Advancements in Health Research

Advancements in Health Research 2026; 3:140
doi:10.4081/ahr.2026.140

EEG-CBF integration in calf slaughter neurophysiology: a narrative review

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Abstract

Loss of consciousness (LOC) during calf slaughter is a neurophysiological process that requires distinction between cortical awareness and reflexive subcortical or spinal activity. Conscious perception depends on intact thalamocortical connectivity and integrated cortical function. Electroencephalography (EEG) and evoked potentials provide indirect measures of cortical activity, while cerebral blood flow (CBF) is the physiological prerequisite for maintaining cortical function. When cerebral perfusion fails, organized cortical electrical activity rapidly ceases. This review synthesizes current evidence on the neurophysiological effects of different slaughter methods in calves by integrating electrophysiological findings with cerebral perfusion data relevant to animal welfare assessment. A structured narrative review was conducted according to SANRA principles. PubMed/MEDLINE was systematically searched from database inception to January 2026 using predefined terms related to calves, cattle, EEG, evoked potentials, cerebral perfusion, stunning techniques, exsanguination without stunning, and religious slaughter. Only peer-reviewed studies reporting direct neurophysiological endpoints in cattle or calves were included. Correctly applied penetrating captive bolt induces rapid cortical changes consistent with loss of cortical function, with pathological evidence also demonstrating disruption of subcortical and brainstem structures. Proper electrical stunning produces an ictal phase followed by post-ictal cortical suppression characterized by EEG patterns incompatible with conscious processing. Non-penetrating captive bolt also achieves electrocortical suppression under controlled conditions, although with greater variability. In contrast, exsanguination without prior stunning causes a rapid reduction in cerebral perfusion pressure and CBF, supported by carotid hemodynamic and computational studies, although direct intracerebral measurements during slaughter are lacking. Electrophysiological studies consistently report EEG patterns compatible with LOC within single-digit seconds. Overall, current evidence indicates that catastrophic loss of cerebral perfusion after carotid transection results in rapid LOC, while available EEG findings during slaughter without stunning are consistent with this conclusion. Future validation of combined EEG-perfusion monitoring under commercial slaughter conditions may reduce uncertainty in neurophysiological welfare assessment.

Key words: calf; cattle; electroencephalography (EEG); cerebral blood flow; cortical responsiveness; unconsciousness; slaughter techniques; animal welfare.

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Introduction

Loss of consciousness (LOC) at slaughter is a welfare-critical endpoint, but defining it operationally remains contested. Conscious perception and the subjective experience of pain in particular, depends on preserved thalamocortical connectivity and integrated processing across distributed cortical networks.[1-3] Electrophysiological measurements, by contrast, address cortical function directly: in cattle, Daly *et al*.[4] showed that visual and somatosensory evoked cortical responses were rapidly abolished after effective captive-bolt stunning, whereas behavioral signs alone lacked sufficient specificity to define cortical state. Such measurements, however, are not currently feasible in commercial settings. The result is a persistent gap between what regulators monitor (animal-based measures) and what neurobiology defines as conscious experience (preserved thalamocortical integration). This gap is most consequential during slaughter without prior stunning, where the timing and certainty of cortical failure remain debated, and during the post-stun exsanguination phase, where the depth and duration of unconsciousness must be guaranteed regardless of stunning method. Behavioral indicators, corneal reflex, gasping, righting attempts and limb movement are easy to observe but are mediated at brainstem or spinal levels and can dissociate from cortical state in either direction.[5,6]

The temporal dynamics of unconsciousness are critical not only at the moment of stunning but throughout the subsequent exsanguination phase. Even when stunning is applied, bleeding is required to ensure adequate meat quality. Therefore, unconsciousness must be reliably induced and maintained during exsanguination, as cerebral perfusion decreases during this period. Within the European Union, slaughter practices are governed by Regulation (EC) No 1099/2009, with scientific interpretation guided by the European Food Safety Authority (EFSA). EFSA opinions recognize that operational monitoring relies primarily on animal-based measures (ABMs), like corneal reflexes, while also emphasizing their inherent limitations and the necessity of combined indicators to infer unconsciousness. Empirical validation in veal calves confirms imperfect concordance between ABMs and EEG-defined cortical states.[7]

Calves differ from mature cattle in cranial morphology and in cerebrovascular anatomy in ways that are directly relevant to the present review.[8,9] Skull thickness, frontal bone density and the position of the brain within the cranial vault influence the mechanical energy actually delivered to the cortex during captive-bolt stunning and have been shown to affect EEG-defined endpoints.[10,11] Cerebrovascular anatomy is equally relevant: in cattle, vertebral and carotid contributions to cerebral perfusion are quantitatively different from those in monogastric species and recent work suggests these differences modulate the rate at which cerebral perfusion falls after carotid section.[12,13] We refer here to the developmental difference between calves and mature cattle, not to senescence.

The present narrative review was therefore undertaken to: summarize the electrophysiological evidence in calves and cattle across slaughter modalities; examine how cerebral hemodynamics relate to cortical electrical failure; and evaluate whether integrating EEG with cerebral perfusion measurements offers a mechanistically coherent framework that could, in principle, narrow the gap between operational welfare assessment and the neurobiology of consciousness. We do not aim to adjudicate the welfare debate around any specific slaughter practice.

Methods

This work is a structured narrative review. We followed the Scale for the Assessment of Narrative Review Articles (SANRA)[14] as a methodological guide for our own writing, that is to ensure transparent justification of the review question, an explicit and reproducible search, critical interpretation of cited studies and coherent synthesis. SANRA was not applied as a quality-rating instrument to the primary studies we cite, because most of those studies are experimental electrophysiological investigations rather than narrative reviews; SANRA is not designed for that purpose.

A structured search of PubMed/MEDLINE, Scopus and Web of Science was conducted from database inception to January 2026. Search blocks were combined using Boolean operators to capture key domains, including species (“calf”, “calves”, “veal”, “bovine”, “cattle”, “cow”), cortical electrophysiology (“electroencephalogram”, “EEG”, “electrocorticography”, “ECoG”, “evoked potential”, “visual evoked”, “somatosensory evoked”), cerebral hemodynamics (“cerebral blood flow”, “cerebral perfusion”, “cerebral oxygenation”, “carotid”, “vertebral artery”, “NIRS”, “transcranial Doppler”), slaughter modalities (“stunning”, “captive bolt”, “electrical stunning”, “exsanguination”, “neck cut”, “religious slaughter”, “shechita”, “halal”, “slaughter without stunning”), and state descriptors (“unconscious”, “insensib*”, “loss of consciousness”, “sentience”). Reference lists of retrieved articles and recent regulatory opinions were also screened manually. Two authors (YVT, PVP) independently screened titles and abstracts, with discrepancies resolved by consensus. A PRISMA-style flow diagram summarizing the selection process is provided as *Supplementary Figure S1*, including the number of records identified, screened, assessed for eligibility and included. Studies were eligible if they met the following criteria: i) peer-reviewed and indexed in PubMed; ii) conducted in cattle or calves; iii) included neurophysiologic measurements, specifically EEG, electrocorticography (ECoG), or evoked potentials, with or without concomitant cerebral perfusion assessment; and iv) published in English. Studies were excluded if they lacked direct cortical or perfusion endpoints, relied solely on behavioral indicators without physiologic validation, or involved non-bovine species without clear translational relevance to cattle or calf neurophysiology. Although the review’s primary focus is calves, the primary literature on bovine slaughter neurophysiology is dominated by studies in adult cattle and bulls. We retained these studies because the underlying neurophysiological principles (thalamocortical integration, ischemic cortical failure) are conserved across age classes. Given substantial heterogeneity in experimental paradigms, EEG criteria, perfusion methodologies, and outcome definitions, findings were synthesized qualitatively, emphasizing mechanistic interpretation and cross-study coherence rather than pooled quantitative estimates. The results were synthesized in 4 different areas of interest: Neurobiological foundations; Electrophysiologic evidence across slaughter modalities; Cerebral blood flow and cortical viability; Comparative mechanistic interpretation.

Neurobiological foundations

Conscious awareness in mammals is predicated upon preserved thalamocortical processing and large-scale cortical network integration.[15] Contemporary neurobiological frameworks emphasize that wakeful experience requires functional connectivity between thalamic relay nuclei and distributed cortical regions, with dynamic synchronization across primary sensory, associative, and integrative networks.[16] Disruption of this coordinated activity, whether through mechanical trauma, electrically induced seizure activity, or ischemia, results in breakdown of thalamocortical communication and consequent loss of cortical function and therefore LOC.[17]

In large-animal models relevant to slaughter pathophysiology, cortical function has been directly interrogated using electroencephalography (EEG), whereas cortical activity can be measured using evoked potential. EPs are present even in states of unconsciousness such as under general surgical anesthesia.[18,19] EEG provides a measure of spontaneous population-level cortical activity, reflecting the integrity of large-scale neuronal synchronization. Evoked potentials, by contrast, assess stimulus-dependent cortical responsiveness and therefore offer more specific evidence of preserved thalamocortical activity. In cattle, Daly *et al*.[4] demonstrated that visual and somatosensory evoked cortical responses were rapidly abolished following effective captive bolt stunning, thereby confirming their utility as sensitive markers of loss of cortical activity.

A comparable mechanistic sequence has been documented during exsanguination. Gregory *et al*.[6] observed rapid disappearance of visual evoked cortical responses in calves subjected to carotid severance, indicating early failure of thalamocortical activity. Cerebral perfusion constitutes the essential metabolic substrate sustaining integrated cortical functions and activity.[20] Experimental data in calves show that catastrophic cerebral hypoperfusion during exsanguination leads to isoelectric EEG within seconds.[5] When cerebral blood flow falls below critical thresholds, cortical function and therefore consciousness is lost, thereby reinforcing the mechanistic linkage between perfusion failure and loss of consciousness.[21]

The principal operational definitions adopted in this review are presented in Table 1 while the interrelated neurophysiologic principles are summarized in Figure 1.

Table 1. Working definitions used in this review.

Term	Definition
Cortical activity	The spontaneous population-level electrical signal generated by cortical neurons, measurable as ongoing EEG. Loss of cortical activity ≈ isoelectric EEG.
Cortical responsiveness	The ability of the cortex to generate a stimulus-locked response (e.g., visual or somatosensory evoked potentials) to a defined sensory input. Loss of cortical responsiveness ≈ abolition of evoked potentials.
Cortical function	Integrated, large-scale processing underlying conscious perception, including preserved thalamocortical communication and dynamic synchronization across distributed cortical networks. Loss of cortical function is the neurobiological correlate of loss of consciousness (LOC).
Isoelectric EEG	An EEG with amplitude below an operational threshold (typically <10% of baseline or <10 µV in the reviewed studies). It implies loss of cortical activity but occurs after loss of cortical function (i.e., after LOC).
Loss of consciousness	An inferred neurobiological state in which integrated cortical processing is no longer supported. It is treated as a clinical/behavioral construct approximated, but not directly measured, by electrophysiological indicators.

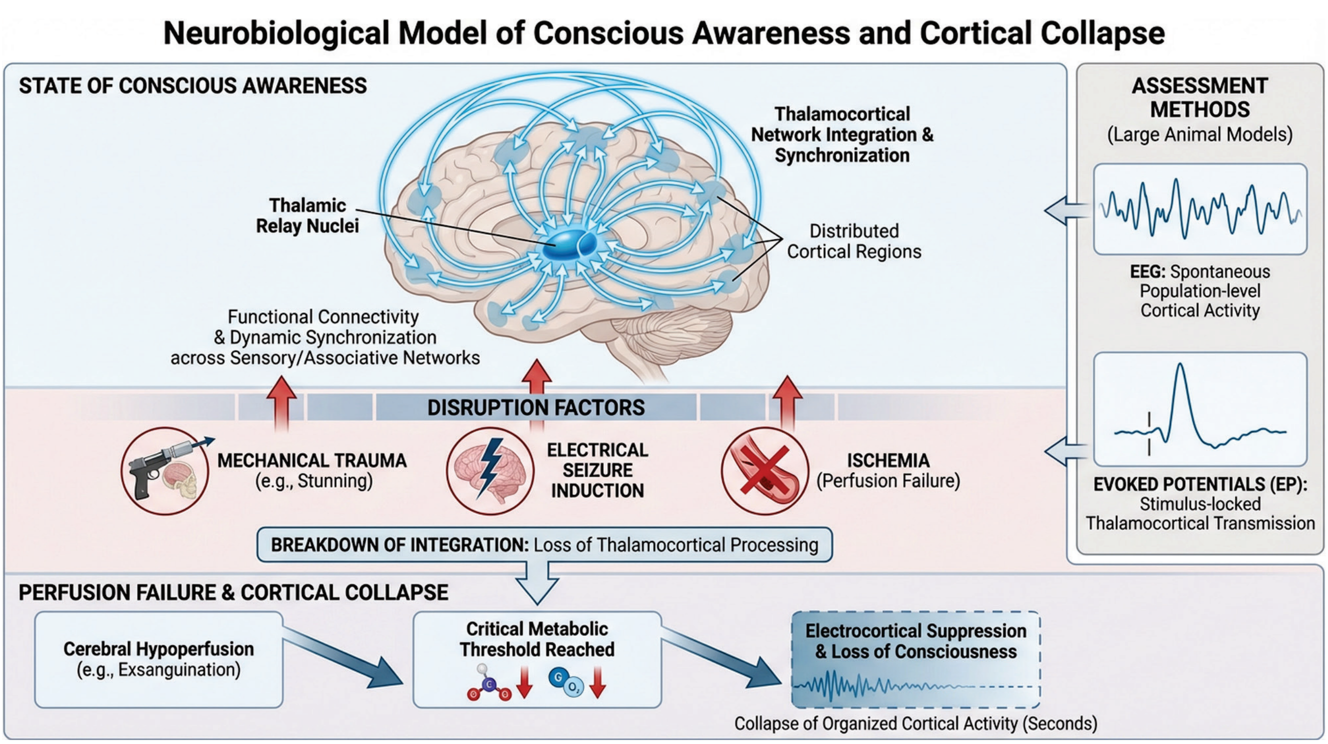

Figure 1. Neurobiological model of conscious awareness and cortical collapse. Conscious awareness is sustained by integrated, dynamically synchronized thalamocortical interactions between thalamic relay nuclei and distributed cortical regions, enabling functional connectivity across sensory and associative networks. Disruption by mechanical trauma (*e.g.*, stunning), electrically induced seizures, or ischemia/perfusion failure produces a breakdown of integration with loss of thalamocortical processing. In the setting of cerebral hypoperfusion (*e.g.*, exsanguination), crossing a critical metabolic threshold rapidly precipitates rapid (within single-digit seconds) loss of cortical function and therefore LOC. Conscious state and network integrity can be assessed using EEG (spontaneous population-level cortical activity).

Electrophysiologic evidence across slaughter modalities

Penetrating captive bolt

Experimental neurophysiologic investigations in calves provide direct cortical evidence supporting the efficacy of penetrating captive bolt (PCB) stunning. In a controlled study employing implanted EEG electrodes in veal calves, Lambooy *et al.*[10] demonstrated that correct frontal or occipital bolt placement produced rapid electrocortical alterations characterized by high-amplitude slow-wave activity swiftly transitioning to marked suppression, consistent with abrupt cortical dysfunction. In contrast, nuchal (neck) application resulted in delayed electrocortical suppression, with a mean interval of approximately 21 seconds before EEG criteria compatible with unconsciousness were achieved. Notably, corneal reflexes persisted until cortical suppression occurred, emphasizing that in cases of delayed cortical failure, such as nuchal placement, reflex persistence during that interval cannot be taken as evidence of preserved conscious awareness. These findings underscore the decisive importance of accurate bolt placement in determining both the immediacy and reliability of cortical failure.

Complementing these observations, Daly *et al.*[4] assessed cortical function in cattle using electrocorticography (ECoG) in combination with visual and somatosensory evoked potentials. They reported rapid and irreversible abolition of evoked cortical responses following effective PCB stunning, indicating immediate interruption of thalamocortical activity at the moment of impact. Because evoked responses represent stimulus-locked cortical activity, their disappearance only provides evidence of loss of cortical activity which implies LOC, but it does not tell us when LOC occurs. Collectively, these data support the conclusion that properly executed PCB induces rapid cortical failure, whereas technical inaccuracies may introduce measurable delay and variability. This interpretation is reinforced by more recent high-quality investigations demonstrating reliable EEG-defined unconsciousness when appropriate energy delivery and positioning criteria are met.[11]

Electrical stunning

Electrical stunning produces a characteristic neurophysiologic sequence consisting of generalized epileptiform activity followed by post-ictal cortical suppression.[5,22] The initial synchronized seizure activity reflects widespread cortical depolarization, while the subsequent suppression phase corresponds to transient disruption of thalamocortical integration. In adult cattle, Devine *et al.*[23] documented marked EEG amplitude reduction and altered spectral patterns shortly after electrical stunning, demonstrating electrocortical states incompatible with conscious processing.

Mechanistically, head-only electrical stunning induces a potentially reversible state;[24] if exsanguination is delayed, cerebral perfusion is maintained and cortical activity may recover. In contrast, head-to-body electrical stunning combines seizure induction with ventricular fibrillation or cardiac arrest, resulting in cessation of cerebral perfusion and elimination of neurologic recovery potential.[25] These mechanistic distinctions are critical for interpreting EEG endpoints and for evaluating the temporal reliability of unconsciousness across electrical stunning modalities.

Non-penetrating captive bolt

Non-penetrating captive bolt (NPCB) stunning relies on concussive transfer of kinetic energy without skull penetration, aiming to induce sufficient cerebral concussion to disrupt cortical integration. In calves, Gibson *et al.*[26] reported electrocortical suppression following NPCB application under controlled conditions, demonstrating that concussive force can induce EEG patterns consistent with unconsciousness. However, subsequent EEG assessments in bovines revealed greater variability and occasional incomplete cortical suppression compared with penetrating methods, highlighting sensitivity to delivered energy, anatomical variability, and application technique.[11] These findings suggest that while NPCB can achieve cortical failure, its reliability may be more dependent on precise technical parameters.

Slaughter without stunning

Experimental studies of exsanguination without prior stunning provide direct insight into loss of cortical function and activity. Gregory *et al.*[6] assessed visual evoked cortical responses during slaughter and observed disappearance of stimulus-locked cortical activity approximately 17 seconds after bilateral carotid severance, indicating rapid cessation of thalamocortical activity. Complementary investigations evaluating spontaneous EEG activity demonstrated suppression within seconds in most calves; however, rare prolonged cases were documented and attributed presumably to carotid occlusion or more likely due to incomplete vessel transection.[5] These cases underscore a critical welfare risk: technical failures in both slaughter with stunning and without stunning due to inter-operator variability in procedure technique may produce outlier cases in which cortical failure is substantially delayed beyond the typical range.

Importantly, validation studies comparing animal-based measures (ABMs) with electrophysiologic endpoints confirm imperfect concordance.[7] Certain behavioral indicators persisted beyond EEG-defined unconsciousness, whereas others ceased before cortical suppression occurred. This discordance underscores the limitations of ABMs as standalone markers of conscious state and highlights the added value of hemodynamic and electrophysiologic validation.

Collectively, the literature delineates two mechanistic pathways to loss of cortical function: abrupt network disruption in stunning modalities due to mechanical tissue trauma and similarly rapid loss of cortical function due to catastrophic loss of cerebral blood flow and pressure during exsanguination without stunning. A synthesis of these findings is presented in Table 2, which summarizes modality-specific mechanisms, temporal profiles, and sources of variability.

Cerebral blood flow and cortical viability

Throughout this section we distinguish between measured endpoints, what the EEG and evoked potentials directly show and mechanistic inference, what physiological reasoning suggests must be happening upstream of those endpoints. The two are not interchangeable. Stunning and non-stunning slaughter modalities ultimately converge on the endpoint of loss of cortical function; however, they achieve this state through fundamentally distinct neurophysiologic pathways. Penetrating captive bolt stunning induces rapid mechanical disruption of cortical and subcortical structures, abolishing stimulus-locked evoked cortical responses at the moment of impact, as demonstrated by Daly *et al.*[4] using electrocorticography and visual and somatosensory evoked potentials in cattle. Electrical stunning, by contrast, generates generalized epileptiform activity followed by post-ictal cortical suppression, with EEG patterns incompatible with conscious processing documented in bovines.[23] In this context, cortical inactivity reflects seizure-induced network disruption rather than primary ischemia.

Table 2. Key studies examined in the research.

Domains	Study (year)	Animal	Group	EEG/ ECoG	EEG criterion for unconsciousness	Key findings
Penetrating captive bolt (PCB)	Lambooy (1981)[10]	Veal calves	Frontal *vs* occipital *vs* nape/neck placement	EEG (implanted electrodes)	Delta/theta tending to isoelectric line; consciousness judged from EEG	Frontal and occipital: 'almost certainly immediate' unconsciousness; nape: mean 21±6 s to unconsciousness; calves conscious until then
	Daly *et al.* (1988)[4]	Cattle	Bolt velocity comparison: 41 *vs* 47 *vs* 55 *vs* 58 m/s	ECoG (spontaneous cortical activity; epidural/dural electrodes) + VER (visual evoked responses)	- Not an EEG-only criterion; - Use loss of VER as primary indicator of insensibility; "severe reduction" of spontaneous of spontaneous ECoG defined as <10% of pre-shot baseline; VER considered "present" if >10% baseline	- Higher bolt velocity (55–58 m/s) ↓ likelihood of VER presence in the first ~8 s and ↑ time to VER recovery; however, VER could persist immediately after stun at all velocities. Spontaneous ECoG suppression (<10%) did not differ significantly by velocity. - 55 m/s as minimum recommended velocity
Electrical stunning	Devine *et al.* (1986)[23]	Adult cattle	Immobilization (n=10) *vs* no immobilization (n=2)	Implanted EEG (frontal bone screw electrodes; transhemispheric bipolar); recorder disconnected during stun/immobilization (no EEG during current)	Uses "window of sensibility" based on LVFA amplitude ~10–40 µV - insensibility inferred when EEG falls <10 µV (toward isoelectric) and LVFA absent/ outside window	EEG amplitude <10 µV at end of immobilization; in non-immobilized animals reached <10 µV at 57 s and 63 s after stun initiation
	Bager *et al.* (1992)[5]	Calves	Head-only electrical stun + slaughter *vs* no-stun slaughter	ECoG+ FFT analysis of power bands 8-30 Hz (sensibility-associated) and 2-8 Hz (insensibility/ anoxia-associated)	Loss of sensibility inferred from electrocortical activity analysis (FFT; HALF etc.)	Stunned calves: permanently insensible at time of stun. Non-stunned: 6 calves lost sensibility within 10 s; one calf with carotid clot had cortical activity >52 s but sensibility not regained (FFT)
Non-penetrating captive bolt (NPCB)	Gibson *et al.* (2009)[26]	Calves	Single group	EEG + FFT to compute total power (Ptot); visual classification: active/ transitional/ high-amplitude low-frequency/ isoelectric	Visual inspection + changes in total power (Ptot)	Changes sufficient to produce insensibility within 0–14 s in conscious animals (inference); biphasic Ptot response in 5 animals

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Table 2. Continued from previous page.

Domains	Study (year)	Animal	Group	EEG/ ECoG	EEG criterion for unconsciousness	Key findings
	Verhoeven *et al.* (2016)[7]	Veal calves	(1) Inverted captive bolt + neck cut (n=25) (2) Upright no-stun (n=7) (3) Inverted no-stun (n=25); (4) Upright no-stun then captive bolt at ~34±8 s (n=25)	Non-invasive EEG electrodes	Visual EEG staging: baseline → transitional → unconscious when low-freq/high-amp dominates → minimal brain activity/flat EEG defined as <10% baseline amplitude	After captive bolt, absence of threat/ withdrawal/corneal/ eyelid reflex indicated unconsciousness per EEG; after non-stun, threat/withdrawal lost before EEG-defined unconsciousness; eyelid/corneal were conservative, persisting until ~76±50 and 85±45 s after EEG loss of consciousness
	Gibson *et al.* (2019)[11]	Bulls	PCB (n=20) *vs* NPCB (n=11)	EEG; visual classification + spectral analysis	Visual EEG states: Normal, Transitional (suppressed; <½ baseline amp), HALF (high-amp low-freq), Isoelectric (amp <1/8 = 12.25% baseline, little/no low-freq)	PCB: 20/20 inconsistent with consciousness; NPCB: 82% complete unconsciousness; 2 bulls showed normal EEG activity periods indicating incomplete concussion
Slaughter without stunning	Gregory and Wotton (1984)[6]	Calves	Exsanguination after cutting carotids/jugulars	ECoG (implanted electrodes) + flash visual evoked cortical responses (VER/EP)	Loss of visual evoked cortical response to flash used as indicator of loss of brain responsiveness	Time from sticking to loss of brain responsiveness: 17 ± 4 s (range 11.5–23 s) “Flat” ECoG occurred later on average (23 ± 11 s), supporting that evoked response loss is a more direct marker than waiting for isoelectric ECoG
	Small *et al.* (2025)[27]	Cattle	EEG recorded in 6; one handled commercially without EEG delay	Non-invasive EEG; offline analysis: RMS power, FFT Ptot (0-30 Hz), SEF50, SEF95, and band power	No fixed numeric EEG threshold; “insensibility duration” operationally based on absence of eye reflexes and awareness, with EEG used to document suppression and frequency shifts (↓SEF50/95, ↓Ptot) during the unconscious period	- Behavioral loss of consciousness within 10 s; EEG-based insensibility duration ~63 s or more; - EEG showed suppression and shift to lower frequencies (↓SEF50/95; ↓Ptot) for ~60-90 s or longer, with gradual recovery - Full behavioral recovery observed

Exsanguination without prior stunning produces a rapid fall in cerebral perfusion. The rate of fall has been estimated indirectly from carotid hemodynamic measurements and computational models: Benchimol *et al*.[12,13] reported substantial reductions in modelled cerebral inflow within the first second after bilateral carotid section, while vertebral artery contributions can partially sustain cortical perfusion in some animals for several seconds.[12,13] Recent *in vivo* work in cattle has refined these estimates and reported time courses on the order of single-digit seconds for clinically meaningful perfusion loss;[28] earlier observations are consistent.[29] Therefore, the rapid reduction in cerebral blood flow is characterized by a decline to electrocortically critical perfusion within approximately 1-10 seconds, with inter-individual variability largely determined by the completeness of vessel transection and the contribution of vertebral collateral flow. This temporal profile reflects ischemia-driven failure of cortical metabolism. Once CBF falls below the threshold required to sustain ATP production, the Na^+/K^+ ATPase fails, ionic gradients collapse and synaptic transmission is no longer supported, producing electrocortical silence.[20,21] Bager *et al*.[5] provided direct biochemical evidence for this sequence in calves, reporting concomitant changes in cerebral lactate and energy charge during exsanguination, alongside the EEG findings already discussed.

Integration of EEG with CBF assessment, as illustrated in Figure 2, permits mechanistic differentiation between concussive suppression (direct mechanical disruption), post-ictal suppression (transient seizure-related cortical inactivity, and ischemic cortical failure (perfusion-dependent collapse).[30] EEG reports cortical electrical activity in real time but is silent on its cause. Concurrent perfusion monitoring identifies whether an observed change in EEG is being driven by failure of the metabolic substrate (cerebral hypoperfusion) or by something else (e.g. mechanical disruption, post-ictal suppression, or anesthetic effect). It is in this specific causal sense, explaining why the EEG is changing, that perfusion data provide context for the cortical electrical signal.[31] Non-invasive approaches such as near-infrared spectroscopy (NIRS) for regional cerebral oxygenation and Doppler ultrasonography for flow velocity measurement offer experimentally feasible methods to correlate perfusion dynamics with electrophysiologic endpoints.[32] To date, however, these modalities have not been systematically validated alongside EEG in calf slaughter research, and their translational utility in this context remains to be established. Such multimodal integration enhances interpretive precision and reduces the likelihood of misclassifying transient cortical suppression as definitive loss of consciousness.[33] Several non-invasive techniques used in human peri-operative neuromonitoring, near-infrared spectroscopy (NIRS) for regional cerebral oxygenation and Doppler ultrasonography for flow velocity, are conceptually transferable to the calf slaughter setting.[32] However, we are not aware of published validation studies that have applied these techniques alongside EEG during commercial calf slaughter and several practical obstacles (sensor coupling on hide-covered skull, motion artefact, signal robustness during exsanguination) are substantial and unresolved. The integration of EEG with cerebral perfusion monitoring should therefore be regarded as a hypothesis-generating proposal that requires validation studies in cattle before it can be recommended for operational use.[33]

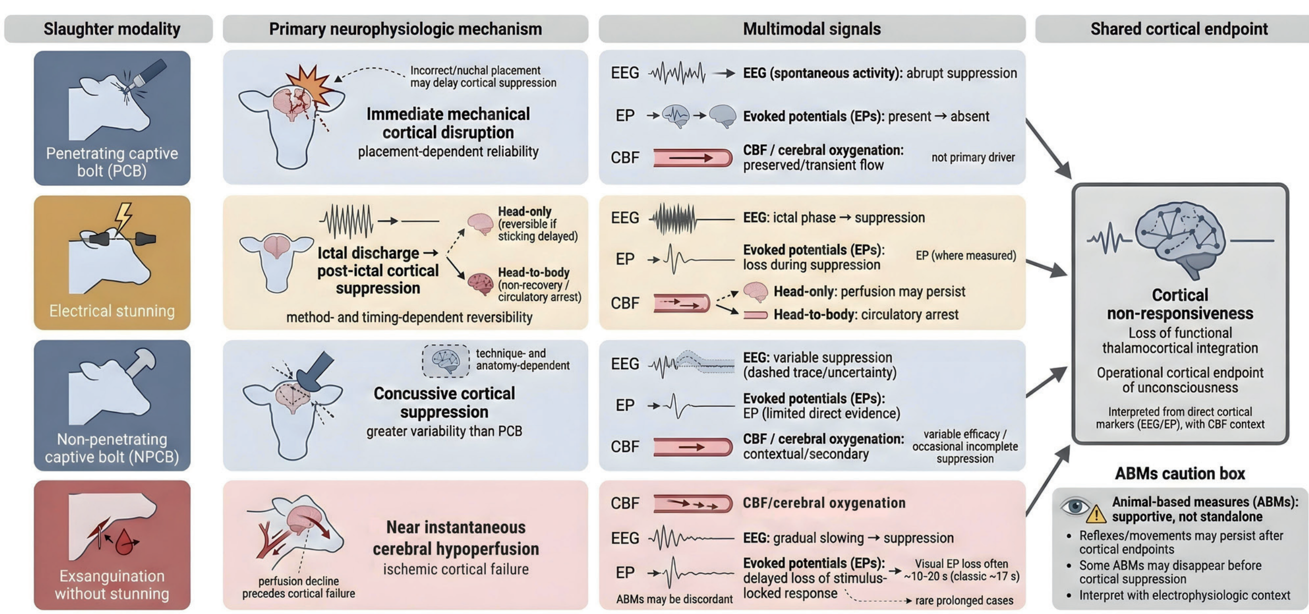

Figure 2. Mechanistic integration of electrophysiologic and cerebral perfusion pathways to cortical unconsciousness across slaughter modalities in calves. The schematic illustrates how PCB, electrical stunning, NPCB, and exsanguination without prior stunning converge on a shared cortical endpoint (cortical non-responsiveness) through distinct neurophysiologic pathways. Mechanical methods primarily induce concussive/mechanical cortical disruption, electrical stunning produces an ictal (seizure-like) phase followed by post-ictal cortical suppression, and exsanguination leads to cerebral hypoperfusion with near instantaneous loss of cortical function. EEG and EPs are shown as the principal direct cortical markers, whereas CBF/cerebral oxygenation provides physiologic context for mechanistic interpretation. Animal-based measures are included as supportive operational indicators but not standalone markers of consciousness. This figure is a conceptual synthesis of mechanisms reported in the cited studies; it is hypothesis-generating and does not present.

Comparative mechanistic interpretation

The principal slaughter modalities differ in their primary neurophysiologic mechanisms, yet all ultimately converge on the endpoint of loss of cortical activity, but this does not equate to time to LOC which is a reflection of loss of integrated cortical functions. Available evidence does not allow LOC to be located at a single threshold of CBF or pressure expressed as a percentage of baseline. The relevant physiological inference is that conscious processing requires perfusion sufficient to sustain cortical electrical activity and that the electrocortical correlates of LOC follow rather than coincide with the perfusion fall. Empirically, in calves subjected to exsanguination without prior stunning, electrocortical patterns compatible with LOC are reported within single-digit seconds, and isoelectric EEG within approximately 17-23 sec, after carotid section.[5,6] The interval between these two endpoints reflects the gradient between perfusion that can no longer sustain organized cortical processing and perfusion that can no longer sustain any cortical electrical activity.

PCB stunning produces rapid mechanical alteration of cortical and subcortical structures. When accurately positioned, abolition of stimulus-locked evoked cortical responses occurs within seconds of impact, indicating abrupt interruption of thalamocortical integration. However, incorrect placement may significantly delay cortical suppression, as demonstrated in calves, thereby introducing meaningful placement-dependent variability in the temporal profile of unconsciousness.[10]

Electrical stunning induces a characteristic sequence of generalized epileptiform activity followed by post-ictal cortical suppression. EEG patterns incompatible with conscious processing have been documented in cattle subjected to properly delivered electrical stunning.[23] The reliability of this modality depends critically on technical parameters, including current density, electrode placement, impedance, and the interval between stunning and exsanguination. NPCB relies exclusively on concussive force. Although EEG suppression consistent with unconsciousness has been observed, variability appears greater than with PCB, reflecting heightened sensitivity to delivered kinetic energy and individual anatomical differences.[26]

By contrast, neck cutting without prior stunning induces rapid cerebral hypoperfusion.[12,13] with loss of visual evoked cortical responsiveness typically occurring within 10-20 seconds, representing isoelectric state or brain death a point where LOC has already occurred.[5,6] Collectively, the available literature supports a high probability of rapid unconsciousness under optimized technical conditions; however, it does not substantiate claims of absolute certainty across all individuals and operational contexts.

Figure 3 provides a graphical synthesis of these modality-specif-

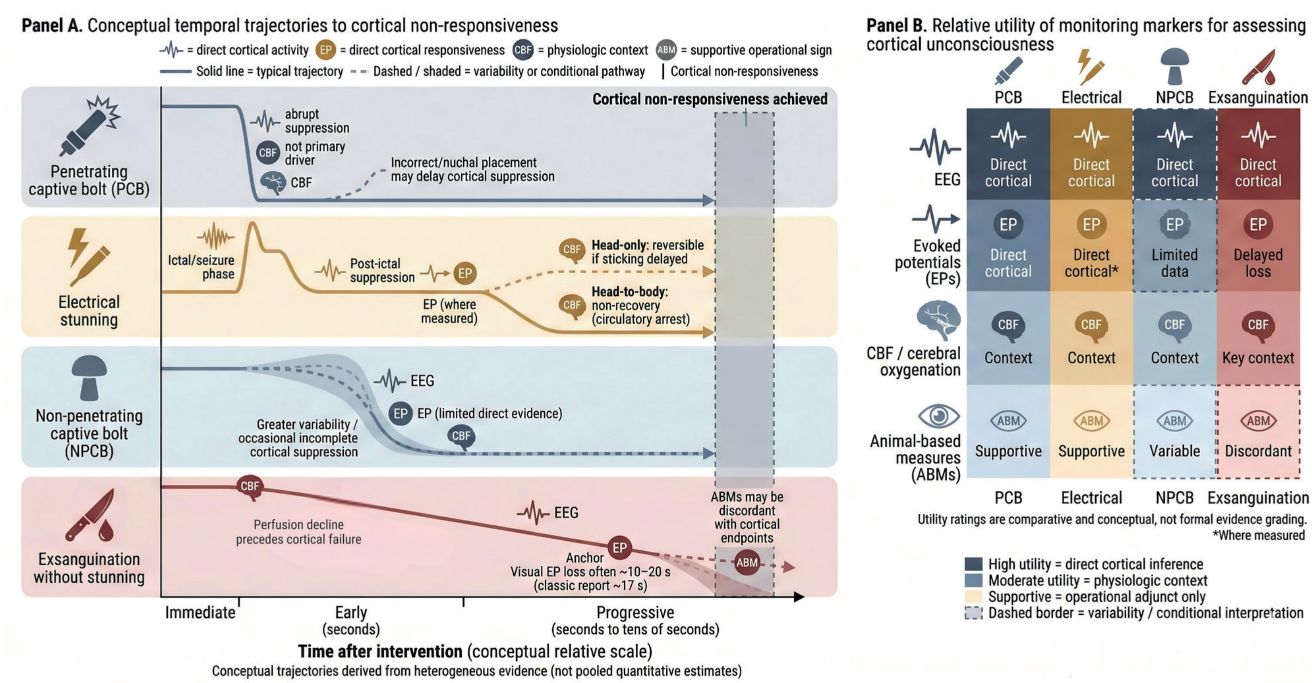

Figure 3. Comparative temporal trajectories and relative utility of monitoring markers for cortical unconsciousness assessment across slaughter modalities in calves. Panel A shows a conceptual, modality-specific timeline to cortical non-responsiveness (relative time scale). PCB typically produces immediate mechanical cortical disruption when correctly positioned, but malposition (e.g., nuchal placement) may delay electrocortical suppression. Electrical stunning follows an ictal (seizure-like) phase with subsequent post-ictal cortical suppression, with reversibility influenced by stunning method and stun-to-stick interval. NPCB can induce cortical suppression but shows greater variability and occasional incomplete suppression. Exsanguination without prior stunning leads to near instantaneous loss of cortical function (LOC) followed by progressive loss of cortical activity to isoelectric state or brain death. Panel B summarizes the relative utility of monitoring markers: EEG and EPs are the principal direct cortical markers (with variable evidence density across modalities), CBF/cerebral oxygenation provides physiologic context (particularly for exsanguination), and ABMs are supportive but not standalone due to imperfect concordance with electrophysiologic endpoints. This schematic is a conceptual synthesis of heterogeneous studies, not a pooled quantitative estimate. This figure is a conceptual synthesis of mechanisms reported in the cited studies; it is hypothesis-generating and does not present pooled quantitative data. The relative time scale is qualitative; the figure should not be read as reporting measured time intervals.

ic mechanisms. Nonetheless, several mechanistic and quantitative aspects remain insufficiently characterized and warrant further rigorous investigation.

Discussion

Electrocorticographic and evoked potential studies have demonstrated rapid and irreversible disruption of thalamocortical transmission following correctly applied penetrating captive bolt stunning.[4] In contrast, during exsanguination without prior stunning, loss of visual evoked cortical responsiveness occurs within seconds as cerebral hypoperfusion occurs, reflecting ischemia-driven collapse of cortical integration while physiology and cerebral blood flow and pressure studies indicate that this is near instantaneous.[5,6] Electrical stunning induces a characteristic sequence of generalized epileptiform activity followed by post-ictal suppression, with EEG patterns incompatible with conscious processing when technical parameters are adequate.[23] Mechanistically, stunning modalities directly compromise cortical network integrity, whereas exsanguination produces progressive metabolic failure secondary to diminished cerebral perfusion. We acknowledge that this topic is contested. The mechanistic framework presented here is intended to clarify which questions are answered by the existing evidence, which are partially answered and which remain open. It is not intended to adjudicate the welfare debate around any specific slaughter practice; that adjudication requires data we explicitly identify as still lacking.

Despite this mechanistic coherence, interpretive certainty is moderated by several limitations. Experimental cohorts are frequently small, restricting robust estimation of rare or tail-risk events. EEG endpoint criteria and analytical methods vary across studies, diminishing cross-study comparability. Multicenter EU-scale data that capture operational heterogeneity remain limited. Moreover, simultaneous EEG-cerebral blood flow (CBF) measurements in calves are uncommon, limiting physiologic triangulation of cortical failure mechanisms.

This topic, previously highlighted in the literature,[34] warrants particularly careful scientific attention, as it lies at the center of ongoing debates surrounding slaughter practices, including those commonly referred to as "religious slaughter". The controversy is frequently fueled by differing interpretations of physiologic evidence and by uncertainty regarding the precise timing of loss of consciousness. A rigorous, mechanistically grounded approach is therefore essential to ensure that policy discussions are informed by objective neurophysiologic data rather than by assumptions or incomplete interpretations.

In alignment with Horizon Europe priorities emphasizing sustainable food systems, technological innovation, and evidence-based regulation, future research should prioritize multicenter, EU-scale validation studies integrating electroencephalography (EEG) and cerebral blood flow (CBF) assessment in calves. The development of field-deployable, multimodal EEG-oxygenation headsets capable of rapid application in commercial slaughter environments would substantially enhance translational feasibility and ecological validity. Moreover, the application of machine-learning algorithms for real-time detection of cortical failure patterns may further improve monitoring precision and standardization across facilities. Quantification of upper-percentile (*e.g.*, 99th percentile) time-to-cortical-failure distributions would strengthen reliability modeling and risk characterization. Finally, harmonization of cortical endpoint definitions across Member States would enhance regulatory coherence and reduce residual epistemic uncertainty in welfare assessment.

Limitations

This structured narrative review is subject to inherent methodological constraints. The primary literature on electrophysiologic determination of unconsciousness in calves remains limited in scope and scale, with many seminal investigations conducted in controlled laboratory environments that may not fully reflect commercial slaughter variability. As a result, the estimation of low-frequency delayed cortical failure events remains constrained. Substantial heterogeneity in EEG acquisition, signal processing, and endpoint definitions complicates direct comparison across studies and precludes quantitative synthesis. Inconsistent application of evoked potential paradigms further limits harmonization of cortical outcomes. Additionally, simultaneous measurement of cerebral perfusion alongside EEG remains sparse in bovine research, so mechanistic inferences linking perfusion decline to cortical electrical failure are often indirect. The calf-specific evidence base is narrower than the broader bovine literature; conclusions that depend on adult-cattle data should be tested in age-matched calf cohorts before being treated as established for the calf. Finally, as a narrative synthesis, even when structured according to SANRA principles, this review does not meet the methodological rigor of a systematic review or meta-analysis and may be influenced by publication bias. Collectively, these considerations underscore the necessity for coordinated, multicenter, multimodal investigations to elucidate the precise relationship between cortical electrophysiology and cerebral perfusion in calves.

Conclusions

Current evidence supports the use of EEG and stimulus-locked evoked potentials as the most direct cortical indicators of LOC available in calves. Across slaughter modalities, electrocortical changes compatible with LOC are reported within single-digit sec when stunning is correctly applied and within approximately 10-20 sec following exsanguination without stunning, with isoelectric EEG occurring later than the operational onset of LOC. Cerebral perfusion is the physiological substrate that ultimately determines cortical electrical activity, but during slaughter it has not been measured directly at the cortex; current inferences rest on carotid hemodynamics and modelling. We therefore propose, but do not yet substantiate, that integrated EEG-perfusion monitoring may improve the precision of welfare assessment. The validation studies required to test this proposal, combined EEG and non-invasive cerebral oxygenation/flow monitoring in calves under controlled and commercial conditions, are the next step.

Abbreviations

ABMs: animal-based measures
CBF: cerebral blood flow
ECoG: electrocorticography
EEG: electroencephalography
EFSA: European Food Safety Authority
EP: evoked potentials
EU: European Union

FFT: fast fourier transform
HALF: high-amplitude low-frequency
LVFA: low voltage fast activity
NIRS: Near-infrared spectroscopy
NPCB: non-penetrating captive bolt
PCB: penetrating captive bolt
Ptot: total power
RMS: root mean square
SANRA: Scale for the Assessment of Narrative Review Articles
SEF50: spectral edge frequency 50
SEF95: spectral edge frequency 95
VER: visual evoked responses

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Online supplementary material:
Figure S1. PRISMA flowchart.

Received: 30 March 2026; Accepted: 23 June 2026.

Contributions: Y Van Tran, formal analysis, writing - original draft preparation, writing - review & editing. Phong Van Pham, formal analysis, writing - original draft preparation, writing - review & editing. Ameen A. Al Alwany, writing - review & editing. Dariusz Myrcik, methodology. Giacomo Fari, methodology. Nicola Cimini, formal analysis, writing - original draft preparation, writing - review & editing. Matteo Luigi Giuseppe Leoni, methodology, formal analysis, writing - original draft preparation, writing - review & editing. Giustino Varrassi, conceptualization, supervision. All authors have read and agreed to the submitted version of the manuscript.

Conflict of interest: the authors declare no competing interests relevant to the content of this article and all authors confirm accuracy.

Data availability: the data that support the findings of this study are available on request from the corresponding author.

Acknowledgments: we extend our sincere gratitude to the Fondazione Paolo Procacci (Rome, Italy) for their invaluable support and assistance throughout the publication process.