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Epileptic Disorders

INVITED REVIEW

Localizing value of disturbances of self-integration, depersonalization, and forced thinking: A systematic review

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Abstract

We performed a systematic review of the localizational value of disturbances of self-integration, depersonalization and forced thinking in focal epilepsy with the aim to summarize the state-of-the-art anatomo-clinical correlations in the field and help guide interpretation of ictal semiology within the framework of pre-surgical evaluation. The review was performed using a PRISMA- and QUADAS2-based approach. Three separate PubMed and EMBASE searches were undertaken using the keywords self-integration, depersonalization and forced thinking, along with synonyms, in combination with terms to identify epileptogenic zone as defined by surgical outcome, MRI-findings or intracranially recorded EEG. Studies published in peer-reviewed journals with an abstract available, limited to English, French, German, Spanish, or Italian were included for review. Abstracts from scientific meetings were included if precise data on semiology in addition to either localization or surgical outcome was presented. Cases were regarded as eligible if data informing on anatomo-clinical correlations were sufficient to allow determination of an epileptogenic zone and evaluate its level of confidence. For disturbances of self-integration, the search identified 18 publications containing 23 eligible cases, with 10 additional cases identified in the literature. For depersonalization, a single case from a two patient study fulfilled inclusion criteria. For forced thinking, the search identified two publications containing four eligible cases, with six additional cases identified through literature searches. The retrieved cases suggest that disturbances of self-integration often reflect an epileptogenic zone centered around the temporoparietal region, where neighboring areas in the parietal lobe, the posterior insula, and likely depending on the type of disturbance also the adjoining occipital lobe, the anterior and middle cingulum, premotor and supplementary motor in addition to medial temporal structures could be involved. When present, lateralized symptomatology reflects a contralateral focus. Depersonalization, as a localizing

ictal phenomenon was quite elusive. Forced thinking either pointed to premotor frontal or temporal epileptogenic zones. Currently, outlined epileptogenic zones of ictal disturbances of self-integration and forced thinking are quite widespread and should be regarded with a low-to-moderate degree of reliability. A focus on such rarer ictal phenomena, in combination with improved imaging techniques and increased use of SEEG, will hopefully lead to an accumulation of cases with better defined epileptogenic zones.

KEYWORDS

anatomo-clinical correlation, depersonalization, disturbances of self-integration, epilepsy surgery, focal epilepsy, forced thinking, ictal semiology, systematic review

1 | INTRODUCTION

Ictal disturbances of self-integration (DSIs), depersonalization, and forced thinking represent rare psychical, experiential, or cognitive semiologic phenomena that exemplify many of the factors that make subjective semiological traits a diagnostical challenge.[1] Still, if correctly identified, they may have a localizing value in the setting of focal epilepsies.[2] The DSIs encompass a variety of different symptoms that collectively can be understood as ictal changes in the experience of being a bodily self, either through an infliction on one of the different aspects that constitute this experience, or through a disrupted synthesis of these aspects. These aspects have been described as falling within five partly overlapping categories: the experience of owning a body; the perception of this body and its parts; of controlling its actions; of occupying a localization in space; and finally, by having a particular, first-person perspective.[3] As these aspects may be affected to different extents and involve either parts of the body, or the body as whole, DSIs constitute a multitude of related symptomatologies. Asomatognosia (illusional limb loss), alien hand experiences (including somatoparaphrenia), limb movement, and positional illusions all represent DSIs affecting parts of the body. Autoscopic phenomena (including autoscopic hallucinations, where one sees a visual representation of one-self outside the body; heautoscopy, where one sees a more or less defined visual representation of one-self outside the body while at the same time partly identifies and perceives a shared localization with the observed body; and lastly out-of-body experience (OBE) where one takes on a removed perspective, perceiving oneself from the outside) represent DSIs related to the whole body or representations of it.[4] Furthermore, the DSIs that simultaneously affect many of the abovementioned dimensions (Multidimensional DSIs) stand out as readily discernable entities. Others may affect only one or a few of these dimensions (paucidimensional DSIs) and can phenomenologically be very similar to, and probably even overlap with various sensory illusional phenomena, both somatosensory (tactile and proprioceptive), visual (complex visual), and vestibular (whole body levitational).

Key Points

- The localizing value of Ictal disturbances of self-integration (DSIs) and forced thinking should be regarded with a low to moderate degree of reliability.
- DSIs often reflect an EZ centered around the temporoparietal region, where additional areas, however, also could be responsible, depending on the type of disturbance.
- Forced thinking often reflects either premotor frontal or a temporal EZ.
- Depersonalization as a localizing ictal phenomenon appears quite elusive and may be better regarded as an aspect occurring alongside other, likely better localizing semiological traits.

Depersonalization is defined within the DSM–5 classification system as "experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions" where it constitutes a part of depersonalization/derealization disorder.[5] The symptom has however also been associated with epileptic seizures already since the last part of the 1800s through the works of Griesinger and Hughlings Jackson.[6]

The last of these terms, forced thinking, was described by Penfield in 1946 as "a feeling of compulsion to think or the occurrence of unusual thoughts."[7] A more recent definition proposed is the "recurrent intrusive thoughts, ideas

or crowding of the mind,"[8] which perhaps brings forward the "forced" aspect more clearly.

Here, we present a systematic review of the localizing value of the DSIs, depersonalization and forced thinking in focal epilepsy, and summarize the state-of-the-art anatomo-clinical correlations. Increased knowledge about and recognition of these more rarely reported symptoms may help guide the interpretation of ictal semiology within the framework of pre-surgical evaluation.

2 | METHODS

2.1 | Search method and eligibility criteria

We conducted a systematic review of the published evidence and report its results according to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) statement.[9] Our systematic review is based on three separate searches aimed at ictal DSIs, depersonalization and forced thinking. To ensure the inclusion of synonyms and furthermore take account of the ambiguity and inherent possibility of overlap with related semiological traits, we included various additional terms for all three searches. The following search string was used for each of the three pubmed searches, initiated by ("Epilepsy"[Mesh] OR "Seizures"[Mesh] OR epilep*[Title/Abstract] OR seizure*[Title/Abstract]) AND ("Surgical Procedures, Operative"[Mesh:NoExp] OR "Neurosurgical Procedures"[Mesh] OR "Special ties, Surgical"[Mesh:NoExp] OR "Neurosurgery" [Mesh] OR "General Surgery"[Mesh] OR "Electro encephalography"[Mesh] OR "surgery" [Subheading] OR surg*[Title/Abstract] OR neurosurg*[Title/Abstract] OR operative*[Title/Abstract] OR electroencephalogra*[Title/Abstract] OR EEG [Title/Abstract] OR stereoelectroence phalogra*[Title/Abstract] OR stereoencephalogra*[Title/Abstract] OR SEEG OR (electric*[Title/Abstract] OR electro[Title/Abstract]) AND encephalograph*[Title/Abstract]) AND, for A) Disturbances of self-integration; ((somatognostic OR somatognosia) OR (asomatognosia) OR (somatoparaphren*)) OR (("body movement" OR "hand movement" OR "limb movement" OR "arm movement" OR "leg movement" OR "limb loss" OR "loss of limb") AND (illusion OR delusion)) OR ("phantom hand" OR "phantom leg" OR "phantom limb") OR ("alien hand" OR "alien limb") OR (autoscop* OR heautoscop* OR "out-of-body") OR ("self- integration" OR self-integration)), for B) Depersonalization; (("Depersonalization"[Mesh]) OR (depersonalization*[Title/Abstract] OR depersonalisation* [Title/Abstract] OR depersonification*[Title/Abstract] OR derealisation*[Title/Abstract] OR derealization*[Title/Abstract])) and lastly for Forced Thinking; ((forced[Title/Abstract] OR intrusive[Title/Abstract]) AND (thinking[Title/Abstract] OR thought*[Title/Abstract] OR agency[Title/Abstract] OR action*[Title/Abstract])) OR hypercognit*[Title/Abstract] OR "hyper cognitive"[Title/Abstract])). Details for similar built EMBASE searches are provided in Data S1. The date of last complete search was January 29th, 2024.

We primarily selected studies published as papers in peer-reviewed journals with an abstract available, limited to English, French, German, Spanish, or Italian. Abstracts from scientific meetings were included if precise data on semiology, in addition to either localization or surgical area and outcome, was presented. Two independent reviewers screened titles, abstracts, and full-text articles to determine eligibility. A third reviewer resolved disagreements at the full-text screening phase and the data abstraction phase. Due to the scarcity of published eligible papers retrieved in the systematic searches, two independent reviewers screened publications referenced by the literature on the subject matter for relevant cases.

As we expected a very low total number of cases, we included both cases from case series and single case publications. Cases where DSIs, depersonalization, or forced thinking were seen upon stimulation only and not reported as part of spontaneous seizure symptomatology, were not included, in accordance with the validity criteria guiding this review.[10]

2.2 | Data extraction

For each identified publication, we extracted all patients with informative data regarding the topic of this review, with special attention to data on anatomo-clinical correlations. Although not easily applicable to the combination of case reports, small case series and literature based identification of single patients, nor to the evaluation of many-tiered anatomo-clinical associations, we performed a QUADAS2-guided assessment regarding both patient enrolment and the assessment of symptoms.[11]

2.3 | Reliability of the reference standard

For each identified case, we assessed our level of confidence in the reported epileptogenic zone (EZ) according to a recently developed method.[10] Based on the findings from MRI, intracerebral EEG (iEEG) and postoperative outcome, four levels of evidence were distinguished:

- "very high" confidence in the reported EZ for patients with Engel class IA[12] after at least one year of postoperative follow-up;

- "high" confidence in the reported EZ for patients with either: (i) well-delineated focal lesion suspected to represent at least part of the EZ (according to the authors of the publication), (ii) well-delineated EZ according to all available iEEG data (according to the authors of the publication), or (iii) an Engel class I (but not specified IA) after at least one year of postoperative follow-up;
- "moderate" confidence in the reported EZ for patients with MRI signs of hippocampal sclerosis or atrophy suspected to be at least part of the EZ;
- "low" confidence in the reported EZ for patients with: (i) normal, multilobar, multifocal, or poorly delineated lesion on MRI; (ii) a poorly delineated EZ according to all available iEEG data (according to the authors of the publication), or (iii) an Engel class II-IV postoperative outcome provided the entire EZ has been entirely removed. Patients who failed surgery, with suspected EZ not completely removed would not be considered for grading.

If several of the above items were available and pointed to different structures, those associated with postoperative outcome prevailed over iEEG conclusions and MRI findings, while iEEG conclusion would prevail over MRI findings. Epileptogenic zones were visualized through Surf Ice (https://www.nitrc.org/plugins/mwiki/index.php/surfice:MainPage) as confidence level weighted surface renderings of cortical parcellations according to the Destrieux-atlas,[13] modified manually by published image data where possible.

Due to the combination of low number of studies with large heterogeneity in study design, quantitative synthesis (meta-analysis) was in general not possible. Statistical evaluations regarding laterality were done using Fisher's exact test, while evaluations regarding dominance or non-dominance were done using a two-tailed sign-test.

3 | OVERALL SUMMARY OF EVIDENCE

Using the GRADE system and its principle domains of risk of bias, consistency, directness, and precision as applicable to diagnostic tests,[14] the summary of evidence was eventually assessed according to the following adapted categories of reliability:

Very low reliability: The true possible epileptogenic zone is probably markedly different from that presented;
Low reliability: The true possible epileptogenic zone might be markedly different from that presented;
Moderate reliability: The authors believe that the true possible epileptogenic zone is probably close to that presented;
High reliability The authors have a lot of confidence that the possible epileptogenic zone is similar to thatpresented

4 | RESULTS

4.1 | Disturbances of self-integration

The PRISMA flow diagram for the search for DSIs (Figure 1) shows that of the 186 citations found and screened for eligibility, 82 records could be excluded immediately (see Data S2 for complete list of records). Of the remaining records, two were not obtainable, 38 were excluded upon reading abstracts and four due to language limitations, leaving 60 articles to be reviewed in full. Of these, 42 had inadequate either diagnostical, anatomical, or semiological information. The 18 publications left contained 23 cases available for inclusion

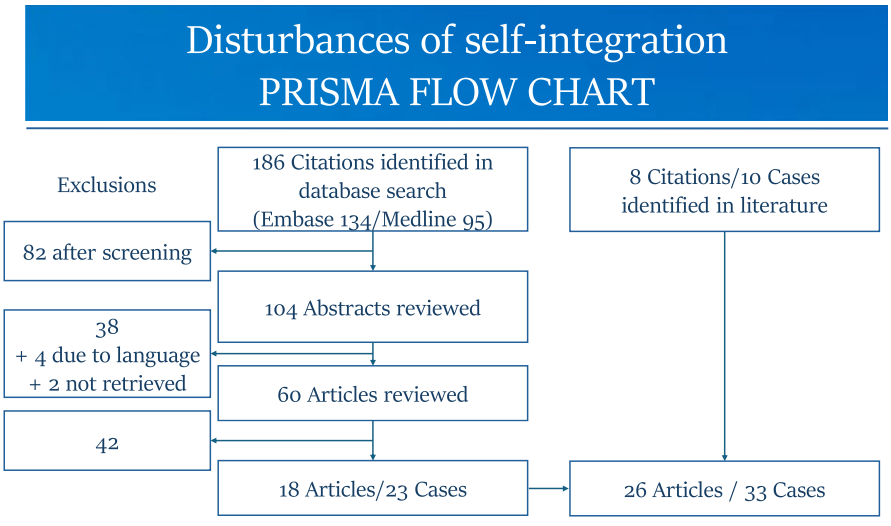

FIGURE 1 Preferred reporting items for systematic review and meta-analysis (PRISMA) flowchart for search on disturbances of self-integration.

in the evidence synthesis. Additionally, search through citations identified 8 articles with 10 cases that fulfilled criteria for inclusion.

In total, we identified 33 eligible cases with DSI. One patient experienced three distinct DSI symptoms, whereas four patients experienced two. All other patients only experienced one DSI symptom. The cases were derived from 26 heterogeneous publications, either small, mainly retrospective, case series specifically devoted to parts of this subject matter or closely related issues (9 studies, 15 patients) or single case studies (14 patients). The last four cases were derived from epilepsy case series where inclusion was defined by an anatomical region in combination with either pathology (tumor),[15] or with examination technique (SEEG).[16,17] Two of the studies identified were classified as case control studies; however, the case–control comparisons did not relate directly to aspects relevant to this review,[18,19] hence reducing some of the bias-risk associated with such studies. All included cases are shown in Table 1.

Nineteen of the 33 cases experienced a DSI affecting only parts of the body (Body part disintegration—BPD). Although most of the patient experiences were highly idiosyncratic, some categorization of the cases appeared warranted. Eleven of the partial disturbances could be regarded as simple, paucidimensional disturbances. Five of these represented asomatognosia with the experience of loss of a limb or detachment of head/face, four had positional, movimental or deformational illusions affecting a body part, and two had micro- or macrosomatognosia, experiencing a change in size of a body part. Nine were however more complex, multidimensional, where a body part was experienced as foreign, strange and/or outside personal control, often with somatoparaphrenic traits. Figure 2A displays epileptogenic zones of BPD-cases according to the evidence confidence grading outlined in Methods. Accordingly both BPD-groups could point to foci in the posterior superior and inferior parietal lobules, the intraparietal sulcus and in some instances the post-central and even precentral gyri. Symptoms with a higher degree of complexity could however also point to the temporoparietal junction (TPJ), the supplementary sensorimotor area (SMA), the middle cingulate gyrus and even anteromedial temporal structures. Interestingly, 16 of the 19 cases showed some type of laterality in the symptomatology, all pointing to a contralateral focus with a high degree of statistical significance (Fisher's exact test $p = .0002$). Thirteen cases had information on hand dominance, where six represented a non-dominant and seven a dominant epileptogenic focus.

Seventeen of the 33 cases experienced DSIs affecting the whole body (WBD). Nine cases had out-of-body experiences (OBE), five experienced autoscopy, two heautoscopy, one case the presence of a "double" and lastly two cases felt illusional levitation (two patients experienced two symptom types). Figure 2B,C display the epileptogenic zones of WBD-cases according to the evidence confidence grading outlined in Methods. Patients with both OBE and autoscopy mostly included the TPJ as a possible focus. However, whereas the former patient group also included the posterior insula and anterior/anterior-middle cingulate gyrus in the possible EZs, the latter in one instance included the posterior insula, and in two either lateral or medial occipital/parietal lobe structures. The two patients with heautoscopy had combined anteromedial temporal and insular foci, whereas the two with levitational experiences pointed to foci in the superior parietal lobule, the precuneus, and the marginal ramus of the cingulate sulcus. In the four patients that experienced a laterality in their WBD-symptoms (for instance a left sided felt presence or autoscopic hallucination[20] or heautoscopy with a person occurring in the right hemifield[18]), it consistently pointed to the contralateral side, however without reaching significance (Fisher's exact test $p = .25$). Hand dominance could be determined in 14 patients with WBD, five with an epileptogenic focus in the dominant, and nine in the non-dominant hemisphere (Two tail sign test $p = .4240$). It should be noted that the WBD-group was heterogeneous, and whereas patients experiencing OBE represented six non-dominant and three dominant foci, all autoscopy cases had a non-dominant focus, while both identified heautoscopy cases had a dominant focus.

Table 1 also shows that in 23 of the patients ictal DSIs could be an initial symptom, whereas in five patients, this always followed other semiology. Two patients lacked information regarding semiological sequential development. In some cases, the DSI symptoms appeared to be congruent with additionally occurring symptoms, where they all possibly were reflecting related symptomatic zones, often close to the central region. In other cases, the presence of DSI appeared to give information very distinct from the other semiology, exemplified by a patient where OBE was occurring alongside seemingly typical temporal seizures initiated by epigastric aura, where an angular gyral xanthoastrocytoma resection gave an Engel IA outcome.[21] Finally, in other cases, additional symptomatology could be taken as indicating an extension of the EZ to areas neighboring the area probably responsible for the DSI-symptom per se, as might have been the case with a patient experiencing either initial OBE *or* visual blurring/deformations before loss of consciousness (LOC), where removal of a dysembryoblastic neuroepithelial tumor in the temporoparietal junction and neighboring lateral occipital gyrus resulted in Engel IA outcome.[22]

TABLE 1 Cases with disturbances of self-integration and depersonalization sorted according to symptom class and epileptogenic zone confidence level.

#	Source	Symptom	Sequence	Hnd	Localizations	EZ confidence	EZ determinant	Engel	Study type
Disturbance of self-integration									
1	Hao et al.[39] #3	BPD—S (ASG—R)	I	R	InsA/pIFG/InsP/ParOp—L	High	RS-fcd/SEEG/stim	I/NA	RCS
2	Nishibayashi et al.[40]	BPD—S (ASG—R)	I	R	SPL—L	High	RS-fcd/iEEG/stim/MRI	IA/NA	SC
3	Bartolomei et al.[17] #5	BPD—S (ASG—L)	I	NA	SPL—R	High	RS-fcd/SEEG	IB/3 years	RCS
4	So and Schauble[41]	BPD—S (ASG—L)	I	NA	SPL—R	High	iEEG/stim	NA	SC
5	Hasegawa et al.[42]	BPD—S (ASG—Head)	I	R	SMG/AG—R	High	RS-fcd/MRI/SEEG	IA/NA	SC
6	Yang et al.[16] #7	BPD—S (mov/pos)	I	NA	ParCntLob/Prec/MargRam—L	Very high	RS-em/SEEG/MRI	IA/26 mo	RCS
7	Ting et al.[43]	BPD—S (mov/pos-R)	I	R	IPS—L	High	MRI-vsc	NA	SC
3	Bartolomei et al.[17] #5	BPD—S (deform—L)	I	NA	SPL—R	High	RS-fcd/SEEG	IB/3 yrs	RCS
8	Gayoso Garcia et al.[44]	BPD—S (mov/pos—L)	I	L	PreCSul, PreCGyr, Csul, PoCGyr—R	Low	RS-lgt/stim/MRI	II/NA	SC
9	Bartolomei et al.[17] #7	BPD—S (MiSG—L)	I	NA	SMG—R	Very high	RS-fcd/SEEG	IA/3 yrs	RCS
10	Maillard et al.[20] #1	BPD—S (MaSG)	I	R	IPL—R	High	MRI-fcd	NA	RCS
11	Wang et al.[45]	BPD—C (AL—L)	I	L	SPL—R	Very high	RS-lgt/MRI	IA/1.5 yrs	RCS
12	Leiguarda et al.[46] #2	BPD—C (AL—L)	I	R	SMA—R	Very high	RS-lym	IA/3 yrs	RCS
13	Brázdil et al.[47]	BPD—C (AL—L)	I	R	aMCG—R	High	MRI-vsc	NA	NA
14	Boesebeck and Ebner[48] #1	BPD—C (AL—R)	I	NA	AMT—L	High	RS-hs/MRI	IA/NA	RCS
15	Boesebeck and Ebner[48] #2	BPD—C (AL—L)	I	NA	pMCG—R	High	MRI-lgt	NA	RCS
16	Carrazana et al.[49]	BPD—C (AL—L)	F	R	SMA/pMCG—R	High	MRI-lgt	NA	SC
17	Heydrich et al.[50]	BPD—C (Phantom—R)	I	R	TPJ—L	High	MRI-fcd	NA	SC
18	Heydrich et al.[23] #1	BPD—C (Invasion—L)	F	L	IPS—R	High	MRI-avm	NA	RCS
19	Heydrich et al.[51]	BPD—C (SPP—L)	F	R	AG/hippocampus—R	Moderate	MRI-vsc/hs	NA	SC
9	Bartolomei et al.[17] #7	WBD—Aut	I	NA	SMG—R	Very high	RS-fcd/SEEG	IA/3 yrs	RCS
20	Fonti et al.[52]	WBD—Aut	I	L	SMG/InsP—L	High	RS-pmg/SEEG/MRI	IA/9 mo	SC
10	Maillard et al.[20] #1	WBD—Aut	I	R	IPL—R	High	RS-fcd/MRI	NA	RCS
21	Maillard et al.[20] #2	WBD—Aut	I	R	IPL/SOG/pSTG—R	Low	MRI-vsc	NA	RCS
22	Maillard et al.[20] #3	WBD—Dbl/Aut	I/F	R	SPL/IPL/SOG/Prec/Cun/LinGyr/ClcSlc—R	Low	MRI-lgt	NA	RCS

(Continues)

TABLE 1 (Continued)

#	Source	Symptom	Sequence	Hnd	Localizations	EZ confidence	EZ determinant	Engel	Study type
23	Heydrich and Blanke[18] #1	WBD—Heaut	F	R	AMT/Ins—L	High	MRI-lgt	NA	RCC
24	Heydrich and Blanke[18] #2	WBD—Heaut	F	R	Hippocampus/(Ins)*—L	High	iEEG/MRI-hs	NA	RCC
25	Salanova et al.[15] #11	WBD—Lev	I	NA	ParCntLob/Prec/MargRam/SPL—L	Very high	RS-mng	IA/NA	RCS
26	Gibbs et al.[53]	WBD—Lev	I	NA	Prec/MargRam—R	High	SEEG/stim/MRI-fcd	NA	SC
27	Fang et al.[33]	WBD-OBE	I	R	TPJ/vPoCGyr—R	Very high	RS-fcd/iEEG	IA/4.5 yrs	SC
28	Brandt et al.[21]	WBD-OBE	I (part)	R	AG—R	Very high	RS-lgt/MRI	IA/5 yrs	SC
29	Blanke et al.[22] #1	WBD-OBE	I (part)	R	AG/pSTG/pMTG/MOG—R	Very high	RS-lgt/MRI	IA/5 yrs	RCS
30	Alwaki et al.[54]	WBD-OBE	I	R	ACG/aMCG/(InsP)*—L	High	RS-fcd/SEEG/stim/MRI	I/NA	SC
31	Hoepner et al.[19] #1	WBD-OBE	NA	NA	InsP—L	High	MRI-fcd	NA	PCC
32	Hoepner et al.[19] #2	WBD-OBE	I (50%)	R	PreCGyr/Csul/PoCGyr/TPJ—L	High	MRI-pmg	NA	PCC
8	Maillard et al.[20] #1	WBD-OBE	I	R	IPL—R	High	RS-fcd/MRI	NA	RCS
19	Heydrich et al.[51]	WBD-OBE	F	R	AG/hippocampus—R	Moderate	MRI-vsc/hs	NA	SC
33	Blanke et al.[22] #2	WBD-OBE	I	R	AG/pSTG/vPoCGyr—L	Low	iEEG/MRI-fcd	II	RCS
Depersonalization									
1	Heydrich et al.[23] #2	Depersonalization	I	L	pSFG/pMFG/SMA—R	High	RS/iEEG	Ia/4 mo	RCS

Abbreviations: ACG, anterior cingulate gyrus; AG, Angular gyrus; AL, Alien Limb; aMCG, anterior part of middle cingulate gyrus; AMT, anteromedial temporal structures; ASG, Asomatognosia; Aut, autoscopic illusion; avm, arteriovenous malformation; C, Complex; ClclSlc, Calcarine sulcus; Csul, central sulcus; Cun, Cuneus; Dbl, Presence of a "double"; deform, deformational; em, encephalomalacia; F, following; fcd, focal cortical dysplasia; Heaut, Heautoscopy; Hnd, Handedness; hs, hippocampal sclerosis; I, Initial; iEEG, non-stereoelectroencephalographic invasive EEG; InsA, Anterior Insula; InsP, Posterior Insula; IPL, Inferior parietal lobulus; IPS, intraparietal sulcus; lev, levitational; lgt, low grade brain tumor; LinGyr, Lingual gyrus; lym, lymphoma; MargRam, Marginal ramus of cingulate sulcus; MaSG, Macrosomatognosia; MFG, Middle frontal gyrus; MiSG, Microsomatognosia; mng, meningeoma; MOG, Middle occipital gyrus; mov/pos, movimental/positional; MRI, magnetic resonance imaging lesion; MTG, Middle temporal gyrus; OBE, Out-of-Body Experience; p, posterior; ParCntLob, Paracentral lobule; PCC, Prospective case control study; PCS, Prospective case series; pMCG, posterior part of middle cingulate gyrus; pmg, polymicrogyria; PoCGyr, Post central gyrus; Prec, Precuneus; PreCGyr, Precentral gyrus; PreCSul, Precentral sulcus; RCC, Retrospective case control study; RCS, Retrospective case series; RS, resective surgery; S, simple; SC, Single case study; SEEG, stereoelectroencephalography; SFG, Superior frontal gyrus; SMA, supplementary motor area; SMG, Supramarginal gyrus; SOG, Superior occipital gyrus; SPL, Superior parietal lobule; SPP, Somatoparaphrenia; STG, Superior temporal gyrus; stim, intracranial stimulation; TPJ, temporoparietal junction; v, ventral; vsc, vascular lesion.

aLocations in parenthesis representing possible symptomatic zone separate from EZ, as identified through invasive EEG.

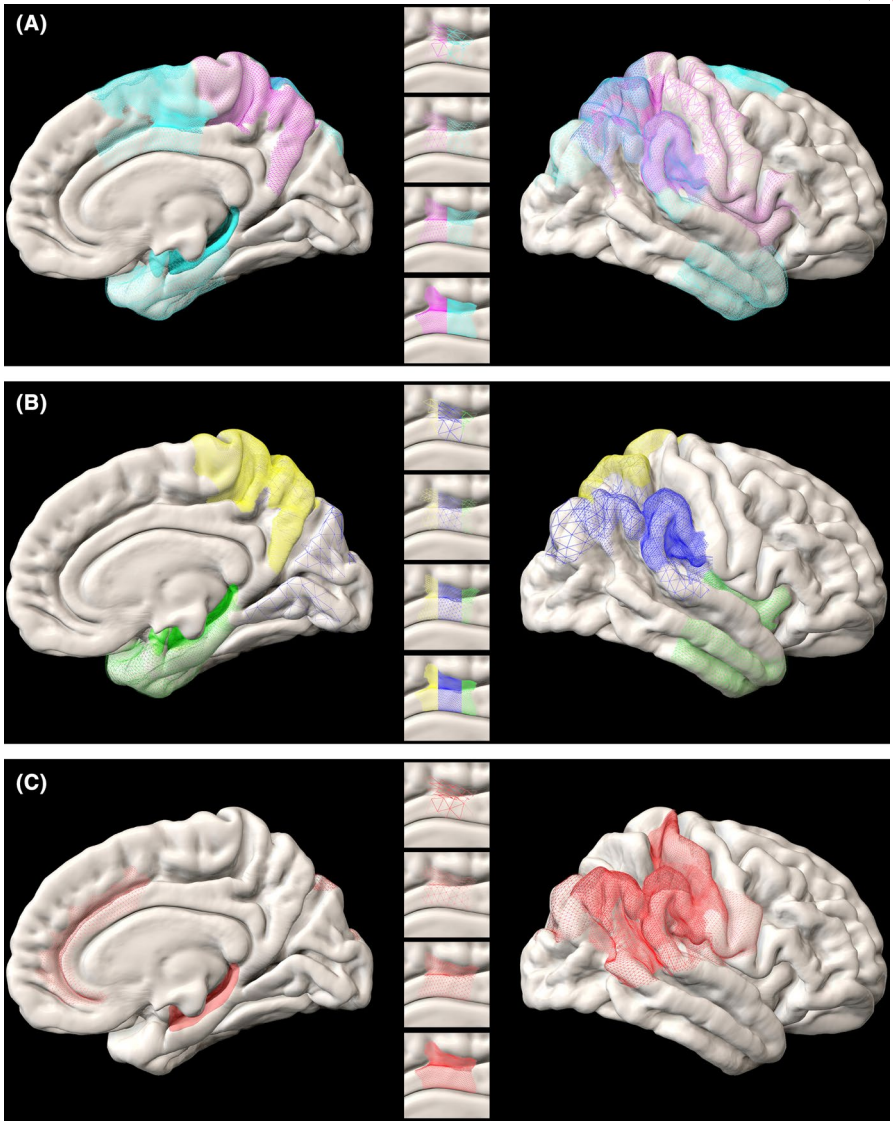

FIGURE 2 (A) Overlaying EZs of patients with simple (Purple) and complex (Cyan) body part disintegration, according to level of confidence. Areas with epileptogenic zones of patients with both BPD-types are depicted as a combination of the two colors. The middle panel shows the surface rendering patterns used to indicate neocortical EZ-confidence level, from top to bottom: Low, Moderate, High and Very High, with an intensity reflecting one case in the given area (Figure S1A shows the corresponding figure with cases of high and very high confidence level only). For simplicity, EZs are displayed as right sided, on a standardized right hemisphere MNI-152 brain. (B) Overlaying EZs of patients with autoscopy (Blue), heautoscopy (Green), and levitational (Yellow) whole body disintegration, according to level of confidence. The middle panel shows the surface rendering patterns used to indicate neocortical EZ-confidence level, from top to bottom: Low, Moderate, High and Very High, with an intensity reflecting one case in the given area (Figure S1B shows the corresponding figure with cases of high and very high confidence level only). For simplicity, EZs are displayed as right sided, on a standardized right hemisphere MNI-152 brain. (C) Overlaying EZs of patients with OBE according to level of confidence. The middle panel shows the surface rendering patterns used to indicate neocortical EZ-confidence level, from top to bottom: Low, Moderate, High and Very High, with an intensity reflecting one case in the given area (Figure S1C shows the corresponding figure with cases of high and very high confidence level only). For simplicity, EZs are displayed as right sided, on a standardized right hemisphere MNI-152 brain.

4.2 | Depersonalization

The PRISMA flow diagram for the depersonalization search (Figure 3) shows that of the 114 citations found, 47 were deemed relevant for retrieval (see Data S3 for complete list of records). Of these, two were not obtainable, whereas six were excluded due to language limitations. Twenty-four were excluded after reading abstracts, whereas 15 articles were reviewed in full text. Of these, only a single case from a two case study fulfilled the criteria for inclusion. This case presented an EZ in the SMA and adjoining part of the superior frontal gyrus with a

high level of confidence (Table 1).[23] It should be added that the second patient of this study and patients from two other studies identified by our search on depersonalization,[21,22] were also identified and included in the material on DSIs. However, the described symptomatology here did not contain any explicit mention of depersonalization, nor did symptoms differ from the other DSI patients, and they were therefore only included in this latter group. The cases in the other identified studies were not included due to lack of a well-defined EZ in terms of both anatomy and confidence level, an inadequate definition of a depersonalization of ictal epileptic origin or most commonly a combination of the two.

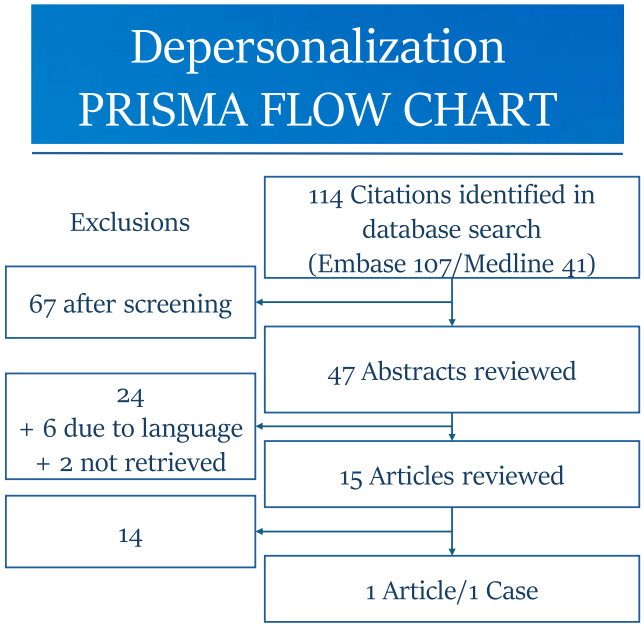

FIGURE 3 Preferred reporting items for systematic review and meta-analysis (PRISMA) flowchart for search on depersonalization.

4.3 | Forced thinking

The PRISMA flow diagram for the search on forced thinking (Figure 4) shows that of 31 citations, 22 were relevant for retrieval (see Data S4 for complete list of records). Twelve publications were deemed ineligible for inclusion upon reading the abstract, whereas eight were deemed ineligible after evaluation of the full manuscript. Two publications were found eligible, containing four cases. Search through relevant literature added five publications, with six cases.

Three of the in total seven publications (six of 10 cases) represented case series devoted to either forced thinking specifically or to a group of ictal phenomena where forced thinking was included (experiential or psychic seizures), and furthermore defined inclusion according to anatomical region, one left-sided frontal (three patients) and two temporal (three patients). Another three publications represented case series with inclusion defined by various epilepsy related criteria, in which one case experienced forced thinking. The last publication was a single case report, identified by our systematic search. Table 2 displays a summary of the included cases with forced thinking whereas Figure 5 displays their EZs according to the evidence confidence grading outlined in Methods. Accordingly six patients had a temporal focus of which three had a very high or high level of confidence (two of which included medial temporal structures). Four had a frontal focus of which two had a high level of confidence (one frontopolar, one middle frontal gyrus).

4.4 | GRADE-reliability evaluation

Evaluating the principle domains of GRADE-reliability points to both weaknesses and strength of the data

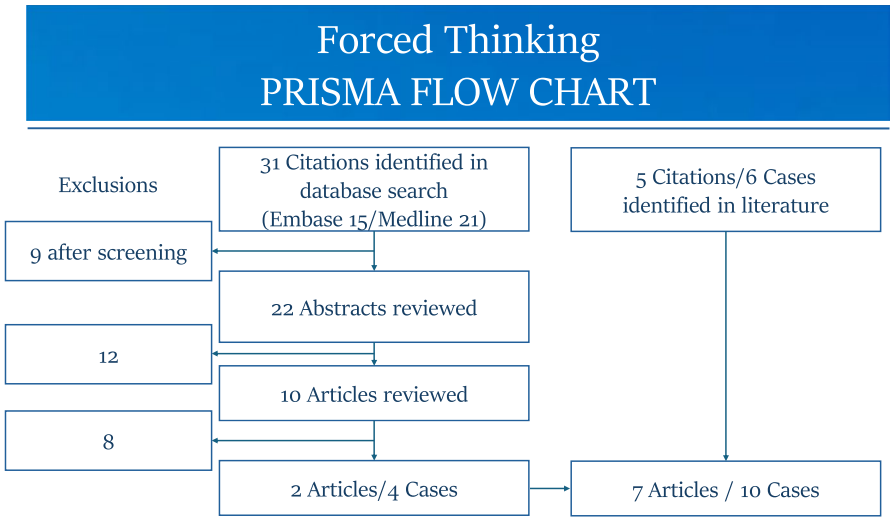

FIGURE 4 Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) flowchart for search on forced thinking.

TABLE 2 Cases with forced thinking sorted according to symptom class and epileptogenic zone confidence level.

Source	Content	Hnd	Localizations	EZ confidence	EZ determinant	Engel	Study type
Cho et al.[24]	"Forgot something i should do"	Amb	ATM—L	Very High	RS-hs/MRI	IA/4.6 years	SC
Siegel et al.[25] #12	Unspecified	NA	Neocortical temporal—R	High	RS-fcd	I/NA	RCS
Mendez et al.[8] #1	"Tell me yes"	R	MFG—L	High	MRI-lgt	I/NA	RCS
Mendez et al.[8] #3	"I need to grab something"	NA	Frontopolar—L	High	MRI-vsc	NA	RCS
Gloor et al.[26] #2	"Thinking of Philomène and Jacuot"	NA	Hippocampus/amygdala—L	High	SEEG/stim	NA	RCS
Di Bonaventura et al.[27] #29	Unspecified	NA	Temporal lobe atrophy—R	Low	MRI[a]	NA	RCS
Thomas[28] #2	Unspecified	NA	Frontopolar—R	Low	MRI-ras[a]	NA	PCC
Mendez et al.[8] #2	"Why don't you tell them how you feel/have a seizure"	R	Frontal anterior—L	Low	MRI-lgt	IA/6 yrs[a]	RCS
Karagulla and Robertson[29] #1	"Two thoughts coming together"	NA	MTG—R	Low	Stim[a]	NA	RCS
Karagulla and Robertson[29] #2	"Rythmic thought"	NA	MTG—L	Low	Stim[a]	NA	RCS

Abbreviations: AMTR, Anteromedial temporal resection; fcd, focal cortical dysplasia; hs, hippocampal sclerosis; iEEG, non-stereoelectroencephalographic invasive EEG; lgt, low grade brain tumor; MFG, Middle frontal gyrus; MRI, magnetic resonance imaging lesion; MTG, Middle temporal gyrus; RCS, Retrospective case series; RS, resective surgery; SC, Single case study; SEEG, stereoelectroencephalography; stim, intracranial stimulation; vsc, vascular lesion.
aMRI/Engel IA/stimulation induced habitual aura could have indicated high confidence level, but diffuse lesions, large resection area and unspecified stimulation parameter/afterdischarge information resulted in inclusion as low confidence level cases.

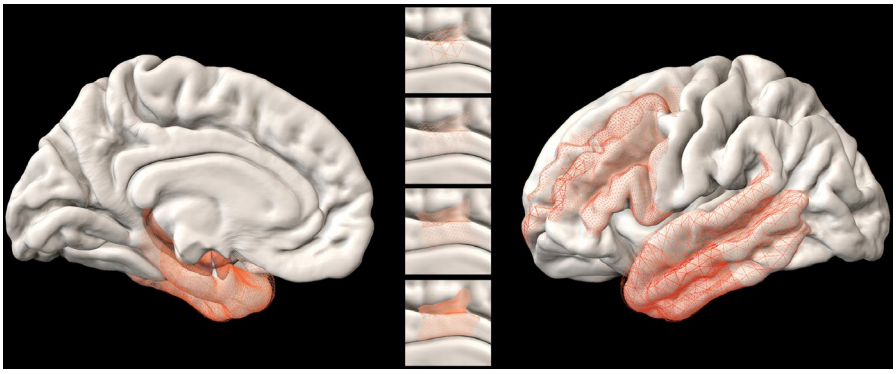

FIGURE 5 Overlaying EZs of patients with Forced Thinking. The middle panel shows the surface rendering patterns used to indicate neocortical EZ-confidence level, from top to bottom: Low, Moderate, High and Very High, with an intensity reflecting one case in the given area (Figure S2 shows the corresponding figure with cases of high and very high confidence level only). For simplicity, EZs are displayed as left sided, on a standardized left hemisphere MNI-152 brain.

retrieved by the systematic search.[14,30] The synthesized EZs of both DSIs and forced thinking were derived from few cases, either single case studies or small and highly selective case series, some of which were even selected according to region. This could imply bias and therefore, a low degree of reliability in the findings. Some of the impact of this bias is however reduced by the fact that the selectivity of the study-populations is also present in the population to which the findings will apply. Furthermore, it appears to be a certain consistency to the findings for the EZs of both phenomena, which may also increase the reliability. Moreover, from the evidence classification guiding the review,[10] it follows that evidence with high, and particularly very high confidence, has a high degree of directness to it, which was more evident for DSIs than for forced thinking. The last principle domain, the precision,

concerns confidence interval of the findings, and although we did not have any quantifications where this could be assessed, the low number of cases, particularly in combination with quite dispersed possible epileptogenic zones must be regarded as a limitation. Thus, there is a low to moderate reliability in the possible anatomical definition of the EZs associated with both DSIs and forced thinking.

5 | DISCUSSION

Our systematic search revealed very few specific reports on seizures with experiential phenomena in the form of disintegration of the bodily self, depersonalization, and forced thinking with an EZ defined by surgery outcome, structural MRI-pathology, or iEEG findings. We identified only 23, 1 and 4 eligible cases for the three different categories mentioned above, respectively, where literature searches added 10 more cases reporting ictal DSIs and six cases with ictal forced thinking. Most eligible cases were derived from case studies or selective case series devoted to either specific symptomatology, defined brain regions or both. Thus, in addition to the low number of reports, both semiological characteristics and the contribution of different potential EZs in our material could be liable to bias.

Several reasons may account for the low number of cases and the uncertainty regarding the EZs of the phenomena in question cannot be discussed properly without taking their prevalence in mind. A publication devoted to autoscopic ictal and post-ictal events, that did not contain any eligible cases for this review, reported a prevalence of 6.3% in their patient population.[31] Although surprisingly high, regarding the few reports on both this and related issues, it is supported by a later study which found a very similar prevalence, however, pointing to the rarity of which the patients in question had such experiences.[32] Another publication on the same topic, although likely representing an underestimation, identified a single patient with autoscopic symptomatology out of 1500 epilepsy surgery patients, even though 31 of these underwent surgery on the highly relevant temporoparietal junction.[33] Other reports give indications on the prevalence of other body integrational symptoms, however, with biases and uncertainties related to the symptom selection, symptom detection, and underlying population. Accordingly, in one publication on somatosensory epilepsy and three publications on parietal lobe epilepsies, 10%, 11%, 11% and 6% of patients, respectively, reported somatognostic dysfunction or body image disturbances, although the definition of the latter in some instances was somewhat unclear.[15,17,34,35] Lastly, in Allen's early work on forced thinking, this feature was present in 2% of an unspecified epilepsy population.[36] Most likely, the fact that these symptoms are relatively rare and therefore not often given attention during diagnostic questioning, contributes to an even lowered detection rate. Furthermore, the patient may even be reluctant to report subjective symptoms if perceived as unfamiliar and perhaps disturbing. If reported, these may be difficult to describe, and in hand, correctly interpreted by physicians.[1] Nonetheless, the rarity of these phenomena appears unquestionable. One may therefore also speculate whether it relates to either these symptoms being overshadowed by other more noticeable semiological traits, or perhaps, that LOC is often imminent when areas that could give rise to these symptoms are involved in ictal activity. This may particularly be true for the disturbances of bodily integration, as although LOC reportedly is relatively uncommon in parietal epilepsy,[37] seizures arising in associative parietal cortices may be more prone to such development.[38]

Keeping the abovementioned limitations in mind, we synthesized the distribution of EZs for both disintegration of bodily self and forced thinking. For the first symptom group we categorized the ictal DSIs as either affecting parts of the body (BPD) or the body as a whole (WBD), and furthermore subdivided the cases in these two groups according to the qualia of the disintegrative symptoms. As Table 1 and Figure 2 show, the different symptom groups vary with regards to both location and extent of EZ distribution. Some of the differences, perhaps particularly regarding differences in extent, may of course rely on variations in number of cases seen in the different groups. However, some of the variation may rather reflect real differences in qualia and perhaps also the complexity of the different symptom groups, where an interplay between epileptogenic zone and symptomatic zone could prove particularly important.[55] For instance, whereas for cases with partial bodily disintegration with pure asomatognostic, dynamic or positional illusional traits a parietal focus was typical, cases with somatoparaphrenic traits had a wider EZ distribution, where also SMA, anteromedial temporal structures or middle portions of the cingulate gyrus could be involved. BPD furthermore strongly suggested a contralateral EZ. For DSIs affecting the whole body, both autoscopic illusion and out-of-body experience included structures in and around the TPJ (posterior parts of superior (STG) and middle (MTG) temporal gyri, posterior insula, supramarginal gyrus and angular gyrus). Autoscopy often included precuneal/cuneal or occipital areas. Patients with OBE also included anterior cingulate, insula and possibly medial temporal structures. Interestingly, the two cases with heautoscopy included insular and medial temporal areas in the possible EZ, without the temporoparietal junction per se. Non-included patients with heautscopy from the same publication have, however, EZs involving also this last region.[18] An increased complexity of both OBE and heautoscopy, when compared with

autoscopy, has been pointed out previously and thought to imply a different and perhaps also a wider regional origin for the first two phenomena, suggesting particular caution with regards to the reliability of a possible EZ of heautoscopy based on only two patients.[22,56–58] Lastly, laterality, when present in the WBD-symptomatology, coincided with a contralateral EZ in all cases. Although few patients were identified by this review, this was in line with previous analyses on lateralized ictal autoscopic phenomena.[59]

For forced thinking, the material of our search showed a quite even distribution between temporal and frontal EZs, with about the same level of confidence for each of the two regions. It should be noted however that selection bias for this symptom-group was even more evident than for the bodily disintegration group. Namely, four of seven publications, contributing with 7 of 10 patients, came from study populations selected for either of these two regions, one of which also selected for the side. Nonetheless, as the centrality of temporal and frontal regions in this ictal phenomenon is supported by additional publications that for various reasons could not be included in our material,[60] some of the concern regarding the effect of this bias on the data presented here may be reduced. Furthermore, the areas of relevance within these two regions should also be affected to a lesser degree, allowing for some increased reliability in the areas that stand out in Figure 5. Although some authors have pointed out differences in qualia of forced thinking deriving from the frontal vs. the temporal lobe,[24,26] the paucity of cases, and the level of description of many of them, made such a distinction difficult here.

Our systematic search on ictal depersonalization gave a very scarce yield with only a single patient from one study, which is somewhat surprising given a reported prevalence of 15%.[61] This patient showed an EZ in the superior frontal gyrus including the SMA.[23] An additional work on depersonalization in epilepsy by the same group presented nine patients, of which 7 underwent epilepsy surgery.[62] Although the anatomical data and surgery outcome-measures provided by this study were not analyzable in terms that allowed inclusion in the present study, several matters are worth noticing. Firstly, although five patients encompassed the abovementioned right mediodorsal premotor cortex in their suggested EZ, all five lobi were included in the possible EZs of at least one of the nine patients in the study. Secondly, at least three of nine patients experienced additional symptoms that might be regarded as DSI, as defined above. It is thus likely that although symptoms of depersonalization *may* point to SMA and premotor areas, the symptom appears to be quite unspecific and may also reflect non-frontal foci. This non-specificity is supported by work pointing to phenomenological similarities between depersonalization and symptomatology usually attributed to epilepsy of temporal origin.[63–65] Furthermore, the material derived through our search on both depersonalization and DSIs suggests that some experience of depersonalization is likely present in many, if not most patients with BPD and especially WBD, where depersonalization has even been regarded as a necessary part for the more complex disturbances.[4] This could also be the case with other experiential or psychic ictal phenomena, and while representing a natural entity within the psychiatric disorders, in the setting of localizing ictal phenomena, depersonalization may be regarded as one aspect that often adjoins other, possibly better localizing semiological traits.

Stimulation studies where DSIs, depersonalization, or forced thinking were elicited without being a part of the patients' habitual seizures were by definition excluded from our material, since the stimulation finding in these cases could not be regarded as pointing to an EZ according to the validity criteria guiding this review.[10] This is in accordance with recent work dedicated to the relationship between SEEG-stimulation and epileptogenic zone determination.[66,67] Nevertheless, the ability to elicit symptoms through stimulation may imply a role of the stimulated area in the generation of similar symptoms also when these occur ictally.[68] A recent systematic review on stimulation-induced disturbances of the bodily-self pointed to the inferior parietal lobule, the precuneus, the posterior insula, the middle cingulum, the SMA and finally the amygdalo-hippocampal complex as the six main areas attributed to bodily self-integration.[3] All these areas stood out as relevant EZs in the material included in this review. Interestingly, the frontal areas were implicated particularly in the sense of agency, in line with our finding on the more complex partial bodily disturbances. Stimulation in the prefrontal cortex, particularly around the middle frontal gyrus, may induce intrusive or forced thoughts, also corroborating some of the findings from our material.[69–71] Caution should be taken however regarding findings from stimulation studies, especially when it comes to very rare ictal phenomena. For instance, although the middle cingulum was indeed implicated in some cases with disturbances in body integration, it appeared to be less central as an EZ than findings from stimulation studies could indicate, being the area where such phenomena most frequently were elicited, and where both whole and partial body integrational disturbances could be seen.[3,72] Furthermore, although autoscopic hallucinations have been shown to be elicited by stimulation in the precuneus and occipitoparietal sulcus,[73] these areas were implicated only in one of four included autoscopy cases, with low degree of reliability. Such discrepancies may yet again reflect simply the difference between symptomatic and epileptogenic zones, but could also be attributable to the low number of cases, to regional factors that make

certain areas less prone to initiation of epileptic seizures, or to regional factors that make other symptoms dominate seizures from these areas.

Lastly, although few cases were found to be eligible for inclusion in this review, there are related cases that for various reasons were excluded. Many of these present data that to a large extent are congruent with the possible EZs outlined by the cases included in this review, both for DSIs,[74–81] and for forced thinking.[7,82–86] Some of the excluded cases point to slightly different locations from those of the eligible patients identified by this review, mostly representing the more complex whole body disintegrational phenomena of out of body experience and heautoscopy, implying for instance that also temporobasal areas could give rise to such phenomena.[87–89]

6 | CONCLUSIONS

In conclusion, our systematic review suggests that ictal disturbances of self-integration often reflect an EZ centered around the temporoparietal region. However, the neighboring areas in the parietal lobe, the posterior insula, and depending on the type of disturbance, also the adjacent occipital lobe, the anterior/middle cingulum, premotor, and SMA, along with medial temporal structures, could also be involved, a variability that likely reflected the type of DSI in question. Furthermore, when present, as was the case in most partial body disturbances, lateralized symptomatology reflected a contralateral focus. Forced thinking often reflected either premotor frontal or a temporal EZ. Depersonalization as a localizing ictal phenomenon was quite elusive and may be better regarded as an aspect occurring alongside other, likely better localizing semiological traits. The conclusions regarding all three symptom groups are however liable to the same limitations, mostly due to the scarcity of cases with a high or very high level of confidence in the EZ, but also due to bias of the studies identified. Additionally, the EZs outlined by this systematic review are quite widespread, both when it comes to DSIs and forced thinking, and one may therefore regard the localizing value of these possible EZs with no more than a low-to-moderate degree of reliability. In the future, an increased focus and an improved categorization of these rare ictal phenomena, in combination with improved imaging techniques and increased use of SEEG will hopefully lead to the accumulation of cases with better defined EZs of a high level of confidence.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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