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Organ Protection During Veterinary Anesthesia Requires More Than Stable Vital Signs: A Physiology-Guided Perioperative Framework Integrating Perfusion, Ventilation, Temperature, Analgesia, Metabolic Demand, and Recovery-Phase Surveillance

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ABSTRACT

Stable heart rate, arterial pressure, oxygen saturation, and end-tidal carbon dioxide can coexist with physiologically important risk because each variable samples only part of oxygen transport, organ blood flow, ventilation, thermal balance, nociceptive stress, and recovery. This article develops an original non-empirical, physiology-guided framework for organ protection during canine and feline anesthesia. The analysis separates measured stability from inferred physiological adequacy and distinguishes pressure from flow, oxygenation from ventilation, global variables from organ-specific vulnerability, an isolated deviation from accumulated exposure, and intraoperative correction from verified recovery. The proposed framework organizes perioperative assessment around parallel domains of oxygen delivery and perfusion, ventilation and gas exchange, temperature and metabolic demand, analgesia and neuroendocrine stress, duration of physiological insult, and recovery-phase surveillance. These domains are interpreted against patient susceptibility, procedure, anesthetic state, measurement credibility, and temporal trajectory. A normal value in one domain does not compensate for unresolved risk in another; corrective action should address the inferred mechanism and be followed by multidomain reassessment. Organ-specific warning states are treated as graded interpretations rather than validated scores or universal thresholds. The framework is intended to improve clinical reasoning, communication, and research design, not to claim prospective prediction or demonstrated outcome improvement. Important boundaries include limited direct veterinary organ-outcome evidence, heterogeneous definitions, device and site effects, confounding by illness and treatment indication, and restricted transferability across species, breeds, procedures, and practice resources. Prospective validation must link defined physiological exposures and responses to organ-specific and recovery outcomes.

Keywords: Veterinary anesthesia, Organ perfusion, Physiological monitoring, Gas exchange, Perioperative hypothermia, Recovery surveillance

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Introduction

Small-animal anesthesia is commonly judged through a compact set of continuously available variables. These measurements are indispensable, but their apparent stability can create false reassurance when interpreted as proof of adequate organ perfusion, oxygen utilization, temperature control, analgesia, or recovery. Contemporary monitoring guidance therefore spans circulation, oxygenation, ventilation, temperature, neuromuscular function,

anesthetic depth, personnel, and the perianesthetic period; large international canine and feline cohorts likewise locate most anesthesia-related deaths after rather than during anesthesia [1–3]. These findings do not show that a particular monitor or intervention prevents death. They establish a narrower point: the physiologic and temporal field relevant to safety is wider than the interval in which conventional vital signs look acceptable.

Risk is also conditional. In primary-care dogs, age, health status, and procedural urgency were associated with anesthesia-related death, demonstrating that identical measurements cannot carry identical meaning across patients [4]. This article therefore proposes an organ-protection framework in which measured values are interpreted through six interacting domains: oxygen delivery and tissue perfusion; ventilation and gas exchange; temperature and metabolic demand; analgesia and neuroendocrine stress; duration and accumulation of insult; and recovery-phase surveillance. The framework separates evidence-supported physiological distinctions from proposed decision logic. It does not define a score, universal threshold, or validated prediction rule. Its purpose is to make unresolved risk visible when a patient appears conventionally stable and to connect intervention with explicit physiological reassessment.

Why stable vital signs may coexist with organ risk

A vital sign is a measurement, not an organ-level conclusion. Arterial pressure does not disclose cardiac output or regional distribution; pulse oximetry can remain high during oxygen administration despite impaired gas exchange; end-tidal carbon dioxide may not equal arterial carbon dioxide; and heart rate can be pharmacologically constrained while stroke volume or vascular resistance changes. The multidomain scope of current monitoring guidance supports treating these signals as complementary rather than mutually substitutable [1].

The proposed distinction is between observed stability and adjudicated adequacy. Observed stability means that selected measurements remain within an accepted or patient-specific range. Adjudicated adequacy requires credible measurements, concordance across relevant domains, no accumulating adverse trajectory, and no unresolved organ-specific warning. Compensation can temporarily preserve a global variable while increasing demand or redistributing flow. Conversely, an abnormal value may reflect artifact, drug effect, or an adaptive response rather than injury. Worldwide canine mortality data associate outcome with patient and procedural context, but cannot determine which apparently stable animals harbored occult organ risk [2]. Accordingly, “stable” should remain a provisional state that is repeatedly tested against baseline disease, anesthetic drugs, procedure, elapsed exposure, and recovery behavior.

Oxygen delivery and tissue perfusion

Oxygen delivery depends principally on blood flow and arterial oxygen content, whereas tissue adequacy additionally depends on regional distribution, diffusion, extraction, and demand. None is captured completely by mean arterial pressure. In anesthetized cats, comparison of echocardiographic and esophageal Doppler estimates with pulmonary artery thermodilution illustrates why correlation, agreement, and trending ability must be distinguished when interpreting cardiac output. Experimental canine evaluation of an arterial-waveform system similarly shows that a derived prediction output requires state- and method-specific validation. A systematic review of dynamic preload indices in ventilated dogs found context-dependent diagnostic performance and high risk of bias [5–7].

These sources support triangulation, not monitor accumulation for its own sake. The clinically relevant questions are sequential: Is the measurement technically credible? Is global flow likely sufficient for the current oxygen content and demand? Is distribution plausibly impaired by vasoconstriction, disease, positioning, or surgical manipulation? Does an intervention improve the targeted state without worsening another domain? Fluid responsiveness answers only whether stroke volume is likely to rise after volume expansion; it does not establish hypovolemia, fluid tolerance, renal benefit, or outcome improvement. The framework therefore keeps pressure, flow, oxygen content, responsiveness, and organ consequence analytically separate.

Arterial pressure and flow

Arterial pressure is valuable because it is accessible, repeatable, and physiologically consequential, but it is the product of flow and vascular resistance rather than a direct measure of either. Cardiac-output method studies reinforce that flow assessment is a separate measurement problem requiring validation [5]. A low pressure can reflect reduced preload, impaired contractility, inappropriate vasodilation, bradycardia, or combined mechanisms;

an acceptable pressure can coexist with low flow when resistance is high. The same mean pressure may therefore imply different organ risks and different corrective actions.

The proposed approach begins with measurement credibility: cuff size and site, waveform quality, patient movement, device limitations, and consistency with pulse quality and other variables. It then asks whether the pressure change is isolated, recurrent, or persistent and whether it coincides with altered flow surrogates, gas exchange, temperature, anesthetic depth, hemorrhage, or surgical events. Dynamic preload indices may assist under specified controlled-ventilation conditions, but their prediction of responsiveness does not establish organ benefit [7]. Pressure should consequently function as an escalation signal within a physiological configuration, not as a solitary treatment target. Correction is incomplete until the presumed mechanism and downstream response have been reassessed.

Ventilation and gas exchange

Ventilation, oxygenation, and respiratory mechanics answer different questions. Ventilation concerns carbon dioxide elimination and alveolar ventilation; oxygenation reflects pulmonary transfer and inspired oxygen; mechanics describe the pressure–volume behavior that conditions both. In healthy anesthetized dogs, driving-pressure-guided versus body-weight-based tidal-volume selection produced different respiratory-mechanics and gas-exchange profiles. A retrospective Air-Test study examined whether a brief reduction in inspired oxygen could expose impaired oxygen uptake and inform recruitment, while another cohort evaluated early oxygenation and compliance measures as predictors of postanesthetic hypoxemia [8–10]. These studies are clinically informative but do not establish a universal ventilator prescription or prove that test-guided intervention improves outcomes.

The central interpretive hazard is masking. High inspired oxygen may preserve pulse-oximetry saturation while atelectasis, shunt, ventilation–perfusion mismatch, or declining compliance develops. Conversely, an abnormal end-tidal carbon dioxide value may arise from ventilation, perfusion, dead space, circuit factors, or sampling error. The proposed framework therefore requires joint reading of inspired oxygen, saturation, capnography, airway pressures, delivered volume, compliance, arterial gas data when justified, and hemodynamic tolerance. Recruitment or ventilator adjustment should be followed by reassessment of both gas exchange and circulation because respiratory improvement achieved at unacceptable cardiovascular cost is not organ protection.

Cerebral protection

The brain is protected neither by arterial pressure alone nor by oxygen saturation considered in isolation. Cerebral oxygen balance depends on arterial oxygen content, cerebral blood flow, metabolic demand, carbon dioxide-dependent vascular tone, venous drainage, and, in vulnerable patients, intracranial compliance. The canine ventilation trial demonstrates that ventilator strategy changes respiratory mechanics and carbon dioxide relationships, but it did not measure cerebral injury or define a cerebral target [8]. The Air-Test evidence similarly concerns systemic gas exchange in selected stable dogs, not cerebral oxygenation [9]. Direct veterinary outcome evidence is therefore insufficient to justify a universal cerebral perfusion or carbon dioxide threshold.

Clinical reasoning should instead identify cerebral vulnerability and avoid preventable combinations: uncertain flow with reduced oxygen content, marked or sustained carbon dioxide disturbance, impaired venous drainage from positioning, excessive metabolic demand, and delayed recovery without an alternative explanation. Normal systemic readings may be less reassuring in patients with intracranial disease, severe anemia, impaired autoregulation, or prolonged prior instability. **Table 1** presents cerebral mechanisms that can remain unresolved despite apparently acceptable routine monitoring. The classifications and reassessment consequences are proposed; they organize available physiological evidence without claiming validated prediction.

Table 1. Cerebral mechanisms that can remain unresolved despite apparently acceptable routine monitoring

Routine monitoring picture	Hidden cerebral mechanism	Patient factors reducing reassurance	What to interrogate	What remains unverified	Mechanism-focused follow-up
Pressure stable; flow evidence absent or discordant	**Pattern:** Pressure-supported, flow-uncertain **Meaning:** Acceptable pressure	High vascular resistance; cardiac disease	Investigate flow mechanism before escalating pressure alone	No validated canine cerebral-flow target	**Recheck:** Recheck flow, oxygen content, positioning, response **Basis:** Proposed synthesis

	does not confirm cerebral flow				
High inspired oxygen with worsening mechanics or gas-exchange discordance	**Pattern:** Oxygenation-masked gas-exchange deficit **Meaning:** High saturation under enriched oxygen may conceal transfer impairment	Pulmonary disease; recumbency; prolonged anesthesia	Address lung state while preserving circulation	Systemic measures do not quantify cerebral oxygenation	**Recheck:** Reassess oxygenation, ventilation, mechanics, hemodynamics **Basis:** Proposed synthesis
Capnographic or arterial carbon dioxide trajectory	**Pattern:** Carbon dioxide-linked vascular shift **Meaning:** Carbon dioxide change may alter cerebral vascular tone despite stable pressure	Intracranial disease; impaired compliance	Correct cause cautiously; avoid abrupt unverified normalization	Direct veterinary cerebral-outcome evidence is limited	**Recheck:** Confirm measurement and neurologic recovery trajectory **Basis:** Proposed synthesis
Stable routine values plus high-risk history or delayed emergence	**Pattern:** Intracranial vulnerability with systemic stability **Meaning:** Conventional stability carries reduced reassurance	Intracranial lesion; anemia; prior instability	Use narrower tolerance for unresolved discordance	Vulnerability is patient-specific	**Recheck:** Extend surveillance and investigate competing causes **Basis:** Proposed synthesis

Myocardial protection

Myocardial protection requires balancing coronary perfusion, cardiac output, oxygen content, wall stress, rate, contractility, and anesthetic demand. Premedication is part of that configuration: a randomized clinical study in healthy dogs showed that acepromazine–methadone with or without dexmedetomidine produced different cardiovascular and anesthetic profiles [11]. Such findings support drug-aware interpretation, not a claim that one regimen prevents myocardial injury.

Treatment studies further show why pressure normalization is insufficient. In isoflurane-anesthetized dogs receiving dexmedetomidine–vatinoxan, dobutamine, norepinephrine, and phenylephrine produced different combinations of pressure, cardiac output, and vascular resistance [12]. In a separate severe isoflurane-hypotension model, dobutamine, norepinephrine, vasopressin, and hetastarch differed in their effects on cardiac output, systemic resistance, and oxygen delivery [13]. These controlled studies in healthy Beagles cannot establish clinical outcome superiority, but they support mechanism-matched intervention. The proposed myocardial warning state is therefore configurational: pressure, rhythm, flow, oxygen content, anesthetic concentration, temperature, and surgical stimulation are interpreted together. A rising pressure achieved through increased resistance may not resolve low-flow risk, while tachycardia that raises output may also increase myocardial demand. Reassessment must examine the intended effect and the physiological cost.

Renal perfusion

Renal blood flow is influenced by cardiac output, perfusion pressure, vascular tone, neurohumoral responses, venous pressure, baseline kidney function, and anesthetic and surgical conditions. Global pressure cannot show regional distribution, and an acceptable cardiac-output estimate cannot confirm cortical or medullary oxygen balance. Evidence comparing cardiac-output methods in anesthetized animals supports the more limited conclusion that flow itself must be measured or inferred with awareness of method error [5].

The framework consequently separates three questions: whether systemic flow is plausibly adequate, whether the kidney is especially susceptible, and whether an early renal warning has emerged. Fluid responsiveness may inform the first question under constrained conditions, but does not answer whether volume administration is needed, tolerated, or renoprotective [7]. Pre-existing renal disease, anemia, venous congestion, nephrotoxic exposure, hemorrhage, and prolonged physiological disturbance may narrow tolerance even when mean pressure is conventionally acceptable. Conversely, transient hypotension does not prove kidney injury. The proposed renal-perfusion state therefore remains an inference until linked to trend-consistent urine output, laboratory change,

injury biomarkers where available, or postoperative renal assessment. This preserves uncertainty while preventing a normal pressure value from closing renal surveillance prematurely.

Hepatic and gastrointestinal perfusion

Hepatic and gastrointestinal risk is difficult to infer from routine monitoring because splanchnic distribution, portal contribution, venous congestion, surgical manipulation, and metabolic demand are poorly represented by global variables. Gastro-oesophageal reflux detected during canine anesthesia illustrates that clinically relevant gastrointestinal disturbance can occur during otherwise routine monitoring and that detection depends strongly on method [14]. Reflux is not evidence of ischemia, but it demonstrates why absence of an overt vital-sign crisis cannot establish gastrointestinal normality.

Outcome evidence is also inconsistent. In a multicentre cholecystectomy cohort, intraoperative hypotension was not independently associated with in-hospital or 28-day mortality after adjustment; postoperative ileus and pancreatitis were more informative [15]. By contrast, a smaller multicentre study of dogs undergoing cholecystectomy for gallbladder mucocele associated severe intraoperative hypotension and increasing age with nonsurvival [16]. Differences in disease definition, sample size, exposure severity, covariates, and postoperative mechanisms could explain the divergence. The proposed splanchnic warning state must therefore integrate susceptibility, surgical context, pressure–flow trajectory, temperature, metabolic disturbance, and postoperative gastrointestinal function. Neither a null adjusted association nor a positive disease-specific association justifies a universal hypotension threshold or causal conclusion.

Temperature and metabolic demand

Temperature modifies demand, drug disposition, vascular tone, coagulation, and recovery. In small dogs undergoing soft-tissue surgery, peripheral warming reduced perioperative heat loss, supporting early warming in susceptible patients [17]. Yet a temperature value cannot reveal the mechanism or prove organ protection. Redistribution, exposure, unwarmed fluids, low ambient temperature, reduced heat production, and measurement-site lag may produce similar trajectories.

Drug context matters: fentanyl combined with medetomidine or acepromazine produced differing oesophageal and rectal patterns in anesthetized dogs [18]. A multicentre study associated canine temperature decline with body size, health status, anesthetic duration, and practice factors [19]. The proposed ledger separates thermal trajectory from metabolic interpretation and requires confirmation of site, trend, susceptibility, exposure, and response. It adjudicates states that an endpoint temperature would collapse. **Table 2** presents perioperative thermal trajectories, heat-flow explanations, and confirmation strategies.

Table 2. Perioperative thermal trajectories, heat-flow explanations, and confirmation strategies

Temperature trajectory	Heat-flow explanation	Conditions producing it	Trend or device signature	Correction and confirmation
Redistribution decline	Heat transfer may precede loss	Small size; vasodilatory drugs	Credible downward trend	**Correction:** Warm early; reduce exposure **Limit:** No injury threshold **Confirm:** Recheck trend and perfusion **Basis:** Proposed synthesis
Procedural cooling	Loss may exceed production	Long procedure; open cavity	Continued decline	**Correction:** Audit warming and losses **Limit:** Site lag **Confirm:** Verify device, skin, core trend **Basis:** Proposed synthesis
Apparent stability	Site or device error possible	Poor contact; peripheral site	Discordance or implausible plateau	**Correction:** Confirm before reassurance **Limit:** Tissue heterogeneity **Confirm:** Repeat at credible site **Basis:** Proposed synthesis
Rewarming, unresolved demand	Other risks persist	Pain; shivering; low flow	Rising temperature; domain discordance	**Correction:** Treat remaining mechanism **Limit:** Warming is not recovery **Confirm:** Reassess demand and circulation **Basis:** Proposed synthesis

Analgesia and neuroendocrine stress

Analgesic adequacy cannot be inferred from heart rate or arterial pressure alone. Nociception, anesthetic depth, autonomic modulation, neuromuscular blockade, vasoactive treatment, and surgical intensity can produce overlapping signs. Conversely, pharmacological suppression of movement or cardiovascular responses does not demonstrate absence of nociceptive processing. Contemporary monitoring guidance supports integrating anesthetic depth, analgesic planning, physiology, and recovery observation rather than treating one signal as a pain monitor [1].

The proposed framework keeps analgesia and neuroendocrine demand as a distinct lane that interacts with circulation, ventilation, temperature, and drug accumulation. Premedication evidence shows that combinations including acepromazine, methadone, and dexmedetomidine alter cardiovascular behavior and anesthetic requirements [11]; it does not establish endocrine protection or superiority for organ outcomes. Clinical adjudication should therefore combine procedural phase, expected nociceptive input, analgesic timing, anesthetic concentration, movement where observable, and multidomain trends. A response to rescue analgesia may support—but does not prove—a nociceptive mechanism. Excess treatment may worsen ventilation, circulation, temperature, or recovery. The aim is not maximum physiological suppression, but proportionate analgesia with explicit reassessment of both analgesic effect and physiological cost.

Hard cases include paralysis, autonomic neuropathy, profound cardiovascular drug effects, and recovery dysphoria. In these settings, discordant observations should widen uncertainty rather than be forced into a binary judgment of adequate or inadequate analgesia.

Duration and Accumulation of Physiological Insult

An isolated nadir and a sustained or recurrent deviation are not equivalent exposures. In a retrospective series of 390 anesthetized dogs, hypotension frequency and associated factors depended on the operational definition, illustrating how persistence changes case classification [20]. This article proposes physiological burden as the interaction of deviation magnitude, duration, recurrence, interdomain concurrence, and patient susceptibility. It is an organizing construct, not a validated dose or score.

Susceptibility also changes over time. Japanese referral-hospital data linked preoperative and perioperative characteristics with anesthesia-related death across a 48-hour window, but older observational data cannot isolate monitoring effects [21]. Body mass, temperature, health status, and procedural context were associated with canine hypotension, while measurement difficulty and case mix remain alternative explanations [22]. A transient abnormality followed by verified recovery may therefore carry different meaning from repeated modest abnormalities accompanied by cooling, impaired gas exchange, or delayed emergence. Documentation should preserve onset, recurrence, corrective response, and unresolved discordance. This temporal record becomes the bridge between intraoperative management and recovery surveillance, without implying that equal exposure produces equal injury across animals.

A proposed organ-protection framework

The framework operates as a repeated adjudication cycle. Establish susceptibility from species, breed, size, age, health, organ disease, procedure, positioning, and prior trajectory; verify measurement credibility; then read six parallel domains: oxygen delivery and perfusion, ventilation and gas exchange, temperature and metabolic demand, analgesia and neuroendocrine stress, accumulated insult, and recovery readiness. Identify organ-specific discordance, select a mechanism-directed correction, and reassess benefit, cross-domain cost, and trajectory.

Two decision boundaries are central. A value crossing a conventional limit is an assessment trigger, not proof of injury. Conversely, normalization in one lane cannot cancel an unresolved warning in another. The cycle continues through transfer and recovery because postoperative mortality timing makes extubation an unsafe endpoint for physiological reasoning [2]. **Figure 1** compresses the proposed state transitions, accumulation channel, and recursive reassessment that prose cannot display simultaneously.

The cycle yields four proposed states: concordant adequacy, compensated vulnerability, active multidomain discordance, and recovery-unverified stability. They are descriptive, not severity grades. Movement depends on trajectory and response; the same measurement may justify observation or escalation. Failure to improve increases uncertainty and reopens measurement, mechanism, and competing-cause assessment.

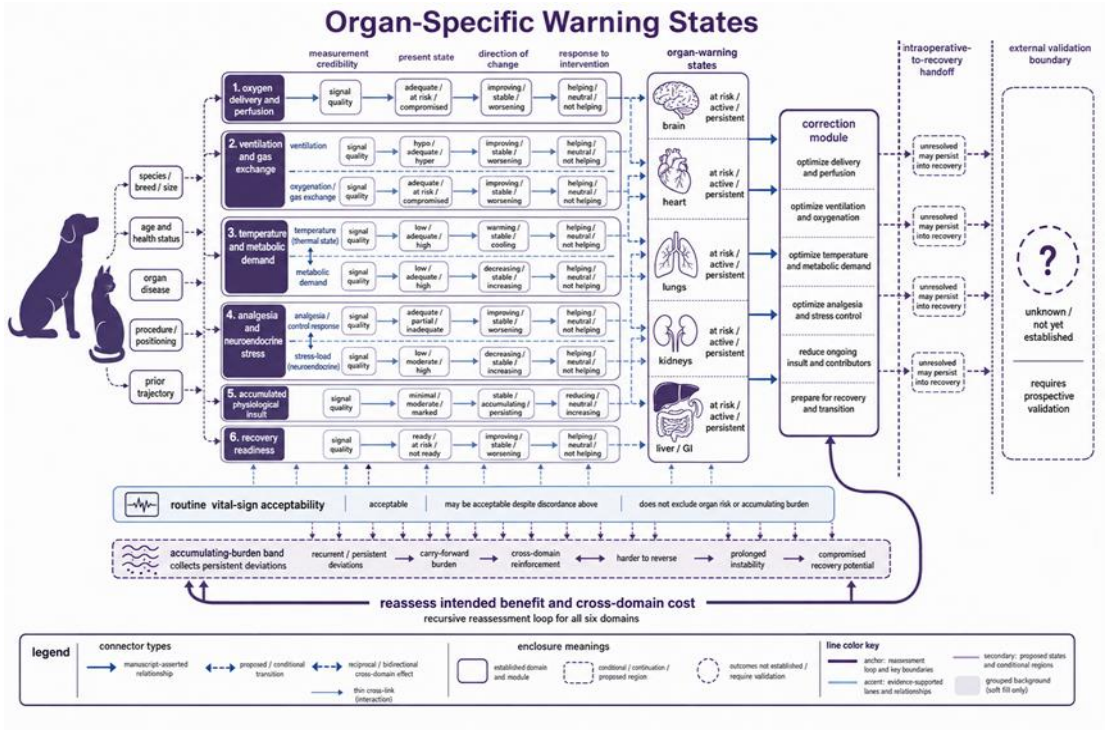

Figure 1. Physiological discordance, accumulated burden, and reassessment across the anesthetic trajectory

Organ-specific warning states

An organ-specific warning state is proposed as a graded inference generated by discordant physiology, patient susceptibility, exposure trajectory, and emerging organ evidence. It is not a diagnosis. In dogs exposed to intraoperative hypotension, urinary injury biomarkers may change before serum creatinine, but associations with pressure exposure were inconsistent and biomarker change did not establish clinically important acute kidney injury [23]. In healthy dogs undergoing desexing, creatinine-defined perioperative acute kidney injury occurred despite routine elective care, yet anesthesia could not be separated from surgery, drugs, fluids, or biological variation [24].

Metabolic warnings must remain distinct. Intra-anesthetic hyperglycemia in nondiabetic dogs was associated with patient and anesthetic factors, but the study did not identify a treatment threshold or show benefit from glucose correction [25]. Warning states should therefore prompt verification, competing-cause review, and surveillance proportional to consequence and reversibility. **Table 3** presents renal, metabolic, neurologic, and respiratory warning signals compared by claim and follow-up.

Table 3. Renal, metabolic, neurologic, and respiratory warning signals compared by claim and follow-up

Warning-signal question	Renal biochemical warning	Metabolic warning	Neurologic recovery warning	Respiratory recovery warning
Candidate interpretation	Possible early injury	Stress, drug, or disease possible	Delayed emergence needs differential	Airway or weakness risk
Patient or procedural influences	Kidney status; dilution	Diabetes status; procedure	Drugs; temperature; instability	Brachycephaly; blockade; opioids
Signal available	Biomarker or creatinine trend	Glucose trajectory	Recovery trend and examination	Breathing, ventilation, oxygenation
Immediate clinical response	Review exposure and nephrotoxins	Investigate; avoid reflex correction	Extend surveillance	Escalate observation
Claim prohibited	Not causal attribution	No treatment trigger	Not proof of cerebral injury	No universal duration
Serial check	Repeat renal assessment	Recheck demand context	Serial systemic review	Confirm functional recovery
Basis	Proposed synthesis	Proposed synthesis	Proposed synthesis	Proposed synthesis

Intraoperative corrective strategies

Correction should follow the inferred mechanism rather than the most conspicuous number. For hypotension, the possibilities include measurement error, excessive anesthetic effect, reduced preload, impaired contractility, bradycardia, vasodilation, obstruction, hemorrhage, or combinations. Comparative canine studies show that vasoactive agents can produce different pressure–flow–resistance configurations [12]. They support physiological matching, not a universal drug hierarchy.

The proposed sequence is: confirm the signal; identify reversible causes; define the mechanism; intervene proportionately; and reassess pressure, flow, oxygen content, ventilation, temperature, analgesic state, and surgery. A fluid bolus is not automatically indicated by low pressure, and predicted responsiveness does not demonstrate fluid tolerance or organ benefit [7]. Recruitment, deeper anesthesia, warming, or vasoconstriction can improve one measure while worsening another. Failure of the expected response should trigger diagnostic revision. When urgent action precedes adjudication, document uncertainty and response so subsequent clinicians can distinguish corrected instability from unresolved risk.

Recovery-phase surveillance

Recovery is a physiological phase, not a timestamp. After vecuronium in dogs, reversal speed differed between sugammadex and neostigmine and depended on dose and timing; objective neuromuscular assessment therefore provides information that elapsed time or visible movement cannot replace [26]. Surveillance should also carry forward accumulated hypotension, ventilation difficulty, hypothermia, analgesic burden, airway vulnerability, and incomplete corrective responses.

Continuity is itself a safety condition. A systems analysis of a fatal canine ventral-slot case described how handoff deficiencies could disconnect prior hypotension and comorbidity from later decisions, although one case cannot establish an effect size [27]. Brachycephalic dogs have shown higher postanesthetic complication risk than matched nonbrachycephalic dogs, but retrospective evidence does not define a universal observation interval [28]. The proposed recovery state therefore requires sustained adequacy across airway, ventilation, oxygenation, circulation, temperature, comfort, neurologic function, and procedure-specific concerns. Deterioration after apparent normalization reopens the relevant intraoperative lane. Discharge from intensive observation should be based on trajectory, phenotype, residual drug effect, and unresolved warnings, not extubation alone.

Clinical implementation

Implementation should be tiered to practice capability while preserving the reasoning sequence. A minimum configuration includes a trained observer, reliable core measurements, trend documentation, escalation authority, and structured transfer of susceptibility, exposures, interventions, responses, residual risks, and surveillance priorities. Current guidance supports dedicated monitoring and continuity, but does not validate this framework's effectiveness [1].

At higher-resource levels, arterial pressure waveform analysis, blood gases, cardiac-output estimation, objective neuromuscular monitoring, or biomarkers may refine adjudication only when users understand method limits. Technology should not displace examination or create false precision. The framework can be embedded in an anesthetic record as linked fields: credible signal, inferred mechanism, action, multidomain response, and handoff status. **Figure 2** shows how implementation preserves or loses physiological history across team and phase transitions.

Low-resource implementation should prioritize dependable basics and escalation pathways. Protocols must specify who observes, who acts, recording intervals, consultation triggers, and transfer of surveillance responsibility. Training should use discordant rather than obviously catastrophic cases because they expose the intended reasoning task.

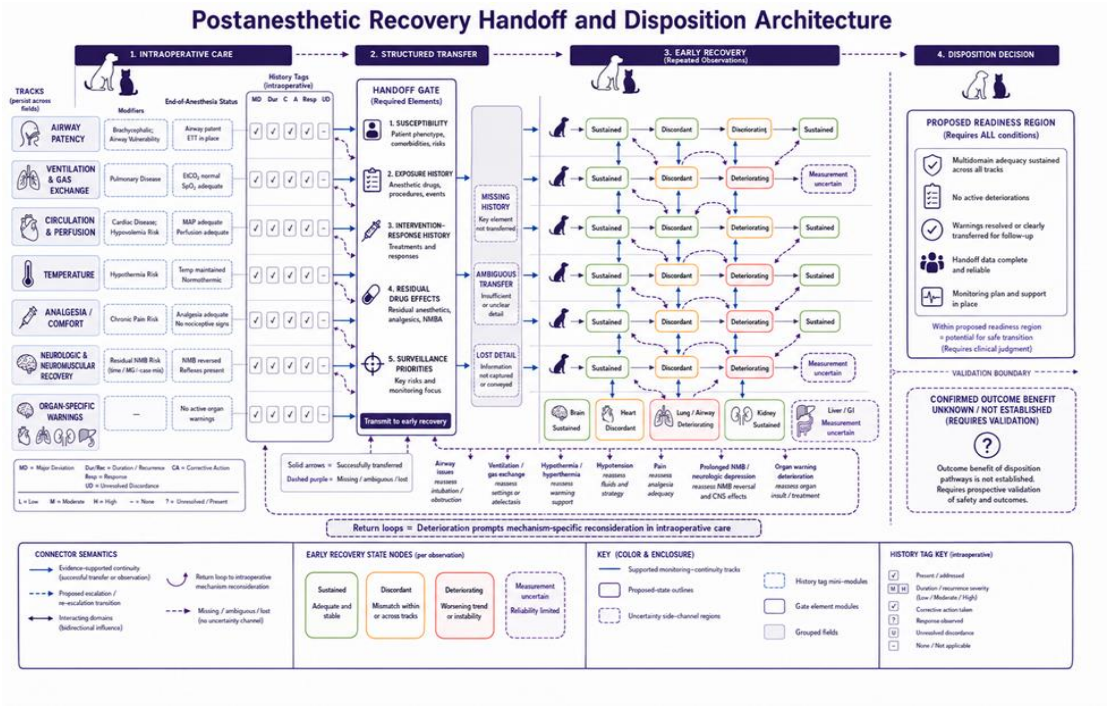

Figure 2. Continuity gates that prevent apparent recovery from erasing unresolved organ risk

Research priorities

Validation should begin with definitions. Human perioperative literature shows that hypotension thresholds, duration rules, relative change, and area-under-threshold constructs are not interchangeable; these methods are useful, but their numerical thresholds cannot be imported into dogs or cats [29]. Large human cohorts further support testing cumulative magnitude–duration exposure, while residual confounding and species differences preclude veterinary causal inference [30].

Veterinary studies should predefine signal quality, baseline, exposure trajectory, intervention timing, competing causes, and organ outcomes. Multicentre cohorts should test transportability across species, breeds, size, disease, procedures, and resources; biomarkers and recovery outcomes should not be treated as injury proof. Pragmatic studies are needed because equipment, staffing, and safety practices vary among veterinary practices [31]. Comparative-strategy research requires stronger control of bias and heterogeneity: a systematic review of canine total intravenous versus inhalant anesthesia did not support a universal technique hierarchy [32]. The framework requires external validation, clinician-agreement testing, and trials of whether mechanism-linked responses improve patient-centered outcomes without new harms.

Outcome sets should include recovery quality, respiratory events, organ measures, supportive-care duration, and meaningful morbidity, with blinded adjudication where feasible. Negative results are essential for identifying states that should be revised or rejected.

Conclusion

Stable vital signs are necessary observations, not sufficient evidence of organ protection. The proposed framework broadens perioperative reasoning from isolated values to credible measurements, pressure–flow distinction, oxygen transport, gas exchange, thermal and analgesic demand, accumulated exposure, organ-specific warnings, mechanism-directed correction, and verified recovery. Its central rule is noncompensatory: normalization in one domain does not erase unresolved risk in another. Its temporal rule is equally important: physiological history must cross the handoff into recovery. This architecture is a structured synthesis, not a validated score, universal threshold set, or demonstrated safety intervention. Species, phenotype, disease, procedure, device, staffing, and outcome-window differences constrain transfer. Prospective veterinary studies must determine whether its states are reproducible, predict meaningful organ and recovery outcomes, and support interventions whose benefits exceed their cross-domain costs.

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