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EEG Correlates of Vection: A Systematic Literature Review

Gaël Van der Lee, François Cabestaing, Hakim Si-Mohammed
Univ. Lille, CNRS, Centrale Lille, UMR 9189
CRIStAL, F-59000, Lille, France

Emails: gael.vanderlee@univ-lille.fr, francois.cabestaing@univ-lille.fr, hakim.simohammed@univ-lille.fr

Abstract—Vection, the illusory sensation of self-motion induced by visual stimuli, is fundamental to understanding sensory integration and has significant implications for virtual reality (VR) and motion sickness research. This review synthesizes findings from 16 peer-reviewed studies that employed electroencephalography (EEG) to investigate the neural correlates of vection. We find that EEG research consistently identifies modulations in the alpha frequency band (α), particularly over parieto-occipital regions, as a key neural signature. Early visual event-related potentials (ERPs), notably the N2 component, also serve as reliable correlates, indexing the initial processing and integration of motion cues sensitive to stimulus properties. Neural activity patterns are further influenced by stimulus characteristics (e.g., field-of-view, motion type), task demands, and individual differences, including handedness and susceptibility to visually induced motion sickness (VIMS). Emerging research explores network connectivity and demonstrates the potential for manipulation of vection via non-invasive brain stimulation targeting α oscillations. Despite these advances, considerable methodological heterogeneity in stimuli, EEG analysis, and vection assessment across studies currently limits direct comparison and the establishment of universally accepted biomarkers. Future research with standardized protocols and larger sample sizes is necessary to solidify these findings. Nonetheless, EEG stands as a valuable tool for exploring the temporal dynamics of vection, offering potential pathways to enhance VR design, mitigate adverse effects like motion sickness, and deepen our understanding of human self-motion perception.

Index Terms—Vection, EEG, Electroencephalography, Virtual Reality, Motion Sickness, Systematic Review, Alpha Band

I. Introduction

Vection, the illusion of self-motion experienced by a stationary observer exposed to visual motion cues [1], has been a subject of scientific inquiry for at least a century [2]. Studying Vection offers a pathway towards understanding the brain’s mechanism for integrating sensory information, particularly how visual input compensates for the vestibular system’s limitations in detecting constant velocity motion [3]. Understanding vection is therefore crucial for understanding the fundamental principles of visuo-vestibular interaction and self-motion perception. While research has expanded into applied domains like simulators and virtual reality [4], [5], a major challenge lies in objectively quantifying the vection experience. Furthermore, the large-field visual motion stimuli that induce vection are often a trigger of visually induced motion sickness (VIMS). These adverse effects can be

accompanied by functional consequences such as postural instability, which has been associated with a higher cognitive workload [6]. Consequently, a related line of inquiry uses EEG to establish objective markers of VIMS [7], often within multimodal assessment paradigms designed to quantify physiological responses to provocative motion [8]. Indeed, Palmisano et al. [1] identified objective vection measurement as one of the major future challenges in the field. Subjective self-reports, though indispensable, are inherently variable and susceptible to bias [9], necessitating complementary objective physiological measures.

Electroencephalography (EEG), with its high temporal resolution, offers a powerful, non-invasive window into the rapid neural dynamics accompanying the onset and maintenance of vection [4], [5]. A growing number of studies have employed EEG to identify potential neural signatures of vection. This paper presents a systematic review of this literature, synthesizing findings from sixteen peer-reviewed studies specifically investigating EEG correlates of vection. Our primary aims are to: (1) characterize the consistent EEG patterns, such as frequency band oscillations (e.g., alpha power) and event-related potentials (ERPs), associated with vection; (2) examine how these patterns are modulated by stimulus properties, individual differences, and task context; and (3) overview the current state of the field regarding methodology and direction. By consolidating these findings, we seek to provide a clearer picture of the neural underpinnings of vection captured by EEG, evaluate the potential for robust biomarkers, and inform both fundamental research on sensory integration and applications in domains such as extended reality (XR) environments.

II. Methods

This systematic review adheres to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) reporting guidelines [10], aiming to provide a transparent account of the study identification, selection, and synthesis process.

A. Search Strategy

A systematic literature search was conducted on May 1, 2025, across three scientific databases: PubMed, Scopus, and Web of Science. These databases were selected for their comprehensive and overlapping coverage of the

biomedical, engineering, and psychological literature relevant to this interdisciplinary topic. No publication start date was applied in order to ensure a comprehensive historical retrieval of all relevant studies. The search included all articles published up to the search date. The following search query was employed uniformly across each database: “Vection” AND (“EEG” OR “Electroencephalogram”): No other databases were systematically searched, as the selected sources were deemed sufficient to capture the relevant literature. This initial search yielded 18 results from PubMed, 27 from Web of Science, and 23 from Scopus, for a total of 68 records before duplicate removal.

B. Study Selection and Eligibility Criteria

Retrieved records were aggregated, and duplicate entries were identified and removed using custom Python scripts, primarily based on Digital Object Identifiers (DOIs) and PubMed IDs (PMIDs) where available. This process identified and removed 36 duplicate records, leaving 32 unique records for screening.

The articles were independently screened using the titles and abstracts of these 32 unique records against predefined eligibility criteria. Full texts were retrieved and assessed when abstract information was insufficient for a decision. The eligibility criteria were defined as follows:

Inclusion Criteria:

- Original research articles (i.e., reporting primary data).
- Studies explicitly investigating visually induced vection (the illusory perception of self-motion).
- Studies utilizing electroencephalography (EEG) as a measurement technique for assessing neural correlates related to vection.

Exclusion Criteria:

- Review articles, meta-analyses, editorials, letters, or conference abstracts without corresponding full papers.
- Studies where the full text was unavailable.
- Studies not utilizing EEG.
- Studies focused solely on motion sickness, simulator sickness, or other phenomena without specifically analyzing EEG correlates of the vection experience itself.

Following the screening process, 15 records were excluded as they did not meet the inclusion criteria. Of the remaining papers, one could not be accessed in its entirety. This resulted in a final selection of 16 studies deemed relevant for inclusion in this systematic review. The study selection process is summarized in a PRISMA flow diagram Figure 1.

C. Data Extraction

A standardized data extraction form was developed. For each included study, we systematically extracted publication details (authors, year, title) and core study design information (objectives, number of subjects). We recorded

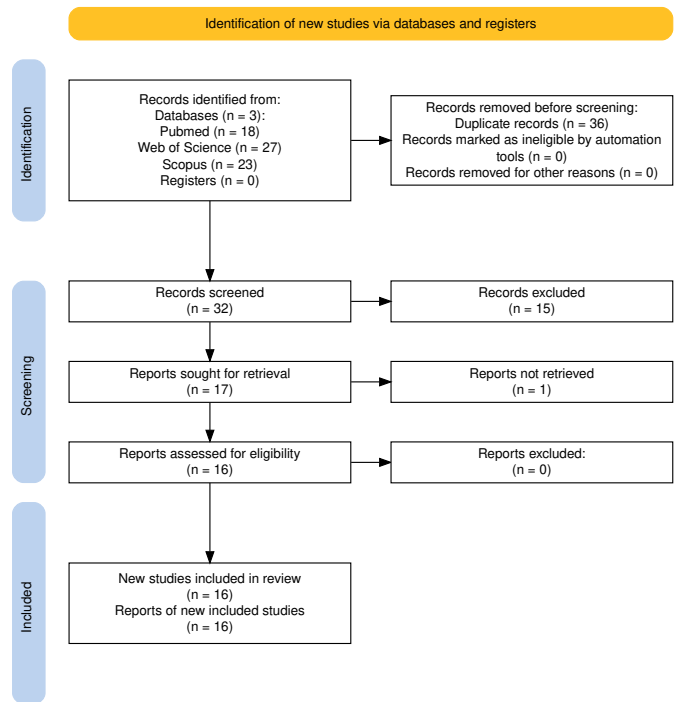


Fig. 1. PRISMA Diagram of the retrieval and selection process

methods related to vection, including induction stimuli (parameters, display system) and assessment techniques. Comprehensive EEG methodology details were extracted, covering the setup, signal processing steps (e.g., filtering, artifact correction), and primary analysis approaches (e.g., spectral analysis, ERPs, connectivity, source localization, statistical tests). Key findings regarding EEG markers of vection were noted, along with influencing factors investigated, such as stimulus characteristics, individual differences (e.g., handedness, motion sickness susceptibility), and task interactions. Finally, reported quantitative results were documented where available. The extracted data were consolidated into a structured table to facilitate synthesis and comparison across studies.

D. Data Synthesis

Given the heterogeneity anticipated in methodologies and reported metrics across studies, a qualitative narrative synthesis approach was employed. The extracted findings were organized and synthesized thematically based on the objectives of the EEG analysis and the research question addressed. The main themes identified a priori were refined during data extraction, resulting in the following five primary categories used to structure the results of this review:

- 1) EEG Correlates of Vection, further subdivided into:
 - a) Rhythms (Power, Frequency)
 - b) Potentials (ERPs, SSEPs)
 - c) Connectivity & Synchronization
 - d) Topography & Source Localization

- 2) Stimulus Factors (e.g., effects of different visual stimuli, field-of-view, motion types, posture).
- 3) Individual Differences (e.g., age, handedness, motion sickness susceptibility).
- 4) Task Interaction (e.g., vection’s impact on concurrent tasks/cognition).
- 5) Neuromodulation (e.g., effects of tACS).

III. Results

A. Overview of the Included Literature

Sixteen peer-reviewed articles satisfied all eligibility criteria for this review. Their temporal distribution (Figure 2) confirms a marked growth of electroencephalographic vection research over the past decade, with more than two-thirds of the papers published after 2018, and a recent shift towards studying individual differences and neuromodulation.

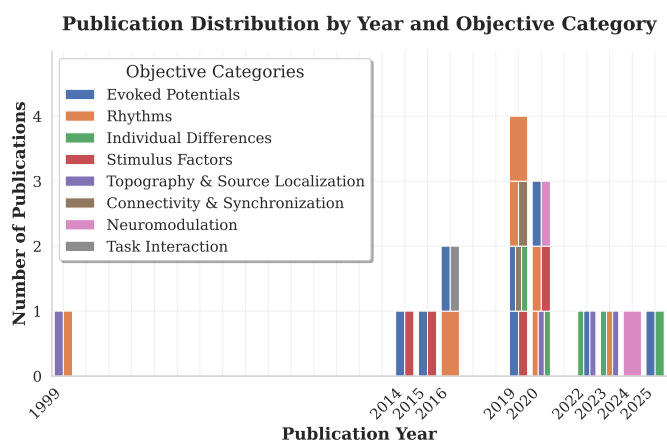


Fig. 2. Distribution of the included studies by publication year and research-objective category. Each block represents one study. For studies assigned to multiple categories, the block is divided into colored stripes, each corresponding to one of the assigned categories.

B. Global Study Characteristics

a) Participants: Sample sizes ranged from $N = 5$ to $N = 57$ (Median = 20). All studies recruited healthy adults; one examined younger versus older cohorts [14], two contrasted left- and right-handers [16], [13], and three separated visually induced motion-sickness (VIMS)-susceptible from resistant participants [25], [12], [17].

b) EEG acquisition: Most vection-EEG studies recorded with medium-density caps (32–64 active electrodes, 10/20–10/10 layout, although sparser montages of 19–28 channels [21], [20] and very dense 96-channel systems [22] also appear. Signals were usually referenced to linked mastoids or earlobes and often re-referenced offline to the common average. Pre-processing almost always applied a 0.1–30/70 Hz band-pass filter plus a 50 or 60 Hz notch for line noise, followed by ocular/muscle artefact removal with ICA or threshold rejection. Data were then resampled to 250–1000 Hz and epoched to stimulus onset or button-marked vection episodes [14],

[25]. Spectral features were extracted with Fast Fourier Transforms [20], [13], [17], Morlet wavelet time–frequency analyses [12], [22], [25], or ERSP/t-f PCA approaches [20]. Event-related potentials were quantified in fixed windows over occipito-parietal sites [21], [15], [14], and some studies also derived connectivity measures such as phase-locking value or cerebro-cerebellar coherence [25], [23].

c) Visual stimulation: All experiments relied on visually induced vection. Eight studies used full-field roll-motion dot patterns [13], [16], [22], [24], [12], [25], [26], [17]; one presented tunnel flow [11] and another simulated constant-velocity forward flow in depth [20]; two manipulated central versus peripheral motion [21], [15]. The duration of visual motion stimulation varies substantially, ranging from brief epochs of 250 ms [11] to prolonged presentations lasting up to 45 seconds [15]. Body posture was seated in all but one study that contrasted upright with supine positions to modulate visuo-vestibular conflict [22].

C. Research Objective Categories

1) Category 1: EEG Correlates of Vection:

a) 1.1 Rhythms (Power and Frequency): Seven studies quantified oscillatory power, predominantly in the α band (8–13 Hz) [20], [13], [22], [18], [12], [17], [23]. Analytical approaches comprised fast-Fourier transforms, Morlet wavelets, and event-related spectral perturbation (ERSP) maps. Table II consolidates the principal rhythm findings.

b) 1.2 Potentials (ERPs, SSVEPs): Eight studies examined potentials time-locked to the visual stimuli used for vection induction, comprising seven studies focusing on Event-Related Potentials (ERPs) [11], [21], [15], [19], [16], [25], [14], [13] and one on Steady-State Visually Evoked Potentials (SSVEPs) [26]. Motion-onset ERPs typically included early sensory components like P1 (~100 ms) and N2 (~200 ms), followed by later P2/P3 complexes. Table III summarizes the findings on ERPs, which generally demonstrate differences in characteristics depending on the specific visual motion parameters presented (e.g., coherence, spatial location). Additionally, one study employed steady-state visually evoked potentials (SSVEPs) elicited by 10 Hz flickering stimuli, finding a shift in amplitude and lateralization depending on whether subjects experienced vection [26].

c) 1.3 Connectivity and Synchronization: Two studies computed functional connectivity metrics. One found that phase-locking values revealed increased parieto-temporal theta synchrony preceding vection in VIMS-resistant individuals [25]. The other that cerebro-cerebellar coherence in the α band increased during vestibular-driven vection [23].

d) 1.4 Topography and Source Localization: Scalp distributions were assessed in four papers, with parieto-occipital regions most frequently implicated. Lateralization depended on handedness [13] and on vection state in SSVEP paradigms [26]. Source estimations (eLORETA /

TABLE I
Experimental parameters of the included EEG-vection studies.

Study	N	Stimulus / Motion	EEG Ch.	Analysis Focus	Category
[11]	29	Tunnel flow (\pm)	12	M-VEP	Potentials, Stimulus
[12]	20	Rotating dots	64	TF, source	Rhythms, Topography, IndividualDiff
[13]	50	Coherent roll	64	FFT, TF	Rhythms, Topography, IndividualDiff
[14]	47	Optokinetic stripes	32	ERPs	Potentials, IndividualDiff
[15]	13	Central/peripheral bars	35	ERPs	Potentials, Stimulus
[16]	57	Coherent vs incoherent roll	64	ERPs, source	Potentials, Topography, IndividualDiff
[17]	6	Rotating circles	20	FFT, topography	Rhythms
[18]	5	Rotating star field	12	Topography, FFT	Topography, Rhythms
[19]	19	Moving bars + oddball	64	ERPs	Potentials, TaskInteraction
[20]	10	Radial forward flow	28	ERSP, t-f PCA	Rhythms
[21]	13	Central/peripheral bars	19	ERPs	Potentials, Stimulus
[22]	21	Roll (upright/supine)	96	TF, cluster	Rhythms, Stimulus
[23]	9	Vestibular + OKN	4 cerebellar	Coherence	Connectivity, Rhythms
[24]	22	Optokinetic roll + tACS	8	Behaviour, tACS	Neuromodulation
[25]	27	Coherent roll	32	ERPs, PLV	Potentials, Connectivity, IndividualDiff
[26]	20	Optic-flow flicker 10 Hz	3	SSVEP	Potentials, Neuromodulation

TABLE II
Summary of rhythm-based findings across studies. \uparrow = increase; \downarrow = decrease.

Study	Band(s)	Direction	Time Window	Topography
[20]	α (10 Hz)	\uparrow	\sim 14 s after onset	Left fronto-central (diffuse)
	β	\downarrow	8–12 s after onset	Parietal
	δ	\downarrow	Later during motion (16–20 s)	Right fronto-central / diffuse
[13]	α	\downarrow (onset); \uparrow (sustained)	Around vection onset / ongoing	Centro-parietal (left-lateral for LH; midline for RH)
[22]	α	\downarrow (onset); \uparrow (upright)	Onset / during vection	Sensorimotor (onset); parieto-occipital (upright)
[18]	High α	\downarrow	During vection	Variable centro-parietal
[12]	δ, θ	\uparrow	During vection	Temporal (sensor-space); superior/middle temporal (source)
	α, β	\uparrow (resistant group only)	During vection	Widespread (sensor-space)
[17]	High α	\uparrow	“Unrecognised” vection period	Right centro-frontal
[23]	α (ECeG)	\downarrow	Vestibular vection	Cerebellum (ECeG); cerebro-cerebellar coherence \uparrow

TABLE III
Summary of ERP findings across studies. \uparrow = larger amplitude / longer latency for the vection condition relative to its control; \downarrow = smaller amplitude / shorter latency.

Study	Component(s)	Modulation	Latency (ms)	Topography
[11]	M-VEP	Direction-dependent difference	\sim 600+	Occipital \rightarrow central
[21]	P1, N2	N2 \uparrow (moving stripes); P1 marginal	120–240	Occipital
[15]	P2, N2	Amplitude varies with motion pattern	130–330	Parieto-occipital
[19]	P1, N2, P3	\downarrow amplitude, P3 \uparrow latency (periphery moving)	P1: 90–130, N2: 150–240, P3: 300–500	Occipital, parietal, frontal
[16]	Early/Late CP	Attenuated for vection-consistent motion	160–300	Central, CSv/retrosplenial
[25]	N223, N290	N223 \uparrow (susceptible), N290 \sim vection	N223: 195–250, N290: 250–310	Parietal
[14]	P1, P2	\uparrow (non-coherent motion)	P1: 100–150, P2: 150–230	Parietal-occipital

sLORETA) mapped early ERP generators to the cingulate sulcus visual area (CSv) and retrosplenial cortex [16], [12]. Tokumaru et al. [18] observed variable high alpha topography during vection, often showing decreases in centro-parietal or mid-frontal regions.

2) Category 2: Stimulus Factors: Four studies manipulated properties of the visual stimulus or body posture. Field-of-view restrictions modulated vection strength and ERP magnitude [15]. Body position (upright vs supine) altered sustained α increases during vection [22]. Forward versus backward optic flow produced distinct motion-

related VEPs [11]. Central versus peripheral motion also affected vection strength and N2 amplitude, with peripheral motion being most effective at inducing both subjective vection and larger occipital N2 responses [21].

3) Category 3: Individual Differences: Handedness studies reported topographical shifts of α power and ERP lateralization without behavioural vection differences [16], [13]. Aging did not significantly affect vection ratings or early ERPs to coherent motion [14]. VIMS susceptibility was linked to enhanced parietal N2 amplitudes and altered θ connectivity [25], as well as differential band-power

changes [12].

4) Category 4: Task Interaction: One study combined a visual oddball task with vection stimulation, showing attenuated sensory (P1, N2) and cognitive (P3) ERPs plus slower reaction times when both central and peripheral fields were moving [19].

5) Category 5: Neuromodulation: Two studies have examined neuromodulatory approaches during visually induced self-motion. One experiment applied individualized α -frequency transcranial alternating current stimulation (tACS) over the superior parietal cortex while inducing vection. Alpha-tACS increased subjective vection intensity compared with θ -tACS and sham conditions [24]. Another study combined right-hemisphere alpha-frequency tACS with SSVEP recording and found that tACS enhanced SSVEP amplitudes during optic-flow-induced self-motion perception; the effect depended on frequency specificity and sufficient current intensity [26].

D. Synthesis of Core Electrophysiological Findings

Across methodologies, α -band activity and early visual ERPs emerged as the most frequently addressed EEG correlates of visually induced self-motion and visual perception. The majority of rhythm investigations reported transient α reductions near vection onset followed by parieto-occipital α enhancements during sustained vection, whereas ERP studies consistently identified N2 amplitude modulations in response to motion-onset configurations. Connectivity and neuromodulation research is less prevalent but provides preliminary evidence for cerebello-cortical coupling and causal influences of parietal α oscillations on vection experience.

IV. Discussion

This systematic review synthesized sixteen peer-reviewed studies investigating the electroencephalographic correlates of visually induced self-motion perception, or vection. The growing body of literature, particularly over the last five years, underscores an increasing interest in leveraging EEG to understand the neural mechanisms underlying this phenomenon. Our analysis reveals consistent patterns of brain activity associated with vection, alongside variability introduced by stimulus parameters, individual traits, and task demands.

The most robust finding across studies centers on the modulation of alpha-band (α) oscillations, primarily over parieto-occipital scalp regions. Several investigations report a transient decrease in α power around the onset of the vection percept [22], [13], potentially reflecting the initial engagement of cortical resources required to re-evaluate sensory inputs and initiate the re-weighting process favoring vision. This is often followed by, or contrasted with, a sustained increase in α power during established vection [20], [22], [13], particularly in upright postures involving visuo-otolithic conflict [22]. Such sustained alpha enhancement is frequently interpreted within the framework of cortical inhibition [27], suggesting a

mechanism for actively suppressing conflicting vestibular or proprioceptive signals once the visual dominance is established [22]. These later oscillatory changes, particularly in the alpha band, have shown direct correlations with subjective vection intensity ratings [20], [23]. The topographical distribution of these alpha changes shows some dependence on individual factors, notably handedness, which influences the lateralization of the effect [13]. Furthermore, the causal relevance of parietal α oscillations was directly demonstrated through tACS, where driving activity at the individual alpha frequency enhanced vection intensity [24].

Evidence concerning other frequency bands and connectivity measures is emerging but currently less extensive. Decreases in beta (β) power sometimes precede or accompany alpha modulations [20], while delta (δ) and theta (θ) activity show associations with vection strength [20] or VIMS susceptibility [12], [25]. Connectivity analyses, though sparse, have pointed towards altered theta-band phase synchronization related to VIMS [25] and increased cortico-cerebellar coherence during vection involving vestibular input [23], highlighting a potential role for the cerebellum in modulating self-motion perception.

The reviewed studies confirm that stimulus characteristics significantly influence both vection and its EEG correlates. Factors such as the type of optic flow (e.g., radial vs. rotational), field-of-view size, and the specific visual field region stimulated (central vs. peripheral) impact the strength of the illusion and associated neural responses [15], [11], [21]. Similarly, manipulating visuo-vestibular conflict through body posture affects brain oscillations [22]. Individual differences beyond handedness and VIMS, such as age, have also been explored, although significant age effects on core vection ERPs were not found in one study [14]. The finding that vection-inducing backgrounds modulate ERPs related to a concurrent visual task [19] indicates that the processing underlying vection consumes attentional resources, impacting performance on other tasks.

Early event-related potentials (ERPs), specifically the visual P1, N2, and P2 components occurring within approximately 250 ms of motion onset, consistently reflect the differential processing of vection-inducing versus non-inducing visual stimuli [15], [14], [21], [16]. The amplitude modulation of these components, particularly the N2, appears sensitive to the spatial configuration of motion (e.g., central vs. peripheral stimulation [21], [15]) and stimulus coherence [14], suggesting they index pre-perceptual stages of sensory evidence integration relevant for self-motion computation. Notably, the N2 amplitude was also found to be larger in individuals more susceptible to visually induced motion sickness (VIMS) [25], hinting at differences in early sensory processing or conflict detection in these populations.

An observation emerging from this review is that, while ERPs effectively distinguish between different stimulus conditions designed to elicit varying levels of vection, find-

ings regarding direct, quantitative correlations between raw ERP components (e.g., amplitude, latency) and the subjective intensity of the vection perceived on a trial-by-trial basis are less consistently reported compared to oscillatory power. This contrasts with oscillatory power findings [20], [23], which appear to correlate more reliably with vection strength. While changes or differences in ERPs might correlate with individual traits related to vection or VIMS susceptibility [25], the current evidence suggests that early ERPs may primarily reflect the brain’s initial processing of the potential for vection inherent in the visual stimulus, rather than the strength of the resulting subjective experience itself [15], [14]. Understanding the precise conditions and components under which ERPs might track subjective vection intensity remains an important open question for future research.

Moreover, certain methodological trends present challenges for synthesis. Sample sizes are often small, potentially limiting statistical power and generalizability. The heterogeneity in visual stimuli (displays, motion parameters), vection measurement techniques (subjective scales, onset latencies), and EEG processing pipelines (referencing, filtering, analysis methods) complicates direct comparisons across studies. Although source localization techniques are beginning to be employed [16], [12], their application remains limited, leaving the precise cortical and subcortical generators of the observed scalp potentials largely inferred.

EEG provides valuable insights into the neural dynamics of vection. Modulations in the alpha band and early visual ERP components stand out as relatively consistent markers, reflecting inhibitory control processes and sensory integration, respectively. While oscillatory activity, particularly alpha power, often shows correlations with subjective vection intensity, findings for early EPs are less consistent in directly tracking subjective strength, suggesting they may be more reliably tied to the processing of stimulus properties predictive of vection. Emerging evidence highlights contributions from other frequency bands, connectivity patterns, and the cerebellum. While methodological variability warrants caution, the existing literature strongly supports the utility of EEG for objectively investigating the temporal course and neural underpinnings of illusory self-motion perception.

V. Conclusion

This review systematically examined the current body of literature employing electroencephalography to investigate the neural correlates of visually induced self-motion (vection). The converging evidence from sixteen studies highlights consistent patterns of brain activity associated with this phenomenon, primarily involving modulations of alpha-band oscillations and early visual event-related potentials. Alpha activity, particularly over posterior scalp regions, appears dynamically regulated, with transient decreases potentially marking the onset of sensory reweighting and sustained increases reflecting inhibitory

processes during established vection. Concurrently, early ERP components such as the P1, N2, and P2 index the initial cortical processing and integration of motion cues critical for triggering the illusion.

The reviewed literature demonstrates that these EEG markers are sensitive to various factors, including the specific characteristics of the visual stimulus, the nature of the induced sensory conflict, task demands, and individual traits like handedness and motion sickness susceptibility. While methodological diversity across studies necessitates careful interpretation, EEG has unequivocally proven to be a valuable tool, offering the temporal resolution required to highlight the rapid neural dynamics underlying the emergence and maintenance of vection. Despite limitations related to sample sizes and standardization, the field provides a growing foundation for understanding the cortical mechanisms of self-motion perception and visuo-vestibular interaction, paving the way for future investigations into objective markers and potential applications in virtual reality and clinical assessment.

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