are affected by vestibular stimulation through passive head movements, while there was only little effect on pattern reversal stimuli.

Organization of visual motion processing

Retinal motion processing

As reviewed by Bach and Hoffmann [164], in many lower vertebrates motion is already processed on a retinal level. Little or no retinal motion processing, as defined by its direction-selectivity, has been reported for higher animals, including primates. In humans, this has been investigated by measuring the electroretinogram (ERG) evoked by motion onset. No evidence for motion-specific processing was found. There is no directional adaptation [164] and the ERG amplitude changes linearly with contrast [165]. The shape of the ERG trace is substantially affected by the stimulus speed [164, 165]. Interestingly, the motion-onset VEP is still recordable at luminance levels below the minimum luminance required for motion ERGs [91].

Cortical areas involved in motion processing

Recent multi-channel studies [27, 28] suggest that the N2 component consists of several subcomponents that are separated by a delay of 20 ms and appear to originate from V3A and MT, respectively. This agrees well with imaging studies [e.g. 31] that report V3A to be involved in motion processing. Based on MEG and electrocorticogram recordings together with data from cortical stimulation, Matsumoto et al. [166] propose that area MT is composed of several subregions that account for the N2 and later responses, and for perceptual phenomena such as motion in depth. Uusitalo et al. [167] identified a complex network of sources accounting for various transient and sustained MEG activations during the processing of motion and pattern onset. The authors suggest that motion-related cortical areas generally respond to any transient stimulus changes. It is interesting to note that the N2 latency agrees much better with the timing of single-unit recordings from the adjacent area MST, rather than with recordings from MT [168].

Eccentricity and cortical magnification

Kubová et al. [14] compared stimulation of the macula with stimulation of the surrounding retina and found a much smaller difference for the

motion VEP N2 (measured as average of P1–N2 and N2–P3 differences) amplitude than for the pattern-reversal VEP response (measured as average of N1–P1 and P1–N2 differences). Schlykowa et al. [38] measured how the response changes when the same motion stimulus is presented at various eccentricities. They found that the amplitude is less reduced for large eccentricities than predicted from V1 cortical magnification, but rather resembles roughly the point image size scaling in MT. There is a possible confounder, though, since the spatial frequency of the stimulus was not adjusted to match cortical magnification. When Müller et al. [169] took cortical magnification of V1 into account for stimulus size, spatial frequency, and speed, they found that amplitudes are constant across eccentricities. Kremláček et al. [170] stimulated at higher eccentricities then most other studies, up to 42° horizontally and 30° vertically. They report that latencies were shorter and amplitudes were larger in the lower visual field. The amplitude effect depended on the electrode position. Consistent with MacKay and Rietveld [13] and Yokoyama et al. [171], but contrary to Takao and Miyata [172], in most recordings there was no amplitude reversal between upper and lower fields as would be expected for V1. N2 latencies decreased with eccentricity. Dagnelie et al. [72] obtained no motion response for foveal stimulation in monkeys. Kremláček et al. [170] found a paradoxical lateralization of amplitude and latency for large eccentricities.

V1–MT hierarchical processing

A few studies have used coherence onset in order to isolate higher-level motion processing. The idea is that local motion is processed in V1, while MT is more specialized in processing global patterns of motion. Coherence onset experiments typically use random dot fields. During incoherent motion phases, each dot has an individual, random, direction. This would only stimulate V1 neurons. During coherent motion phases, all (or a large fraction) of the dots move in the same direction. This would stimulate both V1 and MT neurons. An abrupt transition from incoherent to coherent motion would therefore be an MT-specific motion onset. Some evidence for this is provided by Niedeggen and Wist [173] who investigated the topography of the coherence-onset response. However, a number of studies suggest that MT is activated by both coherent and incoherent motion. Recording multi-unit activity and current source density with a chronically implanted probe, Ulbert et al. [174] identified a complex pattern of activity across MT laminae in humans, reflecting both coherent and incoherent motion. Lam et al. [175] report that MEG responses for the two motion types are similar, though not identical, in most respects including the dependence on stimulus speed. Maruyama et al. [101] found a stronger speed dependence of the MEG amplitude for coherent than incoherent motion. Lam et al. [176] presented incoherent motion on a background of coherent motion. They revealed a complex dependence of the MEG responses on the speeds of the two motions and concluded that there are interactions in the processing of the two motion types.

In a coherence onset experiment, Niedeggen and Wist [177] discovered that orthogonal transparent motion evokes a smaller response than the motion of just one single dot pattern. This is surprising, since one would assume that the two moving patterns in the case of transparent motion should stimulate twice as many MT neurons than the single pattern. Using MEG, Aspell et al. [178] found that a response at around 230 ms after coherence onset originates from around MT or V3. The latency decreased and the amplitude increased with increasing coherence. The study identified a second source in the superior temporal sulcus (STS). In most cases, the sources were lateralized, but MT/V3 and STS activated different hemispheres. Amano et al. [179] investigated whether coherence-onset responses in the MEG predict reaction times. They found that the time at which the temporally integrated motion response reaches a threshold correlates well with the reaction time (regression slope ≈ 1), better than peak time or the time when the non-integrated signal reaches a threshold.

Direct pathways to area MT

ffytche et al. [180] investigated the spatio–temporal characteristics of very early (35–105 ms)

cortical responses to motion. Their findings suggest that slow motion activates first V1 and subsequently MT, while fast motion activates MT first. This indicates the presence of a direct pathway to MT, bypassing V1. The time course agrees well with Buchner et al. [78], who found that pattern-reversal stimuli activate MT starting from 30 ms, clearly before the onset of activity in the striate cortex at around 50 ms. On the other hand, Azzopardi et al. [168] did not find a selective reduction in response magnitude in MT neurons for slow speeds when V1 was lesioned. The conclusion by ffytche et al. is supported by Schoenfeld et al. [181], who used a bright flash to temporarily deactivate V1. A subsequently presented motion stimulus evoked an N2 component that appeared to originate from MT. A simple luminance stimulus did not evoke a sizable response. Further evidence comes from Benson et al. [182], who recorded motion-onset potentials from a blindsight patient with V1 lesions who was unaware of the motion, but could guess the direction of motion accurately.

Clinical research and diagnosis

Most applications in clinical research and diagnosis have not distinguished between directional and non-directional responses. For diagnostic purposes this is not necessarily a problem. What counts here is sensitivity and specificity as well as practicability in clinical routine use. Whether the stimulation of motion-processing mechanisms is selective or not is irrelevant if the other criteria are met. Of course, there might be cases where the use of direction selective motion-VEPs is beneficial even for purely diagnostic purposes.

For all proposed diagnostic applications discussed below, longitudinal studies are missing, and most studies only included a relatively small number of patients. In many cases, the motivation for using motion VEPs is an assumed involvement of magnocellular or dorsal-stream neural mechanisms.

Glaucoma

Evidence from animal models regarding the magnocellular system being particularly affected by glaucoma is contradictory [183] and psychophysical evidence for a specific effect of glaucoma on motion processing is weak (see review in [184]). Nevertheless, the idea that glaucoma might be detected using motion VEPs attracted interest. Results by Kubová et al. [110] suggest that the amplitude of the N2 is hardly affected by chronic glaucoma, while the latency appears to be a more sensitive indicator of glaucoma than the pattern-reversal P100 latency. In a study by Korth et al. [123] both amplitude and latency were affected in patients with open-angle glaucoma. In a multivariate approach, they found a sensitivity of 77% at a specificity of 90% when combining several stimulus conditions and both latency and amplitude information. It is yet unclear whether motion VEPs could compete with the pattern electroretinogram, for which prospective studies suggest a high predictive value [185, 186].

Optic neuritis and multiple sclerosis

Kubová and Kuba [187] report that the motion VEP exhibits less latency increase in retrobulbar neuritis than the pattern-reversal VEP. However, motion VEP latency appeared to be more sensitive in the detection of multiple sclerosis. A subsequent study [188] suggests that both VEP types, pattern reversal and motion onset, can be affected independently in multiple sclerosis while retrobulbar neuritis is always associated with an increased latency in the pattern-reversal VEP. The diagnostic specificity of the motion VEP in these studies is unclear, though, and other authors report less promising results. In a study by Herbst et al. [189], the mean latency of the N2 (measured at the Pz electrode) was significantly increased in multiple sclerosis compared to a control group, but only few individual patients had latencies beyond the normal range as defined by the control group. This is consistent with results by Tobimatsu and Kato [190], who had the lowest detection rate for optic neuritis and multiple sclerosis for VEPs evoked by apparent motion stimuli, compared to various other achromatic and chromatic stimuli. In their sample of patients, all abnormalities detected with the

motion stimuli could also be detected with some of the other stimulus types.

Amblyopia

Kubová and Kuba [187] and Kubová et al. [191] found that motion-onset N2s obtained with the amblyopic eye differ neither in amplitude nor in latency from those obtained with the normal fellow eye. Motion-onset VEPs did not change with visual acuity. The pattern-reversal P100, on the other hand, showed a decrease in amplitude and an increase in latency, both correlating with visual acuity. In these studies, both motion and pattern-reversal stimuli had a contrast of 90%. There are at least four possible interpretations for this result:

1. The motion system (and possibly the magnocellular system and dorsal stream in general) might be relatively spared by amblyopia, as suggested by Kubová et al. [191]. It is unclear how this could be reconciled with psychophysical studies that report the motion system to be affected [e.g., 192, 193]
2. For the motion system, amblyopia might be equivalent to a reduced stimulus contrast. Since the response characteristic of the motion system saturates for relatively low contrasts, the amblyopia-induced contrast reduction would not have an effect.
3. Area MT and the motion VEP seem relatively insensitive to the details of the stimulus pattern. It might therefore make little difference if earlier processing stages provide only a degraded stimulus representation to the higher stages of the motion processing system.
4. In recent psychophysical study, Ho et al. [194] found that motion processing is not only impaired for the amblyopic eye, but also for the fellow eye. Thus, studies comparing both eyes might fail to reveal an effect even if it was actually present. Furthermore, Ellemberg et al. [192] showed that monocular deprivation results in a smaller deficit in motion perception than binocular deprivation. Thus, collaborative interactions between the inputs from the two eyes during development, rather than sparing of the motion system as such, might partly account for lack of an effect in the motion VEPs in monocular amblyopia.

Strabismus and nystagmus

There might be interconnections between the development of binocular vision and motion processing that manifest themselves as a directional bias in motion VEPs [195]. Using an oscillating grating, Norcia et al. [118] found that adults whose strabismus onset was before 6 months of age show the same VEP asymmetries between temporalwards and nasalwards motion as infants of less than 6 months of age. This was confirmed by Shea et al. [196], who also showed that there is no asymmetry in pattern-reversal stimuli. Anteby et al. [197] found that VEP asymmetries are not caused by nystagmus, thus ruling out a possible confounder. Wilson et al. [198] came to the same conclusion after recording VEPs in monkeys with paralyzed eyes. Norcia et al. [199] report that VEP asymmetries are lower in patients with esotropia who received aligning surgery before 2 years of age than in those who received surgery later in life. This agrees well with Tychsen et al. [200], who used prisms to simulate strabismus in monkeys. Kommerell et al. [201] investigated the correlation between optokinetic nystagmus (OKN) and VEP in patients with infantile strabismus, but found that only 8 out of 20 patients with OKN asymmetries exhibited a VEP asymmetry, and for these eight no correlation was found between the strengths of the two asymmetries. This makes it unlikely that the motion VEP reflects the defect that causes the OKN asymmetry. Brosnahan et al. [202], on the other hand, found such a correlation, and explained the discrepancies to the aforementioned study by the different scales used for rating the OKN and by the more diverse population in their own study. Mason et al. [203] showed that nasal-to-temporal displacements evoked larger responses, as opposed to OKN which is larger for the opposite direction, supporting the view that there is no simple connection between asymmetries in OKN and motion VEPs. Data by Birch et al. [121] indicates that normal and strabismic children do not differ

immediately after the onset of strabismus, suggesting that asymmetries in patients with strabismus ''may not represent a persistence of the normal infantile state but, rather, a pathologic disruption of motion pathways as a result of prolonged abnormal binocular sensory experience''. Fawcett and Birch [204] found a high correlation between motion VEP asymmetry and the absence of bifoveal fusion and proposed the motion VEP as an objective diagnostic tool.

Dyslexia

The question whether dyslexia possibly reflects a more general deficit in magnocellular processing is under intense debate [e.g., 205–207]. Motion VEPs have been used to verify this hypothesis. Kubová et al. [208] found that N2 latencies are clearly reduced in many dyslexic children. This effect seems to become less when the children get older [209]. Schulte-Körne et al. [210] compared 'static' and motion VEPs and found that differences between dyslexic children and controls occurred as early as 100 ms. In another study [211] they compared coherent and non-coherent motion-onsets. VEPs to non-coherent motion onsets did not differ between the two groups, while the coherent motion onsets resulted in differences around 300–800 ms. In the latter two studies, VEP traces are dominated by positivities at 100 ms and 200 ms in both dyslexics and controls. Since it is not clear to what degree motion is exclusively processed by the magnocellular system, in particular if stimuli have a high contrast as in the studies by Schulte-Körne, the conclusion that the VEP findings confirm a link between magnocellular deficits and dyslexia has received criticism [212]. Furthermore, as some parameters of the stimulus sequence are not provided and therefore the influence of adaptation is unclear, the VEPs are difficult to compare with usual motion-onset VEPs. In addition, effects around 300–800 ms seem rather late for the involvement of basic visual processing stages.

Other diseases

In hepatic and portosystemic encephalopathy, the latency of motion-onset VEPs is delayed and has been suggested as a diagnostic tool in combination with an assessment of the dominant cortical frequency [213]. Motion N2 latency is increased in spinocerebellar degeneration, but not in Parkinson's disease, while the pattern-reversal N2 is unaffected in both cases [214]. In congenital stationary night-blindness, motion VEPs can only be recorded with a luminance of at least 0.06–0.1 cd/m^2, compared to less than 0.003 cd/m^2 in normal subjects [92]. A relatively low stimulus contrast of 10% was used. For high-contrast pattern-reversal, the authors found similar luminance limits, though the curve shape changed with decreasing luminance. In a subject with Williams syndrome, Nakamura et al. [215] found that motion-evoked MEG responses are within the normal range. For neuroborreliosis, Kubová et al. [216] report that in some patients only motion VEPs, but not pattern-reversal VEPs, exhibit an increase in latency. However, the authors frequently found a discrepancy between the patients' subjective vision problems and the VEP findings. In many patients motion VEP latencies did not exceed the normal range (mean + 2.5 standard deviations).

Comparison to fMRI

fMRI provides a much higher spatial resolution while VEPs are better for tracking neural activity on a fine temporal scale. This has implications on the design of the stimuli and consequently on the type of experiments that can be performed with either method. Adaptation experiments where the effects of different adaptors on the neural response to a probe stimulus are tested are one example. If adaptation and probe stimuli are interleaved, fMRI is not able to separate the differences in the responses to various adaptors from differences in the response to the probe stimulus unless these are several seconds apart or rather complicated paradigms are used. Furthermore, the underlying physiology of fMRI measurements are not yet understood in all respects. In a clinical context, using VEPs might be more economical than fMRI and there are less contraindications.

Due to differences in signal generation, with fMRI reflecting changes in blood oxygenation

and VEPs assessing the electrical activity of large numbers of neurons, the two methods might tap different aspects of motion processing. This has been proposed by Henning et al. [53], but differences in adaptation between condition make their data difficult to interpret.

Summary and conclusion

The above overview shows how motion VEPs have been applied to a broad range of questions in basic research and clinical diagnosis. In spite of a considerable body of research, there are several unresolved issues and open questions, including the following:

- In spite of some promising preliminary results, motion VEPs have not yet found a widespread use in clinical diagnostics. First of all, studies with a larger number of patients are needed to verify the diagnostic value relative to established methods, assessing both sensitivity and specificity. Second, it would be helpful to devise stimulation schemes that shorten the duration of an exam without suffering from motion adaptation.
- Most studies used the N2 as a correlate of motion processing. As discussed in the introduction, the majority of evidence currently points towards MT and possibly V3A as the origin. On the other hand there is little doubt from single-cell studies that V1 is involved in motion processing. Is this reflected by the VEP?
- The N2 occurs rather late compared to the onset of activity in area MT as reported by animal studies [e.g., 217]. So far, only very few studies, e.g. by ffytche et al. [180], have investigated earlier components originating from MT.
- In some cases, different studies seem to report contradictory results. It is beyond the scope of this review to discuss possible reasons in each single case, but it seems plausible that simple differences in stimulation, such as contrast, speed, and timing, account for some of these discrepancies. Critical aspects might be how motion-specific the evoked response is or how selectively the magnocellular system and the dorsal stream were activated. This needs more investigation.

In spite of these open questions, motion VEPs have considerably contributed to our understanding of human motion processing and helped bridging the gap between our knowledge about the healthy visual system and the need to better understand mechanisms of disease.

References

1. Snowden RJ, Freeman TCA (2004) The visual perception of motion. Curr Biol 14:R828–R831
2. Livingstone MS, Hubel DH (1987) Psychophysical evidence for separate channels for the perception of form, color, movement, and depth. J Neurosci 7:3416–3468
3. Merigan WH, Maunsell JH (1993) How parallel are the primate visual pathways? Annu Rev Neurosci 16:369–402
4. Anderson SJ, Holliday IE, Singh KD, Harding GFA (1996) Localization and functional analysis of human cortical area V5 using magneto-encephalography. Proc R Soc Lond B Biol Sci 263:423–431
5. Clifford CWG, Ibbotson MR (2003) Fundamental mechanisms of visual motion detection: models, cells and functions. Prog Neurobiol 68:409–437
6. Purkinje J (1820) Beyträge zur näheren Kenntniss des Schwindels aus heautognostischen Daten. Medicinische Jahrbücher des kaiserlich-königlichen österreichischen Staates 6:79–125
7. Addams R (1834) An account of a peculiar optical phænomenon seen after having looked at a moving body. Lond Edinb Phil Mag J Sci 5:373–374
8. Gastaut H, Régis H (1951) Un signeélectroéncephalographique peu connu: les pointes occipitales survenant pendant l'ouverture des yeux. Rev Neurol (Paris) 84:640–643
9. Evans CC (1953) Spontaneous excitation of the visual cortex and association areas—lambda waves. Electroenceph Clin Neurophysiol 5:69–74
10. Barlow JS, Cigánek L (1969) Lambda responses in relation to visual evoked responses in man. Electroenceph Clin Neurophysiol 26:183–192
11. Billings RJ (1989) The origin of the occipital lambda wave in man. Electroenceph Clin Neurophysiol 72:95–113
12. Marshall C, Harden C (1952) Use of rythmically varying patterns for photic stimulation. Electroenceph Clin Neurophysiol 4:283–287
13. MacKay DM, Rietveld WJ (1968) Electroencephalogram potentials evoked by accelerated visual motion. Nature 217:677–678
14. Kubová Z, Kuba M, Hubacek J, Vit F (1990) Properties of visual evoked potentials to onset of movement on a television screen. Doc Ophthalmol 75:67–72
15. Kuba M, Kubová Z (1992) Visual evoked potentials specific for motion onset. Doc Ophthalmol 80:83–89

16. Kuba M (2006) Motion-onset visual evoked potentials and their diagnostic applications. Nucleus HK, Hradec Králové
17. Heinrich SP, Renkl AEH, Bach M (2005) Pattern specificity of human visual motion processing. Vision Res 45:2137–2143
18. Müller R, Göpfert E (1988) The influence of grating contrast on the human cortical potential visually evoked by motion. Acta Neurobiol Exp (Warsz) 48:239–249
19. Kubová Z, Kuba M, Spekreijse H, Blakemore C (1995) Contrast dependence of motion-onset and pattern-reversal evoked potentials. Vision Res 35:197–205
20. Bach M, Ullrich D (1997) Contrast dependency of motion-onset and pattern-reversal VEPs: interaction of stimulus type, recording site and response component. Vision Res 37:1845–1849
21. Göpfert E, Müller R, Breuer D, Greenlee MW (1999) Similarities and dissimilarities between pattern VEPs and motion VEPs. Doc Ophthalmol 97:67–79
22. McKeefry DJ (2001) Visual evoked potentials elicited by chromatic motion onset. Vision Res 41:2005–2025
23. Probst T, Plendl H, Paulus W, Wist ER, Scherg M (1993) Identification of the visual motion area (area V5) in the human brain by dipole source analysis. Exp Brain Res 93:345–351
24. Nakamura H, Kashii S, Nagamine T, Matsui Y, Hashimoto T, Honda Y, Shibasaki H (2003) Human V5 demonstrated by magnetoencephalography using random dot kinematograms of different coherence levels. Neurosci Res 46:423–433
25. Amano K, Kuriki I, Takeda T (2005) Direction-specific adaptation of magnetic responses to motion onset. Vision Res 45:2533–2548
26. Kaneoke Y, Watanabe S, Kakigi R (2005) Human visual processing as revealed by magnetoencephalography. Int Rev Neurobiol 68:197–222
27. Bundo M, Kaneoke Y, Inao S, Yoshida J, Nakamura A, Kakigi R (2000) Human visual motion areas determined individually by magnetoencephalography and 3D magnetic resonance imaging. Hum Brain Mapp 11:34–45
28. Schellart NAM, Trindade MJG, Reits D, Verbunt JPA, Spekreijse H (2004) Temporal and spatial congruence of components of motion-onset evoked responses investigated by whole-head magneto-electroencephalography. Vision Res 44:119–134
29. Chawla D, Phillips J, Buechel C, Edwards R, Friston KJ (1998) Speed-dependent motion-sensitive responses in V5: an fMRI study. Neuroimage 7:86–96
30. Smith AT, Greenlee MW, Singh KD, Kraemer FM, Hennig J (1998) The processing of first- and second-order motion in human visual cortex assessed by functional magnetic resonance imaging (fMRI). J Neurosci 18:3816–3830
31. Orban GA, Fize D, Peuskens H, Denys K, Nelissen K, Sunaert S, Todd J, Vanduffel W (2003) Similarities and differences in motion processing between the human and macaque brain: evidence from fMRI. Neuropsychologia 41:1757–1768
32. Dupont P, Orban GA, De Bruyn B, Verbruggen A, Mortelmans L (1994) Many areas in the human brain respond to visual motion. J Neurophysiol 72:1420–1424
33. Shulman GL, Schwarz J, Miezin FM, Petersen SE (1998) Effect of motion contrast on human cortical responses to moving stimuli. J Neurophysiol 79:2794–2803
34. Born TR, Bradley DC (2005) Structure and function of visual area MT. Annu Rev Neurosci 28:157–189
35. Göpfert E, Müller R, Hartwig M (1984) Effects of movement adapation on movement visual evoked potentials. Doc Ophthal Proc Ser 40:321–324
36. Göpfert E, Müller R, Markwardt F, Schlykowa L (1983) Visuell evozierte Potentiale bei Musterbewegung. EEG EMG Z Elektroenzephalogr Elektromyogr Verwandte Geb 14:47–51
37. Müller R, Göpfert E, Hartwig M (1986) The effect of movement adaptation on human cortical potentials evoked by pattern movement. Acta Neurobiol Exp (Warsz) 46:293–301
38. Schlykowa L, van Dijk BW, Ehrenstein WH (1993) Motion-onset visual-evoked potentials as a function of retinal eccentricity in man. Cogn Brain Res 1:169–174
39. Bach M, Ullrich D (1994) Motion adaptation governs the shape of motion-evoked cortical potentials. Vision Res 34:1541–1547
40. Wist ER, Gross JD, Niedeggen M (1994) Motion aftereffects with random-dot chequerboard kinematograms: relation between psychophysical and VEP measures. Perception 23:1155–1162
41. Hoffmann MB, Unsöld A, Bach M (2001) Directional tuning of motion adaptation in the motion-onset VEP. Vision Res 41:2187–2194
42. Heinrich SP, Bach M (2003) Adaptation characteristics of steady-state motion visual evoked potentials. Clin Neurophysiol 114:1359–1366
43. Müller R, Göpfert E, Leinweber M, Greenlee MW (2004) Effect of adaptation direction on the motion VEP and perceived speed of drifting gratings. Vision Res 44:2381–2392
44. Maurer JP, Heinrich TS, Bach M (2004) Direction tuning of human motion detection determined from a population model. Eur J Neurosci 19:3359–3364
45. Heinrich SP, van der Smagt MJ, Bach M, Hoffmann MB (2004) Electrophysiological evidence for independent speed channels in human motion processing. J Vision 4:469–475
46. Maurer JP, Bach M (2003) Isolating motion responses in visual evoked potentials by pre-adapting flicker-sensitive mechanisms. Exp Brain Res 151:536–541
47. Andreassi JL, Juszczak NM (1982) Hemispheric sex differences in response to apparently moving stimuli as indicated by visual evoked potentials. Int J Neurosci 17:83–91
48. Göpfert E, Schlykowa L, Müller R (1988) Zur Topographie des Bewegungs-VEP am Menschen. EEG EMG Z Elektroenzephalogr Elektromyogr Verwandte Geb 19:14–20
49. Hollants-Gilhuijs MAM, de Munck JC, Kubová Z, van Royen E, Spekreijse H (2000) The development

of hemispheric asymmetry in human motion VEPs. Vision Res 40:1–11
50. Ahlfors SP, Simpson GV, Dale AM, Belliveau JW, Liu AK, Korvenoja A, Virtanen J, Huotilainen M, Tootell RBH, Aronen HJ, Ilmoniemi RJ (1999) Spatiotemporal activity of a cortical network for processing visual motion revealed by MEG and fMRI. J Neurophysiol 82:2545–2555
51. Odom JV, de Smedt E, van Malderen L, Spileers W (1999) Visually evoked potentials evoked by moving unidimensional noise stimuli: effects of contrast, spatial frequency, active electrode location, reference electrode location, and stimulus type. Doc Ophthalmol 95:315–333
52. Kuba M, Toyonaga N, Kubová Z (1992) Motion-reversal visual evoked responses. Physiol Res 41:369–373
53. Henning S, Merboldt K, Frahm J (2005) Simultaneous recordings of visual evoked potentials and bold mri activations in response to visual motion processing. NMR Biomed 18:543–552
54. Chakor H, Bertone A, McKerral M, Faubert J, Lachapelle P (2005) Visual evoked potentials and reaction time measurements to motion-reversal luminance- and texture-defined stimuli. Doc Ophthalmol 110:163–172
55. Clarke PGH (1972) Visual evoked potentials to sudden reversal of the motion of a pattern. Brain Res 36:453–458
56. Clarke PGH (1974) Are visual evoked potentials to motion-reversal produced by direction-sensitive brain mechanisms? Vision Res 14:1281–1284
57. Wattam-Bell J (1991) The development of motion-specific cortical responses in infants. Vision Res 32:287–297
58. Snowden RJ, Ullrich D, Bach M (1995) Isolation and characteristics of a steady-state visually-evoked potential in humans related to the motion of a stimulus. Vision Res 35:1365–1373
59. Tyler CW, Kaitz M (1977) Movement adaptation in the visual evoked response. Exp Brain Res 27:203–209
60. Lounasmaa OV, Williamson SJ, Kaufman L, Tanenbaum R (1985) Visual evoked responses from non-accipital areas of human cortex. In: Weinberg H, Stroink G, Katila T (eds) Biomagnetism: applications & theory, 5th world conference on biomagnetism. Pergamon Press, New York, pp 348–353
61. Priebe NJ, Churchland MM, Lisberger SG (2002) Constraints on the source of short-term motion adaptation in macaque area MT. I. The role of input and intrinsic mechanims. J Neurophysiol 88:354–369
62. Bair W, Cavanaugh JR, Smith MA, Movshon JA (2002) The timing of response onset and offset in macaque visual neurons. J Neurosci 22:3189–3205
63. Bair W (2004) No doubt about offset latency. Vis Neurosci 21:671–674
64. Kobayashi Y, Yoshino A, Ogasawaram T, ans Nomura S (2002) Topography of evoked potentials associated with illusory motion perception as a motion aftereffect. Cogn Brain Res 13:75–84
65. Pinilla T, Cobo A, Torres K, Valdes-Sosa M (2001) Attentional shifts between surfaces: effects on detection and early brain potentials. Vision Res 41:1619–1630
66. Kreegipuu K, Allik J (in press) Detection of motion onset and offset: reaction time and visual evoked potential analysis. Psychol Res. DOI: 10.1007/s00426-006-0059-1
67. Spileers W, Mangelschots E, Maes H, Orban GA (1996) Visual evoked potentials elicited by a moving unidimensional noise pattern. Electroenceph Clin Neurophysiol 100:287–298
68. Di Russo F, Pitzalis S, Aprile T, Spitoni G, Patria F, Stella A, Spinelli D, Hillyard SA (in press) Spatiotemporal analysis of the cortical sources of the steady-state visual evoked potential. Hum Brain Mapp. DOI: 10.1002/hbm.20276
69. Barnikol UB, Amunts K, Dammers J, Mohlberg H, Fieseler T, Malikovic A, Zilles K, Niedeggen M, Tass PA (2006) Pattern reversal visual evoked responses of V1/V2 and V5/MT as revealed by MEG combined with probabilistic cytoarchitectonic maps. Neuroimage 31:86–108
70. Kulikowski JJ (1978) Pattern and movement detection in man and rabbit: separation and comparison of occipital potentials. Vision Res 18:183–189
71. Spekreijse H, Dagnelie G, Maier J, Regan D (1985) Flicker and movement constituents of the pattern reversal response. Vision Res 25:1297–1304
72. Dagnelie G, de Vries MJ, Maier J, Spekreijse H (1986) Patternreversal stimuli: motion or contrast? Doc Ophthalmol 61:343–349
73. Heinrich SP, Bach M (2001) Adaptation dynamics in pattern-reversal visual evoked potentials. Doc Ophthalmol 102:141–156
74. Di Russo F, Pitzalis S, Spitoni G, Aprile T, Patria F, Spinelli D, Hillyard SA (2005) Identification of the neural sources of the pattern-reversal VEP. Neuroimage 24:874–886
75. Hoffmann MB, Seufert PS, Bach M (2004) Simulated nystagmus suppresses pattern-reversal but not pattern-onset visual evoked potentials. Clin Neurophysiol 115:2659–2665
76. Clarke PGH (1973) Comparison of visual evoked potentials to stationary and moving patterns. Exp Brain Res 18:156–164
77. Mackie RT, McCulloch DL, Bradnam MS, Glegg M, Evans AL (1996) The effect of motion on pattern-onset visual evoked potentials in adults and children. Doc Ophthalmol 91:371–380
78. Buchner H, Gobbele R, Wagner M, Fuchs M, Waberski TD, Beckmann R (1997) Fast visual evoked potential input into human area V5. Neuroreport 8:2419–2422
79. Patzwahl DR, Zanker JM, Altenmüller EO (1994) Cortical potentials reflecting motion processing in humans. Vis Neurosci 11:1135–1147
80. Kuba M, Kremláček J, Kubová Z (1998) Cognitive evoked potentials related to visual perception of motion in human subjects. Physiol Res 47:265–270

81. Kubová Z, Kremláček J, Szanyi J, Chlubnová J, Kuba M (2002) Visual event-related potentials to moving stimuli: normative data. Physiol Res 51:199–204
82. Jiang Y, Luo YJ, Parasuraman R (2002) Neural correlates of perceptual priming of visual motion. Brain Res Bull 57:211–219
83. Cochin S, Barthelemy C, Roux S, Martineau J (2001) Electroencephalographic activity during perception of motion in childhood. Eur J Neurosci 13:1791–1796
84. Krishnan GP, Skosnik PD, Vohs JL, Busey TA, O'Donnell BF (2005) Relationship between steady-state and induced gamma activity to motion. Neuroreport 16:625–630
85. Patzwahl DR, Zanker JM (2000) Mechanisms of human motion perception: combining evidence from evoked potentials, behavioural performance and computational modelling. Eur J Neurosci 12:273–282
86. Jokisch D, Daum I, Suchan B, Troje NF (2005) Structural encoding and recognition of biological motion: evidence from event-related potentials and source analysis. Behav Brain Res 157:195–204
87. Hassenstein B, Reichardt W (1956) System-theoretische Analyse der Zeit-, Reihenfolgen- und Vorzeichenauswertung bei der Bewegungsperzeption des Rüsselkäfers *Chlorophanus*. Z Naturforsch 11B:513–524
88. Borst A, Egelhaaf M (1989) Principles of visual motion detection. Trends Neurosci 12:297–306
89. Göpfert E, Müller R, Simon EM (1990) The human motion onset VEP as a function of stimulation area for foveal and peripheral vision. Doc Ophthalmol 75:165–173
90. Markwardt F, Göpfert E, Müller R (1988) Influence of velocity, temporal frequency and initial phase position of grating patterns on motion VEP. Biomed Biochim Acta 47:753–760
91. Dodt E, Kuba M (1995) Simultaneously recorded retinal and cerebral potentials to windmill stimulation. Doc Ophthalmol 89:287–298
92. Kubová Z, Kremláček J, Kuba M, Chlubnová J, Svěrák J (2004) Photopic and scotopic VEPs in patients with congenital stationary night-blindness. Doc Ophthalmol 109:9–15
93. Kremláček J, Kuba M, Kubová Z, Chlubnová J (2004) Motion-onset VEPs to translating, radial, rotating and spiral stimuli. Doc Ophthalmol 109:169–175
94. Holliday IE, Meese TS (2005) Neuromagnetic evoked responses to complex motions are greatest for expansion. Int J Psychophysiol 55:145–157
95. Delon-Martin C, Gobbelé R, Buchner H, Haug BA, Antal A, Darvas F, Paulus W (2006) Temporal pattern of source activities evoked by different types of motion onset stimuli. Neuroimage 31:1567–1579
96. Nishiike S, Nakagawa S, Nakagawa A, Uno A, Tonoike M, Takeda N, Kubo T (2002) Magnetic cortical responses evoked by visual linear forward acceleration. Neuroreport 13:1805–1808
97. Beardsley SA, Vaina LM (2005) Psychophysical evidence for a radial motion bias in complex motion discrimination. Vision Res 45:1569–1586
98. Tobimatsu S, Goto Y, Yamasaki T, Tsurusawa R, Taniwaki T (2004) Non-invasive evaluation of face and motion perception in humans. J Physiol Anthropol Appl Human Sci 23:273–276
99. Maffei L, Campbell FW (1970) Neurophysiological localization of the vertical and horizontal visual coordinates in man. Science 167:386–387
100. Müller R, Göpfert E, Hartwig M (1985)VEP-Untersuchungen zur Kodierung der Geschwindigkeit bewegter Streifenmuster im Kortex des Menschen. EEG EMG Z Elektroenzephalogr Elektromyogr Verwandte Geb 16:75–80
101. Maruyama K, Kaneoke Y, Watanabe K, Kakigi R (2002) Human cortical responses to coherent and incoherent motion as measured by magnetoencephalography. Neurosci Res 44:195–205
102. Nakamura Y, Ohtsuka K (1999) Topographical analysis of motion-triggered visual evoked potentials in man. Jpn J Ophthalmol 43:36–43
103. Kawakami O, Kaneoke Y, Maruyama K, Kakigi R, Okada T, Sadato N, Yonekura Y (2002) Visual detection of motion speed in humans: spatiotemporal analysis by fMRI and MEG. Hum Brain Mapp 16:104–118
104. Chlubnová J, Kremláček J, Kubová Z, Kuba M (2005) Visual evoked potentials and event related potentials in congenitally deaf subjects. Physiol Res 54:577–583
105. Lorteije JAM, Kenemans JL, Jellema T, van der Lubbe RHJ, de Heer F, van Wezel RJA (2006) Delayed response to animate implied motion in human motion processing areas. J Cogn Neurosci 18:158–168
106. American Encephalographic Society (1994) Guideline thirteen: guidelines for standard electrode position nomenclature. J Clin Neurophysiol 11:111–113
107. Hoffmann M, Dorn TJ, Bach M (1999) Time course of motion adaptation: motion onset visual evoked potentials and subjective estimates. Vision Res 39:437–444
108. Heinrich SP, Schilling AM, Bach M (2006) Motion adaptation: net duration matters, not continuousness. Exp Brain Res 169:461–466
109. Odom JV, Bach M, Barber C, Brigell M, Marmor MF, Tormene AP, Holder GE, Vaegan (2004) Visual evoked potentials standard (2004). Doc Ophthalmol 108:115–123
110. Kubová Z, Kuba M, Hrochova J, Sverak J (1996) Motion-onset visual evoked potentials improve the diagnosis of glaucoma. Doc Ophthalmol 92:211–221
111. Skrandies W, Jedynak A, Kleiser R (1998) Scalp distribution components of brain activity evoked by visual motion stimuli. Exp Brain Res 122:62–70
112. Kremláček J, Kuba M, Holčík J (2002) Model of visually evoked cortical potentials. Physiol Res 51:65–71

113. Maier J, Dagnelie G, Spekreijse H, van Dijk BW (1987) Principal components analysis for source localization of VEPs in man. Vision Res 27:165–177
114. Kremláček J, Kuba M (1999) Global brain dynamics of transient visual evoked potentials. Physiol Res 48:303–308
115. Victor JD, Mast J (1990) A new statistic for steady-state evoked potentials. Electroenceph Clin Neurophysiol 78:378–388
116. Meigen T, Bach M (1999) On the statistical significance of electrophysiological steady-state responses. Doc Ophthalmol 98:207–232
117. De Vries M, van Dijk B, Spekreijse H (1989) Motion onset-offset VEPs in children. Electroenceph Clin Neurophysiol 74:81–87
118 Norcia AM, Garcia H, Humphry R, Holmes A, Hamer RD, Orel-Bixler D (1991) Anomalous motion VEPs in infants and in infantile esotropia. Invest Ophthalmol Vis Sci 32:436–439
119. Braddick O, Birtles D, Wattam-Bell J, Atkinson J (2005) Motion- and orientation-specific responses in infancy. Vision Res 45:3169–3179
120. Hamer RD, Norcia AM (1994) The development of motion sensitivity during the first year of life. Vision Res 34:2387–2402
121. Birch EE, Fawcett S, Stager D (2000) Co-development of VEP motion response and binocular vision in normal infants and infantile esotropes. Invest Ophthalmol Vis Sci 41:1719–1723
122. Mitchell TV, Neville HJ (2004) Asynchronies in the development of electrophysiological responses to motion and color. J Cogn Neurosci 16:1363–1374
123. Korth M, Kohl S, Martus P, Sembritzki O (2000) Motion-evoked pattern visual evoked potentials in glaucoma. J Glaucoma 9:376–387
124. Langrová J, Kuba M, Kremláček J, Kubová Z, Vít F (2006) Motion-onset VEPs reflect long maturation and early aging of visual motion-processing system. Vision Res 46:536–544
125. Coch D, Skendzel W, Grossi G, Neville H (2005) Motion and color processing in school-age children and adults: an ERP study. Dev Sci 8:372–386
126. Müller R, Göpfert E, Breuer D, Greenlee MW (1999) Motion VEPs with simultaneous measurement of perceived velocity. Doc Ophthalmol 97:121–134
127. Müller R, Bochmann G, Greenlee MW, Göpfert E (2003) Relationship between motion VEP and perceived velocity of gratings: effects of stimulus speed and motion adaptation. Doc Ophthalmol 107:115–126
128. Wang L, Kaneoke Y, Kakigi R (2003) Spatiotemporal separability in the human cortical response to visual motion speed: a magnetoencephalography study. Neurosci Res 47:109–116
129. Gallichio JA, Andreassi JL (1982) Visual evoked potentials under varied velocities of continuous and discrete apparent motion. Int J Neurosci 17:169–177
130. Kaneoke Y, Bundou M, Koyama S, Suzuki H, Kakigi R (1997) Human cortical area responding to stimuli in apparent motion. Neuroreport 8:677–682
131. Kawakami O, Kaneoke Y, Kakigi R (2000) Perception of apparent motion is related to the neural activity in the human extrastriate cortex as measured by magnetoencephalography. Neurosci Lett 285:135–138
132. Clifford CWG, Langley K (1996) Psychophysics of motion adaptation parallels insect electrophysiology. Curr Biol 6:1340–1342
133. Hammett ST, Thompson PG, Bedingham S (2000) The dynamics of velocity adaptation in human vision. Curr Biol 10:1123–1126
134. Uusitalo MA, Jousmäki V, Hari R (1997) Activation trace lifetime of human cortical responses evoked by apparent visual motion. Neurosci Lett 224:45–48
135. Amano K, Nishida S, Takeda T (2006) MEG responses correlated with the visual perception of velocity change. Vision Res 46:336–345
136. Greenlee MW, Heitger F (1988) The functional role of contrast adaptation. Vision Res 28:791–797
137. Kremláček J, Kuba M, Chlubnová J, Kubová Z (2006) Visual mismatch negativity elicited by magnocellular system activation. Vision Res 46:485–490
138. Näätänen R, Jacobsen T, Winkler I (2005) Memory-based or afferent processes in mismatch negativity (MMN): a review of evidence. Psychophysiol 42:25–32
139. McKeefry DJ (2001) Chromatic visual evoked potentials elicited by fast and slow motion onset. Col Res Appl 26:S145–S149
140. McKeefry DJ (2002) The influence of stimulus chromaticity on the isoluminant motion-onset VEP. Vision Res 42:909–922
141. McKeefry DJ (2001) Motion adaptation in chromatic motion-onset visual evoked potentials. Doc Ophthalmol 103:229–251
142. Morand S, Thut G, de Peralta RG, Clarke S, Khateb A, Landis T, Michel CM (2000) Electrophysiological evidence for fast visual processing through the human koniocellular pathway. Cereb Cortex 10:817–825
143. Victor JD, Conte MM (1992) Evoked potential and psychophysical analysis of fourier and non-fourier motion mechanisms. Vis Neurosci 9:105—123
144. Ellemberg D, Lavoie K, Lewis TL, Maurer D, Lepore F, Guillemot JP (2003) Longer VEP latencies and slower reaction times to the onset of second-order motion than to the onset of first-order motion. Vision Res 43:651–658
145. Sofue A, Kaneoke Y, Kakigi R (2003) Physiological evidence of interaction of first- and second-order motion processes in the human visual system: a magnetoencephalographic study. Hum Brain Mapp 20:158–167
146. Thier P, Haarmeier T, Chakraborty S, Lindner A, Tikhonov A (2001) Cortical substrates of perceptual stability during eye movements. Neuroimage 14:533–539
147. Hoffmann MB, Bach M (2002) The distinction between eye and object motion is reflected by the motion-onset visual evoked potential. Exp Brain Res 144:141–151

148. Tikhonov A, Haarmeier T, Thier P, Braun C, Lutzenberger W (2003) Neuromagnetic activity in medial parietooccipital cortex reflects the perception of visual motion during eye movements. Neuroimage 21:590–600
149. Armington JC, Bloom MB (1974) Relations between the amplitudes of spontaneous saccades and visual responses. J Opt Soc Am 64:1263–1271
150. Kleiser R, Skrandies W (2000) Neural correlates of reafference: evoked brain activity during motion perception and saccadic eye movements. Exp Brain Res 133:312–320
151. Torriente I, Valdes-Sosa M, Ramirez D, Bobes MA (1999) Visual evoked potentials related to motion-onset are modulated by attention. Vision Res 39:4122–4139
152. Pazo-Alvares P, Amenedo E, Cadaveira F (2004) Automatic detection of motion direction changes in the human brain. Eur J Neurosci 19:1978–1986
153. Lorenzo-López L, Amenedo E, Pazo-Alvares P, Cadaveira F (2004) Pre-attentive detection of motion direction changes in normal aging. Neuroreport 15:2633–2636
154. Anllo-Vento L, Hillyard SA (1996) Selective attention to the color and direction of moving stimuli: electrophysiological correlates of hierarchical feature selection. Percept Psychophys 58:191–206
155. Martín-Loeches M, Hinojosa JA, Rubia FJ (1999) Insights from event-related potentials into the temporal and hierarchical organization of the ventral and dorsal streams of the visual system in selective attention. Psychophysiology 36:721–736
156. Valdes-Sosa M, Bobes MA, Rodriguez V, Pinilla T (1998) Switching attention without shifting the spotlight: object-based attentional odulation of brain potentials. J Cogn Neurosci 10:137–151
157. Niedeggen M, Sahraie A, Hesselmann G, Milders M, Blakemore C (2002) Is experimental motion blindness due to sensory suppression? An ERP approach. Cogn Brain Res 13:241–247
158. Niedeggen M, Hesselmann G, Sahraie A, Milders M, Blakemore C (2004) Probing the prerequisites for motion blindness. J Cogn Neurosci 16:584–597
159. Rodríguez V, Valdés-Sosa M (2006) Sensory suppression during shifts of attention between surfaces in transparent motion. Brain Res 1072:110–118
160. Fort A, Besle J, Giard MH, Pernier J (2005) Task-dependent activation latency in human visual extrastriate cortex. Neurosci Lett 379:144–148
161. Beer AL, Röder B (2005) Attending to visual or auditory motion affects perception within and across modalities: an event-related potential study. Eur J Neurosci 21:1116–1130
162. Armstrong BA, Neville HJ, Hillyard SA, Mitchell TV (2002) Auditory deprevation affects processing of motion, but not color. Cogn Brain Res 14:422–434
163. Probst T, Wist ER (1990) Electrophysiological evidence for visual-vestibular interactions in man. Neurosci Lett 108:255–260
164. Bach M, Hoffmann MB (2000) Visual motion detection in man is governed by non-retinal mechanisms. Vison Res 40:2379–2385
165. Korth M, Rix R, Sembritzki O (2000) The sequential processing of visual motion in the human electroretinogram and visual evoked potential. Vis Neurosci 17:631–646
166. Matsumoto R, Ikeda A, Nagamine T, Matsuhashi M, Ohara S, Yamamoto J, Toma K, Mikuni N, Takahashi J, Miyamoto S, Fukuyama H, Shibasaki H (2004) Subregions of human MT complex revealed by comparative MEG and direct electrocorticographic recordings. Clin Neurophysiol 115:2056–2065
167. Uusitalo MA, Virsu V, Salenius S, Näsänen R, Hari R (1997) Activation of human V5 complex and rolandic regions in association with moving visual stimuli. Neuroimage 5:241–250
168. Azzopardi P, Fallah M, Gross CG, Rodman HR (2003) Response latencies of neurons in visual areas MT and MST of monkeys with striate cortex lesions. Neuropsychologia 41:1738–1756
169. Müller R, Göpfert E, Schlykowa L, Anke D (1990) The human motion VEP as a function of size and eccentricity of the stimulation field. Doc Ophthalmol 76:81–89
170. Kremláček J, Kuba M, Chlubnová J, Kubová Z (2004) Effect of stimulus localisation on motion-onset VEP. Vision Res 44:2989–3000
171. Yokoyama M, Matsunaga I, Yonekura Y, Shinzato K (1979) The visual evoked response to a moving pattern. In: Tazawa Y (ed) Proceedings of the XVIth Symposium of the International Society for Clinical Electrophysiology of Vision—Morioka, 24–28 May 1978, Jpn J Ophthalmol, Tokyo, pp 207–214
172. Takao M, Miyata Y (2001) Spatial property of motion visual evoked potentials. Percept Mot Skills 93:735–738
173. Niedeggen M, Wist ER (1999) Characteristics of visual evoked potentials generated by motion coherence onset. Cogn Brain Res 8:95–105
174. Ulbert I, Karmos G, Heit G, Halgren E (2001) Early discrimination of coherent versus incoherent motion by multiunit and syaptic activity in human putative MT+. Hum Brain Mapp 13:226–238
175. Lam K, Kaneoke Y, Gunji A, Yamasaki H, Matsumoto E, Naito T, Kakigi R (2000) Magnetic response of human extrastriate cortex in the detection of coherent and incoherent motion. Neuroscience 97:1–10
176. Lam K, Kaneoke Y, Kakigi R (2003) Human cortical response to incoherent motion on a background of coherent motion. Neurosci Lett 247:41–44
177. Niedeggen M, Wist ER (1998) Motion evoked brain potentials parallel the consistency of coherent motion perception in humans. Neurosci Lett 246:61–64
178. Aspell JE, Tanskanen T, Hurlbert AC (2005) Neuromagnetic correlates of visual motion coherence. Eur J Neurosci 22:2937–2945
179. Amano K, Goda N, Nishida S, Ejima Y, Takeda T, Ohtani Y (2006) Estimation of the timing of human

visual perception from magnetoencephalography. J Neurosci 26:3981–3991
180. ffytche DH, Guy CN, Zeki S (1995) The parallel visual motion inputs into areas V1 and V5 of human cerebral cortex. Brain 118:86–96
181. Schoenfeld MA, Heinze HJ, Woldorff MG (2002) Unmasking motion-processing activity in human brain area V5/MT+ mediated by pathways that bypass primary visual cortex. Neuroimage 17:769–779
182. Benson PJ, Guo K, Hardiman MJ (1999) Cortical evoked potentials due to motion contrast in the blind hemifield. Neuroreport 10:3595–3600
183. Rasmussen CA, Kaufman PL (2005) Primate glaucoma models. J Glaucoma 14:311–314
184. Shabana N, Pérès VC, Carkeet A, Chew PTK (2003) Motion perception in glaucoma patients: a review. Surv Ophthalmol 48:92–106
185. Bach M (2001) Electrophysiological approaches for early detection of glaucoma. Eur J Ophthalmol Suppl 11:S41–S49
186. Bach M, Unsoeld AS, Philippin H, Staubach F, Maier P, Walter HS, Bomer TG, Funk J (2006) Pattern erg as early risk indicator in ocular hypertension—a long-term prospective study. Invest Ophthalmol Vis Sci 47:4881–4887
187. Kubová Z, Kuba M (1992) Clinical application of motion-onset visual evoked potentials. Doc Ophthalmol 81:209–218
188. Kubová Z, Kuba M (1995) Motion-onset VEPs improve the diagnostics of multiple sclerosis and optic neuritis. Sbor věd Prací LF UK Hradec Králové 38:89–93
189. Herbst H, Ketabi A, Thier P, Dichgans J (1997) Comparison of psychophysical and evoked potential methods in the detection of visual deficits in multiple sclerosis. Electroenceph Clin Neurophysiol 104:82–90
190. Tobimatsu S, Kato M (1998) Multimodality visual evoked potentials in evaluating visual dysfunction in optic neuritis. Neurology 50:715–718
191. Kubová Z, Kuba M, Juran J, Blakemore C (1996) Is the motion system relatively spared in amplyopia? Evidence from cortical evoked potentials. Vision Res 36:181–190
192. Ellemberg D, Lewis TL, Maurer D, Brar S, Brent HP (2002) Better perception of global motion after monocular than after binocular deprivation. Vision Res 42:169–179
193. Constantinescu T, Schmidt L, Watson R, Hess RF (2005) A residual deficit for global motion processing after acuity recovery in deprivation amblyopia. Invest Ophthalmol Vis Sci 46:3008–3012
194. Ho CS, Giashi DE, Boden C, Doughterty R, Cline R, Lyons C (2005) Deficient motion perception in the fellow eye of amblyopic children. Vision Res 45:1615–1627
195. Norcia AM (1996) Abnormal motion processing and binocularity: infantile esotropia as a model system for effects of early interruptions of binocularity. Eye 10:259–265
196. Shea SJ, Chandna A, Norcia AM (1999) Oscillatory motion but not pattern reversal elicits monocular motion biases in infantile esotropia. Vision Res 39:1803–1811
197. Anteby I, Zhai HF, Tychsen L (1998) Asymmetric motion visually evoked potentials in infantile strabismus are not an artifact of latent nystagmus. J AAPOS 2:153–158
198. Wilson JR, Noyd WW, Aiyer AD, Norcia AM, Mustari MJ, Boothe RG (1999) Asymmetric responses in cortical visually evoked potentials to motion are not derived from eye movements. Invest Ophthalmol Vis Sci 40:2435–2439
199. Norcia AM, Hamer RD, Jampolsky A, Orel-Bixler D (1995) Plasticity of human motion processing mechanisms following surgery for infantile esotropia. Vision Res 35:3279–3296
200. Tychsen L, Wong WMF, Foeller P, Bradley D (2004) Early versus delayed repair of infantile strabismus in macaque monkeys: II. Effects on motion visually evoked responses. Invest Ophthalmol Vis Sci 45:821–827
201. Kommerell G, Ullrich D, Gilles U, Bach M (1995) Asymmetry of motion VEP in infantile strabismus and in central vestibular nystagmus. Doc Ophthalmol 89:373–381
202. Brosnahan D, Norcia AM, Schor CM, Taylor DG (1998) OKN, perceptual and VEP direction biases in strabismus. Vision Res 38:2833–2840
203. Mason AJ, Braddick OJ, Wattam-Bell J, Atkinson J (2001) Directional motion asymmetry in infant VEPs–which direction? Vision Res 41:201–211
204. Fawcett SL, Birch EE (2000) Motion VEPs, stereopsis, and bifoveal fusion in children with strabismus. Invest Ophthalmol Vis Sci 41:411–416
205. Livingstone MS, Rosen GD, Drislane FW, Galaburda AM (1991) Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. Proc Natl Acad Sci USA 88:7943–7947
206. Skottun BC (2000) The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. Vision Res 40:111–127
207. Stein J, Talcott J, Walsh VV (2000) Controversy about the visual magnocellular deficit in developmental dyslexics. Trends Cogn Sci 4:209–211
208. Kubová Z, Kuba M, Peregrin J, Nováková (1995) Visual evoked potential evidence for magnocellular system deficit in dyslexia. Physiol Res 44:87–89
209. Kuba M, Szanyi J, Gayer D, Kremláček J, Kubová Z (2001) Electrophysiological testing of dyslexia. Acta Medica (Hradec Králocé) 44:131–134
210. Schulte-Körne G, Bartling J, Deimel W, Remschmidt H (2004) Motion-onset VEPs in dyslexia. evidence for visual perceptual deficit. Neuroreport 15:1075–1078
211. Schulte-Körne G, Bartling J, Deimel W, Remschmidt H (2004) Visual evoked potential elicited by coherently moving dots in dyslexic children. Neurosci Lett 357:207–210
212. Skottun BC, Skoyles JR (2004) Some remarks on the use of motion VEPs to assess magnocellular sensitivity. Clin Neurophysiol 115:2834–2836
213. Kuba M, Kremláček J, Hulek P, Kubová Z, Vít F (1996) Advanced electrophysiological diagnostics of

hepatic andportosystemic encephalopathy. Acta Medica (Hradec Králocé) 39:21–26
214. Arakawa K, Tobimatsu S, Kato M, Kira J (1999) Parvocellular and magnocellular visual processing in spinocerebellar degeneration and Parkinson's disease: an event-related potential study. Clin Neurophysiol 110:1048–1057
215. Nakamura M, Kaneoke Y, Watanabe K, Kakigi R (2002) Visual information process in williams syndrome: intact motion detection accompanied by typical visuospatial dysfunctions. Eur J Neurosci 16:1810–1818
216. Kubová Z, Szanyi J, Langrová J, Kremláček J, Kuba M, Honegr K (2006) Motion-onset and pattern-reversal visual evoked potentials in diagnostics of neuroborreliosis. J Clin Neurophysiol 23:416–420
217. Smith AM, Majaj NJ, Movshon JA (2005) Dynamics of motion signalling by neurons in macaque area MT. Nat Neurosci 8:220–228