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When a Clinically Stable Veterinary Patient Is Not Truly Stable: A Trajectory-Based Decision Architecture for Detecting Early Physiological Deterioration Before Conventional Diagnostic and Monitoring Thresholds Are Crossed

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ABSTRACT

Veterinary patients are commonly classified as clinically stable when vital signs, laboratory values, imaging findings, and observable behavior remain within accepted limits or show no single abnormality that compels escalation. Yet apparent stability can coexist with declining physiological reserve, asynchronous organ-specific change, early injury signals, or discordance among clinical domains. This article develops an original non-empirical clinical decision architecture for interpreting such patients before conventional diagnostic or monitoring thresholds are crossed. The analysis distinguishes isolated measurements from patient-specific trajectories; population reference limits from meaningful within-patient change; physiological compensation from durable stability; and cross-domain discordance from confirmed deterioration. It further proposes that serial evidence should be adjudicated across parallel clinical, laboratory, behavioral, and organ-specific domains while preserving measurement uncertainty, alternative explanations, and disease-specific boundaries. Four decision states—Stable, Vulnerable, Deteriorating, and Rescue-Required—are proposed as action-oriented categories rather than validated prognostic classes. Movement among states is intended to depend on direction and persistence of change, reserve, concordance or discordance among domains, confirmation of unexpected signals, and the time available before plausible harm. The architecture does not provide universal thresholds, monitoring intervals, scores, or treatment triggers. Its principal value is as a testable reasoning structure for deciding when apparent stability warrants continued observation, closer reassessment, diagnostic escalation, or urgent intervention while keeping uncertainty explicit. Prospective validation is required across species, diseases, settings, measurement systems, and clinically meaningful outcomes.

Keywords: Clinical deterioration, Veterinary monitoring, Physiological reserve, Longitudinal trajectories, Diagnostic uncertainty; Reassessment

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Introduction

Clinical stability in veterinary internal medicine is often inferred from a snapshot: acceptable vital signs, laboratory results that have not crossed familiar thresholds, no obvious new organ dysfunction, and an animal whose behavior or examination does not compel immediate escalation. This approach is practical, but it can confuse absence of a threshold crossing with absence of meaningful change. Long-term canine monitoring demonstrates that resting heart and respiratory rates have interpretable patient-specific longitudinal patterns,

illustrating why repeated measurements can reveal information that a single clinic observation cannot represent [1]. The central problem addressed here is therefore not whether thresholds are useful, but whether threshold status alone is sufficient to characterize a changing patient.

Continuous acquisition also does not solve interpretation automatically. In hospitalized dogs, a wireless harness could track heart-rate and respiratory-rate trends while agreement limitations constrained direct substitution for clinical measurement, and temperature performance was less reliable [2]. In intensive-care dogs and cats, respiratory measurements were influenced by observation technique, clinical condition, and time point [3]. Thus, a trajectory architecture must separate signal acquisition, confirmation, and clinical actionability rather than treating repeated data as inherently superior data.

Risk stratification adds another layer but remains non-deterministic at the individual level. In dogs with diabetic ketoacidosis, the APPLEfull score provided only moderate prognostic discrimination, supporting use of composite severity information as one input rather than a stand-alone state classifier [4]. This article consequently proposes a trajectory-based decision architecture that asks five linked questions: Is the patient changing relative to its own recent course? Is reserve likely to be limited? Are clinical domains converging or diverging? Could measurement or context explain the apparent change? And how rapidly must uncertainty be reduced before plausible harm becomes unacceptable? The available evidence is dominated by dogs and cats in monitored clinical settings, so broader species and practice-setting transfer remains a validation problem. The architecture is a proposed synthesis, not a validated prediction model, and preserves disease-, species-, setting-, and measurement-specific limits throughout.

The clinical problem of apparent stability

Apparent stability is best treated as a provisional clinical interpretation rather than a physiological fact. A patient can remain normotensive, nonazotemic, ambulatory, interactive, or free of overt respiratory distress while an organ-specific injury process is evolving. In dogs with spontaneous acute kidney injury, urinary clusterin and cystatin B provided renal-injury information that was not equivalent to conventional renal-function measures [5]. The implication is narrow but important: preserved or only mildly changed function does not necessarily exclude contemporaneous structural injury.

The reverse problem is equally important. A novel or sensitive signal can be abnormal without proving acute deterioration. In cats, urinary cystatin B was higher in acute kidney injury but also overlapped with chronic kidney disease, demonstrating that a biologically plausible injury marker can remain diagnostically ambiguous without clinical context [6]. In dogs, prospective assessment of cell-cycle arrest markers and neutrophil gelatinase-associated lipocalin distinguished some acute kidney injury cases from chronic kidney disease, critical illness without acute kidney injury, and healthy controls, but these findings still did not establish a universal escalation trigger [7].

The proposed architecture therefore treats apparent stability as a confidence statement that must be continually updated. Confidence should fall when new data are inconsistent with the patient's prior trajectory, when a secondary organ system begins to deviate, or when the observed state depends heavily on a single uncertain measurement. Conversely, one isolated abnormal result should not automatically reclassify the patient. The analytical task is to determine whether the signal is persistent, physiologically coherent, reproducible, and consequential within the time available for reassessment.

Why conventional threshold-based monitoring misses early deterioration

Thresholds compress complex distributions into operational categories. They are indispensable for communication and rapid decisions, yet they can be insensitive to clinically meaningful movement that remains within the same category. Their limitation becomes more obvious when measurement precision is imperfect: a device may reproduce a direction of change while lacking sufficient agreement for an absolute treatment decision [2]. In such circumstances, the relevant question is not merely whether a value is above or below a cut point, but whether the observed change exceeds plausible measurement noise and is consistent with other information.

Renal biomarker studies illustrate a second limitation. Structural-injury or cellular-stress signals and conventional renal-function categories are related but non-equivalent, so discordance between them requires interpretation rather than automatic escalation [5-7]. A threshold-only approach can therefore fail in two directions: it may delay attention to a patient whose trajectory is worsening before conventional criteria are met, or it may overreact to a sensitive but nonspecific signal whose meaning is unresolved.

Context can also shift the apparent value of a measurement without a corresponding physiological transition. Respiratory rate and breathing pattern in intensive-care dogs and cats vary with observation method, condition, and time point [3]. The architecture proposed here therefore places every threshold inside a broader interpretive frame: patient-relative change, persistence, cross-domain support, measurement credibility, and time-to-harm. A threshold crossing may sharply increase concern, but absence of one cannot by itself certify stability. Conversely, prethreshold movement is not labeled deterioration until alternative explanations and reproducibility have been considered.

Physiological reserve and compensated disease states

Physiological reserve describes the capacity to absorb stress without overt loss of function. In this article it is used as a clinical concept rather than a directly measured latent variable. Evidence from systemic disease supports the possibility that organ stress can emerge outside the primary presenting system. Dogs with immune-mediated hemolytic anemia had increased urinary neutrophil gelatinase-associated lipocalin relative to healthy controls, although the studied marker was not significantly associated with survival [8]. This pattern is consistent with cross-organ stress but does not establish a quantitative measure of reserve.

Serial biomarker behavior in critically ill dogs further argues against a binary intact-versus-failed view. Neutrophil gelatinase-associated lipocalin and tissue inhibitor of metalloproteinase-2 showed heterogeneous trajectories, with some measures relating to outcome while others were not uniformly prognostic [9]. Such findings support dynamic interpretation of organ-injury burden, but they do not justify mapping any biomarker directly onto a proposed decision state.

Reserve also cannot be inferred from population reference intervals alone. Weekly biological-variation data for urine protein-to-creatinine ratio and urine specific gravity in healthy dogs demonstrate why within-patient change can be clinically different from simply remaining inside or outside a population interval [10]. The proposed architecture therefore treats reserve as a modifier of the meaning of change. A small persistent deviation may deserve greater concern in an animal with limited compensatory capacity, advanced comorbidity, or recent decompensation than in an otherwise robust patient. Because reserve is rarely measured directly, this judgment must remain explicit, revisable, and separated from claims of validated risk prediction.

Temporal trajectories versus isolated clinical measurements

A trajectory is more than a sequence of values. Clinically, it is the direction, persistence, patient-relative magnitude, and cross-domain coherence of change over a relevant time horizon. In dogs with suspected pancreatitis, serial pancreatic lipase immunoreactivity, C-reactive protein, ultrasonography, and clinical severity supplied nonidentical information over time [11]. This supports a trajectory view in which disagreement among modalities is itself interpretable rather than treated as analytical failure.

Daily monitoring in hospitalized dogs with acute pancreatitis similarly showed that clinical signs did not consistently parallel lipase measurements [12]. Such discordance can arise because biomarker kinetics, organ recovery, treatment effects, and observable behavior operate on different time constants. It should not be assumed that the slower or faster domain is necessarily more truthful. Instead, the clinician must ask whether divergence is expected for that disease and treatment phase, or whether it signals a new process requiring reassessment.

Longitudinal echocardiographic and cardiac biomarker analysis in dogs with atrial fibrillation further demonstrates that organ-specific phenotypes are time-dependent rather than reducible to baseline status [13]. The proposed architecture therefore prioritizes trajectory features that can be examined without inventing universal numerical slopes: direction of change, persistence across repeated observations, departure from the patient's recent baseline, and convergence or divergence among domains. The clinically relevant time window is likewise contextual: an informative trajectory in an acutely deteriorating inpatient may require much closer sampling than one in compensated chronic disease. An isolated abnormality can still be decisive when severe, but apparently acceptable values should carry less reassurance when several domains are moving coherently toward deterioration.

Cross-domain discordance as an early warning signal

Cross-domain discordance occurs when different evidence streams imply different clinical states. It is not inherently pathological, because signs, imaging, biomarkers, and organ function can change on different schedules. Its value lies in lowering confidence that a simple stable-versus-unstable label is adequate. In dogs presenting with respiratory signs, vertebral left atrial size and vertebral heart size added organ-specific information

for detecting congestive heart failure, illustrating how a nonspecific presenting sign can require adjudication with a more mechanistically linked domain [14].

Discordance is also relevant during treatment. In cats receiving toceranib, proteinuria, azotemia, and hypertension were tracked as partly independent monitoring domains [15]. A patient can therefore appear stable by one measure while another domain develops a clinically relevant abnormality. The appropriate response is not to assume drug toxicity or deterioration, but to investigate whether the new signal is persistent, treatment-related, disease-related, or artifactual.

A more explicit cross-organ example is acute pancreatitis. In affected dogs, urinary neutrophil gelatinase-associated lipocalin was increased even in some animals without conventional acute kidney injury classification, whereas fractional-excretion measures were less convincing as early markers [16]. This pattern supports discordance as an early-warning cue but not as a rescue trigger. The proposed architecture uses cross-domain discordance to shorten the distance to verification: repeat the questionable measure when feasible, examine a mechanistically related domain, reassess the animal sooner, and escalate only when persistence, concordance, organ dysfunction, or time-to-harm makes continued uncertainty unsafe.

Table 1 maps each mismatch to the evidence streams in conflict and the discriminator most likely to reduce uncertainty without assuming that either stream is automatically correct.

Table 1. Cross-domain mismatches classified by the evidence streams in conflict and the verification question they create

Mismatch being reconciled	First evidence stream	Second evidence stream	Why disagreement may occur	Most informative next discriminator
Symptom-organ mismatch	Nonspecific clinical sign	Organ-specific assessment	Competing organ sources or imperfect discrimination in either stream	Confirm a mechanistically linked organ discriminator promptly
Function-injury mismatch	Conventional functional classification	Injury or cellular-stress signal	Injury may precede functional change; chronic disease or marker overlap may also contribute	Repeat or triangulate the injury signal with function and clinical trajectory
Treatment-domain divergence	One monitored domain remains stable	Another domain develops an abnormality	Drug exposure, comorbidity, and disease effects may evolve independently	Investigate each domain separately and shorten follow-up if divergence persists
Multimodal kinetic lag	Improving clinical, laboratory, imaging, or behavioral stream	A different stream remains abnormal	Modalities may index different processes and time constants	Follow the unresolved high-consequence stream on its appropriate timescale

Note: The mismatch classes organize verification; they are not validated scores, thresholds, or state-transition rul

Integrating vital signs, laboratory trends, behavior, and organ-specific indicators

Integration should not mean collapsing every observation into a single undifferentiated impression. Serial pancreatitis data show that clinical severity, inflammatory markers, pancreatic biomarkers, and ultrasonographic findings can change on different schedules [11]. The proposed architecture therefore retains separate evidence lanes long enough to determine whether each domain is improving, stable, worsening, or uncertain. Vital signs and behavior provide immediate clinical context; laboratory trends characterize selected physiological processes; and organ-specific tests can clarify mechanisms that general observations cannot resolve.

This separation matters when a sign is nonspecific. In dogs with respiratory signs, cardiac radiographic indices added information for adjudicating congestive heart failure [14]. Organ-specific evidence can therefore change confidence in the interpretation of a general sign without becoming a universal arbiter. Likewise, renal injury signals during pancreatitis illustrate how a secondary organ domain may diverge from the primary syndrome [16]. Such discordance should prompt confirmation and appropriately earlier reassessment rather than automatic reclassification.

The proposed integration rule is qualitative: establish measurement credibility, compare each domain with its own recent course, and then evaluate convergence or divergence. Behavior deserves explicit consideration because changes in interaction, mobility, appetite, posture, respiratory effort, or ordinary activity may contextualize numerical measurements, while hospitalization, stress, analgesia, and observer effects can modify those

observations. No fixed weighting is proposed. A signal should exert greater influence when it is reproducible, mechanistically relevant, temporally coherent, and consequential for near-term harm; uncertainty should remain explicit when those conditions are not satisfied.

A proposed trajectory-based deterioration architecture

The proposed architecture treats deterioration as a time-dependent adjudication problem rather than a single threshold event. In dogs with systemic inflammation, new-onset organ dysfunction provided distinct screening and prognostic information, supporting newly emerging functional loss as a high-concern trajectory feature [17]. Host-response biomarkers such as apolipoprotein A1 and serum amyloid A represent a different information layer related to systemic inflammation and illness severity [18]. Endothelial and inflammatory biomarker studies further demonstrate that biological activation signals can accompany septic organ dysfunction without establishing that every measured marker is specific, prognostic, or directly actionable [19].

Three dimensions are therefore kept analytically separate. Observed trajectory concerns repeated vital, behavioral, laboratory, and organ-specific change relative to the patient's recent course. Vulnerability concerns the inferred capacity to tolerate additional stress, potentially modified by age, comorbidity, compensated disease, prior decompensation, and treatment burden. Decision urgency concerns how long uncertainty can reasonably persist before plausible harm favors confirmation, diagnostic escalation, or rescue over continued observation. Cross-domain concordance strengthens confidence; discordance triggers adjudication; and measurement concerns divert a signal into verification.

Movement among states is proposed to be reversible rather than linear. A vulnerable patient may return toward stable status after a transient or artifactual abnormality resolves, whereas an apparently stable patient may move rapidly toward deterioration when independent domains worsen coherently. Rescue-Required is not defined by a universal number; it represents a condition in which delay for routine observation is no longer proportionate to plausible immediate harm. Reassessment feeds back into each dimension, permitting escalation or de-escalation as new information changes confidence.

The simultaneous roles of time, parallel evidence domains, verification, reserve, state transitions, and feedback are therefore represented visually rather than as a one-directional sequence **(Figure 1)**.

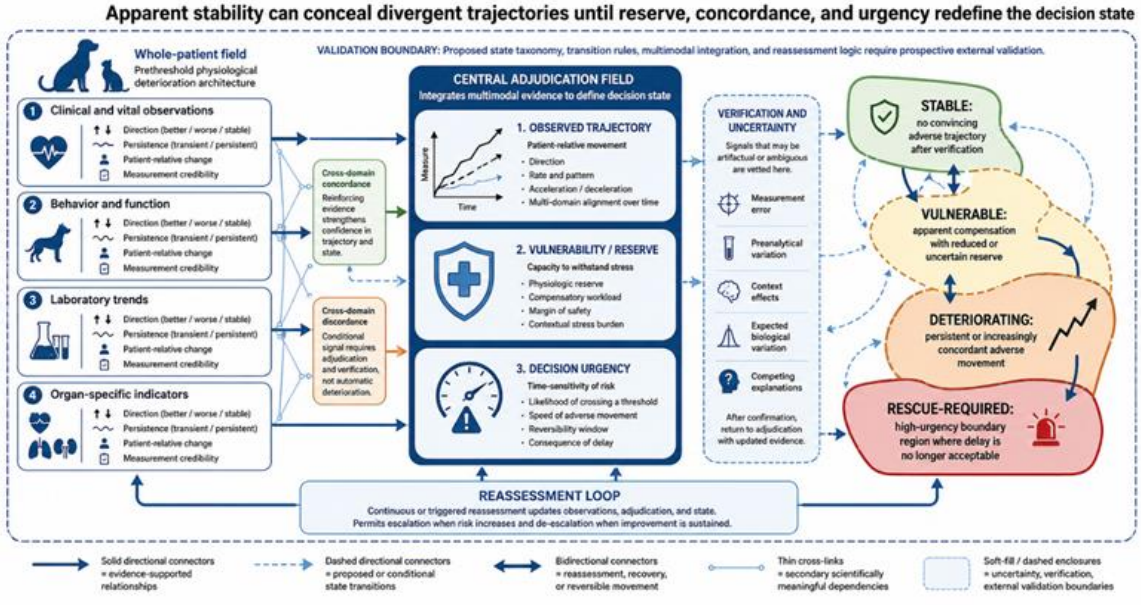

Figure 1. Apparent stability can conceal divergent trajectories until reserve, concordance, and urgency redefine the decision state

Stable, vulnerable, deteriorating, and rescue-required decision states

The four decision states are proposed action-oriented categories rather than diagnostic labels or validated prognostic strata. Vulnerability is included because reduced reserve can be clinically meaningful before overt instability. A canine frailty phenotype has been prospectively evaluated in aging dogs and was associated with subsequent mortality, demonstrating that vulnerability can be operationalized in one bounded population [20].

The proposed Vulnerable state is broader than geriatric frailty: it describes an apparently compensated patient whose capacity to tolerate additional physiological stress is reduced or uncertain.

Deteriorating denotes a persistent or increasingly coherent adverse trajectory warranting active diagnostic or therapeutic escalation even if no universal rescue threshold has been crossed. Etiologic certainty need not be complete. In dogs with suspected primary immune thrombocytopenia, prospective serial assessment characterized clinically relevant bleeding severity despite the absence of a single definitive diagnostic test [21]. Current decision state and diagnostic certainty should therefore remain separate.

Stable denotes no convincing adverse trajectory after expected variation, measurement credibility, and contextual modifiers have been considered. Compensated disease does not automatically imply durable stability: longitudinal observations in dogs with preclinical myxomatous mitral valve disease demonstrate measurable change before overt congestive failure [22]. Rescue-Required denotes a proposed high-urgency condition in which plausible immediate harm makes delay for routine observation inappropriate. It is defined by action necessity and time-to-harm, not a universal laboratory or vital-sign value.

Table 2 compares the four proposed states across the dimensions that actually distinguish them, making the change in clinical posture visible without converting the architecture into a score.

Table 2. How trajectory, reserve, concordance, and time-to-harm differentiate the four proposed decision states

Decision dimension	Stable	Vulnerable	Deteriorating	Rescue-Required
Patient-relative trajectory	No convincing adverse movement	Compensated or subtly changing	Persistent or increasingly coherent worsening	Severe or rapidly worsening dysfunction
Physiological reserve	Not evidently limited	Limited or uncertain	Being depleted or clinically consequential	Insufficient for safe delay
Cross-domain pattern	Reproducibly nonworsening	Subtle or discordant concern	Concordant or consequential worsening	Immediate-threat pattern; full concordance is not required
Tolerance for unresolved uncertainty	Context-appropriate observation remains acceptable	Lower tolerance; confirmation should occur earlier	Etiology may remain uncertain, but action cannot remain routine	Delay for routine observation is disproportionate to plausible harm
Clinical posture	Continue monitoring	Verify sooner and protect reserve	Escalate evaluation or treatment	Stabilize urgently
Reassessment cadence	Set by disease and context	Earlier than routine	Shortened as the trajectory evolves	Immediate or continuous

Note: Stable, Vulnerable, Deteriorating, and Rescue-Required are proposed action-oriented states, not validated prognostic classes or universal treatment thresholds.

Reassessment frequency and escalation thresholds

Reassessment frequency should reflect the expected rate of clinically meaningful change rather than follow one schedule across diseases. Serial blood biomarkers in dogs with pneumonia, septic peritonitis, and pyometra demonstrate dynamic disease-course kinetics visible only through repeated measurement [23]. The proposed architecture therefore increases reassessment intensity when an adverse trajectory steepens, discordance persists, reserve appears limited, or uncertainty concerns an outcome with little tolerance for delay.

Reassessment must also respect modality-specific resolution. In dogs treated for aspiration pneumonia, clinical findings and C-reactive protein could improve on a different timeline from thoracic imaging abnormalities [24]. Persistent imaging change should not automatically prevent de-escalation, but neither should it be dismissed without considering disease phase and the consequence of misclassification. The unresolved high-consequence domain should guide what requires verification next.

The principle extends to chronic disease. Longitudinal assessment of dogs with steroid-responsive chronic inflammatory enteropathy followed microbial dysbiosis, fecal bile acids, and clinical disease activity as distinct response domains [25]. Reassessment should therefore specify which domain is expected to change, in which direction, and over what clinically plausible time frame.

Serial pancreatitis and aspiration-pneumonia evidence further demonstrates that clinical, biochemical, and imaging domains may normalize asynchronously [11, 12, 24]. The proposed escalation rule is therefore

conditional rather than numerical: worsening direction, persistence, increasing concordance, new organ dysfunction, reduced reserve, or shrinking time-to-harm should intensify action; reproducible improvement, a credible benign explanation, or resolution of discordance may support de-escalation. Universal hourly, daily, or percentage-change thresholds are not established by this evidence.

Application across veterinary internal-medicine settings

The architecture is intended as a reasoning scaffold rather than a universal score. In geriatric dogs, frailty provides one empirical example of clinically meaningful vulnerability before obvious acute instability [20]. In other patients, vulnerability might instead reflect comorbidity, depleted organ reserve, recent decompensation, or treatment burden; these broader applications remain proposed rather than validated extensions.

Systemic inflammatory disease demonstrates why evidence layers should remain distinct. New organ dysfunction, host-response biomarkers, and endothelial or inflammatory signals provide nonidentical information in canine sepsis-related settings [17-19]. A biomarker abnormality, an organ-function change, and an overt bedside transition therefore should not be treated as interchangeable. Concordance may strengthen concern, whereas divergence should prompt verification and consideration of competing explanations.

Clinical time scale also changes the architecture's use. Preclinical myxomatous mitral valve disease illustrates longitudinal compensated change before overt decompensation [22]. Acute infectious disease requires a different temporal posture because serial biomarker and physiological trajectories can evolve over a shorter clinical course [23]. Similar reasoning can be applied cautiously to renal disease, pancreatitis, respiratory presentations, endocrine or metabolic emergencies, gastrointestinal disease, and treatment monitoring represented by the evidence base.

The transfer boundary remains substantial. Most supporting evidence concerns dogs and cats in specialty or hospital environments. Application to large animals, exotic species, production systems, general practice, or resource-constrained settings has not been established. The architecture therefore specifies questions—what is changing, how credible is it, how much reserve may remain, whether domains agree, and how long uncertainty can safely persist—rather than species-independent actions.

Failure modes and sources of diagnostic overconfidence

A trajectory system can fail by mistaking measurement variation for physiological change. Paired canine samples obtained by jugular venipuncture and peripheral intravenous catheters showed that collection route can influence some biochemical or hematologic measurements sufficiently to affect interpretation [26]. Unexpected worsening should therefore prompt a preanalytical plausibility check when sampling, contamination, handling, or assay conditions could explain the discrepancy. Verification, however, should not delay intervention when a serious clinical threat is independently evident.

Binary interpretation can also conceal distributional overlap. In cats with acute kidney injury and chronic kidney disease, symmetric dimethylarginine differed among groups yet showed clinically relevant overlap, including results that did not align neatly with categorical expectations [27]. Reference-limit status should consequently remain one evidence feature rather than an absolute statement about trajectory.

A third failure is assuming biological plausibility guarantees useful discrimination. Serum phosphorylated neurofilament heavy chain was evaluated as a candidate biomarker for progressive myelomalacia in dogs but did not provide adequate intended diagnostic discrimination [28]. Although neurologic disease is outside this article's main internal-medicine examples, the methodological lesson is transferable: a biologically attractive candidate may still fail as a decision tool.

Further overconfidence can arise from treating correlated domains as independent confirmation, ignoring treatment effects, interpreting delayed imaging resolution as continuing deterioration, or inferring low risk from a population-normal value despite patient-relative decline. The proposed architecture therefore retains explicit uncertainty and permits reclassification in both directions as measurements, competing explanations, and the observable course change.

Validation and research priorities

The architecture requires prospective testing before predictive or clinical utility can be claimed. One relevant design precedent is the prospective evaluation of urinary biomarkers in nonazotemic hospitalized dogs followed for subsequent acute kidney injury [29]. Validation of the present architecture should similarly enroll patients

before conventional deterioration thresholds are crossed, define the index assessment prospectively, and adjudicate subsequent clinically meaningful deterioration independently of the proposed state assignment.

More sensitive measurement must also demonstrate incremental value rather than merely detect smaller abnormalities. Two-dimensional speckle-tracking echocardiography has been evaluated in dogs with systemic inflammatory response syndrome to characterize left ventricular systolic function beyond conventional assessment [30]. Analogous testing should determine whether patient-relative trajectories, cross-domain discordance, or vulnerability assessments add information beyond standard threshold monitoring and clinician judgment.

Comparative longitudinal validation is equally important. High-sensitivity cardiac troponin I has been compared with a first-generation assay in Doberman Pinschers with, without, and subsequently developing dilated cardiomyopathy [31]. The proposed architecture should likewise be compared with existing monitoring approaches using prespecified measurement schedules, external cohorts, multiple centers, and clinically meaningful future states.

Priority analyses include discrimination, calibration, false-alert burden, stability of state assignment, interobserver reproducibility, and clinical decision consequences. Species, disease, age, setting, caregiver observation, cost, monitoring burden, and welfare consequences require separate evaluation rather than assumed equivalence. Null or nonincremental findings must be capable of revising or rejecting state definitions, transition logic, or the architecture itself.

Conclusion

Clinical stability should not be equated automatically with absence of physiological deterioration. The principal contribution of this article is a proposed decision architecture that places patient-relative trajectory, physiological reserve, cross-domain concordance or discordance, measurement credibility, and decision urgency alongside conventional thresholds. Its purpose is not to replace reference intervals, diagnostic criteria, severity scores, or clinical judgment, but to explain why an apparently acceptable snapshot can deserve different levels of confidence in different patients.

The proposed Stable, Vulnerable, Deteriorating, and Rescue-Required states organize action around direction and persistence of change, capacity to tolerate additional stress, and the time available to resolve uncertainty. They are not validated prognostic classes, universal thresholds, or treatment rules. Measurement error, asynchronous recovery, disease-specific kinetics, competing diagnoses, and incomplete diagnostic certainty can alter state interpretation.

The architecture is therefore best regarded as a falsifiable clinical reasoning structure. Its usefulness depends on prospective evidence showing that trajectory-based adjudication adds reproducible and actionable information beyond conventional monitoring without unacceptable false alarms, intervention, or monitoring burden. Until such evidence exists, apparent stability should be interpreted with calibrated confidence rather than assumed to represent a durable physiological state.

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