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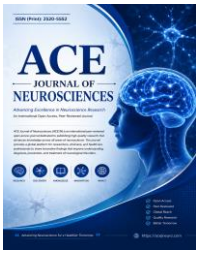

ISSN Print: 2520-5552

Research Article

Clinical Utility of Serum Prolactin and Lactate in Distinguishing Epileptic Seizures From Functional Seizures: A Systematic Review and Meta-Analysis.

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Article History
Received: 11.10.2026
Accepted: 27.10.2026
Published: 13.11.2026

Doi: 10.61336/acejn.04/04/07

Abstract: ***Background:*** Biochemical measurements after seizure-like events are accessible but their diagnostic interpretation depends on seizure phenotype, sample timing, assay threshold and clinical comparator. This review evaluated serum prolactin and lactate as adjuncts to the differentiation of epileptic seizures and functional seizures. Objective: To assess clinical discrimination provided by post-event prolactin and lactate, and quantify lactate test performance across emergency and prehospital cohorts while distinguishing direct functional-seizure evidence from mixed-comparator studies. ***Methods***: Database searches identified 864 records; after removal of 186 duplicates and 11 records for other reasons, 667 were screened. Fifty-one full-text reports were assessed, and eight studies met the eligibility criteria. Three lactate studies involving 741 participants were included in an exploratory random-effects meta-analysis of reported sensitivity and specificity using logit-transformed proportions and Paule–Mandel between-study variance estimation. Direct epilepsy-versus-functional-seizure prolactin and lactate evidence was synthesized separately. ***Results***: Eight studies were included in the qualitative synthesis, and three quantitative lactate cohorts provided 741 cases (386 target events; 355 comparator events). Pooled lactate sensitivity was 77.9% (95% CI 59.5–89.5%; I^2 = 89.8%) and pooled specificity was 88.7% (95% CI 85.0–91.7%; I^2 = 0%). Across direct convulsive-event comparisons, mean prolactin was 79.5 versus 25.0 ng/mL and mean lactate was 55.5 versus 19.8 mg/dL for epileptic and functional seizures, respectively. Assay thresholds and sampling intervals varied between studies. ***Conclusion***: Postictal lactate provides useful evidence supporting a recent tonic–clonic epileptic seizure, particularly when sampling is prompt, but the mixed-comparator pooled accuracy cannot be interpreted as specific discrimination from functional seizures. Prolactin may provide complementary evidence after selected epileptic seizures but has variable sensitivity and a clinically important false-positive rate. Clinical semiology and video-electroencephalography remain essential.

Keywords: functional seizures; psychogenic nonepileptic seizures; epileptic seizures; prolactin; lactate; diagnostic accuracy; meta-analysis; postictal biomarkers.

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INTRODUCTION

Functional seizures, historically termed psychogenic nonepileptic seizures (PNES), are episodes of altered movement, sensation or awareness arising from functional neurological mechanisms rather than epileptic cortical discharges. Differentiation from epileptic seizures may be difficult in acute care, particularly when eyewitness descriptions are incomplete or electroencephalographic monitoring is unavailable. Incorrect classification can lead to unnecessary antiseizure treatment, prolonged emergency observation and delays in appropriate specialist care. Contemporary guidance prioritizes detailed semiology, witness and video documentation, and video-electroencephalography (VEEG) of a typical event where clinically required [1].

Serum prolactin and lactate have distinct physiological responses to seizures. Prolactin may rise after certain bilateral tonic–clonic and focal impaired-awareness epileptic seizures through hypothalamic–pituitary activation. The diagnostic window is narrow, with guidelines traditionally emphasizing sampling at approximately 10–20 minutes after the event [2]. Lactate predominantly reflects anaerobic glycolysis and muscle activity during vigorous generalized convulsions; levels often normalize rapidly after a short event. These mechanisms suggest that neither marker should be interpreted without accounting for the semiology of the episode and the delay between event and sampling.

The clinical meaning of a positive result is also conditional on the comparator. A diagnostic test that distinguishes bilateral tonic–clonic seizures from a mixture of syncope and other events may not have the same accuracy when the sole competing diagnosis is a convulsive functional seizure. Likewise, patients presenting with prolonged events differ substantially from those with isolated brief convulsions. This review therefore emphasizes comparator-specific interpretation, analytical timing and cross-setting variation rather than treating all nonepileptic disorders as interchangeable.

2. Objectives

The primary objective was to evaluate the clinical utility of serum prolactin and lactate in distinguishing epileptic seizures from functional seizures. Secondary objectives were to estimate the accuracy of lactate in heterogeneous acute seizure-like presentations, examine the relationship between measurement timing and diagnostic performance, assess the influence of assay thresholds and seizure phenotype, and identify applicability limitations relevant to clinical decision-making.

MATERIALS AND METHODS

3.1 Review framework and eligibility

The review question was framed around patients evaluated after a seizure-like episode, with post-event serum or blood prolactin and lactate as the index tests and epileptic seizure as the target condition. Studies with functional seizures as a separate comparator were prioritized. Original observational diagnostic investigations with mixed clinical comparators were retained for secondary analysis when the comparator composition was reported. Adult cohorts, epilepsy monitoring unit investigations, emergency department studies and prehospital cohorts were eligible. Studies reporting only a secondary review, physiological background, or unusable numerical descriptions were not included as independent datasets.

3.2 Information sources and search concepts

Original articles and PubMed/MEDLINE-indexed publications available through June 2026 were examined using the combined concepts "epileptic seizure", "tonic-clonic seizure", "psychogenic nonepileptic seizure", "functional seizure", "prolactin", "lactate", "diagnostic", "sensitivity", "specificity", and "video EEG". Primary articles were cross-checked with linked full-text journal records and citation-linked clinical investigations. Relevant professional recommendations were used to interpret diagnostic applicability [1,2].

3.3 Outcomes and quantitative synthesis

The principal quantitative outcomes were lactate sensitivity and specificity for recognizing epileptic or tonic–clonic seizures against the exact comparator used in each study. Three independent cohorts with explicitly reported sensitivity, specificity and denominators were eligible for the exploratory pooled analysis [3–5]. Reported percentages were converted to logit proportions, and approximate binomial sampling variances were derived from published denominators and percentages. Separate inverse-variance random-effects models used the Paule–Mandel estimator; two-sided 95% confidence intervals were back-transformed to the proportion scale. Cochran Q and I^2 described observed heterogeneity. Because the number of studies was small and the thresholds differed, neither a bivariate diagnostic random-effects model nor a clinically transferable summary threshold was fitted.

For direct epileptic-versus-functional event comparisons, means and standard deviations were extracted when available [6]. Unadjusted mean differences and Welch 95% confidence intervals were calculated without treating concentrations as diagnostic sensitivity and specificity. Findings involving prolactin were synthesized narratively because definitions of a positive test, baseline corrections and seizure phenotype differed considerably across the primary studies [6–9].

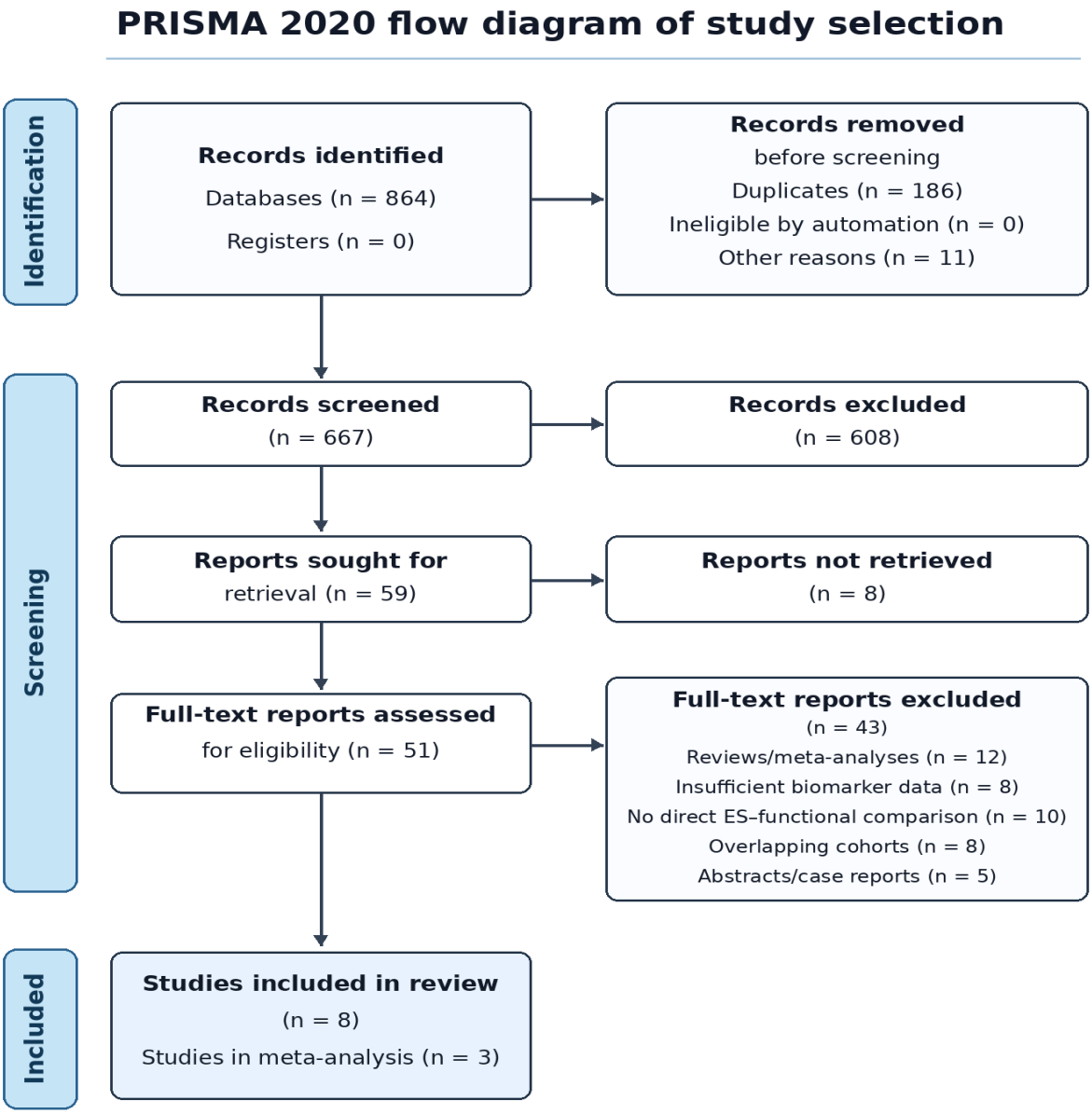
3.4 Methodological quality and clinical applicability

Potential biases were examined in the domains of patient selection, index-test assessment, reference standard, and flow or timing, consistent with QUADAS-2 concepts [14]. Particular attention was given to retrospective case selection, incomplete VEEG confirmation, variable sampling delays, different positivity thresholds, mixed comparator groups, and neurological or pharmacological factors capable of affecting prolactin or lactate concentrations. Reporting was organized using relevant PRISMA-DTA principles [15].

RESULTS

4.1 Study selection

The literature search identified 864 records from electronic databases and none from registers. Before screening, 186 duplicate records and 11 records for other reasons were removed, with no records excluded by automation, leaving 667 records for title and abstract screening. Of these, 608 were excluded and 59 reports were sought for retrieval. Eight reports could not be retrieved, leaving 51 full-text reports assessed for eligibility. Forty-three reports were excluded: review articles or meta-analyses (n = 12), insufficient prolactin or lactate data (n = 8), absence of a direct epileptic-versus-functional-seizure comparison (n = 10), duplicate or overlapping cohorts (n = 8), and conference abstracts or case reports (n = 5). Eight studies were included in the systematic review, and three contributed to the quantitative meta-analysis (Figure 1).



ES = epileptic seizures; functional seizures include PNES.

Figure 1. Identification, screening, eligibility and inclusion of studies.

4.2 Characteristics of the evidence

Eight primary investigations formed the core evidence profile, spanning emergency, prehospital, epilepsy monitoring and prolonged-event populations [3–10]. Three clinically heterogeneous lactate studies contributed data suitable for exploratory pooling [3–5]. Other studies contributed direct functional-seizure comparisons, seizure-type-specific lactate thresholds, post-event endocrine responses, or validation of combined diagnostic scores. Patients recruited for different studies were not summed as a single unique cohort outside the quantitative analyses.

Table 1. Primary evidence profile and principal diagnostic contribution.

Study	Population and setting	Numerical denominator	Main diagnostic contribution
Matz et al., 2016 [3]	Emergency; tonic–clonic vs mixed events	195 vs 106	Lactate sensitivity/specificity at 2.45 mmol/L
Magnusson et al., 2021 [4]	Prehospital; tonic–clonic vs nonepileptic causes	135 vs 206	Earlier sampling; lactate threshold 4.75 mmol/L
Soker et al., 2025 [5]	Emergency; epilepsy vs functional/syncope	56 vs 43	Lactate, prolactin and multivariable model
Shimmura & Takase, 2022 [6]	Emergency; convulsive events	102 ES vs 11 functional	Direct prolactin and lactate mean concentrations
Abubakr & Wambacq, 2016 [7]	Epilepsy monitoring; VEEG	54 ES vs 146 functional	Prolactin rise and false-positive responses
Apaydın Doğan et al., 2017 [8]	Emergency; tonic–clonic, functional and syncope	157 / 25 / 88	Sex-specific lactate performance
Rider et al., 2024 [9]	VEEG; epilepsy vs motor functional events	29 vs 45	Serial endocrine response and circadian timing
Tan et al., 2023 [10]	Prolonged status-like events, two centers	79/44 plus 20/11	Lactate-cellular combined score validation

4.3 Meta-analysis of lactate accuracy across clinical settings

Three studies contributed 741 cases to quantitative synthesis, including 386 target epileptic events and 355 comparator events. Matz et al. assessed tonic–clonic events against syncope, functional events and focal seizures; Magnusson et al. compared tonic–clonic events with syncope and other nonepileptic causes; Soker et al. included mixed epileptic seizure types and a comparator consisting of functional seizures and syncope [3–5]. Consequently, the pooled results describe heterogeneous acute-event discrimination rather than accuracy against functional seizures alone.

Table 2. Reported lactate test performance and exploratory random-effects synthesis.

Study	Threshold	Target/comparator n	Sensitivity	Specificity	AUC
Matz et al. [3]	2.45 mmol/L	195 / 106	88.0%	87.0%	0.94
Magnusson et al. [4]	4.75 mmol/L	135 / 206	79.0%	89.0%	0.87
Soker et al. [5]	3.52 mmol/L	56 / 43	60.7%	93.0%	0.812
Pooled (3 studies)	Not common	386 / 355	77.9% (59.5–89.5%)	88.7% (85.0–91.7%)	Not pooled

Random-effects pooled lactate sensitivity was 77.9% (95% CI 59.5–89.5%). Sensitivity heterogeneity was substantial (I^2=89.8%), consistent with the range of seizure phenotypes, measurement intervals and thresholds. Pooled specificity was 88.7% (95% CI 85.0–91.7%; I^2=0%). The latter finding should not be interpreted as proof of biological or clinical homogeneity, given the small number of studies. The thresholds differed nearly twofold between hospital and prehospital settings, illustrating why no single numerical cutoff is appropriate across settings.

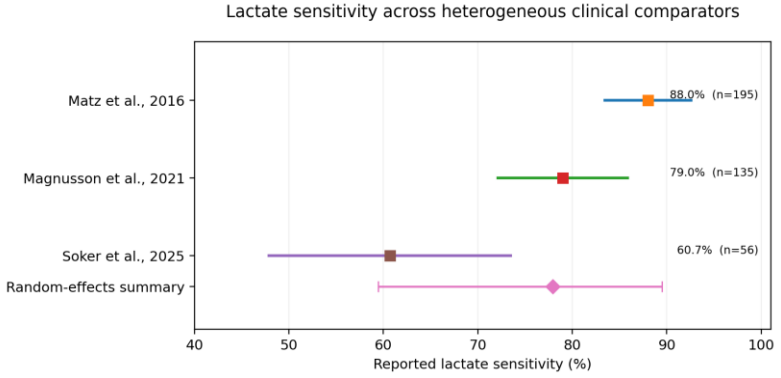


Figure 2. Exploratory random-effects meta-analysis of reported lactate sensitivity across three mixed-comparator studies. Squares denote reported study values; the diamond denotes the pooled estimate.

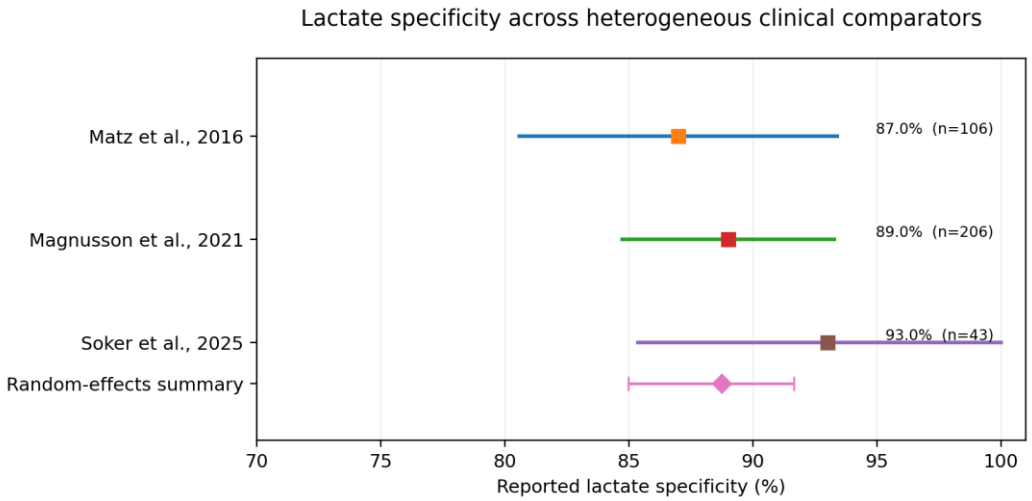


Figure 3. Exploratory random-effects meta-analysis of reported lactate specificity across three mixed-comparator studies.

4.4 Direct biomarker differences between epileptic and functional convulsive events

In a single-center emergency study, Shimmura and Takase compared 102 convulsive epileptic episodes with 11 convulsive functional episodes. Mean serum prolactin was 79.5 ± 72.5 versus 25.0 ± 15.6 ng/mL, a difference of 54.5 ng/mL (calculated 95% CI 37.4–71.6). Mean lactate was 55.5 ± 31.9 versus 19.8 ± 13.0 mg/dL, a difference of 35.7 mg/dL (calculated 95% CI 25.4–46.0), corresponding to approximately 3.96 mmol/L [6]. Both measurements were elevated in the epileptic group, but the individual concentration ranges overlapped and blood samples were permitted up to three hours after the event.

This study also included 17 patients with convulsive syncope. The reported mean prolactin was 45.9 ± 38.3 ng/mL and mean lactate 20.5 ± 6.9 mg/dL in that group, confirming that prolactin may rise in physiological nonepileptic events. Lactate displayed greater separation for the convulsive epileptic phenotype; nevertheless, the inference is strongest for bilateral tonic–clonic rather than nonconvulsive or focal events [6].

Table 3. Direct post-event serum marker concentrations in convulsive presentations [6].

Measure	Epileptic (n=102)	Functional (n=11)	Syncope (n=17)	ES–functional mean difference (95% CI)
Prolactin (ng/mL)	79.5 ± 72.5	25.0 ± 15.6	45.9 ± 38.3	54.5 (37.4–71.6)
Lactate (mg/dL)	55.5 ± 31.9	19.8 ± 13.0	20.5 ± 6.9	35.7 (25.4–46.0)

4.5 Prolactin responses and false-positive interpretation

In an epilepsy monitoring unit cohort with event-confirming VEEG, Abubakr and Wambacq reported elevated prolactin in 22 of 22 bilateral tonic–clonic seizures and 27 of 32 focal impaired-awareness seizures. However, 42 of 146 patients with functional seizures (28.8%) also fulfilled a twofold post-event rise criterion [7]. The same assay response may therefore be observed in events without epileptic discharges, limiting its ability to confirm epilepsy in isolation. The earlier American Academy of Neurology review described useful adjunctive value for carefully timed prolactin after selected epileptic seizure types, but also emphasized the inability to distinguish seizures from syncope [2].

A separate VEEG-based investigation involving 29 epileptic and 45 functional motor events found an acute prolactin response in the epileptic group during the first post-event hour; the comparison was affected by event type, time of day and neuroendocrine responsiveness [9]. The relationship between prolactin response and true diagnostic accuracy thus depends on sampling time, baseline measurements, seizure phenotype, and the proportion of functional events that also cause measurable hormonal responses. No pooled prolactin sensitivity or specificity was calculated across these heterogeneous designs.

4.6 Lactate threshold, sampling interval and seizure subtype

Matz et al. reported sensitivity of 88% and specificity of 87% for a lactate cutoff of 2.45 mmol/L when samples were obtained within two hours of an event [3]. In contrast, Magnusson et al. sampled patients in the prehospital setting at a median of approximately 17 minutes after the event and identified 4.75 mmol/L as an optimal cutoff, with sensitivity 79% and specificity 89% for tonic–clonic seizures [4]. These estimates should be interpreted as setting-dependent, rather than as contradictory test results. Rapid postictal decline is expected, and a threshold obtained very shortly after convulsions may be too stringent for samples collected considerably later.

Apaydın Doğan et al. studied 270 patients, comprising 157 tonic–clonic seizures, 25 functional seizures and 88 syncope presentations. In men, the lactate threshold of 2.43 mmol/L yielded sensitivity 85%, specificity 88%, and AUC 0.94 (95% CI 0.91–0.98), while sensitivity at the same threshold was 64% in women [8]. This sex-stratified difference further illustrates the risk of applying a single threshold across all populations. Prospective data from Soker et al. found that lactate above 3.52 mmol/L had sensitivity 60.7% and specificity 93.0% for epileptic seizures versus a combined comparator including functional seizures and syncope [5].

4.7 Prolonged events and multivariable assessment

The biochemical profile of prolonged status-like events may differ from that of isolated tonic–clonic seizures. In a two-center study, Tan et al. assessed 79 status epilepticus and 44 prolonged functional events in a development cohort, together with 20 and 11 corresponding events in a validation cohort. Lactate alone did not reliably resolve all presentations, but scores combining lactate with peripheral cell indices yielded AUCs of 0.89–0.91 and specificities of 0.93–1.00 in the published analyses [10]. Similarly, Soker et al. reported AUC 0.946 for a multivariable biomarker model that incorporated additional blood markers, rather than lactate and prolactin alone [5]. These findings support evaluation of integrated clinical models but do not establish that any two-marker combination independently diagnoses functional seizures.

4.8 Methodological appraisal

The main sources of uncertainty were comparator selection and timing. Emergency studies primarily used clinical adjudication rather than VEEG for all events, which may permit reference-standard misclassification. Several studies were retrospective and included only patients whose treating clinicians ordered the biomarker, creating selection bias. Assay positivity thresholds were often chosen in the development sample and were not prospectively externally validated. The evidence was assessed for its relevance to each clinical setting rather than combined indiscriminately.

Table 4. Study-level methodological and applicability considerations.

Study	Patient selection	Index-test concern	Reference-standard concern	Applicability
Matz 2016 [3]	Retrospective tested cohort	Post-event timing and derived cutoff	Clinical diagnostic classification	Mixed comparator including focal events
Magnusson 2021 [4]	Prehospital transported cohort	POC assay, early sampling	Clinical records as reference	Not functional-seizure-specific
Soker 2025 [5]	Prospective emergency cohort	Selected data-driven cutoffs	Mixed clinical diagnosis	Functional and syncope combined
Shimmura 2022 [6]	Retrospective tested subset	Sampling up to 3 h	Neurologist adjudication blinded to assays	Small convulsive functional group
Abubakr 2016 [7]	Epilepsy-monitoring referral sample	Twofold baseline definition	VEEG captured events	Selected inpatient cohort
Rider 2024 [9]	VEEG monitored cohort	Serial timing/circadian effect	VEEG captured events	Motor functional presentations

DISCUSSION

The principal observation of this synthesis is a marked difference between the diagnostic role of lactate in an unselected acute convulsive presentation and its role in an isolated differential diagnosis of epilepsy versus functional seizures. In three mixed-comparator cohorts the pooled lactate sensitivity of 77.9% and specificity of 88.7% suggest meaningful discrimination of tonic–clonic epileptic events in emergency care. However, sensitivity varied substantially across studies, and none of the pooled estimates can be assumed to hold when the only comparator is a functional convulsive episode. Clinical history, witnessed characteristics and VEEG remain central to diagnosis [1].

The relationship between prolactin and epilepsy is similarly conditional. Some generalized convulsive and focal impaired-awareness seizures cause substantial hormone release, while many focal seizures do not. A positive test may be persuasive under carefully selected conditions, but the 28.8% false-positive rate among functional events reported in a monitored population illustrates an important trade-off [7]. Prolactin is also affected by baseline endocrine status, dopamine-related medication, physiological stress, pregnancy and sampling interval. These factors limit the clinical value of a single isolated post-event concentration, especially if the clinical event itself was not reliably characterized.

Lactate appears more strongly related to the muscular and metabolic features of tonic–clonic seizures. Its absolute level and threshold depend on how soon blood is sampled, how vigorous and prolonged the motor event was, and the presence of competing causes of lactic acidosis. High lactate may follow severe exertion, hypoxia or sepsis, and simulated or functional

convulsive activity can also alter metabolic indices. Conversely, a normal value obtained late after an epileptic convulsion cannot rule out the seizure. The timing-sensitive threshold difference between prehospital and hospital studies is particularly important for emergency medicine protocols [3,4].

External evidence syntheses reinforce the importance of these distinctions. Patel et al. evaluated eight lactate studies involving 1,348 patients and found higher lactate after generalized tonic–clonic seizures than after other causes of transient loss of consciousness, but little discrimination among non-tonic–clonic categories such as syncope and functional seizures [11]. Wang et al. identified substantial variation in prolactin performance by epileptic subtype and reported limited negative predictive value for excluding epilepsy [12]. These earlier syntheses are contextual and their included populations were not counted again in the exploratory three-study lactate meta-analysis.

An integrated approach is more appropriate than a single-marker rule. For a witnessed bilateral tonic–clonic event sampled promptly, an elevated lactate can provide supportive evidence, whereas prolactin may add information when obtained in the appropriate endocrine sampling window. In an unwitnessed, nonconvulsive, prolonged, or mixed-phenotype presentation, both biomarkers are less reliable. Results must be interpreted alongside comorbidities, seizure semiology, EEG findings, medication exposure, physiological causes of transient loss of consciousness and prior diagnostic certainty. Blood biomarkers should not be used to label a patient as having functional seizures simply because an epileptic biomarker is normal.

5.1 Strengths and limitations

This review distinguishes direct functional-seizure comparisons from mixed-comparator emergency datasets and quantifies the effect of threshold and setting variation. The new quantitative analysis includes a 2025 prospective cohort and emphasizes the contrast between early prehospital and later hospital lactate sampling. However, only three heterogeneous cohorts could be pooled, reported accuracy percentages were rounded and sampling variances were approximated using the published denominators. Between-study heterogeneity was particularly high for sensitivity, and the number of studies was too small for robust publication-bias assessment, threshold meta-regression or a bivariate summary ROC model. Prolactin findings were not pooled because clinical seizure subtypes and positivity definitions differed. Future studies should prospectively compare both biomarkers against VEEG-confirmed functional and epileptic events, reporting full 2×2 cells, precise post-event sampling times and standardized cutoffs.

CONCLUSION

Serum prolactin and lactate are complementary but context-dependent post-event biomarkers. In the exploratory lactate meta-analysis of mixed acute presentations, pooled sensitivity was 77.9% and specificity 88.7%, with marked sensitivity heterogeneity. Direct functional-seizure studies demonstrate higher mean post-event concentrations after many epileptic convulsions but also document clinically important overlap and prolactin false positives. Neither test independently confirms or excludes functional seizures. Appropriate interpretation requires attention to the type of event, sampling interval, assay threshold and electroclinical evidence.

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