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Perioperative Risk Is Dynamic Rather Than Preoperative: An Adaptive Veterinary Anesthesia and Surgical-Care Architecture Linking Physiological Reserve, Procedural Stress, Intraoperative Instability, Organ Vulnerability, and Recovery

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ABSTRACT

Veterinary perioperative risk is commonly summarized before anesthesia using patient characteristics, comorbidity, physical-status classification, laboratory findings, and anticipated procedural complexity. These variables are clinically important, but they describe vulnerability before the physiological consequences of anesthesia, surgery, instability, intervention, and recovery have unfolded. This article develops an original non-empirical framework in which perioperative risk is treated as a time-dependent clinical state rather than a fixed preoperative attribute. The synthesis distinguishes baseline physiological reserve and organ vulnerability from procedural stress, anesthetic exposure, evolving hemodynamic and respiratory instability, thermal and coagulation disturbances, cumulative physiological insult, response to rescue, and postoperative recovery. The proposed architecture interprets risk through repeated state updating: an animal with substantial baseline vulnerability may remain stable when stress is limited and abnormalities are rapidly reversible, whereas an apparently low-risk animal may acquire clinically important risk through sustained or interacting perioperative disturbances. Measurement quality and clinical context are treated separately from the physiological state being measured, and isolated thresholds are not assumed to represent equivalent risk across animals or procedures. The framework is intended to organize perioperative observation, interpretation, escalation, and reassessment rather than provide a validated risk score or universal intervention algorithm. Its applicability is constrained by predominantly canine evidence, heterogeneous surgical populations, observational outcome studies, limited feline data, device- and method-dependent measurements, and uncertain transferability across practice settings. Prospective multisite validation is required before predictive or decision-support use.

Keywords: Veterinary anesthesia, Perioperative risk, Physiological reserve, Surgical stress, Intraoperative instability, Postoperative recovery

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Introduction

Preoperative assessment remains indispensable in veterinary anesthesia because systemic disease, physiological compromise, and anticipated procedural demands influence anesthetic planning. The American Society of Anesthesiologists physical-status classification has reproducible prognostic value at the population level, yet it

describes the patient's condition before subsequent exposures occur. Veterinary anesthesia also contains medication-process hazards, while intra-anesthetic abnormalities may provide information about hospitalization, cost, or mortality that was unavailable at initial assessment [1–3]. These observations support a broader distinction between baseline vulnerability and perioperative risk as it actually evolves.

Mortality data likewise indicate that clinically important events are not confined to induction or the operating period. In a university cohort of dogs and cats, anesthesia-related deaths occurred across the peri-anesthetic course and were associated with patient and procedural characteristics [4]. A preoperative label therefore cannot, by itself, describe whether an animal will tolerate an expected stressor, develop instability, respond promptly to correction, accumulate organ injury, or deteriorate during recovery.

This article proposes that perioperative risk should be conceptualized as an adaptive state generated by interactions among physiological reserve, organ vulnerability, procedural stress, anesthetic exposure, current physiological instability, cumulative insult, rescueability, and recovery trajectory. The purpose is not to replace established preanesthetic classification, but to specify when and why the risk estimate should change as clinically meaningful new information appears.

Limitations of static preoperative risk classification

Static classification is valuable precisely because it compresses complex clinical information into an interpretable baseline estimate. Evidence supporting ASA physical status demonstrates an association between greater systemic disease burden and increased anesthesia-related mortality [1]. The limitation is therefore not that baseline classification is meaningless; it is that the classification answers a narrower question than the complete perioperative problem. It indicates vulnerability at assessment, not the subsequent trajectory of exposure and response.

Two animals assigned a similar baseline category may experience different procedures, anesthetic drug effects, airway challenges, durations of hypotension, degrees of hypoxemia, thermal losses, hemorrhage, or recovery disturbances. Conversely, a patient considered high risk before anesthesia may encounter a limited stressor, remain physiologically stable, and recover without accumulating additional abnormalities. Static classification cannot encode these transitions because they have not yet occurred.

The distinction proposed here is between preoperative susceptibility and current perioperative state. The former should remain visible throughout care, but the latter must be updated when physiological conditions, procedural demands, therapeutic interventions, or recovery behavior change. This formulation also avoids interpreting an ASA category as a continuously calibrated probability of harm. It is a clinically useful classification whose prognostic meaning remains conditional on species, case mix, clinician judgment, and the subsequent course of anesthesia and surgery.

Baseline physiological reserve

Physiological reserve is used here to mean the latent capacity of an animal to absorb perioperative stress while maintaining adequate function. It is not equivalent to chronological age, a normal laboratory panel, or the absence of a named diagnosis. In dogs and cats older than 8 years, routine preanesthetic blood testing infrequently changed the assigned ASA status or anesthetic plan, demonstrating that additional measurements do not automatically produce a different estimate of clinical vulnerability [5]. Conversely, very large elective-procedure datasets indicate that laboratory variables, physical status, signalment, and procedural information can contribute jointly to baseline risk stratification [6].

Reserve should therefore be inferred from convergent clinical information rather than represented by a single surrogate. Relevant information may include functional status, severity and compensation of disease, hydration and circulatory condition, respiratory capacity, hematologic or biochemical abnormalities, and the expected ability to respond to physiological perturbation. These observations do not establish a validated reserve index.

Referral-hospital mortality evidence further supports separating initial vulnerability from later events: preoperative characteristics were prognostically relevant, but intraoperative abnormalities also contributed outcome information [7]. The proposed architecture consequently treats baseline reserve as the starting capacity against which subsequent stress is interpreted, not as the final perioperative risk state.

Comorbidity and organ vulnerability

Comorbidity becomes perioperatively important when disease reduces tolerance to a stressor that the procedure or anesthetic course can plausibly generate. This distinction is more informative than counting diagnoses. A renal disorder may increase concern about hypoperfusion, pulmonary disease may narrow tolerance for impaired gas exchange, cardiovascular disease may limit compensation for changes in vascular tone or preload, and disorders of hemostasis may alter tolerance for surgical bleeding. The relevant construct is therefore organ vulnerability: the susceptibility of a particular physiological system to deterioration under a specific exposure.

Evidence used to estimate baseline mortality risk already demonstrates that systemic disease severity, laboratory abnormalities, and perioperative characteristics provide partly different information [6]. Multicenter canine mortality data similarly indicate that preoperative vulnerability and subsequent physiological abnormalities cannot be assumed to represent the same dimension of risk [7]. The framework proposed here extends that observation by treating comorbidity as a modifier of the consequence of later instability.

This approach does not imply that every diagnosis predicts an anesthetic complication or that different organ systems can be placed on a common numerical scale. The clinical question is narrower: if a particular disturbance occurs, does this animal have reduced capacity to tolerate it or recover from it? That conditional interpretation permits the same measured abnormality to carry different significance in animals with different underlying vulnerabilities.

Procedural magnitude and surgical stress

Procedural magnitude should describe the physiological challenge actually imposed, rather than rely solely on a broad surgical label. Dogs undergoing surgery for brachycephalic obstructive airway syndrome, invasive adrenal neoplasia requiring adrenalectomy and cavotomy, or thoracic procedures illustrate distinct combinations of airway vulnerability, vascular risk, anatomical invasiveness, hemorrhagic potential, and gas-exchange disturbance [8–10]. These contexts cannot be reduced to an interchangeable category of "major surgery."

The same operation may also impose different effective stress according to patient reserve, duration, blood loss, airway anatomy, positioning, ventilation requirements, surgical manipulation, and complications arising during the procedure. Thus, procedural stress is proposed as a relative exposure: the interaction between what the procedure demands and what the patient can physiologically accommodate.

This framing is deliberately non-numeric. The available evidence does not establish a universal veterinary scale on which surgical stress can be added to ASA status or other baseline variables. Instead, procedural magnitude identifies foreseeable pathways through which risk can become active. Airway surgery may make airway obstruction and recovery particularly consequential; thoracic surgery may increase the relevance of oxygenation and ventilation; highly vascular surgery may increase the importance of hemorrhage, perfusion, and rescue capacity. Anticipating these domains changes monitoring priorities without predetermining the patient's eventual trajectory.

Anesthetic exposure as a dynamic modifier

Anesthesia should not be treated as a neutral interval between preoperative assessment and surgical outcome. Anesthetic and sedative drugs alter vascular tone, heart rate, myocardial performance, ventilatory control, airway behavior, and the requirements for additional anesthetic agents. In healthy dogs, combinations incorporating medetomidine-vatinoxan or dexmedetomidine altered alfaxalone induction requirements and mean arterial pressure patterns during sevoflurane anesthesia [11]. In another randomized clinical trial, labetalol administered to dexmedetomidine-treated dogs produced measurable changes across several hemodynamic variables [12].

These studies establish physiological responsiveness to pharmacological exposure, not improved or worsened clinical outcome. Their importance for the proposed framework is that the anesthetic regimen can move the patient from one physiological state to another after baseline assessment has already occurred.

Patient phenotype also modifies interpretation. In brachycephalic dogs undergoing surgery for obstructive airway syndrome, dexmedetomidine- and acepromazine-based premedication strategies produced different peri-anesthetic physiological profiles [13]. Such evidence argues against regarding drug exposure as an isolated pharmacological property. The clinically relevant unit is the drug–patient–procedure interaction over time. An exposure that is tolerated under one combination of reserve and procedural stress may acquire different significance when airway, cardiovascular, or other vulnerabilities narrow the margin for compensation.

Hemodynamic instability

Hemodynamic instability is often operationalized through arterial pressure, but a single low value does not adequately describe its clinical meaning. The proposed distinction is between an isolated observation, an exposure-linked hemodynamic transition, and a persistent or recurrent trajectory. Pharmacological studies demonstrate that anesthetic regimens can change mean arterial pressure and related cardiovascular variables over short intervals [11]. Interventions directed at cardiovascular state can likewise produce multidimensional changes rather than a simple binary shift between hypotension and normotension [12].

Accordingly, a hemodynamic abnormality should be interpreted in relation to its onset, persistence, recurrence, plausible cause, patient vulnerability, concurrent physiological findings, intervention, and subsequent response. This does not establish that any particular combination predicts organ injury or mortality, nor does it justify a universal veterinary blood-pressure threshold. It specifies what information would be necessary to judge whether apparent instability is transient, evolving, or insufficiently corrected.

Table 1 follows each hemodynamic episode through verification and corrective response, making persistence and reversibility visible rather than classifying risk from a single pressure value.

Table 1. How a hemodynamic episode changes meaning from first detection through verification and response

Hemodynamic episode	At first detection	After verification and context review	After corrective action	Risk meaning
Isolated low-pressure observation	Possible transient disturbance or measurement problem	Repeat the measure and review anesthetic depth, procedure phase, and baseline reserve	Resolution supports a limited event; persistence moves the episode into a different category	A single value does not establish perfusion failure or cumulative insult
Exposure-linked transition	Directional change follows anesthesia, a drug, or an intervention	Assess temporal fit and reversible contributors	The physiological response becomes new evidence about correctability	Association identifies a plausible driver but does not itself establish harm
Persistent or recurrent hypotensive trajectory	Repeated or sustained abnormality raises concern	Evaluate continuing stress, organ vulnerability, and interacting disturbances	Incomplete or short-lived improvement implies unresolved burden	Persistence and recurrence carry more concern than an isolated breach; no universal pressure-time boundary is proposed
Pressure discordant with the broader physiological picture	The number and clinical state do not align	Seek convergence across measurement quality, perfusion-related observations, and procedure context	Interpret response only after the discordance is reduced	Pressure alone cannot fully represent the hemodynamic state

Respiratory instability

Respiratory instability also comprises several separable states. Ventilatory mechanics, oxygenation, airway patency, and the validity of the measurement used to describe them should not be collapsed into a single category. In mechanically ventilated healthy dogs, selection of tidal volume according to driving pressure altered respiratory mechanical behavior compared with body-weight guidance, demonstrating that the ventilatory strategy itself can change the observed respiratory state [14]. This does not establish superiority for postoperative outcomes.

Monitoring further introduces interpretive uncertainty. In anesthetized dogs, the oxygen reserve index was associated with arterial oxygenation but could not be regarded as a direct substitute for arterial partial pressure of oxygen [15]. A seemingly precise monitor value therefore remains a measurement of a physiological construct under defined operating conditions, not the construct itself.

Airway instability constitutes another domain. Veterinary anesthesiologists report clinically important complications associated with endotracheal intubation, emphasizing that airway instrumentation can generate hazards that are analytically distinct from measured gas exchange [16]. Because these data arise from clinician reporting, they do not provide patient-level incidence estimates.

The proposed architecture consequently keeps airway patency, ventilation, gas exchange, and observability separate while allowing them to interact. A respiratory state becomes more concerning when abnormalities persist, converge across domains, interact with patient vulnerability, or fail to normalize after corrective action; those transition rules remain to be prospectively validated.

Temperature, metabolism, and coagulation

Temperature, metabolic balance, and coagulation illustrate why perioperative state cannot be represented adequately by cardiovascular and respiratory measurements alone. Each domain may change during anesthesia through altered heat production and loss, fluid administration, hemorrhage, tissue injury, drug exposure, environmental conditions, or underlying disease. Their importance is not that every deviation carries equivalent prognostic weight, but that deterioration may evolve while attention is focused on more immediately visible variables.

Within the proposed architecture, temperature is treated as a trajectory rather than a terminal recovery measurement. Metabolic observations are interpreted as contextual indicators of substrate use, perfusion, electrolyte balance, or acid–base disturbance rather than combined into a single "metabolic risk" category. Coagulation remains separate because hemorrhage, dilution, disease, and surgical source control can alter hemostatic state through different mechanisms.

These domains function as modifiers of current tolerance and recovery, not interchangeable additions to a composite score. An isolated abnormality may remain clinically limited, whereas persistence, convergence with another unstable domain, or failure to improve after correction may change its importance. The framework does not assign universal thresholds or assume equivalent measurement validity across devices, species, procedures, or settings.

Cumulative physiological insult

A dynamic model requires more than determining whether an abnormality occurred; severity, duration, recurrence, and interaction with other disturbances also matter. Experimental canine evidence demonstrates that severe hypotension can be followed by early renal injury signals, while a clinical canine cohort associated greater depth and duration of intraoperative hypotension with subsequent urinary biomarker change [17, 18]. These findings support an exposure concept but do not establish a universally applicable pressure–time threshold for clinical acute kidney injury.

Thermal disturbance provides another example. Peripheral warming in small dogs undergoing soft-tissue surgery improved preservation of body temperature, demonstrating that part of intraoperative thermal burden is modifiable [19]. The proposed synthesis defines cumulative physiological insult as unresolved burden created when disturbances persist, recur, interact, or leave residual organ consequences.

Cumulative insult is not a mathematical sum. Hypotension, hypoxemia, hypothermia, hemorrhage, and related abnormalities have different mechanisms and clinical meanings. Their convergence may nevertheless narrow remaining reserve. The architecture therefore preserves the trajectory and response of individual domains while recognizing cross-domain deterioration as potentially more consequential than a brief, isolated abnormality that rapidly resolves.

Recovery as a continuation of perioperative risk

Completion of surgery does not reset perioperative risk to its preoperative value. Recovery removes some exposures, including surgical manipulation and maintenance anesthesia, but creates another physiological context in which residual drug effects, airway compromise, pain, agitation, hypothermia, oxygenation abnormalities, hemorrhage, or organ dysfunction may become apparent. Veterinary mortality evidence demonstrates that clinically important outcomes may occur after the intraoperative period [4]. Multicenter canine evidence similarly supports retaining postoperative time within the peri-anesthetic risk horizon [7].

Recovery is therefore treated as another state transition. An animal unstable during surgery may normalize after correction of its dominant disturbance; another may remain apparently compensated while carrying unresolved burden. Conversely, an uneventful intraoperative course does not preclude a newly developing recovery-phase abnormality. Thermal status may remain relevant because intraoperative heat loss is modifiable but not necessarily abolished by warming [19].

The proposed architecture consequently requires deliberate recovery reassessment rather than assuming extubation signifies resolution of risk. The question is whether relevant physiological domains are returning toward an acceptable patient-specific state, remaining abnormal, or revealing new dysfunction. Later outcome prediction remains a separate question requiring validation.

A dynamic perioperative-risk architecture

The central contribution of this article is a proposed dynamic perioperative-risk architecture in which risk is repeatedly updated through interactions among baseline reserve, organ vulnerability, procedural stress, anesthetic exposure, current instability, unresolved physiological insult, rescueability, and recovery trajectory. No numerical weights are assigned. Instead, new information is evaluated according to whether it changes the animal's current state, expected tolerance, or need for intervention.

Dynamic measurements are useful only when their operating conditions are respected. Pulse-pressure and systolic-pressure variation can provide information about fluid responsiveness in mechanically ventilated dogs under defined conditions, illustrating that a dynamic variable is itself conditional [20]. Canine dynamic-compliance literature demonstrates substantial methodological heterogeneity [21]. Even end-inspiratory pause duration can alter calculated respiratory-system compliance in anesthetized dogs with healthy lungs [22]. Measurement state must therefore remain analytically separate from biological state.

Three recurrent questions organize updating: What changed? Does that change matter in this patient under the present stressor? What happened after response or rescue? Risk may remain stable, escalate, partially reverse, or accumulate unresolved burden. Reassessment then feeds back into subsequent interpretation.

Table 2 organizes risk by perioperative phase, showing which information is inherited from baseline and which can emerge only after exposure, instability, rescue, or recovery begins.

Table 2. Perioperative risk components as they are updated across preoperative assessment, anesthesia, surgery, and recovery

Risk component	Preoperative assessment	Induction and early anesthesia	Active procedure	Emergence and recovery
Reserve and organ vulnerability	Starting tolerance and susceptible systems are identified	Observed tolerance may confirm or challenge the baseline estimate	Reserve can be depleted by sustained or interacting stress	Residual vulnerability remains relevant after the procedure ends
Dominant exposure	Anticipated procedural and anesthetic demands	Drug effects, airway transition, and early stabilization	Surgical magnitude, duration, hemorrhage, ventilation, and thermal loss	Residual drugs, pain, airway risk, hypothermia, hemorrhage, or new dysfunction
Information that updates risk	Physical status, comorbidity, function, and planned procedure	New sustained or corroborated physiological change	Persistence, recurrence, multidomain convergence, and rescue response	Direction of recovery, treatment requirement, and newly apparent organ dysfunction
Pattern that prevents de-escalation	Unresolved baseline instability	Failure to stabilize after initial correction	Accumulating burden or only transient rescue	Apparent emergence without a stable multidomain recovery trajectory
Primary reassessment question	What stress can this patient tolerate?	What changed after exposure?	Is instability resolving, recurring, or accumulating?	Are relevant domains returning toward an acceptable patient-specific state?

The temporal architecture in **Figure 1** is required to show how these states interact across organs, exposures, escalation boundaries, and repeated reassessment rather than forming a one-way sequence.

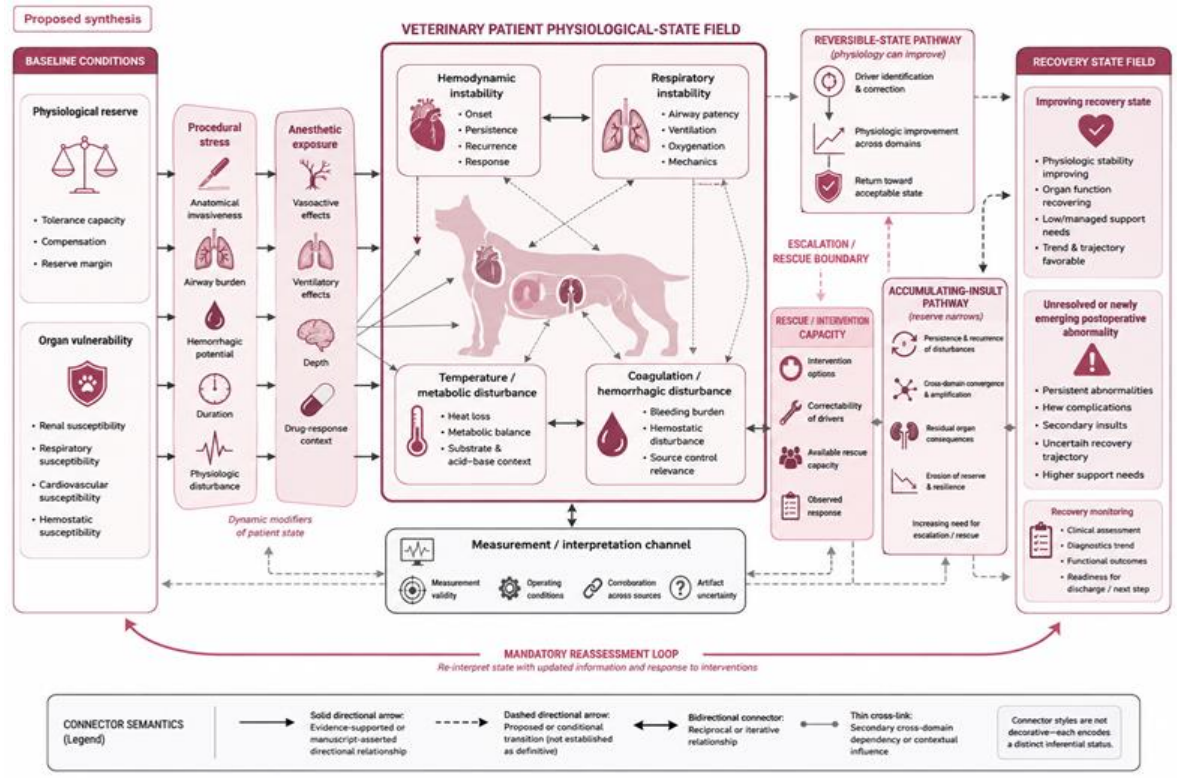

Figure 1. When perioperative risk changes state: reserve depletion, instability, rescue, and recovery across the anesthetic–surgical trajectory

Trigger points for intervention and rescue

A trigger is clinically useful only when it links an observed change to an interpretable state and a feasible response. Veterinary practice does not provide a universal rescue rule. Emergency and critical-care specialists vary in vasopressor selection and treatment of vasodilatory hypotension in dogs and cats [23]. Such variation may reflect differences in physiology, evidence interpretation, case context, clinician preference, and resources.

Rescue interventions are also vulnerable to confounding by indication. Dogs and cats receiving red blood cell transfusions around general anesthesia differ in illness severity and intervention timing, so associations between transfusion timing and outcome cannot establish causal superiority of one strategy [24]. Introduction of cell salvage can similarly change available options for surgical hemorrhage without demonstrating a universal outcome advantage [25].

The proposed architecture therefore treats triggers as conditional escalation boundaries. A threshold breach becomes more consequential when persistent, corroborated, physiologically plausible, relevant to a vulnerable organ, or accompanied by deterioration elsewhere. Rescueability modifies operational urgency because definitive correction may be immediately available in one setting and delayed in another. Crucially, intervention does not terminate the risk episode: the physiological response becomes new information. Failure to improve supports continued escalation, whereas convincing reversal may justify de-escalation and transition toward recovery surveillance.

Reversible versus accumulating risk states

The distinction between reversible and accumulating risk is a proposed clinical interpretation, not a binary biological taxonomy. A state is provisionally reversible when its dominant driver can be corrected and relevant physiology moves toward an acceptable condition without evidence of continuing dysfunction. Risk is considered accumulating when exposure persists, abnormalities recur, several domains deteriorate, or organ consequences remain after the initiating disturbance has been corrected.

Hypotension-related renal evidence illustrates the temporal distinction. Severe experimental hypotension can be followed by early renal injury signals [17]. Greater hypotensive exposure in clinical dogs has also been associated with subsequent urinary biomarker change [18]. Neither observation means that every episode causes kidney

injury, but both argue against equating restoration of an arterial-pressure value with complete resolution of biological consequences.

The architecture therefore separates correction of the initiating disturbance from resolution of its consequences. Restored pressure, oxygenation, temperature, or control of bleeding may indicate successful immediate rescue while still warranting surveillance for residual dysfunction. Rapid normalization without emerging abnormalities may instead support de-escalation. No numerical penalty is assigned for persistence and no recovery bonus is proposed; the distinction exists to preserve clinically meaningful trajectory information.

Postoperative reassessment

Postoperative reassessment determines whether the patient has entered a stable recovery trajectory rather than merely whether anesthesia has ended. A prospective study of dogs recovering from general anesthesia identified poor recovery quality as a distinct outcome with associated risk factors [26]. In specialty veterinary dentistry, mortality evaluated through 14 days after general anesthesia demonstrates that the clinically relevant outcome horizon can extend beyond immediate emergence [27]. Neither endpoint is interchangeable with intraoperative instability.

Serial physiology can also document reversal. In dogs with spontaneous hemoperitoneum, hyperfibrinolysis frequently resolved rapidly after surgical control of hemorrhage, supporting the principle that removal of a dominant driver can change the postoperative physiological state [28]. Outcome interpretation nevertheless depends on measurement definitions: comparison of postoperative complication-grading systems in dogs demonstrated differences in classification reliability [29].

Reassessment therefore has two functions. First, determine whether physiology, behavior, organ function, and treatment requirements are improving, persisting, or deteriorating. Second, determine whether an apparent change represents biology or differences in measurement and classification. Both are necessary before risk is considered resolved, monitoring is intensified, or a postoperative abnormality is attributed to an earlier intraoperative event.

Clinical workflow integration

A dynamic architecture is useful only if it can be incorporated without continuous rescoring or excessive documentation. The proposed workflow therefore centers on clinically meaningful transitions: preanesthetic assessment, induction and early stabilization, substantial procedural changes, emergence, immediate recovery, and sustained or recurrent instability. Additional updates occur when a new organ-specific concern develops or rescue materially changes the patient's state.

At each transition, clinicians would record a compact sequence: relevant vulnerability, active stressors, observed instability, confidence in the measurement, response taken, and subsequent physiology. This preserves trajectory without creating an exhaustive checklist. Medication-process evidence supports including care delivery itself among potential peri-anesthetic hazards [2]. Variation in vasopressor practice demonstrates the contextual nature of rescue decisions [23]. Availability of cell-salvage technology further illustrates that rescue capacity can depend on practice resources [25].

The architecture is intended to support rather than replace clinical judgment. Frequent non-actionable alerts could increase workload without improving care, while classifying every transient deviation as escalation would destroy discrimination. Implementation should therefore prioritize state changes that alter interpretation, intervention, monitoring intensity, or postoperative planning and permit low-consequence resolved events to remain documented without permanently inflating perceived risk.

Validation priorities

Validation should proceed sequentially because a dynamic framework cannot be clinically credible if its component measurements are unreliable. Oscillometric arterial-pressure measurement in anesthetized adult dogs demonstrates device-specific agreement and limitations relative to invasive measurement [30]. Point-of-care coagulation testing likewise shows assay-dependent analytical performance, emphasizing that rapid availability does not itself establish measurement validity [31].

After measurement and operational definitions are standardized, prospective studies should determine whether temporally defined variables add prediction beyond simpler baseline assessment. Early postinduction respiratory variables have been associated with subsequent postanesthetic hypoxemia in dogs, illustrating a candidate temporal prediction relationship that requires external validation before universal threshold use [32]. ASA

physical-status assignment itself also contains observer-dependent uncertainty, including disagreement in clinically ambiguous canine scenarios [33].

The architecture therefore requires prospective multisite testing with prespecified definitions of instability, persistence, recurrence, rescue response, recovery, and outcomes. Candidate models should be compared with baseline-only approaches, assessed for calibration and discrimination, externally validated across species and practice settings, and tested for measurement and missing-data sensitivity. Only after incremental predictive value is established should studies address decision utility, alert burden, intervention consequences, welfare implications, and implementation feasibility.

Conclusion

Perioperative risk in veterinary anesthesia is better conceptualized as an evolving clinical state than as a property determined once before induction. Baseline physical status, physiological reserve, and organ vulnerability establish starting conditions, but procedural stress, anesthetic exposure, instability, cumulative insult, rescue response, and recovery continually alter their clinical meaning. The proposed architecture preserves established preoperative assessment while adding explicit temporal updating, measurement boundaries, reversible and accumulating states, and postoperative reassessment.

Its purpose is organizational rather than predictive: it supplies neither a score nor a universal intervention threshold. Current evidence is predominantly canine, heterogeneous, and frequently observational or context specific. Prospective validation must therefore address measurement reliability, state definitions, temporal relationships, incremental prediction, external generalizability, and decision utility. A dynamic approach becomes valuable only if it improves interpretation and action without transforming ordinary perioperative variability into unsupported risk escalation.

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