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Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

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Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls

Narrative Literature Review

Abstract—Attention problems are common; ADHD is a specific developmental diagnosis. Misdiagnosis occurs when current distractibility, restlessness, poor initiation, or impulsivity is accepted without establishing developmental onset, pervasiveness, impairment, and exclusion of better explanations. The reverse error also occurs when genuine ADHD is attributed entirely to anxiety, depression, personality, poor motivation, or modern technology. This narrative review examines diagnostic pitfalls in adults, including symptom-list matching, anchoring on a self- or prior diagnosis, overreliance on rating scales or medication response, failure to assess impairment, and neglect of collateral or developmental evidence. Mimics and modifiers include sleep deprivation and sleep disorders, anxiety, trauma, depression, bipolar disorder, substance use, medication effects, medical and neurological conditions, learning disorders, autism, and environmental overload. Comorbidity is common, so identifying an alternative does not automatically exclude ADHD. A structured differential uses chronology, episode pattern, context specificity, functional analysis, and response to environmental change. Treatment trials should have predefined outcomes and cannot serve as diagnostic proof. The review proposes language for communicating uncertainty and correcting a label without invalidating the person's difficulties. Accurate diagnosis replaces moral explanations with a testable account while avoiding use of one diagnostic label to explain every difficulty.

Index Terms—ADHD, misdiagnosis, adult ADHD, differential diagnosis, screening, sleep, mood disorders, diagnostic error

I. INTRODUCTION

ADHD is simultaneously underrecognized and vulnerable to overextension. Adults who were missed in childhood may finally receive a coherent explanation; others may adopt the label because online examples accurately describe distress but not its cause. Both realities can exist. The ethical task is not to defend a preferred prevalence, but to apply the developmental and differential criteria consistently. European consensus emphasizes a comprehensive lifespan assessment and the frequent presence of comorbidity (Kooij et al., 2019). NICE guidance likewise requires diagnosis by an appropriately qualified professional on the basis of a full clinical, developmental, psychosocial, and mental-state assessment rather than rating scales alone (National Institute for Health and Care Excellence, 2018).

JPPC articles on ADHD and depression illustrate why treatment difficulty should prompt reassessment of diagnosis, comorbidity, communication, and functional targets (Haverkampf, 2017a; Haverkampf, 2017b; Haverkampf, 2017c). This review focuses on where assessment goes wrong and how to correct it without returning to moral language such as laziness or lack of discipline.

II. SCOPE AND REVIEW METHOD

This article is a narrative literature review with a literature-search cutoff of 30 August 2022, one month before the nominal issue date of 2022 Sep 30. Searches covered adult ADHD diagnostic standards, developmental persistence, comorbidity, symptom screening, differential diagnosis, late-onset debate, medication evidence, and diagnostic error. Priority was given to professional guidance available by 30 August 2022, systematic or umbrella reviews, meta-analyses, longitudinal studies, diagnostic research, and clinically informative original studies. Bibliographic details were checked against PubMed records, the Crossref DOI registry, or the issuing organization's page. Relevant JPPC articles were read in the local archive and used where they materially informed the present question.

The review is not a systematic review and does not estimate a pooled effect. It is intended to support recognition, formulation, and shared clinical reasoning. Absence of a citation does not imply absence of evidence, and the applicability of group findings to one person depends on setting, development, culture, medical status, exposure, and the quality of the assessment. Guidance cited here should be checked for later updates before clinical use.

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Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

Core propositions (author-developed clinical synthesis; not a validated diagnostic instrument)

- Current attention difficulty is real even when ADHD is not the best explanation.
- Developmental chronology and cross-setting impairment are essential discriminators.
- Comorbidity means that finding anxiety, depression, trauma, or substance use does not automatically settle the case.
- Rating scales and stimulant response cannot confirm diagnosis.
- Correcting a label should preserve support for the functional problem and explain the new evidence.

III. THE FOUR QUESTIONS A SYMPTOM LIST CANNOT ANSWER

A symptom list cannot establish onset, pervasiveness, impairment, or cause. An adult can endorse losing things, procrastinating, interrupting, restlessness, and poor concentration during a depressive episode, chronic insomnia, trauma, stimulant use, or an impossible workload. ADHD becomes more likely when related difficulties form a persistent developmental pattern across settings and produce clinically important consequences not better explained by another condition.

The ASRS is a validated screener, not a diagnostic instrument (Kessler et al., 2005). Its positive predictive value changes with the population tested. In a specialty clinic, endorsement means something different from an unselected online audience. Item examples should be explored: what was lost, how often, in which setting, under what level of structure, and with what consequence?

Impairment is sometimes inferred from distress alone or from a high-achieving person's effort. Both deserve attention, but diagnostic impairment should be concrete. Conversely, good grades or employment do not exclude ADHD when support and compensation conceal cost. The assessor must describe the pathway between symptom and consequence, not merely count achievements. Diagnostic criteria organize the decision, but symptom resemblance alone is insufficient: developmental, functional, and exclusion evidence is still required (American Psychiatric Association, 2013).

Minimum evidence domains before assigning adult ADHD (author-developed clinical synthesis; not a validated instrument)

Domain	Insufficient shortcut	Stronger evidence
Development	'I have always felt different'	Specific early examples and converging records or accounts
Pervasiveness	Difficulty in one disliked role	Related pattern across relevant settings or demands
Impairment	High symptom score	Concrete safety, role, relationship, or autonomy consequences
Differential	No obvious medical illness	Active review of mood, anxiety, trauma, sleep, substances, medicine
Context	Symptoms worsen with remote work	Determine whether structure reveals or causes the difficulty
Validity	Improves with stimulant	Diagnosis established independently of treatment response

IV. DEVELOPMENTAL AND 'LATE-ONSET' PITFALLS

Adult recall is fallible, and childhood documentation can be biased toward disruptive behavior. Requiring a report-card phrase can miss quiet inattention; accepting a reconstructed childhood solely because the current diagnosis feels explanatory risks confirmation bias. The solution is graded evidence: early routines, repeated comments, unfinished work, forgetfulness, peer patterns, family descriptions, and the structures that may have compensated.

Longitudinal research complicates a simple continuity story. Moffitt et al. (2015) identified an adult ADHD group with little overlap with the cohort's childhood group, while Sibley et al. (2017) demonstrated that persistence estimates depend on definition. These findings justify research and careful assessment; they do not eliminate developmental criteria. Apparent adult onset should trigger a particularly thorough search for sleep, mood, substance, medical, cognitive, and environmental explanations.

Rising demands can expose a longstanding vulnerability. A student may manage under parental structure but struggle after moving to independent living; a worker may change from an active role to unstructured administration. This is not truly new onset if related patterns can be found earlier. The chronology should distinguish first impairment, first recognition, and first symptom—three dates that are often different.

V. MOOD, ANXIETY, AND TRAUMA

Depression can produce reduced initiation, slowed thinking, forgetfulness, and loss of sustained effort. These often track a mood episode and improve with recovery. Anxiety can make attention highly efficient for threat but unavailable for the task; checking and reassurance consume working memory. Trauma-related hypervigilance, intrusions, avoidance, and dissociation can also appear as distractibility or inconsistent presence. A dimensional review of adult ADHD comorbidity shows why these overlapping domains must be assessed rather than treated as exclusions by default (Katzman et al., 2017).

Bipolar activation is a high-consequence differential when distractibility, talkativeness, activity, risk, and reduced sleep

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

occur in a distinct episode. Reduced need for sleep is different from wanting sleep but being unable to obtain it. ADHD and bipolar disorder can co-occur, so the question is not which label wins but which features are trait-like and which are episodic.

Communication-Focused Therapy for depression may generate hypotheses about how reduced connection and information exchange accompany impaired action (Haverkampf, 2017b); it does not establish the cause of executive complaints. The empirical question is whether developmental and cross-setting evidence for ADHD persists after mood and context are adequately described. Differential assessment should examine depressive and bipolar course, anxiety severity, and trauma exposure rather than assuming that distractibility identifies its own cause (Malhi et al., 2018; Daveney et al., 2019; Singh et al., 2017; Craske et al., 2017; Spitzer et al., 2006; Koenen et al., 2017).

Trait, episode, and threat-linked patterns (author-developed clinical synthesis; not a validated instrument)

Pattern	Chronology	Examples
ADHD-like trait	Developmental, persistent, demand-sensitive	Forgetting, time estimation, inhibition across roles
Depressive episode	Onset with mood/interest/energy change	Slowed initiation and concentration
Bipolar activation	Distinct departure with reduced sleep need	Accelerated activity, risk, speech, confidence
Anxiety	Worsens around uncertainty or evaluation	Threat capture, checking, avoidance
Trauma	Linked to reminders, hyperarousal, dissociation	Scanning, intrusion, shutdown

VI. SLEEP, SUBSTANCES, MEDICATION, AND MEDICAL CONDITIONS

Insufficient sleep impairs vigilance, working memory, inhibition, and emotional regulation. Obstructive sleep apnea, restless legs, circadian disruption, shift work, and insomnia should be assessed. A person may also use caffeine or stimulants to compensate, creating a cycle of activation and poorer sleep. Improvement after sleep treatment does not exclude coexisting ADHD, but it may substantially change the presentation.

Alcohol, cannabis, sedatives, stimulants, and other substances affect attention during intoxication, withdrawal, and recovery. Prescribed medication may activate, sedate, or impair cognition. The timeline should include dose changes and nonprescribed use without assuming misconduct. Cardiovascular, endocrine, neurological, sensory, pain, and post-infectious conditions are considered according to history and examination.

Neuropsychological testing may help characterize learning, memory, intellectual, or neurological questions, but no cognitive profile is specific enough to serve as an ADHD laboratory test. Faraone et al. (2015) review ADHD's multifactorial basis; routine diagnosis remains clinical. A normal performance under quiet, novel, one-to-one conditions does not automatically reproduce daily demands, while poor performance may have many causes. Chronic stress can impair prefrontal functions, and chronic insomnia has a specific evidence-based treatment literature; both observations support active assessment but neither is a diagnostic shortcut (McEwen, 2007; Trauer et al., 2015).

VII. AUTISM, LEARNING, AND ENVIRONMENTAL FIT

Autism and ADHD can co-occur, and both can involve executive difficulties, sensory regulation, intense interests, or social consequences. The developmental social-communication pattern and restricted or repetitive features require their own assessment. Learning disorders can produce task-specific avoidance and apparent inattention; language, hearing, vision, and educational opportunity also matter.

Digital interruption and fragmented work can make attention unreliable without causing ADHD. Constant alerts, rapid switching, unclear priorities, and surveillance reduce the conditions for sustained focus. An environmental trial—notifications changed, instructions clarified, tasks sequenced—can reveal modifiable contributors. It is not a diagnostic test, because people with ADHD also benefit from structure.

A fair assessment does not use context to explain away disability, nor diagnosis to excuse a harmful context. It specifies the interaction. This opens more interventions: accommodations, role redesign, sleep treatment, trauma care, substance support, or ADHD treatment as indicated.

Differential diagnosis: evidence to seek (author-developed clinical synthesis; not a validated instrument)

Alternative or comorbidity	Evidence beyond 'poor focus'
Sleep disorder	Sleep opportunity, snoring/apnea, schedule, daytime sleepiness
Depression/anxiety	Episode or threat-linked time course and core symptoms
Bipolar disorder	Distinct activation, reduced sleep need, consequences
Trauma	Intrusions, hyperarousal, avoidance, dissociation, exposure
Autism/learning	Developmental social, sensory, restricted, or task-specific pattern
Substance/medication	Temporal relationship to use, withdrawal, or dose
Environment	Workload, interruptions, ambiguity, opportunity and fit

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

VIII. TREATMENT RESPONSE IS NOT A DIAGNOSTIC TEST

Subjective improvement after stimulant exposure cannot confirm ADHD; medication response does not establish diagnostic validity. Expectation, novelty, and concurrent structure may affect perceived change. A medication trial should follow a defensible assessment, include safety review and monitoring, and define observable target behaviors before treatment. Cortese et al. (2018) synthesize comparative medication efficacy and tolerability, not diagnostic validity.

Nonresponse has many explanations: an incorrect target or diagnosis, an inadequate trial, nonadherence, adverse effects, sleep loss, comorbidity, or substance use. Haverkampf's (2017c) treatment-resistant adult ADHD article similarly emphasizes comorbidity and monitoring. Diagnostic review should precede reflexive escalation. Borrowed or non-prescribed stimulants introduce additional cardiovascular, sleep, mood, anxiety, misuse, and substance-use risks; disclosure should prompt nonjudgmental safety assessment, not be treated as diagnostic evidence. Medication should not be started, stopped, or altered without the prescriber.

Psychological and environmental interventions can help regardless of whether every symptom is attributed to ADHD. External cues, task decomposition, protected focus, explicit agreements, and emotional-regulation work address real functional problems. Their usefulness does not prove or disprove etiology.

IX. CORRECTING A DIAGNOSIS WITHOUT WITHDRAWING HELP

When evidence no longer supports ADHD, the clinician should name what remains true: concentration, organization, shame, or impairment. Explain which criterion was not met and what alternative formulation better fits. A correction should come with a care plan, not an empty space. If records or medication are affected, responsibilities and safe transition must be explicit.

When ADHD was previously missed, acknowledge how stereotypes or limited information contributed. The new diagnosis should not rewrite every life event as a symptom. It is one explanatory model that organizes some patterns, supports accommodations or treatment, and remains open to comorbidity.

Diagnostic humility can be communicated directly: ‘The current difficulties are clear. The evidence for childhood onset is incomplete, and sleep and depression could account for part of the pattern. We will address those and continue the developmental assessment.’ This is more informative than either instant certainty or indefinite ambiguity.

X. CONTEXT, EQUITY, AND ACCESS

Clinical thresholds are not culturally or socially weightless. Opportunity, exposure, power, language, disability, service design, and prior treatment affect both symptoms and the evidence available to an assessor. Equity does not mean applying less rigorous criteria; it means collecting better contextual evidence and avoiding an interpretation that mistakes structural constraint for individual pathology.

Stereotypes. Gendered and racialized expectations can produce both missed diagnosis and overinterpretation of behavior. Assessment should ask how referral, discipline, compensation, and perceived impairment were shaped by those expectations while applying the same developmental, functional, and differential standards to the final decision.

Digital information. Online communities can improve recognition and give language to hidden effort, but symptom matching without prevalence or differential context can also create false certainty. Clinicians should ask what the person found recognizable, test those propositions collaboratively, and distinguish resonance from screening and diagnosis.

Records. People with disrupted schooling, migration, care experience, or estranged family may lack collateral evidence. The record should distinguish missing information from evidence of absence, state the resulting uncertainty, and identify a fair next step. Limited access should not be treated as proof against ADHD, treatment resistance, or lack of motivation.

XI. MEASUREMENT, FOLLOW-UP, AND REVISION

Follow-up should begin before intervention by defining what will be observed, at what interval, and by whom. Symptom measures can be useful, but they should be paired with function, safety, adverse effects, and the person's priorities. The aim is not to accumulate data; it is to notice whether the working formulation predicts the course and whether the chosen action produces a benefit worth its cost.

XII. A DETAILED INTERVIEW GUIDE

The interview should test the diagnosis without asking the patient to defend it. For each criterion, obtain an example, its setting, and its earliest credible date; then separate first symptom, first impairment, and first recognition. Compare lifelong patterns with changes linked to sleep, mood, trauma, substances, illness, or environmental overload. Records and collateral history can clarify development when available and consented, but absence of childhood documentation is not itself disproof. If evidence conflicts, name the uncertainty and agree on the next discriminating step instead of converting a screening score or medication response into a verdict.

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

Each domain below can alter diagnosis, urgency, intervention, or the interpretation of outcome. The prompts are not a script that must be completed in one sitting. They identify information to obtain at a pace compatible with safety, development, capacity, consent, and the clinical setting. When collateral information is useful, its purpose and limits should be discussed, and the source's opportunity, relationship, and possible bias should be considered.

Criteria audit. Review what evidence was obtained for each current criterion and which conclusions were inferred from a screening score or global impression. Ask for a concrete example, setting, frequency, and consequence rather than accepting terms such as distractible or impulsive as self-explanatory. Note when the behavior is absent and which structure, interest, urgency, or support changes it. Separate the patient's report, clinician observation, records, and consented collateral accounts, because each source answers a different question and has different limits. The audit should make missing evidence visible and preserve examples that contradict the preferred formulation rather than counting only endorsements.

Three dates. Distinguish the first related symptom, first clinically important impairment, and first recognition of the pattern. Rising adult demands can expose longstanding difficulty, while a genuinely new problem may begin with mood, sleep, substance use, illness, or environmental overload. Reconstruct school, home, friendships, hobbies, early work, and the structures that may have compensated. Childhood documentation can strengthen the account but is not the only form of evidence, and its absence should not decide the diagnosis. Record how confident each date is, what source supports it, and whether further records or collateral history can clarify onset without pressuring an estranged or unsafe relationship.

Trait and episode. Compare a persistent developmental pattern with changes tied to depression, bipolar activation, anxiety, trauma, sleep disruption, exposure, or medical illness. Build separate timelines for attention, activity, mood, arousal, sleep, and recovery, noting whether symptoms cluster into a distinct departure from baseline. Reduced need for sleep differs from insomnia, and threat-captured attention differs from broad cross-situational inconsistency, although comorbidity can produce both. Abrupt onset, marked fluctuation, psychosis, focal neurological signs, or full remission after another condition is treated should widen the differential. State which observations support ADHD and which are better explained by an episode or exposure.

Setting. Determine whether difficulty is cross-situational, task-specific, or linked to one environment. Compare work or study, home, relationships, finances, driving, health routines, and preferred activities, and ask which supports reduce impairment. A single hostile workplace, learning mismatch, or constantly interrupted role can generate real executive difficulty without establishing ADHD; conversely, a structured setting may conceal a broader developmental pattern. Describe the sequence from intention through competing information to action and consequence. An environmental change can test a contributor, but benefit from structure is not diagnostic. Record the actual change, its implementation, and the outcome; a retrospective impression that things were better is insufficient.

Alternatives. For each major mimic or comorbidity, record evidence that supports and weakens it. Review sleep disorder, depression, anxiety, trauma, bipolarity, autism, learning difficulty, substance use, medication effects, sensory problems, and relevant medical or neurological conditions. Finding one alternative does not automatically exclude ADHD, and an ADHD label should not make the alternative disappear. Ask which features are developmental, episodic, threat-linked, exposure-related, or confined to a particular task. If the evidence remains mixed, identify the next discriminating history, examination, targeted assessment, or period of observation and continue support for established functional needs while diagnostic confidence remains graded.

Treatment inference. Establish whether medication response, a psychological strategy, or an accommodation was incorrectly used as proof of diagnosis. Ask what target was defined before treatment, whether exposure and adherence were known, and what changed in missed obligations, safety, relationships, sleep, appetite, mood, and substance risk. Subjective alertness after a stimulant is not diagnostic, while nonresponse can reflect dose, implementation, adverse effects, comorbidity, the wrong target, or an incorrect formulation. Marked activation, reduced need for sleep, psychosis, severe anxiety, cardiovascular symptoms, or misuse requires prompt prescriber-led reassessment. Educational material should not direct unsupervised medication change.

Next test. Convert uncertainty into a dated plan rather than an indefinite provisional label. The next step may be collateral history, school or work records, sleep care, mood or substance treatment, targeted medical review, or observation under a defined environmental change. State what question the step is intended to answer, who is responsible, what outcome will be recorded, and when the diagnosis will be revisited. With consent, another person's account may clarify development or function, but it should have a stated purpose and should not overrule the patient or expose them to harm. A correction should preserve care for the real functional difficulty and explain what evidence changed.

Consent, collateral evidence, and record repair should form one transparent process. Explain why another source may help, what information will be requested, and how disagreement will be represented; do not contact a person whose involvement could compromise safety or autonomy. When a diagnosis changes, preserve the earlier conclusion as

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

part of treatment history but label it as superseded, correct factual errors, and update medication indications. The patient should receive a plain-language account of what remains established, what is uncertain, and which practical supports continue regardless of the label.

Follow-up should also test the consequences of correction. Monitor whether sleep, mood, substance care, learning support, accommodations, or medication changes improve the predefined functional problem, and review adverse effects and loss of services. State who holds responsibility during transitions between pathways. A missed appointment or unavailable assessment should not silently turn a provisional decision into a permanent one. If new developmental evidence, activation, psychosis, neurological change, or unsafe stimulant use emerges, reopen the formulation promptly and address urgent risk before completing the diagnostic debate.

Interpret the domains together and distinguish absent evidence from evidence of absence. One response may support several explanations, while silence may reflect memory, shame, language, fear, or how the question was asked. Record the source and confidence of material findings, including contradictions, and state what would increase or decrease diagnostic confidence. When the course does not fit, revise the formulation before escalating treatment by inertia. Accurate repair means neither defending nor withdrawing a label reflexively; it means matching confidence, support, medication safety, and follow-up to the evidence available.

XIII. CRITICAL APPRAISAL AND EVIDENCE TRANSFER

Evidence transfer requires attention to design. Professional guidance integrates evidence and expert judgment for defined services and dates; it is not a substitute for reading the recommendation's population, exclusions, and later updates. Systematic reviews reduce selective citation but inherit the definitions and biases of included studies. Original studies can supply detail and chronology while remaining vulnerable to sampling, measurement, confounding, and chance.

Measurement choices matter because interviews, symptom screens, developmental records, behavioral observations, and treatment outcomes capture different aspects of an ADHD assessment. They cannot be treated as interchangeable: a statistically reliable group difference may be imperceptible to an individual, while a meaningful functional change may be missed by a narrow symptom total. The measure should therefore be named when it affects interpretation and read alongside onset, settings, impairment, alternatives, and adverse effects.

Causal language should follow the research design. Cross-sectional association cannot establish which variable came first. Longitudinal association improves temporal ordering but may retain confounding and measurement error. Randomized allocation supports causal inference for the tested intervention under its trial conditions, not for every mechanism proposed to explain the result. Mechanistic accounts in this review are therefore treated as models that organize testable observations, not as established facts about one person’s hidden cause.

Heterogeneity is clinically informative. Average effects can conceal groups that benefit, do not respond, deteriorate, or could not access the intervention. Trials often exclude people with medical complexity, active substance problems, acute risk, unstable housing, language barriers, or multiple diagnoses. Before applying an estimate, the assessor should compare the study population, treatment competence, dose, outcome, and setting with the decision at hand.

The nominal publication date of this manuscript is 2022 Sep 30. Sources were selected for availability through 30 August 2022, and guidance is identified by the version then available. This updated cutoff supports the reassigned issue date but remains a deliberate limitation: a future reader must check for new safety warnings, revised diagnostic systems, superseded recommendations, retractions, and later comparative evidence before using the review clinically.

Benefits should not be separated from burden and harm. Assessment itself can stigmatize, alarm, disclose sensitive information, or delay more appropriate care. Treatment can cause adverse effects, opportunity cost, dependency on reassurance, financial strain, or deterioration. Monitoring should therefore include unwanted effects and reasons for discontinuation, and the absence of benefit should prompt a review of fit, not an accusation that the person did not try hard enough.

Local JPPC articles are used as prior clinical and theoretical work, not as replacements for external evidence. Their case-based or communication-oriented propositions should be read at the level their methods support. Where they generate a useful question, the present review pairs that question with guidance, systematic evidence, or original research and makes the author's interest in Communication-Focused Therapy explicit.

XIV. DOCUMENTING CONFIDENCE, SECOND OPINIONS, AND RECORD REPAIR

Diagnostic uncertainty should be communicated as a set of propositions rather than a vague reservation. The clinician can state that current executive difficulty is established, specify which developmental or cross-setting criterion remains uncertain, identify the alternatives still being assessed, and name the next source of evidence. This language validates impairment without asking the patient to defend a preferred label. It also permits correction: the explanation can change while the person's difficulty, need for support, and earlier effort remain real.

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

Communication with other clinicians should distinguish a confirmed diagnosis, a working hypothesis, a positive screen, and a historical label. Referral letters and problem lists that collapse these categories can make a provisional impression effectively permanent. When a conclusion changes, the record should explain the evidence and practical consequences in plain language, identify which treatment or accommodation continues, and specify responsibility for medication review or transfer. A person should not lose all care merely because one diagnostic account has narrowed.

The conversation should also anticipate disagreement. A patient may reasonably attach relief, identity, community, or access to the diagnosis, while a clinician may be concerned about missing developmental evidence or a safer alternative explanation. Inviting the patient to correct examples, review the evidential threshold, and see what would increase or decrease confidence can preserve collaboration. If a second opinion is sought, its purpose should be explicit and the reviewer should examine original examples and records rather than simply endorse or reject the prior summary.

A defensible ADHD assessment separates criterion evidence from global impression. The record should show examples for current symptoms, evidence about childhood onset, settings in which the pattern appears, functional impairment, informant or document sources, and active alternatives. Missing evidence should remain visible rather than being converted into a confident negative or positive conclusion. This structure makes it possible for another clinician to understand why the diagnosis was assigned, deferred, or rejected and prevents later notes from copying a label without its original evidential basis.

Confidence can differ across components. Current executive impairment may be clear while developmental onset remains uncertain; ADHD may be probable while a sleep disorder still needs assessment; or ADHD may be established with depression accounting for recent deterioration. A single categorical checkbox cannot communicate these distinctions. The formulation should state what is well supported, what is provisional, and what finding would materially change the decision. Treatment and accommodation can then be matched to the supported component rather than waiting for impossible certainty.

A second opinion is useful when stimulant treatment carries substantial risk, childhood evidence is contested, a previous assessment omitted major alternatives, or diagnosis has significant educational, occupational, or identity consequences. The second clinician should review original examples and records rather than relying on the first report's summary. Agreement adds confidence only if assessments are genuinely independent; disagreement should identify the proposition at issue—for example onset, impairment, or episodicity—and the evidence capable of resolving it.

Diagnostic repair also requires attention to records. Factual errors should be corrected, superseded conclusions labeled as such, and medication indications updated. The earlier diagnosis should not be erased because it forms part of treatment history, but it should not remain the first unqualified label copied into every encounter. The person should receive a plain-language explanation and a copy or route to the revised assessment where appropriate. Transparent documentation reduces both repeated misdiagnosis and the need to relive the entire history at each new service.

XV. SAFETY, URGENCY, AND BOUNDARIES

Misdiagnosis can create direct risk through inappropriate medication, missed mania or psychosis, untreated sleep or substance problems, and loss of care when a label is removed. Safety review should therefore accompany diagnostic revision. Medication changes require prescriber oversight, and urgent states are managed before the full diagnostic debate is resolved.

Urgent and safeguarding considerations (author-developed clinical synthesis; apply current local emergency and safeguarding requirements)

- Assess stimulant misuse or diversion, cardiovascular symptoms, severe insomnia, and interactions.
- Escalate urgently for episodic activation with little need for sleep, psychosis, severe depression, intoxication or withdrawal, dangerous impulsivity, a new focal neurological sign, delirium, catatonia, marked behavioral change, severe medical symptoms, or a rapidly deteriorating mental state.
- If ADHD is not confirmed, continue to address the functional complaint and provide a safe transition to the appropriate pathway.
- Ask directly about suicidal thoughts, self-harm, violence, exploitation, inability to care for basic needs, severe intoxication or withdrawal, and safeguarding concerns when clinically indicated.
- This educational review cannot determine an individual's diagnosis, fitness for work, medication plan, or level of care; those decisions require an appropriately qualified clinician with access to the person and relevant records.

XVI. LIMITATIONS AND RESEARCH NEEDS

Adult ADHD has no diagnostic biomarker, retrospective developmental history is fallible, and several mimics may also be comorbid. Screening performance varies by setting, research on apparently late-onset presentations remains contested, and referral or treatment samples cannot estimate diagnostic error in the wider population. Evidence from children and adolescents should not be generalized automatically to adults, although a systematic scoping review found evidence of overdiagnosis and major gaps about

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

benefits and harms in milder cases (Kazda et al., 2021). These limitations prevent a clean estimate of over- or underdiagnosis from the evidence reviewed here.

Future studies should report how each criterion, developmental onset, and impairment were established, what evidence sources were available, and whether alternatives were tested over time. The most useful outcome is not agreement with an initial label but a formulation that predicts function, preserves safety and support, and changes when new evidence does not fit. This review therefore proposes a diagnostic process rather than a frequency estimate.

XVII. CONCLUSION

ADHD is misdiagnosed when symptom resemblance replaces developmental and differential reasoning. A defensible adult diagnosis requires evidence of a persistent developmental pattern, relevant settings, functional impairment, and active testing of alternatives. Sleep, mood, anxiety, trauma, substances, medication, medical illness, learning, autism, and environmental overload may mimic or modify ADHD, and comorbidity prevents a simple winner-takes-all differential. Screening results and medication response are evidence, not verdicts. Whether confirming or correcting a label, the clinician should preserve support for the person's real difficulty, document what is established and provisional, make the next diagnostic test explicit, and maintain safe continuity of care.

XVIII. CONFLICTS OF INTEREST AND DISCLOSURES

Conflicts of interest—The author developed and writes about Communication-Focused Therapy (CFT). CFT is discussed as a formulation perspective and not as a substitute for established diagnostic assessment, evidence-based treatment, or independent replication. No other conflict of interest was reported.

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Patient material—No identifiable patient material is presented. Any brief examples are generic composites created to illustrate clinical reasoning and are not case reports.

Haverkampf, C. J. (2022). Misdiagnosis of ADHD: Mimics, Comorbidity, and Assessment Pitfalls. J Psychiatry Psychotherapy Communication, 2022 Sep 30;11(3):71-80

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