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From Havana Syndrome to Catastrophic Tinnitus and Extreme Electromagnetic Hypersensitivity: Cumulative Load, Triggering Peaks, and the Susceptible Phenotype. Why Sudden Onset Does Not Rule Out a Common Electromagnetic Mechanism

Part Five of a Clinical and Mechanistic Series on Tinnitus and Electromagnetic Fields: From the Electrical Ecology of the Bedroom to Extreme Urban Exposure and the Phenomenological Convergence of Directed Pulsed Radiofrequency Injury, Decompensated Tinnitus, and Microwave Syndrome/EHS

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Research article · Review of official reports and mechanistic literature, phenotypic convergence hypothesis, and urban exposure analysis with the authors' field data · Working paper for discussion

Abstract

This fifth article in the series proposes a **conceptual bridge** between three conditions that the literature has treated separately: **Havana syndrome** (officially termed anomalous health incidents, AHI), **catastrophic or decompensated tinnitus**, and **extreme electromagnetic hypersensitivity (EHS / microwave syndrome)**. All three share a strikingly similar descriptive profile: the abrupt onset of an intracranial sound or pressure, intense tinnitus, vertigo, headache, and cognitive, sleep, and mood disturbances. The National Academies report (2020) concluded that the most plausible mechanism for the acute phase of AHI is **directed, pulsed radiofrequency (RF) energy** [1], and the GAO report (2024) and the clinical studies in JAMA (2018–2019) document its sudden-onset phenomenology [2,3,4]. Our central thesis, presented as the authors' hypothesis, is that **the sudden onset of a critical episode does not imply the absence of a prior cumulative load**: the acute event would be the outcome of a model of **cumulative load plus a triggering peak** acting on a **susceptible phenotype**. Within this framework, a sonic component may coexist, but the electromagnetic component (pulsed RF / near fields) can by itself account for the chain of mechanisms, including the microwave auditory effect described by Frey [7,8,9,10], and higher levels of stimulation could shorten and precipitate such critical episodes. This reasoning has a direct civilian implication: sudden-onset cases in the general population could follow the same mechanism of accumulation and peak triggering. Drawing on the authors' field evidence, we document how public urban exposure subjects people to severe fields, on the order of hundreds of V/m of electric field and tens to more than 100 mG of alternating magnetic field, in parks, at bus stops, and in transit areas located near electrical substations, high-voltage lines that cross parks, and electrical rooms adjoining bedrooms in universities, hotels, and resorts, as well as in school zones. The authors' field investigation at Downtown Doral Charter Upper School (DDCUS, 2022) recorded peaks of 200 V/m of ELF electric field and 31 mG of magnetic flux on the sidewalks adjacent to a school [35,36]. We draw a strict distinction between what has been demonstrated and what is proposed: the Artificial Quantum Noise (AQN) framework is a

conceptual hypothesis of the authors, not an established scientific category; and the convergence between AHI, catastrophic tinnitus, and extreme EHS is a phenomenological hypothesis, not a proven etiological equivalence.

Keywords: Havana syndrome; anomalous health incidents (AHI); directed pulsed radiofrequency; microwave auditory effect (Frey effect); catastrophic tinnitus; electromagnetic hypersensitivity (EHS); microwave syndrome; cumulative load; triggering peak; susceptible phenotype; urban ELF exposure; substations and high-voltage lines; electric field; alternating magnetic field; electromagnetic hygiene; Artificial Quantum Noise (AQN).

1. Introduction: Three Conditions, One Descriptive Profile

The four previous articles in this series built, step by step, a pathophysiological framework for tinnitus centered on the redox-environmental axis and on near-field electromagnetic exposure: the first placed oxidative stress and glutathione at the center of cochlear damage [37]; the second developed the concepts of central gain and phantom sound [38]; the third integrated both lines into a two-axis strategy [39]; and the fourth carried that framework into the high-exposure bedroom and nocturnal sympathetic activation [40]. This fifth article takes a step toward the **extremes of the spectrum**: the most severe conditions, with the most abrupt onset, which the literature usually treats as separate and even enigmatic entities.

Our starting point is a phenomenological observation. When the clinical descriptions of **Havana syndrome** (now officially termed anomalous health incidents, AHI), **catastrophic or decompensated tinnitus**, and **extreme electromagnetic hypersensitivity (EHS) or microwave syndrome** are placed side by side, the overlap is striking. The National Academies report describes the acute onset of AHI as "the sudden onset of a perceived loud sound, a sensation of intense pressure or vibration in the head, and pain in the ear or more diffusely in the head," followed by tinnitus, vertigo, unsteadiness, and cognitive difficulties [1]. The same description (intrusive sound, cranial pressure, disabling tinnitus, vertigo, cognitive fog, insomnia, depressed mood) could apply equally to a patient with catastrophic tinnitus [22,23] or to an extreme EHS case of microwave syndrome [11,12].

We state the thesis at the outset to guide the reader. We propose, as the authors' hypothesis, that this resemblance is not coincidental but points to a **common final mechanism** of abnormal neural excitability induced or modulated by electromagnetic fields, and that the three conditions differ in **intensity, directionality, and exposure context** rather than in nature. The central argument of the article follows from this hypothesis: the sudden onset of a critical episode **does not imply** the absence of a prior cumulative load; the acute event may be the visible outcome of a silent accumulation capped by a triggering exposure peak acting on a susceptible phenotype.

2. Havana Syndrome (AHI) and the Directed Pulsed Radiofrequency Hypothesis

Framing note. AHI is a phenomenon whose clinical, geopolitical, and intelligence dimensions are still under discussion. Here we address it solely in its clinical and mechanistic dimension and on the basis of official and peer-reviewed sources, without taking a position on attributions of responsibility or on whether the exposures were deliberate or accidental.

2.1. The Documented Sudden-Onset Phenomenology

Beginning in 2016, U.S. government personnel at the embassy in Havana reported "a sudden onset of symptoms, usually following a loud sound": headache, tinnitus, visual problems, vertigo, and cognitive difficulties [2]. The initial clinical study in JAMA described persistent neurological manifestations in the affected personnel [3], and the subsequent advanced neuroimaging study documented differences in white and gray matter volume, connectivity, and cerebellar structures compared with controls [4]. In its 2024 report, the GAO consolidated the term anomalous health incidents (AHI) and described how they have affected employees and family members both abroad and within the United States [2].

2.2. The Most Plausible Mechanism: Directed, Pulsed RF Energy

The most frequently cited conclusion of the National Academies committee (2020) is unequivocal both in its caution and in its direction: "the committee found the unusual presentation of acute, directional or location-specific early phase signs… to be consistent with the effects of directed, pulsed radio frequency (RF) energy," and "overall, directed pulsed RF energy… appears to be the most plausible mechanism" [1]. In an independent analysis, Golomb had already argued that the clinical picture closely matches the known effects of pulsed radiofrequency/microwave radiation [5]. It is crucial to emphasize that this mechanism is not exotic: it rests on more than half a century of literature on the interaction of pulsed RF with the nervous system, including the microwave auditory effect.

2.3. The Microwave Auditory Effect (Frey Effect) as a Physical Bridge

In 1962, Frey showed that humans can "hear" pulsed electromagnetic energy as a buzzing, clicking, or hissing sound perceived inside the head, without any external acoustic sound [7]. Later work confirmed and characterized this thermoelastic effect: absorption of an RF pulse produces a minute but abrupt thermal expansion in brain tissue that launches an acoustic pressure wave detected by the cochlea [9,10], and the same group even described light perceptions induced by pulsed RF [8]. The Frey effect is therefore an **established physical bridge** between pulsed electromagnetic exposure and the perception of intracranial sound, which is exactly the kind of initial symptom described by people affected by AHI and by many patients with sudden-onset tinnitus and extreme EHS. That a single physical stimulus can produce a perceived "sound" with no acoustic source is the cornerstone of our convergence hypothesis.

3. Phenomenological Convergence: AHI, Catastrophic Tinnitus, and Extreme EHS

A systematic comparison of the three conditions reveals a shared symptomatic core. We summarize it in the following table, with the caveat that this is a **descriptive** comparison, not a demonstration of etiological identity.

Clinical feature	Havana syndrome (AHI)	Catastrophic tinnitus	Extreme EHS / microwave syndrome
Intracranial sound/pressure at onset	Yes (characteristic)	Yes (intrusive tinnitus)	Yes (buzzing, pressure)
Tinnitus	Yes	Yes (defining feature)	Yes (frequent)
Vertigo / unsteadiness	Yes	Frequent	Yes
Headache / cranial pressure	Yes	Frequent	Yes
Cognitive dysfunction / brain fog	Yes	Yes	Yes
Insomnia / sleep disturbance	Yes	Yes	Yes
Anxiety / depression / severe distress	Yes	Yes (suicide risk)	Yes
Link to RF/EMF exposure	Proposed (pulsed RF)	Authors' hypothesis	Proposed (microwaves)

Catastrophic tinnitus is not a figure of speech: the clinical literature uses the term for tinnitus of maximal severity and decompensation, with disabling distress [22], and a meta-analysis documents a pooled prevalence of suicidal ideation close to 20% in tinnitus populations, which places the severe end of the spectrum in a category of seriousness comparable to that of major neuropsychiatric conditions [23]. Microwave syndrome/extreme EHS, for its part, has a historical basis (the microwave irradiation of the U.S. embassy in Moscow between 1953 and 1975) and has been the subject of a review of mechanisms linking it to RF exposure [11,12]. In our view, the overlap in the symptomatic core of the three conditions is too close to be ignored.

The shared mechanistic basis is plausible. Low-intensity RF/microwave exposure produces widespread neuropsychiatric effects, including depression, through non-thermal pathways [6], which have been proposed to be mediated by activation of voltage-gated calcium channels (VGCC) [14], with associated oxidative stress [27,28]. The same cascade (altered neuronal excitability, oxidative stress, autonomic dysregulation) may underlie both the central gain of tinnitus [20,21] and the autonomic-cognitive picture of EHS and AHI.

4. Sudden Onset ≠ Absence of Load: The Accumulation-and-Trigger Model

The most important argument of this article is also the most counterintuitive: **the fact that an episode appears suddenly does not mean that it originated suddenly**. We propose, as a hypothesis, that the critical presentations of AHI, catastrophic tinnitus, and extreme EHS follow a model of **cumulative load plus a triggering peak** acting on a susceptible phenotype.

4.1. The Silent Cumulative Load

In his review of microwave syndrome, Carpenter highlights a key idea: prior exposure sensitizes. He describes how "excessive exposure to EMFs, even for a brief period of time, can induce the syndrome," and how pulsed exposure (smart meters, nearby towers) lowers the symptom threshold [11]. The distinction between peak power and average power, central to Frey's work, explains why a pulsed exposure can be biologically active even when its average appears low [7,10]. The load builds silently: sustained oxidative stress [27,28], background autonomic dysregulation [24,25,26], and chronic suppression of nocturnal repair prepare the ground without yet producing an overt episode.

4.2. The Triggering Peak

On this sensitized ground, an **exposure peak** (a directed RF pulse in the case of AHI, a sudden and intense environmental exposure in the civilian case) acts as the trigger for the critical episode. This explains the paradox of sudden onset: the peak is acute, but it meets a system already loaded up to its threshold. The clinical observation that higher levels of stimulation can **shorten and precipitate** critical episodes of EHS and catastrophic tinnitus, gathered by the authors in their field experience with more than 1,000 EHS cases, is consistent with a system in which dose (cumulative and peak) modulates both the likelihood and the speed of the outcome.

4.3. The Susceptible Phenotype

The same peak does not produce the same effect in everyone. The National Academies report acknowledges this when it attributes the clinical heterogeneity to "variability of exposure dosage conditions… and anatomical differences that could influence individual exposure and/or response" [1]. The literature on human variability in response to EMF points in the same direction [15], and metabolic and genetic screening studies in individuals with EHS have identified polymorphisms (e.g., CYP2C19, GSTT1/GSTM1) associated with differential susceptibility, together with biomarkers proposed to characterize the condition [16,17]. We acknowledge that reviews of provocation studies do not always replicate conscious perception of the fields [18], which is consistent with our thesis: the mechanism we propose is physiological and cumulative, not necessarily conscious detection in real time. In this framework, the susceptible phenotype (whether by genetics, oxidative load, autonomic state, or anatomy) is the one that crosses the critical threshold first.

5. The Civilian Implication: Sudden-Onset Cases in the General Population

If the model of cumulative load plus a triggering peak acting on a susceptible phenotype is correct, it has a consequence that extends beyond the diplomatic or intelligence sphere: **sudden-onset cases of catastrophic tinnitus and extreme EHS in the civilian population could follow exactly the same mechanism**. The difference

would lie in the source of the peak (not a directed beam, but an intense environmental exposure) and in its recognition (civilian cases are rarely investigated as electromagnetic exposures).

This hypothesis reframes a public health problem. Tinnitus shows a high global prevalence and an upward trend, with increasing reports among younger generations [19]. The associated neuropsychiatric conditions (anxiety, depression, insomnia) are also on the rise. The conventional reading attributes them to the psychosocial use of technology; without denying that factor, we propose that **physical exposure to electromagnetic fields** (chronic background exposure punctuated by environmental peaks) is a systematically unmeasured cofactor. The clinical question that follows is concrete: when a patient presents with sudden-onset tinnitus or abrupt-onset EHS with no conventional cause, has the electromagnetic exposure of the patient's environment been characterized? In practice, almost never, even though the EUROPAEM guideline makes exposure reduction the primary measure for EMF-related illness [13].

6. Extreme Urban Exposure: The Environmental Peaks No One Measures

The link that completes the argument is the finding that exposure peaks capable of acting as triggers **are not exceptional and do not require a directed weapon**: they are distributed throughout the city. Field evidence collected by the authors documents how public urban exposure subjects people to severe fields, on the order of hundreds of volts per meter (V/m) of electric field and tens to more than 100 milligauss (mG) of alternating magnetic field, in everyday locations: parks, bus stops, and transit areas, and, of particular concern, near electrical substations, under high-voltage lines that cross parks, in electrical rooms adjoining bedrooms in universities, hotels, and resorts, and in school zones.

6.1. The DDCUS Case (Doral, 2022): The Authors' Field Data

The electromagnetic evaluation of Downtown Doral Charter Upper School (DDCUS), carried out by the authors' team under the IGEF and the NOXTAK Electromagnetic Research Center (1st edition, July 5, 2022), illustrates the problem with concrete data [35,36]. The school is located less than 600 feet from a telecommunications tower, with high-voltage lines running over the adjacent streets. On the outside sidewalks, where children walk and wait, the following peak values were recorded:

Measured quantity	Peak	Average
Multiband RFR (μW/cm²)	7.35	5.86
ELF electric field (V/m)	**200**	170
ELF magnetic flux (mG)	**31**	28

The highest values were recorded between 9:30 and 11:30 a.m., coinciding with the presence of children around the school [35,36]. The symptoms reported in the school community (fatigue, sleep disturbance, nervousness, skin problems, tachycardia, brow pain, difficulty concentrating, pressure and numbness in the head, skin tingling, and chest pressure) correspond to the same autonomic-cognitive cluster that we have described for EHS [11,12]. The report documents a peak of 200 V/m of ELF electric field and 31 mG of magnetic flux on the sidewalk adjacent to 54th Street; the most extreme urban values observed by the authors at other locations (under high-voltage lines and next to substations) reach even greater magnitudes, on the order of hundreds of V/m and more than 100 mG.

6.2. Consistency with the ELF Exposure Literature

These values are not anomalous for the physical environment described. The European review of low-frequency field exposure places typical outdoor urban ELF magnetic fields at 0.05–0.2 μT (0.5–2 mG), but documents increases to several μT (tens of mG) directly beneath high-voltage lines and at the boundaries of substations and transformers [30]. Occupational measurements in high-voltage substations record fields of up to 70 μT (700 mG) in switchgear areas [31,32]. The ELF electric field, for its part, couples capacitively to the body and depends strongly on the distance

from the source [33,34]. The precautionary guidelines of building biology consider levels far below those measured around DDCUS to be anomalous for areas of prolonged occupancy [34]. In other words, urban environmental peaks exist, are measurable, and well exceed precautionary thresholds, yet they are not part of any routine health assessment.

7. Integrated Thesis

Bringing these elements together, we formulate the thesis in the highlighted block below:

Havana syndrome (AHI), catastrophic tinnitus, and extreme electromagnetic hypersensitivity share a descriptive profile so close that it suggests a common final mechanism of abnormal neural excitability modulated by electromagnetic fields, with the microwave auditory (Frey) effect and directed pulsed RF as an established physical bridge. The sudden onset of a critical episode does not imply the absence of a cumulative load: we propose a model of cumulative load plus a triggering peak acting on a susceptible phenotype, in which a sonic component may coexist, but the electromagnetic component is sufficient to explain the chain of mechanisms, and in which greater stimulation shortens and precipitates episodes. It follows that sudden-onset cases in the civilian population may follow the same mechanism, fueled by severe urban exposure peaks (on the order of hundreds of V/m and tens to more than 100 mG) present in parks, bus stops, substations, high-voltage lines, electrical rooms, and school zones, as documented by the authors' field evidence (DDCUS, 2022: peaks of 200 V/m and 31 mG on school sidewalks).

To preserve rigor, we distinguish what the evidence demonstrates from what we propose as hypotheses:

Elements established in the literature:

- According to the National Academies, the most plausible mechanism of the acute phase of AHI is directed, pulsed RF energy [1]; its sudden-onset phenomenology is documented by the GAO and by clinical studies in JAMA [2,3,4].
- The microwave auditory (Frey) effect allows pulsed RF to be perceived as intracranial sound through a characterized thermoelastic mechanism [7,8,9,10].
- RF/microwave exposure produces neuropsychiatric effects and autonomic dysregulation through non-thermal pathways [6,14,24,25].
- There is individual variability and genetic-metabolic susceptibility in the response to EMF [15,16,17].
- Urban environments next to high-voltage lines and substations have elevated ELF fields that are measurable and far higher than urban background levels [30,31,32]; the authors documented peaks of 200 V/m and 31 mG on school sidewalks (DDCUS, 2022) [35,36].

Elements proposed as the authors' hypotheses:

- That AHI, catastrophic tinnitus, and extreme EHS share a common final mechanism of EMF-modulated neural excitability (a phenomenological convergence, not a proven etiological equivalence).
- That sudden onset follows a model of cumulative load plus a triggering peak acting on a susceptible phenotype, and that greater stimulation shortens and precipitates critical episodes.
- That sudden-onset civilian cases follow the same mechanism, fueled by urban exposure peaks that are currently neither measured nor considered.
- That the Artificial Quantum Noise (AQN) framework describes the coherent/polarized component of this exposure [29] (a conceptual framework proposed by the authors, not an established scientific category).

8. Limitations

This work has limitations that define the scope of its conclusions. The convergence between AHI, catastrophic tinnitus, and extreme EHS is a phenomenological hypothesis based on symptom overlap; it does not constitute a

demonstration of etiological identity or of a single mechanism. The mechanism of AHI itself remains under scientific debate, and no official source considers the question settled. The model of cumulative load plus a triggering peak is a proposed explanatory framework that has not been measured prospectively. The authors' field data (DDCUS and urban observations) are point-in-time technical measurements that have not been peer reviewed, and the experience with EHS cases is observational and uncontrolled; these data establish the existence and magnitude of exposures, not a causal relationship with the clinical conditions. The most extreme urban figures cited by the authors (hundreds of V/m and more than 100 mG) correspond to field observations at specific locations and should not be confused with the specific values in the DDCUS report (peaks of 200 V/m and 31 mG). The AQN concept is a proposed theoretical framework that has not yet been validated or independently measured. We therefore present a plausible and coherent framework that integrates official sources, peer-reviewed literature, and our own data, not a demonstration of causality.

9. Proposed Lines of Research

- Structured clinical comparison (symptoms, audiology, neuroimaging, autonomic biomarkers) across AHI, catastrophic tinnitus, and extreme EHS cohorts to test the hypothesis of a shared phenotype.
- Systematic urban dosimetry studies (ELF electric field, ELF magnetic field, RF) in parks, bus stops, school zones, and areas around substations and high-voltage lines, with public exposure maps.
- Screening for the susceptible phenotype (genetic polymorphisms, oxidative load, autonomic profile) in patients with sudden-onset tinnitus or abrupt-onset conditions without a conventional cause.
- Modeling of the trigger threshold: the relationship between cumulative load, peak magnitude, and the probability/latency of the critical episode, as the basis for a testable hypothesis.
- Protocols for electromagnetic characterization of the patient's environment as part of the differential evaluation of sudden-onset tinnitus and new-onset EHS.

10. Conclusion

This fifth installment takes the series to the extremes of the spectrum and proposes a bridge between three conditions that the literature has kept separate. The descriptive resemblance among Havana syndrome, catastrophic tinnitus, and extreme electromagnetic hypersensitivity is too close to be coincidental, and the official conclusion that directed pulsed RF is the most plausible mechanism of AHI, together with Frey's microwave auditory effect, provides a physical bridge that makes the convergence plausible. Our central thesis, presented with due caution, is that the sudden onset of a critical episode does not rule out a prior cumulative load: the acute event is the outcome of a silent accumulation capped by a triggering peak acting on a susceptible phenotype, and greater stimulation shortens and precipitates these episodes. An unavoidable civilian implication follows: sudden-onset cases in the general population may follow the same mechanism, fueled by severe urban exposure peaks that no one currently measures (hundreds of V/m and tens to more than 100 mG in parks, bus stops, substations, high-voltage lines, electrical rooms, and school zones), as documented by the authors' field evidence. Beginning to measure the electromagnetic load of public spaces and of the patient's environment, and to recognize the susceptible phenotype, may be the step that turns an enigma into a testable hypothesis and, above all, into a preventable exposure.

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