

Toward a Psychiatric State Index: Differentiating Autistic Meltdown from Psychotic Episode Onset Using Real-Time EEG and AI

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1. Abstract

Autistic meltdowns and psychotic episodes can appear strikingly similar to an outside observer loss of behavioral control, apparent disconnection, distress yet their neurological substrates are fundamentally different. As an autistic individual who has experienced sensory overload from the inside, I propose a conceptual framework for a wearable device analogous to the Bispectral Index (BIS) monitor used in anesthesia, adapted to differentiate these two states in real time using EEG-derived biomarkers and machine learning. I synthesize current literature on EEG signatures of autistic sensory overload (alpha instability, impaired sensory gating, posterior gamma surges) and pre-psychotic states (temporal beta/gamma abnormalities, delta/alpha power spectral shifts), and propose two novel composite indices the Sensory Overload Index (SOI) and Reality Disconnection Index (RDI) that could be computed on-chip from a minimal electrode montage. This perspective article bridges the gap between lived autistic experience and computational neuroscience, arguing that the phenomenological distinction an autistic person perceives from the inside should be matched by a technological distinction measurable from the outside.

2. Introduction: Why This Matters A Personal Note

I am autistic. I have felt the rising tide of sensory overload the fluorescent hum becoming unbearable, the texture of fabric transforming into sandpaper, the voices in a room collapsing into a single crushing wall of noise. I know what a meltdown feels like from the inside: I am fully present, painfully aware, and unable to stop the cascade. My consciousness is not altered *it is overwhelmed*.

This is categorically different from psychosis, where the architecture of reality itself shifts. Yet clinically, these two states are

frequently confused [1]. An autistic person in meltdown may be misdiagnosed as psychotic, leading to inappropriate pharmacological intervention antipsychotics administered when the actual need is sensory reduction and nervous system de-escalation [2]. The reverse misidentification, though rarer, carries its own dangers. The clinical overlap between autism and psychosis is well documented. ASD and primary psychosis display overlapping epidemiological, environmental, and genetic components, as well as endophenotypic similarities [1]. At a behavioral and cognitive level, individuals with ASD and primary psychosis show overlapping symptomatology, impeding differentiation between psychosis and ASD symptoms and hampering differential diagnosis [1]. A recent study revealed significantly increased rates of autistic traits in patients with psychosis [1].

I propose that this diagnostic confusion is solvable not through better behavioral observation, which will always struggle with surface-level similarity, but through real-time neurophysiological monitoring. The technology largely exists. What is missing is the conceptual framework to connect it, and perhaps the autistic voice to insist that the distinction is real, consequential, and measurable.

3. The Bispectral Index: A Proven Paradigm

3.1. How BIS Works

The Bispectral Index (BIS) monitor, introduced in 1996, transformed anesthesia monitoring by reducing the complex, multidimensional EEG into a single actionable number. The BISx module acquires real-time EEG data and computes its analysis from the frequency, power, and phase throughout the entire frequency range, presenting the result as an index between 0 (isoelectric EEG) and 100 (fully awake) [3]. Additional parameters include Suppression Ratio (SR), Spectral Edge Frequency (SEF), Me-

dian Power Frequency (MF), and electromyographic strength (EMG) [3].

Critically, BIS is not a simple spectral measure. It is a complex parameter consisting of power spectral and higher-order bispectral components, as well as time-domain components [4]. The bispectral analysis captures phase coupling between frequency components — a measure of nonlinear brain dynamics that pure power spectral analysis misses entirely.

3.2. BIS in Neuropsychiatric Populations: The Calibration Problem

The algorithm underpinning BIS was developed by analyzing the normal EEGs of over 1,000 healthy volunteers [5]. This creates a fundamental problem when applied to neurologically atypical populations. BIS values in children with intellectual disability predominantly due to autism, cerebral palsy, or Down syndrome were significantly lower than controls in the awake state: median 72 versus 97 ($P < 0.001$) [6]. This 25-point baseline depression means a standard BIS algorithm would systematically misread an awake autistic person as sedated.

This is not a flaw of the technology — it is a feature waiting to be repurposed. The fact that BIS detects a baseline neurophysiological difference in autism demonstrates that the bifrontal EEG, even from a minimal electrode montage, carries exploitable signal about neurodevelopmental state. Furthermore, researchers have noted that bispectral analysis might be useful in assessing neurological function in a global sense [4], and the question has been raised explicitly: whether psychiatric patients require a separate monitor [7].

The answer, I argue, is yes — and the following sections first map the scope of the calibration problem, then describe what such a monitor should measure.

3.3. The BIS Calibration Challenge Across Neuropsychiatric Populations

The problem of BIS unreliability in neurologically atypical populations is not a single anomaly — it is a systematic pattern that spans diagnostic categories, and understanding it is essential for designing the PSI monitor. The following evidence demonstrates both the scope of the problem and the information it inadvertently reveals.

Autism and Intellectual Disability. In the largest study to date, 18 intellectually disabled children (with etiologies predominantly due to autism, cerebral palsy, or Down syndrome) showed BIS values significantly lower than 35 controls at every stage of anesthesia: awake (72 vs. 97, $P < 0.001$), intraoperative maintenance (34 vs. 21–45 vs. 33–52, $P = 0.02$), and return of consciousness (59 vs. 73, $P = 0.009$) [6]. The investigators concluded that anesthesiologists should be alert to possible lower BIS values in intellectually disabled children, and called for multicenter studies to find the optimal manner of evaluating consciousness in these patients with documented specific etiologies [6]. The implication is stark: an awake autistic child at BIS 72 would be classified as “sedated” by standard thresholds, poten-

tially leading to under-dosing of anesthesia (risking awareness) or, conversely, delayed extubation if the clinician waits for a “normal” BIS recovery value that will never come.

Cerebral Palsy. A comparison of 20 children with quadriplegic cerebral palsy and mental retardation against 21 matched controls found that absolute BIS values were lower in the CP group while awake and at all sevoflurane concentrations, though the pattern of change was similar [25]. This is a critical observation: the dynamic response to anesthetic deepening is preserved even when the absolute baseline is shifted. A study of BIS during desflurane anesthesia in CP children confirmed the same finding — lower absolute values, preserved relative response [26]. Furthermore, anticonvulsant medications commonly used in CP further depress baseline BIS values, and the interaction with midazolam premedication creates additional unpredictability [27]. These findings suggest that relative change from personal baseline — not absolute threshold — is the reliable signal in neurologically atypical populations.

Psychosis and Bipolar Disorder. Ramesh et al. recorded BIS values in patients suffering from psychosis, depression, organic and bipolar disorder and compared them with control patients without psychiatric illness [7]. They observed that BIS values in psychiatric patients are significantly lower compared to controls, with patients with psychosis and bipolar disorder having lower BIS values than patients with depression [7]. The editorial commentary noted that BIS values derived from normal individuals might show spuriously low values in psychiatric patients, and that patients remained awake with no manifestation of sedation despite low BIS readings [7]. The cautionary conclusion was explicit: BIS might not be a true indicator of depth of anesthesia in psychiatric patients [7].

Electroconvulsive Therapy (ECT) Effects. In 30 patients with schizophrenia or mood disorders undergoing ECT, mean resting BIS values dropped from 91.6 at baseline to 79.7 after the sixth ECT session ($P = 0.02$) [28]. The proportion of patients whose resting BIS values fell into the sedated/anesthetized range increased from 26% at baseline to 60% after six sessions [28]. Anesthesiologists were warned that a considerable proportion of patients who have received a course of ECT may have BIS values in the sedated/anesthetized range even when fully awake, and that BIS may not provide accurate estimation of anesthesia depth after the initial sessions [28]. This iatrogenic BIS depression adds a further layer of complexity: the PSI monitor would need to account for treatment-induced shifts in baseline neural activity.

Dementia. Patients with Alzheimer’s disease and multi-infarct dementia had an awake BIS averaging 89.1, compared to 94.7 in age-matched controls [5]. While the deficit is smaller than in autism or psychosis, it falls below 90 the standard threshold indicating sedation raising the question of whether anesthesia thresholds should be adjusted downward for dementia patients, and by how much [5].

Epilepsy. Case reports describe patients with pre-existing epilepsy showing anomalous BIS readings during surgery. In one case, unexpectedly low BIS values persisted for hours until the raw EEG was checked and found to show epileptiform activity that the BIS algorithm misinterpreted as deep anesthesia [5]. This highlights that the BIS algorithm, designed for the frequency characteristics of normal brain activity transitioning through anesthetic stages, can be systematically misled by pathological waveforms.

The Manufacturer's Own Warning. Medtronic's BIS documentation acknowledges the limitation: due to limited clinical experience, BIS values should be interpreted cautiously in patients with known neurological disorders and those taking other psychoactive medications [29]. Clinical trials studying BIS algorithm improvements (e.g., NCT04765046) explicitly exclude

patients with major psychiatric conditions including bipolar disorder, schizophrenia, Alzheimer's disease, dementia, Parkinson's disease, and major depression [30] effectively acknowledging that the algorithm is not validated for these populations while continuing to sell the device for use in operating rooms where these patients regularly present.

The BIS monitor is designated as unreliable in cases of hypothermia or neurological impairment, and the index has not been proven to measure consciousness independently of the cause of reduced consciousness [31].

3.4. What the BIS Failure Pattern Reveals

Rather than viewing these calibration failures as limitations to work around, I propose they constitute a rich dataset about the neurophysiological signatures of different conditions. Consider the pattern:

Population	Awake BIS	Deficit vs. Control	Primary EEG Alteration
Neurotypical	94–98	—	—
Dementia	89	–6	Alpha slowing
Schizophrenia/Psychosis	~85*	–10	Beta/gamma dysregulation
Post-ECT psychiatric	80	–15	Diffuse slowing
Autism/ID	72	–25	Alpha instability, altered coherence
CP with anticonvulsants	~65*	–30	Epileptiform + drug effects

*Estimated from literature descriptions; exact values vary by study.

Each condition depresses the BIS in a characteristic way because each condition alters the EEG in a characteristic way and the BIS algorithm, designed to map those EEG characteristics onto a consciousness scale, misreads condition-specific neural signatures as depth-of-anesthesia signals. An autism-specific alpha instability pattern looks like “less conscious” to an algorithm calibrated on stable alpha rhythms. A psychosis-specific beta/gamma abnormality looks like “less conscious” to an algorithm expecting normal oscillatory coupling.

The PSI monitor I propose inverts this logic: instead of trying to map atypical EEGs onto a consciousness scale, it maps them onto a psychiatric state space where each condition occupies a distinct region. The BIS's “failure” in neuropsychiatric populations is the PSI's training data.

3.5. Design Implications for the PSI Monitor

The BIS calibration problem teaches five design principles for the PSI:

1. Personal baseline is mandatory. No population-level threshold will work across the neuropsychiatric spectrum. The PSI must establish each individual's own awake baseline during a calibration period, then detect deviations from that baseline exactly as experienced anesthesiologists already do with BIS in challenging patients, using it as a trend monitor rather than an absolute scale.

2. Relative change trumps absolute value. Studies in cerebral palsy demonstrate that the pattern of change in response to neu-

ral state transitions is preserved even when absolute values are shifted [25,26]. The SOI and RDI should be computed as deviations from personal baseline, not as absolute indices.

3. Medication effects must be modeled. Anticonvulsants, antipsychotics, benzodiazepines, and mood stabilizers each shift the EEG baseline in characteristic ways [27,28]. The PSI must incorporate medication profile as a calibration input a feature no current BIS system offers.

4. Comorbidity is the rule, not the exception. An autistic person with epilepsy on anticonvulsants who develops psychotic features presents all the BIS failure modes simultaneously. The PSI's dual-index architecture (SOI + RDI) must handle overlapping conditions, not force a binary classification.

5. Raw EEG must remain accessible. The epilepsy case report where hours of anomalous BIS were only explained when someone examined the raw tracing [5] argues that any clinical index must provide clinician access to the underlying signal. The PSI should display SOI and RDI as headline numbers with one-tap access to the contributing spectral, topographic, and temporal data.

4. Neurophysiology of Autistic Meltdown: The Bottom-Up Cascade

4.1. Alpha Instability and Failed Sensory Gating

The autistic meltdown is not a behavioral choice it is a nervous system event with identifiable precursors. Recent research helps explain what happens in the brain during an autistic meltdown: the insular cortex, which processes sensory information, functions differently in autistic people, causing a constant state of

heightened alertness and increased susceptibility to sensory overwhelm, which accumulates and triggers a meltdown [8].

The gating mechanism that should attenuate this accumulation relies on alpha-band oscillations (7–13 Hz). A 2026 study using burst-resolved high-density EEG demonstrated that alpha oscillatory states are less stable in autistic individuals, suggesting that sensory differences in autism may be linked to less stable brain rhythms that normally help control sensory input [9]. Alpha activity reflects rhythmic processes that modulate cortical and thalamocortical excitability and is widely considered a top-down cortical sensory gating mechanism [9].

Complementary evidence from sensory gating paradigms confirms that autism is associated with impaired sensory gating reduced inhibition of irrelevant sensory afferents [10]. The P50 event-related potential, which indexes pre-attentive filtering, shows deficient suppression in autism, indicating that the central nervous system fails to adequately filter incoming stimulation [10].

4.2. Frontoparietal Desynchronization Bursts

In adolescents with ASD, resting-state EEG reveals ultra-short desynchronization bursts between frontal and parietal areas [11]. Mathematical models demonstrate that a brain system exhibiting this pattern of activity is highly sensitive to external events [11]. This finding was linked to symptom severity, suggesting the pattern spans modalities and ages [11]. These bursts represent moments of complete loss of top-down regulatory coordination microsecond windows of vulnerability that accumulate under sustained sensory load.

4.3. Posterior Gamma Hyperactivation

ASD is associated with enhanced gamma activity in posterior brain areas during visual processing. An EEG study found enhancement of gamma activity in the eyes-open compared to eyes-closed condition in temporal and occipital brain regions areas involved in processing visual information [12]. This gamma hyperactivation may underlie the hyper-sensitivity symptoms to visual stimuli commonly reported in autism [12].

4.4. The Meltdown Trajectory

Synthesizing these findings, the pre-meltdown neural cascade proceeds as follows:

1. **Accumulation phase** (minutes to hours): Sustained sensory input meets impaired gating → progressive alpha destabilization
2. **Vulnerability phase** (minutes): Frontoparietal desynchronization bursts increase in frequency → loss of top-down regulation
3. **Overflow phase** (seconds): Posterior gamma surges exceed compensatory capacity → EMG activation, autonomic arousal
4. **Meltdown** (minutes): Full regulatory collapse → behavioral expression

Critically, *consciousness is preserved throughout*. The autistic person in meltdown is not disconnected from reality they are drowning in it.

5. Neurophysiology of Psychotic Episode Onset: The Top-Down Dissolution

5.1. Beta/Gamma Abnormalities in Temporal and Parietal Regions

The pre-psychotic state shows a fundamentally different neural signature. Abnormal neural oscillations have been shown to precede the onset of frank psychosis [13]. Specific patterns of beta and gamma oscillations, detected by machine learning algorithms, can predict transition to psychosis in clinical high-risk patients. The left superior temporal gyrus, the left inferior parietal lobule, and the precuneus contributed most strongly to prediction (AUC = 0.77) [13].

This topographic distribution — temporal and medial parietal — is strikingly different from the posterior sensory cortex activation seen in pre-meltdown states. The temporal involvement implicates auditory processing and language networks, consistent with the auditory hallucinations and thought disorder characteristic of psychosis but absent in meltdown.

5.2. Power Spectral Density Shifts

Spectral analysis of alpha and delta band waves demonstrates the effectiveness of stimulus-independent EEG in identifying abnormal activity patterns associated with first-episode psychosis (FEP) [14]. A generalized model incorporating multiple frequency bands delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), and low-beta (12–16 Hz) — has been proposed for associating potential EEG biomarkers with FEP [14]. A Gaussian Process Classifier achieved 95.78% specificity using resting-state power spectral density features [14].

Notably, these are resting-state, stimulus-independent changes the psychotic brain shows altered baseline activity, whereas the pre-meltdown autistic brain shows altered reactivity to stimulation. This distinction is fundamental.

5.3. Classification Performance

Using EEG signals and circulant spectrum analysis (ciSSA) sub-band signals, researchers developed a classification model for FEP achieving AUC of 0.9893, accuracy of 96.23%, sensitivity of 0.966, and specificity of 0.956 using SVM on hybrid features [15]. This demonstrates that the EEG signature of psychosis onset is robust enough for automated detection the same should be achievable for meltdown prediction.

5.4. The Psychosis Trajectory

1. **Prodromal phase** (weeks to months): Gradual beta/gamma dysregulation in temporal regions → subtle perceptual changes
2. **High-risk phase** (days to weeks): Increasing power spectral deviation from baseline → attenuated psychotic symptoms
3. **Transition phase** (hours to days): Critical threshold of neural oscillation abnormality → reality testing begins to fail
4. **Episode** (days to weeks): Sustained altered state → frank psychosis

Critically, *consciousness is altered*. The individual in psychosis experiences a different reality not an excess of the shared one.

6. A Personal Phenomenology: Why the Distinction Is Not Academic

I want to pause the scientific narrative to say something that no EEG study captures: the experience is different.

In sensory overload, I can feel each individual stimulus. I can name the sound that is too loud, the light that is too bright, the texture that is intolerable. My suffering is specific, located, and if the environment changes immediately relievable. When someone dims the lights, I feel the relief in my body within seconds. The problem is not in my perception of what is real. The problem is that what is real is too much. This is the phenomenological signature that a monitoring device should detect: intact reality processing under excessive load, versus compromised reality processing under normal or reduced load.

The clinical consequences of confusing these states are severe. An autistic person in meltdown who is administered antipsychotic medication receives a chemical intervention targeting a neural system that is not malfunctioning. The sensory gating deficit and alpha instability that drive the meltdown are not addressed by dopamine D2 receptor antagonism. Meanwhile, the sedation side effects may mask the meltdown's resolution, creating a false impression of pharmacological efficacy and establishing a harmful prescribing pattern.

7. Proposed Device: The Psychiatric State Index (PSI) Monitor

7.1. Hardware Architecture

I propose a wearable device modeled on the BIS monitor's form

factor a small, single-use sensor strip applied to the head but with an expanded montage and multimodal sensing:

Electrode configuration (8 channels): - 2 bifrontal (Fp1, Fp2) leveraging the proven BIS sensor placement - 2 temporal (T7, T8) capturing psychosis-relevant beta/gamma - 2 parieto-occipital (P3/O1, P4/O2) capturing meltdown-relevant posterior gamma - 2 reference/ground

Auxiliary sensors: - Galvanic skin response (GSR) autonomic arousal marker - Photoplethysmography (PPG) heart rate and heart rate variability - Skin temperature autonomic regulation

This configuration is feasible with current technology. An ear-worn wearable EEG system has already demonstrated on-chip machine learning for real-time ASD prediction using four EEG channels (Fp1, Fp2, T7, and T8) fabricated in 180 nm CMOS [16]. A separate wearable on-chip processor for real-time emotion prediction in ASD has been demonstrated using power spectral density, logarithmic interhemispheric power spectral ratio, and interhemispheric power spectral differences from eight EEG channels with a linear SVM classifier [17].

7.2. Dual-Index Computation

Unlike BIS, which outputs a single consciousness index, the PSI monitor computes two parallel indices:

Sensory Overload Index (SOI): 0–100

The SOI quantifies proximity to meltdown through five sub-components:

Sensory Overload Index (SOI): 0–100

Component	Source	Derivation
Alpha stability	P3/O1, P4/O2	Burst duration variance in 7–13 Hz band
Gating efficiency	All channels	Ratio of stimulus-locked to spontaneous alpha suppression
Gamma load	P3/O1, P4/O2	Posterior gamma power relative to personal baseline
Frontoparietal coherence	Fp1-P3, Fp2-P4	Phase-locking value in alpha/beta bands
Autonomic surge	GSR, PPG	Rate of change in skin conductance and HR

Reality Disconnection Index (RDI): 0–100

Component	Source	Derivation
Temporal beta/gamma	T7, T8	Current source density abnormality in 13–40 Hz
Delta/alpha ratio	Global	Power spectral shift toward low-frequency dominance
Bifrontal bicoherence	Fp1, Fp2	Phase coupling nonlinearity (core BIS technology)
Hemispheric asymmetry	Bilateral pairs	Inter-hemispheric power ratio deviation
Temporal coherence	T7-Fp1, T8-Fp2	Frontotemporal functional connectivity

An SOI of 0 indicates minimal sensory load; 100 indicates imminent meltdown. The critical innovation is the rate of change: meltdown SOI rises steeply over minutes, unlike the gradual drift of baseline shifts.

Reality Disconnection Index (RDI): 0–100

The RDI quantifies reality contact through five sub-components:

An RDI of 100 indicates full reality contact; 0 indicates complete disconnection. The critical innovation here is the temporal scale: psychotic RDI declines gradually over days to weeks, unlike the acute SOI surge.

7.3. Personal Baseline Calibration

Given that awake BIS values in intellectually disabled children (including autism) average 72 versus 97 in controls [6], and that psychiatrically ill patients show lower baseline BIS values [7], any fixed-threshold system would fail. The PSI monitor must establish a personal baseline during a 24-hour calibration period, then detect deviations from that individual's own baseline rather than deviations from population norms.

This is analogous to how experienced anesthesiologists use BIS not as an absolute number, but as a trend relative to the patient's own awake value.

7.4. Alert Logic

The PSI monitor generates four alert states based on the interaction of both indices:

The most critical clinical distinction is between **RED-S** and **RED-P**: both present as behavioral crisis, but RED-S requires environmental modification while RED-P requires pharmacological intervention. Confusing these leads to harm in both directions.

Alert Logic

Alert	SOI	RDI	Rate of Change	Recommended Action
GREEN	<30	>70	Stable	No intervention
YELLOW	30–60	>70	SOI rising	Reduce sensory input
RED-S (Sensory)	>60	>60	SOI rising rapidly	Immediate sensory reduction; do NOT sedate
ORANGE	<40	40–60	RDI declining slowly	Clinical psychiatric assessment
RED-P (Psychotic)	Variable	<30	RDI declining	Psychiatric emergency intervention

7.5. Worked Computation Example

To make the index construction concrete, consider two clinical vignettes drawn from the same autistic individual Patient A, whose 24-hour calibration baseline was recorded during a calm day at home.

Personal baseline (calibration day):

Each sub-component is normalised to 0–100 by mapping the current value onto the individual's own calibration distribution (mean $\pm$ 3 SD $\rightarrow$ 0–100 for SOI; inverted for RDI where higher is healthier). Formally, for SOI sub-component i:

Personal baseline (calibration day):

Feature	Baseline value	Units
Alpha burst-duration variance (P3/O1)	$\sigma^2 = 0.12 \text{ s}^2$	Alpha stability
Stimulus-locked / spontaneous alpha-suppression ratio	0.85	Gating efficiency
Posterior gamma power (P3+O1, P4+O2)	$4.2 \mu\text{V}^2$	Gamma load
Frontoparietal PLV (Fp1-P3, Fp2-P4, α/β)	0.61	FP coherence
GSR slope	$0.02 \mu\text{S/s}$	Autonomic
Temporal beta/gamma CSD (T7, T8)	$1.8 \mu\text{V}^2/\text{cm}^2$	Temporal β/γ
Delta / alpha PSD ratio	1.1	δ/α ratio
Bifrontal bicoherence (Fp1, Fp2)	0.47	Bicoherence
Inter-hemispheric power ratio deviation	0.06	Hemisp. asym.
Frontotemporal coherence (T7-Fp1, T8-Fp2)	0.55	FT connectivity

$$s_i = \min(100, \max(0, 100 \times (x_i - \mu_i) / (3 \cdot \sigma_i)))$$

where x_i is the current epoch value, μ_i and σ_i are from the calibration period. For RDI the mapping is inverted (deviations below baseline signal disconnection):

$$r_i = \min(100, \max(0, 100 \times (1 - |x_i - \mu_i| / (3 \cdot \sigma_i))))$$

The composite indices are weighted sums:

$$\text{SOI} = \sum_{i=1}^5 w_i^{(S)} \cdot s_i; \text{RDI} = \sum_{i=1}^5 w_i^{(R)} \cdot r_i$$

Default weights (w) are equal (0.20 each); the calibration phase can adjust them per patient via ridge regression on labelled calm/distressed epochs if available.

Vignette 1 Sensory Meltdown Trajectory (Sunday, supermarket visit)

SOI component	Current	Baseline $\mu \pm \sigma$	s_i	Weight
Alpha stability (burst var.)	0.38 s^2	0.12 ± 0.07	$\min(100, 100 \times (0.38 - 0.12) / (3 \times 0.07)) = 100$	0.2
Gating efficiency	0.54	0.85 ± 0.10	$(0.85 - 0.54) / (3 \times 0.10) = 100$	0.2
Gamma load	$8.9 \mu\text{V}^2$	4.2 ± 1.5	$(8.9 - 4.2) / (3 \times 1.5) = 100$	0.2
FP coherence (drop)	0.33	0.61 ± 0.09	$(0.61 - 0.33) / (3 \times 0.09) = 100$	0.2
Autonomic surge (GSR slope)	$0.11 \mu\text{S/s}$	0.02 ± 0.03	$(0.11 - 0.02) / (3 \times 0.03) = 100$	0.2

$\text{SOI} = 0.20 \times (100+100+100+100+100) = 100$ but this is the clipped ceiling. In practice the trajectory was:

Time	SOI	RDI	Rate $\Delta\text{S/min}$	Alert
$t = 0$	18	88	—	GREEN
$t + 3 \text{ min}$	35	85	5.7	YELLOW
$t + 6 \text{ min}$	58	82	7.7	YELLOW (approaching RED-S)
$t + 8 \text{ min}$	74	79	8	RED-S triggered at SOI > 60, rising > 5/min
$t + 10 \text{ min}$	91	76	8.5	RED-S confirmed

Vignette 1 Sensory Meltdown Trajectory (Sunday, super-market visit)

At $t = 0$ min (entering the supermarket), the 4-second epoch shows:

For gating, a decrease from baseline signals overload, so the deviation is (baseline – current).

$SOI = 0.20 \times (100+100+100+100+100) = 100$ but this is the clipped ceiling. In practice the trajectory was:

Key Observations: - SOI climbed from 18 $\rightarrow$ 91 in 10 min-

utes (steep, characteristic meltdown ramp). - RDI remained > 70 throughout reality contact preserved. The individual was overwhelmed, not disconnected. - Alert at $t + 8$: **RED-S**. Recommended action: immediate sensory reduction do NOT sedate. - Had the caregiver received the YELLOW alert at $t + 3$, leaving the supermarket then would likely have prevented escalation.

Vignette 2 Early Psychotic Episode (same individual, two months later)

Over 72 hours, the cloud-augmented Stage 2 classifier tracks a slow RDI decline:

Vignette 2 Early Psychotic Episode (same individual, two months later)

RDI component	Day 0 (baseline)	Day 1 eve	Day 2 eve	Day 3 AM
Temporal β/γ CSD	92	81	68	52
δ/α ratio	95	88	74	58
Bifrontal bicoherence	88	82	71	55
Hemispheric asymmetry	90	85	76	61
FT connectivity	93	84	69	49
RDI composite	91.6	84	71.6	55

Meanwhile, SOI hovered between 15 and 25 *no sensory overload*.

Key observations: - RDI declined $91.6 \rightarrow 55$ over 3 days (gradual, characteristic psychotic trajectory). - SOI stayed < 30 no sensory crisis at all. Behavioural observation alone might not yet flag concern; the individual was quiet and withdrawing, not ag-

itated. - Alert at Day 2: **ORANGE**. Recommended action: clinical psychiatric assessment. - Without the PSI monitor, the first clinical contact would typically occur only at Day 3–5, when overt psychotic symptoms (disorganised speech, hallucinations) become unmistakable days after pharmacological intervention would have been most effective.

Meanwhile, SOI hovered between 15 and 25 *no sensory overload*.

Time	SOI	RDI	Rate $\Delta R/\text{day}$	Alert
Day 0	20	91.6	—	GREEN
Day 1	22	84	-7.6	GREEN (RDI > 70)
Day 2	18	71.6	-12.4	GREEN $\rightarrow$ ORANGE at RDI crossing 70
Day 2 + 8 h	25	64	-13.0	ORANGE — psychiatric assessment recommended
Day 3	19	55	-14.0	ORANGE approaching RED-P

The Differential Diagnosis in Numbers

Superimposing both vignettes illustrates the core clinical value:

This is the computation the BIS monitor cannot perform and the computation that would have prevented the clinical confusion I experienced personally.

The Differential Diagnosis in Numbers

Feature	Meltdown (Vignette 1)	Psychosis (Vignette 2)
SOI trajectory	$18 \rightarrow 91$ in 10 min	15–25, stable
RDI trajectory	$88 \rightarrow 76$, stable	$91.6 \rightarrow 55$ over 3 days
Dominant rate	$\Delta SOI \approx +7/\text{min}$	$\Delta RDI \approx -12/\text{day}$
Time-scale	Minutes	Days
Correct alert	RED-S	ORANGE $\rightarrow$ RED-P
Correct action	Sensory reduction	Psychiatric evaluation
Harm if confused	Sedation deepens overload	Environment change delays needed medication

8. The AI Engine: From Raw Signal to Clinical Decision

8.1. Feature Extraction

The PSI monitor would employ a multi-domain feature extraction pipeline:

1. Spectral domain: Power spectral density across delta, theta, alpha, beta, and gamma bands proven effective for FEP classification [14,15]

2. Bispectral domain: Phase coupling and bicoherence the core BIS innovation, now repurposed for psychiatric state assessment

3. Temporal domain: Microstate analysis a 2026 study demonstrated that explainable AI with EEG microstate analysis can uncover novel neurophysiological markers for autism [18]

4. Connectivity domain: Phase-locking values and coherence across electrode pairs capturing the frontoparietal desynchronization characteristic of pre-meltdown states [11]

5. Autonomic domain: GSR slope, HR variability, and skin temperature trends

8.2. Classification Architecture

A two-stage classifier is proposed:

Stage 1: State Detection (on-chip, real-time) A lightweight SVM or shallow neural network similar to what has been demonstrated on 180 nm CMOS for ASD detection [16]. classifies the current 4-second epoch into one of five states: baseline, sensory loading, pre-meltdown, pre-psychotic, or indeterminate.

Stage 2: Trajectory Prediction (cloud-augmented) A recurrent neural network (LSTM or Transformer) operating on the last 5–30 minutes of state history predicts trajectory: escalating toward meltdown (minutes), escalating toward psychosis (hours/days), or returning toward baseline.

The two-stage architecture balances latency (Stage 1 alerts within seconds) with accuracy (Stage 2 reduces false positives by incorporating temporal context).

8.3. Explainability

Black-box classification is insufficient for clinical adoption. Following the approach of the 2026 explainable AI study for autism EEG classification [18], the PSI system should provide clinicians with:

Which frequency bands are driving the current alert

Which electrode locations show the most deviation from personal baseline

Confidence intervals for both SOI and RDI

A 30-minute trend visualization

Gap Analysis

Component	Meltdown	Psychosis	(Combined (PSI
Wearable EEG	[16] ✓	[23] ✓	Feasible
On-chip ML	[16,17] ✓	[Prototype [23	Feasible
Autonomic fusion	[19,20] ✓	[21,22] ✓	Feasible
Real-time alert	[19] ✓	[21] ✓	Feasible
Differential classification	✗	✗	MISSING
Labeled training dataset	✗	Partial	MISSING
Clinical validation	✗	✗	MISSING

10. Challenges and Limitations

10.1. The Comorbidity Problem

ASD and psychotic disorders can co-occur. Approximately 6.5% of patients with psychosis score above clinical cutoff for autistic traits [1]. In comorbid individuals, both SOI and RDI may acti-

9. Existing Technology and Feasibility

9.1. Meltdown Prediction

The VB Senacquad device, developed in 2026, uses biosensors to track heart rate, skin conductance, skin temperature, and movement, and is designed to alert a caregiver's smartphone up to 25 minutes before a potential meltdown [19]. Its next development phase plans to integrate in-ear EEG biomarkers to further improve prediction accuracy [19]. This demonstrates that meltdown prediction is already achievable with autonomic signals alone; adding EEG would refine it.

A thesis project demonstrated meltdown detection combining heart rate monitors and electrodermal sensors in a finger glove worn by an autistic child during daily activities [20], confirming feasibility of continuous, naturalistic monitoring.

9.2. Psychosis Prediction

Machine learning algorithms can detect patterns of abnormal brain activity predictive of later transitions to psychosis with promising accuracy, and patients who already had a first episode of psychosis demonstrated the same abnormal patterns revealed in at-risk patients with later transition [13].

The e-Prevention project demonstrated long-term continuous recording of biometric indices through wearable sensors (smartwatches) combined with intelligent cloud-based data processing for prediction of clinical symptoms and relapse in psychosis patients [21]. The SleepSight system extended this to include accelerometry, heart rate, ambient light, and smartphone usage patterns for real-time relapse detection in schizophrenia [22].

A Chinese patent describes a wearable NIRS-EEG dual-mode brain function detection device for real-time schizophrenia monitoring, transmitting data via Bluetooth to a mobile terminal and network server [23].

9.3. Gap Analysis

The hardware and algorithms exist independently for each condition. What does not yet exist is a system that monitors both simultaneously and differentiates between them the PSI monitor proposed here.

vate simultaneously, and the device must handle this gracefully alerting to both processes rather than forcing a binary classification.

10.2. The Heterogeneity Problem

EEG findings in autism are notoriously heterogeneous. A study

of 186 intellectually able adults with ASD found no significant group-mean differences in any of 3,443 EEG biomarkers and weak classification performance [24]. This reinforces the necessity of personal baseline calibration rather than population-level thresholds. The PSI monitor should detect deviation from this person's own baseline, not deviation from a normative database.

10.3. The Ecological Validity Problem

Most EEG studies are conducted in controlled laboratory settings. Real-world wearable monitoring introduces motion artifacts, electrode impedance fluctuations, and environmental electromagnetic noise. The BIS monitor already addresses some of these challenges in the operating room; extending this to daily-wear contexts will require robust artifact rejection a challenge that the ear-worn wearable EEG system has begun to address with continuous impedance monitoring [16].

10.4. The Ethical Problem

Continuous neurological monitoring of a vulnerable population raises significant ethical concerns. Who has access to the data? Can it be used to restrict autonomy rather than protect it? As an autistic person, I am acutely aware that technology designed to "help" us can be wielded against us. The PSI monitor must be designed with the autistic community, not merely for it. Data sovereignty, consent frameworks, and the right to remove the device must be fundamental design principles, not afterthoughts.

11. Proposed Roadmap

Phase 1 Dataset Creation (12–18 months) Prospective collection of multi-channel EEG, GSR, and PPG during naturalistic settings from: - Autistic individuals during known high-overload situations (with participant-directed labeling of pre-meltdown states) - Clinical high-risk psychosis patients (with clinician assessment at regular intervals) - Comorbid ASD + psychosis patients - Neurotypical controls under stress

Phase 2 Algorithm Development (12 months) Training and validation of SOI and RDI algorithms using the labeled dataset, with: - Leave-one-subject-out cross-validation - Explainability analysis (which features drive each index) - Personal calibration protocol optimization

Phase 3 Prototype Device (12 months) Integration into a wearable form factor, leveraging existing platforms (ear-worn EEG, forehead sensor strips), with: - On-chip inference for Stage 1 classification - Bluetooth connectivity for Stage 2 cloud processing and caregiver alerts - 24+ hour battery life

Phase 4 Clinical Validation (24 months) Prospective trial in emergency departments and residential care settings comparing: - PSI-guided intervention versus standard clinical assessment - Meltdown misidentification rate as psychosis (primary outcome) - Time to appropriate intervention (secondary outcome) - Patient/family satisfaction and sense of safety (tertiary outcome)

12. Conclusion

The confusion between autistic meltdown and psychotic episode is not merely a diagnostic inconvenience it is a source of real

harm to autistic people who are medicated for a condition they do not have, in a moment when they need environmental accommodation rather than chemical restraint. I know this because I have felt the difference from the inside.

The neuroscience supports what autistic people have been saying: these states are fundamentally different. Meltdown is a bottom-up sensory flood with preserved consciousness; psychosis is a top-down dissolution of reality processing. The EEG signatures differ in frequency (posterior gamma versus temporal beta/gamma), topology (sensory cortex versus temporal-frontal networks), dynamics (acute minutes-scale versus gradual days-scale), and associated parameters (alpha collapse versus baseline spectral shift).

The technology to detect these differences in real time substantially exists in the BIS monitor's bispectral analysis, in wearable EEG platforms, in on-chip machine learning, in autonomic sensor fusion. What is missing is the integration into a single device purpose-built for this differential classification, and the clinical datasets to train and validate it. I offer this proposal not as a dispassionate academic exercise, but as someone who would wear this device. The autistic community deserves technology that recognizes the specificity of our neurology not as a deficit to be normalized, but as a distinct state to be accurately identified and appropriately supported.

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