


SYSTEMATIC REVIEW

Received: 26 February 2025 *Accepted : 02 November 2026*
Last revision: 20 October 2026 *Published: 19 November 2026*

Suspected Vagal Reflex-Mediated Death in Forensic Practice: A Systematic Review of Autopsy and Clinical Case Reports

Mohammad Tegar Indrayana[1], Bima Diokta Alparisi[2*], Regina Trisya Arenja[2], Najmi Khairussyifa[2], Patricia Dean Ully Marbun[2], Shalsabila Ghaasani Amanda[2]

[1]Department of Forensic Medicine, Faculty of Medicine, University of Riau, Pekanbaru, Indonesia
[2]Clinical Clerkship Program, Faculty of Medicine, University of Riau, Pekanbaru, Indonesia

ABSTRACT

Introduction: Fatal vagal inhibition is physiologically plausible but lacks a pathognomonic postmortem marker. This review critically examines reported fatal events in which vagal or related cardioinhibitory reflexes were proposed. **Methods:** Five databases were searched for English- or Indonesian-language case reports and case series published from 2015 through 2024. Eligible reports described a fatal outcome with relevant forensic or perioperative evidence. Reporting followed PRISMA 2020, and quality was assessed using the JBI case-report checklist. A post hoc descriptive framework classified confidence in reflex involvement as probable, possible, or indeterminate. **Results:** Thirteen publications described 16 decedents, including 12 males, aged 15 months to 81 years. Seven deaths occurred in accidental or therapeutic circumstances, five followed assaults, two were suicidal, and two were natural. Reported stimuli included cervical vagal injury, neck compression, airway manipulation, craniofacial impact, and disease-associated autonomic dysfunction. Four reports were classified as probable reflex involvement, five as possible, and four as indeterminate. Competing explanations included mechanical asphyxia, occult central nervous system injury, pulmonary edema, and structural disease. Confidence in reflex involvement did not establish it as the exclusive fatal mechanism. **Conclusion:** Forensic attribution requires converging temporal, anatomical, and physiological evidence, together with systematic assessment of alternative causes. Opinions should qualify reflex involvement and distinguish the initiating cause, terminal mechanism, and legal manner of death.

Keywords: Autopsy, cause of death, forensic medicine, sudden death, vagal inhibition, vagal reflex.

© 2026 Mohammad Tegar Indrayana, Bima Diokta Alparisi, Regina Trisya Arenja, Najmi Khairussyifa, Patricia Dean Ully Marbun, Shalsabila Ghaasani Amanda
Cermin Dunia Kedokteran is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

https://doi.org/10.55175/g9k9z116
e-ISSN: 2503-2720

INTRODUCTION

Sudden death presents a substantial clinical and medicolegal challenge because a rapidly fatal event may leave limited structural evidence and permit several competing explanations. Cardiovascular disease predominates, although neurological, respiratory, gastrointestinal, toxicological, and reflex-mediated mechanisms also require consideration.[1–3] Forensic interpretation must distinguish the injury or disease that initiated the fatal sequence from its terminal physiological mechanism and the legal manner of death.

The vagus nerve provides the principal parasympathetic connection between the brainstem and thoracoabdominal viscera. Its predominantly sensory fibers convey mechanical, chemical, inflammatory, and metabolic information to the nucleus tractus solitarius, whereas cardiac efferents modulate sinoatrial and atrioventricular nodal activity.[4–10] Excessive activation of a cardioinhibitory reflex can cause abrupt bradycardia, atrioventricular block, hypotension, and, in severe circumstances, asystole. Associated sympathetic withdrawal may produce vasodilation, while some reflex circuits also suppress respiration. Experimental studies have identified a genetically defined population of cardiac vagal sensory neurons that can reproduce the Bezold–Jarisch triad of bradycardia, hypotension, and respiratory suppression.[11,12]

The term "vagal reflex" encompasses anatomically distinct circuits. Direct stimulation of the cervical vagal trunk differs from carotid sinus–mediated cardioinhibition, whose afferent limb is predominantly glossopharyngeal. Craniofacial stimulation may engage a trigeminocardiac reflex, whereas cardiac vagal afferents participate in the Bezold–Jarisch reflex. These circuits can converge on brainstem autonomic networks and share vagal cardiac efferents, but their afferent pathways and initiating stimuli are not interchangeable.[1,7,10,12] The terminal reflex is transient and cannot be directly demonstrated at autopsy; pulmonary edema, visceral congestion, and fluid blood are nonspecific findings.

***Correspondence Author** bimadiokta21@gmail.com*





A forensic attribution therefore requires convergence of circumstances, temporal sequence, anatomical plausibility, and a sufficiently comprehensive investigation of alternative causes. Misattributing death to a vagal reflex may conceal homicidal neck trauma or mechanical asphyxia, distort the assigned manner of death, and undermine subsequent legal proceedings.[13,14] This review critically reassesses reported fatal events in which vagal or related cardioinhibitory reflexes were proposed, focusing on causal evidence, competing explanations, and the distinction between the cause, mechanism, and manner of death.

METHODS

Review Design and Reporting

This systematic review was reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 statement.[15] The review was not prospectively registered. The evidence comprised heterogeneous case reports and small case series; quantitative pooling was neither planned nor considered appropriate.

Eligibility Criteria

Eligibility was structured using a population–phenomenon of interest–context framework. The population comprised human decedents, including patients who experienced cardiac arrest and subsequently died. The phenomenon of interest was suspected excessive vagal or related cardioinhibitory reflex activation. Eligible reports provided forensic autopsy findings, postmortem imaging, histopathology, scene reconstruction, or detailed perioperative physiological observations. English- or Indonesian-language case reports and case series published from January 2015 through December 2024 were considered. Reviews, animal studies, therapeutic vagus nerve stimulation studies, nonfatal events, and reports lacking sufficient clinical or forensic information were excluded.

Information Sources and Search Strategy

Three reviewers independently conducted a systematic search across five electronic databases: PubMed, ScienceDirect, Google Scholar, Taylor & Francis, and ProQuest. The search was restricted to English- and Indonesian-language case reports and case series published from January 2015 through December 2024. The core search strategy combined terms related to fatal outcomes, vagal pathways, and forensic investigation using the following Boolean search syntax: ("death" OR "sudden death" OR fatal) AND ("vagal reflex" OR "vagal inhibition" OR "vagus nerve" OR "nervus vagus" OR "cranial nerve X" OR "cardioinhibitory reflex") AND (forensic OR autopsy OR postmortem). Additionally, the reference lists of all eligible reports and relevant review articles were manually examined to identify additional relevant cases. The search and reporting processes were conducted in accordance with the PRISMA 2020 statement.

Study Selection and Data Extraction

Three reviewers participated in title-and-abstract screening, full-text assessment, and data extraction against prespecified eligibility criteria. Disagreements were resolved by consensus. Extracted variables comprised publication year, country of the reported event, number of decedents, age, sex, precipitating event, survival interval, physiological observations, postmortem investigations, the certified or proposed cause and manner of death, anatomical evidence involving the vagus nerve or a related reflex circuit, and plausible competing mechanisms. Critical Appraisal and Causal Interpretation

Critical Appraisal and Causal Interpretation Reporting quality was evaluated using the Joanna Briggs Institute (JBI) critical appraisal checklist for case reports.[16] Critical appraisal findings informed the qualitative synthesis and interpretation rather than serving as a rigid numerical threshold for study exclusion. To evaluate causal attribution, a post hoc descriptive framework was established to distinguish confidence in reflex involvement from certainty that a reflex acted as the sole fatal mechanism. Reflex involvement was classified into three categories: probable, when an immediate, anatomically credible trigger was supported by documented bradycardia, asystole, or focal neural injury, and the report favored a reflex contribution after evaluating alternative causes; possible, when a reflex contribution remained physiologically plausible but unconfirmed, particularly in the presence of a competing mechanism; and indeterminate, when temporal or anatomical evidence was insufficient to evaluate a reflex contribution. An independently sufficient alternative cause precluded attributing death solely to a reflex mechanism, regardless of the involvement category assigned. These categories represent qualitative interpretive judgments rather than validated diagnostic criteria or numerical probabilities.

RESULTS

Study Selection

Electronic database searching identified 295 records. After removing 18 duplicates, 277 records underwent title and abstract screening, leading to the exclusion of 250 records. All 27 reports sought for retrieval were successfully retrieved and assessed in full text. Fourteen reports did not meet the eligibility criteria and were excluded. Consequently, 13 publications describing 16 decedents were included in the qualitative synthesis **(Figure 1)**.

Characteristics of the Included Reports

The 13 publications described events in Canada, India, France, Sri Lanka, Italy, Japan, China, Nigeria, and Tunisia. Twelve of the 16 decedents were male, and ages ranged from 15 months to 81 years. Most reports incorporated a medicolegal autopsy; one perioperative report relied primarily on contemporaneous physiological observations. Accidental or therapeutic circumstances accounted for seven deaths, assaults for five, suicide for two, and natural disease for two. These descriptive circumstances were not treated as interchangeable with a formally certified legal manner of death. The principal trigger categories comprised direct cervical vagal injury, neck compression or carotid sinus loading, upper-airway stimulation, craniofacial impact, and disease-associated autonomic dysfunction. **Table 1** summarizes the included reports.[13,14,17–27]

Forensic Patterns and Strength of Causal Evidence

The strongest support for reflex involvement occurred in reports documenting focal cervical vagal injury or an immediate physiological response during manipulation. Potenza, *et al.*, Barranco, *et al.*, and Watanabe, *et al.*, described hemorrhage or perineural injury in close anatomical relation to the vagal trunk. Omokanye, *et al.*, documented





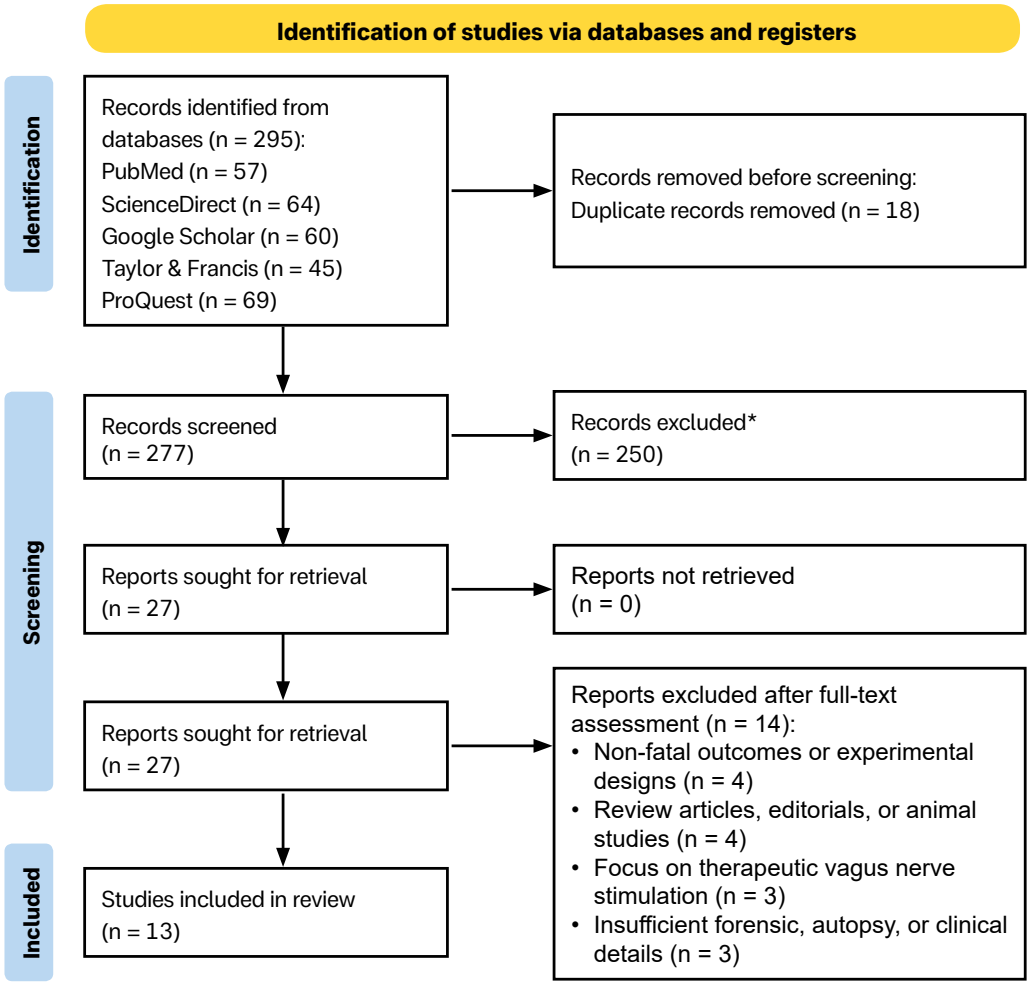


Figure 1. Study identification and selection flow diagram following the PRISMA 2020 structure.[15]
Note: *Records excluded during title and abstract screening based on predefined eligibility criteria (n = 250).

Table 1. Characteristics of the included reports and fatal events.[13,14,17–27]

Study; Country	Year	n	Age/ Sex	Precipitating Event and Clinical Course	Postmortem or Physiological Evidence	Authors' Principal Cause-of-Death Interpretation
Mattu and Orde, Canada.[17]	2024	1	53 y/M	Road-traffic collision followed by resuscitation; chewing gum was identified in the larynx.	Autopsy documented blunt-force injuries and laryngeal chewing gum; the report emphasized uncertainty regarding aspiration versus resuscitation-related displacement.	Primary traumatic death; laryngeal gum considered a possible contributor or iatrogenic artifact rather than a proven reflex death.
Kumar, *et al.*, India.[18]	2023	1	16 y/F	Suicidal near-hanging; rescued alive; severe respiratory failure and death approximately 24 h later.	Marked pulmonary congestion and edema at autopsy; clinical hypoxemia and tachypnea.	Delayed death from post-obstructive/negative-pressure pulmonary edema following hanging.
Hickey, *et al.*, Canada.[19]	2022	1	15 mo/M	Unwitnessed vomiting followed by collapse and persistent asystole despite resuscitation.	Postmortem radiography and CT identified a push pin; autopsy showed complete glottic/tracheal occlusion at the vocal cords.	Choking due to a push pin; vagal inhibition was discussed only as a possible coexisting response.





Study; Country	Year	n	Age/ Sex	Precipitating Event and Clinical Course	Postmortem or Physiological Evidence	Authors' Principal Cause-of-Death Interpretation
Tuchtan, *et al.*, France.[20]	2022	3	14, 32, 42 y/M	Immediate or rapidly fatal collapse after facial impacts during assaults.	Routine autopsy was insufficient; neuropathological examination demonstrated brainstem and/or axonal lesions.	Fatal craniofacial trauma with possible reflex neural conduction and/or occult central nervous system injury.
Vadysinghe, *et al.*, Sri Lanka.[21]	2022	1	27 y/F	Breathlessness and rapid collapse 48 h postpartum, approximately 1 h after a meal while supine.	Large volume of gastric contents in the airway; bronchiolar eosinophilic material with neutrophils; toxicology and biochemical testing were noncontributory.	Airway obstruction from choking with aspiration of gastric contents.
Benevento, *et al.*, Italy.[22]	2021	1	72 y/M	Complex suicide involving a neck ligature attached to a stone followed by entry into the sea.	Autopsy showed findings of both drowning and strangulation; toxicology was essentially negative.	Combined asphyxia from self-strangulation and drowning.
Potenza, *et al.*, Italy.[13]	2021	1	Early 20s/M	Immediate collapse after the neck was grasped during a domestic altercation.	Only superficial nail abrasions externally; focused neck dissection showed a perineural hematoma around the left cervical vagal trunk and no sufficient alternative injury.	Neurally mediated cardiac arrest attributed to direct compression of the left vagus nerve.
Barranco, *et al.*, Italy.[14]	2018	1	38 y/M	Assault with a broken-glass stab wound to the left neck followed by rapid death.	Major cervical vessels and airway were intact; hematoma was present around the vagal trunk and branches.	Deep neck stab wound causing vasovagal inhibition and parasympathetic cardioinhibition.
Aoki, *et al.*, Japan.[23]	2016	1	81 y/F	Takotsubo cardiomyopathy in the setting of severe systemic illness; fatal outcome followed autonomic and myocardial dysfunction.	Autopsy described myocardial injury and pathological changes involving the autonomic nervous system, including the vagus nerve.	Takotsubo cardiomyopathy with autonomic involvement; the terminal contribution of a vagal reflex remained inferential.
Liang, *et al.*, China.[24]	2016	1	42 y/M	Neurofibromatosis type 1; acute neck pain and swelling, progressive dyspnea, loss of consciousness, and death despite resuscitation.	Rupture of the right internal carotid artery caused a massive cervical hematoma and airway compression; lesion was adjacent to the carotid baroreflex region.	Asphyxia from airway obstruction by cervical hematoma; vagal cardiac inhibition proposed as an additional possible mechanism.
Omokanye, *et al.*, Nigeria.[25]	2016	1	4 y/M	Eleven-day impaction of a bent nail in the laryngotracheal lumen; manipulation during removal provoked abrupt bradycardia and cardiac arrest with subsequent mortality.	Direct temporal association between tracheal manipulation and bradycardia/arrest; no medicolegal autopsy was reported.	Fatal perioperative cardiac arrest attributed to vagal reflex stimulation during foreign-body disimpaction, potentiated by hypercarbia/sepsis.
Nouma, *et al.*, Tunisia.[26]	2016	2	7 y/F; 35 y/M	Accidental partial hanging: a child entangled in a curtain cord and an adult trapped between metal bars.	Scene reconstruction and autopsy demonstrated ligature compression and asphyxial findings; one case had injury near the carotid sinus.	Accidental hanging with mechanical asphyxia and vascular compression; reflex cardioinhibition was physiologically plausible but not directly documented.
Watanabe, *et al.*, Japan.[27]	2015	1	Late 40s/M	Collapsed immediately after acupuncture needle manipulation in the right cervical region.	Medicolegal autopsy showed severe hemorrhage around the right vagus nerve; other organs and laboratory studies were not explanatory.	Severe vagally mediated bradycardia and/or arrhythmia following direct vagus nerve injury during acupuncture.





abrupt bradycardia and cardiac arrest during laryngotracheal manipulation.[13,14,25,27] These four reports were classified as probable reflex involvement. The perioperative case nevertheless lacked autopsy confirmation and retained potentially contributory airway, anesthetic, and systemic factors.

Five reports were classified as possible reflex involvement because the precipitating stimulus was credible but the contribution remained unproven: facial impact with occult central nervous system injury, choking with aspiration of gastric contents, combined self-strangulation and drowning, an expanding cervical hematoma, and accidental hanging.[20–22,24,26] Four reports were classified as indeterminate: laryngeal chewing gum potentially displaced during resuscitation, delayed post-obstructive pulmonary edema after hanging, complete airway occlusion by a push pin, and autonomic pathology in Takotsubo cardiomyopathy without documentation of the terminal rhythm.[17–19,23] **Figure 2** illustrates the distinct pathways and the evidence required for forensic attribution. **Table 2** presents the report-level interpretation, including competing mechanisms and confidence in reflex involvement.

DISCUSSION

Principal Findings

The review identifies substantial variation in the evidence supporting suspected vagal-reflex–mediated death. Reports of direct cervical neural injury and immediate collapse differ fundamentally from cases in which airway obstruction, delayed pulmonary edema, trauma, or structural disease already provided a sufficient explanation. Four of the 13 publications offered comparatively persuasive temporal or anatomical support for reflex involvement. The remaining reports supported, at most, an unconfirmed contribution or did not permit meaningful causal assessment. These proportions describe the selected literature and should not be interpreted as estimates of the incidence or diagnostic accuracy of fatal vagal inhibition.

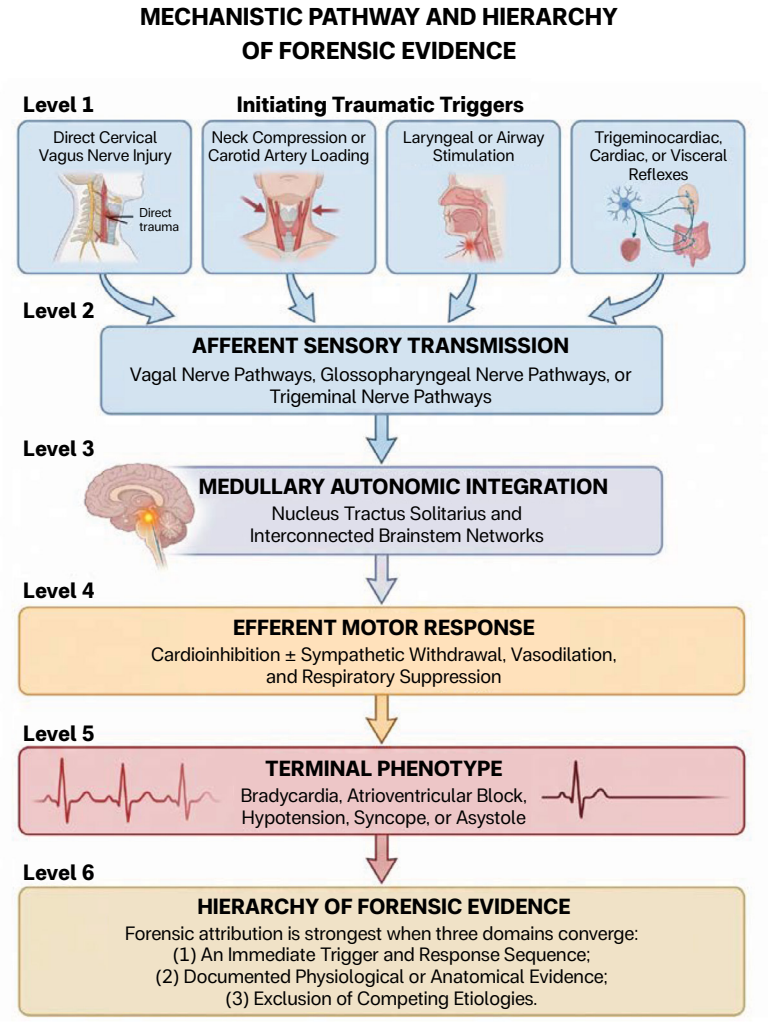


Figure 2. Anatomically distinct pathways that may produce cardiac vagal effects and the evidence required for forensic attribution. Original schematic synthesis based on vagal physiology and the included reports.[1,4,5,7,10,12–14,17–27]

Direct Cervical Vagal Injury

Reports by Potenza, *et al*., Barranco, *et al*., and Watanabe, *et al*., provide the clearest anatomical support.[13,14,27] Each described a precipitating event involving the cervical course of the vagus nerve, immediate or near-immediate collapse, and hemorrhage or perineural injury identified during focused dissection. The absence of a sufficient alternative structural lesion strengthened the proposed association. Focal hemorrhage establishes injury near the nerve; however, without independently prove the terminal rhythm. A defensible opinion should qualify the inference, for example, "death consistent with neurally mediated cardioinhibition following cervical vagal stimulation."

Neck Compression and Carotid Sinus Reflexes

Hanging, strangulation, and expanding cervical hematoma can produce simultaneous airway, vascular, hypoxic, and neural effects. The afferent limb of the carotid sinus reflex is predominantly glossopharyngeal, with vagal efferent output to the heart; labeling every neck-compression death as direct vagal stimulation is therefore anatomically imprecise. In the reports by Nouma, *et al*., and Benevento, *et al*., the circumstances and postmortem findings supported mechanical asphyxial processes, while a reflex contribution remained unconfirmed.[22,26] In the case described by Liang, *et al*., the cervical hematoma was adjacent to the carotid baroreflex region, but airway compression provided a sufficient independently explanation on its own.[24]

Airway Stimulation and Mechanical Asphyxia

Vagal branches supplying the upper airway can mediate abrupt bradycardia during instrumentation or foreign-body manipulation. The report by Omokanye, *et al*., is informative because bradycardia and arrest occurred during disimpaction of the





Table 2. Forensic interpretation of the included reports and confidence in reflex involvement.[13,14,17–27]

Study	Year	Manner or Context	Primary Cause or Initiating Condition	Proposed Reflex Pathway	Evidence Supporting Vagal Involvement	Competing or Independently Sufficient Mechanism	Confidence in Reflex Involvement
Mattu[17]	2024	Accident	Road-traffic trauma; uncertain laryngeal gum	Superior laryngeal/ vagal response to laryngeal stimulus	Anatomically plausible laryngeal stimulus only; no documented bradycardia or focal nerve injury	Multiple blunt-force injuries; gum may have been displaced during resuscitation	Indeterminate
Kumar[18]	2023	Suicide	Near-hanging with delayed post-obstructive pulmonary edema	Carotid sinus or laryngeal reflex sometimes proposed in hanging	No contemporaneous reflex documentation; delayed course is inconsistent with instantaneous vagal arrest	Negative-pressure/ post-obstructive pulmonary edema and hypoxic injury	Indeterminate
Hickey[19]	2022	Accident	Complete glottic obstruction by a push pin	Laryngeal afferent activation with vagal cardioinhibition	Laryngeal foreign body and focal trauma; persistent asystole was nonspecific and did not establish reflex arrest	Complete mechanical airway occlusion fully explains death	Indeterminate
Tuchtan[20]	2022	Assault-related	Craniofacial impact with occult brainstem/axonal injury	Trigeminocardiac/ trigeminovagal reflex and medullary autonomic disruption	Immediate collapse after facial impact; reflex mechanism physiologically plausible	Demonstrated brainstem and axonal injury may independently explain death	Possible
Vadysinghe[21]	2022	Accident	Choking and aspiration of gastric contents	Laryngeal vagal inhibition during airway stimulation	Rapid collapse and extensive airway contents; no other disease identified	Mechanical airway obstruction and aspiration	Possible
Benevento[22]	2021	Suicide	Combined self-strangulation and drowning	Carotid sinus loading and vagal efferent cardioinhibition	Neck compression was anatomically capable of engaging a cardioinhibitory reflex; no direct physiological documentation	Drowning, venous/ arterial compression, and mechanical asphyxia	Possible
Potenza[13]	2021	Assault-related	Direct left vagus compression during neck grasping	Direct cervical vagal stimulation	Immediate collapse; perineural hematoma around left vagus; no sufficient alternative lesion	No independently sufficient structural cause identified	Probable
Barranco[14]	2018	Homicide	Deep left cervical stab wound	Direct vagal trunk/ branch stimulation	Hematoma adjacent to vagus; major vessels and airway intact; rapid death	Limited competing structural injury	Probable





Study	Year	Manner or Context	Primary Cause or Initiating Condition	Proposed Reflex Pathway	Evidence Supporting Vagal Involvement	Competing or Independently Sufficient Mechanism	Confidence in Reflex Involvement
Aoki[23]	2016	Natural	Takotsubo cardiomyopathy with severe systemic illness	Disease-associated autonomic dysregulation; specific reflex pathway unproven	Autonomic pathology and myocardial injury	Takotsubo cardiomyopathy and systemic illness; terminal rhythm not established	Indeterminate
Liang[24]	2016	Natural	Ruptured internal carotid artery in NF1 with massive cervical hematoma	Carotid baroreflex activation and secondary vagal cardioinhibition	Hematoma adjacent to carotid sinus; sudden deterioration	Airway obstruction by hematoma was the certified cause	Possible
Omokanye[25]	2016	Therapeutic complication	Manipulation of an impacted laryngotracheal nail	Laryngocardiac vagal reflex	Abrupt bradycardia and arrest during foreign-body disimpaction; immediate temporal association	Hypercarbia, sepsis, airway compromise, and anesthetic factors were contributory	Probable
Nouma[26]	2016	Accident	Partial hanging in two unusual circumstances	Carotid sinus/ glossopharyngeal afferent input with vagal efferent response	Neck compression and one lesion near carotid sinus	Mechanical asphyxia and cervical vascular compression	Possible
Watanabe[27]	2015	Therapeutic complication	Acupuncture-related right cervical vagus injury	Direct vagal stimulation	Immediate collapse; severe hemorrhage around right vagus; negative alternative investigation	No sufficient competing cause reported	Probable

laryngotracheal foreign body.[25] The report by Hickey, *et al*., by contrast, demonstrated complete airway occlusion at the vocal cords by a push pin, establishing a sufficient mechanical explanation.[19] Postpartum choking with gastric-content aspiration presents a similar interpretive problem: rapid collapse is compatible with a laryngocardiac response, but airway obstruction and aspiration prevent designation of a reflex as the sole mechanism.[21]

A 2025 series of nine medicolegal café-coronary autopsies provides additional context beyond the prespecified publication window. The deaths were attributed to complete upper-airway obstruction by food; vagal inhibition remained a theoretical contributor.[28] This series was not included in the systematic synthesis or its numerical totals. Sudden collapse alone cannot establish a primary reflex mechanism when mechanical asphyxia is demonstrable.

Craniofacial and Cardiac Reflex Pathways

Facial stimulation may activate trigeminal afferents and vagal cardiac efferents. The series by Tuchtan, *et al*., nevertheless identified brainstem or axonal lesions that were not evident during routine gross examination.[20] Microscopic central nervous system injury is therefore an important competing explanation after apparently limited craniofacial trauma. Targeted neuropathological examination, including the brainstem and craniocervical region when indicated, strengthens the investigation before accepting a reflex mechanism.

The Bezold–Jarisch reflex establishes biological plausibility for profound cardioinhibition. Experimental activation of NPY2R-expressing cardiac vagal sensory neurons produces bradycardia, hypotension, respiratory suppression, cerebral hypoperfusion, and syncope.[11,12] Extrapolation from animal physiology to an individual human death nevertheless requires caution. In the report by Aoki, *et al*., autonomic and myocardial abnormalities supported dysregulation but did not establish that a reflex, rather than cardiomyopathy-associated arrhythmia or another complication of systemic illness, caused the terminal event.[23]

Contemporaneous Physiological Evidence

Contemporaneous observations can support a transient reflex more directly than nonspecific postmortem findings. A 2026 report described abrupt cardiac arrest during renal calyceal manipulation in percutaneous nephrolithotomy, followed by recovery after resuscitation.[29] This nonfatal event was outside the eligible population and publication window and was used only as contextual evidence. A closely observed trigger, recorded rhythm disturbance, assessment of alternative causes, and response to intervention support a reflex





hypothesis more strongly than congestion, pulmonary edema, or petechiae alone. The temporal association must still be interpreted alongside anesthetic, hypoxic, embolic, and other competing explanations.

Forensic Diagnostic Framework

Assessment of suspected fatal reflexes should integrate scene reconstruction, temporal sequence, anatomical correlation, physiological observations, and investigation of alternative causes. **Table 3** presents an original practical framework derived from the physiological literature and included reports.[1,4,10,12–14,17–27] Its purpose is to make the reasoning explicit and proportionate to the evidence, particularly where an unsupported attribution could obscure trauma, asphyxia, intoxication, cardiac disease, or a treatment complication.

Role of Postmortem Imaging

Postmortem computed tomography (PMCT) can strengthen the assessment of structural alternatives by documenting injuries and foreign bodies before dissection. In the push-pin case, postmortem imaging helped localize the obstructing object, which was subsequently confirmed at autopsy.[19] Comparative studies support integrating PMCT with conventional examination, toxicology, and histology rather than treating a negative scan as proof of a reflex death.[30,31] PMCT can guide targeted dissection, but it cannot demonstrate a transient vagal response or exclude intoxication, microscopic neural lesions, or primary electrical cardiac disease. Its value in this setting is therefore complementary and exclusionary, with the extent of exclusion determined by the suspected alternative and the investigations actually performed.

Cause, Mechanism, and Manner of Death

The cause of death should identify the initiating injury or disease, such as a cervical stab wound, hanging, foreign-body aspiration, or internal carotid artery rupture. The mechanism describes the resulting physiological derangement, including mechanical asphyxia, cerebral hypoxia, pulmonary edema, hemorrhagic shock, or neurally mediated cardioinhibition. The manner of death is a separate medicolegal classification, commonly natural, accident, suicide, homicide, or undetermined, subject to jurisdictional practice. Therapeutic complication describes a circumstance and does not independently determine the legal manner of death. Where a source report does not provide formal certification, the synthesis should retain descriptive circumstances rather than infer an unsupported legal classification.

Methodological Quality and Limitations

The evidence is vulnerable to publication and selection bias because unusual, dramatic events are preferentially reported. Reporting varied accross for scene reconstruction, cardiac rhythm monitoring, toxicology, examination of the cardiac conduction system, molecular autopsy, and targeted neuropathology. Several publications proposed vagal involvement without establishing it as the principal fatal mechanism. Language restrictions and the 2015–2024 publication window further limit coverage. The post hoc involvement categories are unvalidated, do not provide calibrated probabilities, and cannot establish diagnostic accuracy. Case reports provide no denominator from which to estimate the population frequency of fatal reflexes.

One eligible perioperative report did not include a medicolegal autopsy, limiting direct comparison with the postmortem literature. The title therefore encompasses both autopsy and clinical case reports. Contextual publications outside the eligibility

Table 3. Proposed framework for evaluating suspected fatal vagal or related cardioinhibitory reflexes. Original synthesis of physiological principles and case-based evidence .[1,4,10,12–14,17–27]

Domain	Questions to Address	Findings that Strengthen Attribution	Findings that Weaken or Limit Attribution
Trigger and temporality	Was there a physiologically credible stimulus? Did collapse occur immediately or within seconds?	Witnessed trigger-response sequence; monitored bradycardia, AV block, or asystole	Long survival interval; delayed deterioration; uncertain chronology
Anatomical correlation	Was the relevant neural pathway directly injured or stimulated?	Focal injury in the relevant pathway supports anatomical exposure; documented airway manipulation supports a credible trigger	Nonspecific lesions or uncertain anatomical correlation; absence of a lesion does not exclude a transient reflex
Competing causes	Is another disease or injury independently sufficient to cause death?	Absence of lethal trauma, complete obstruction, major hemorrhage, toxic exposure, or structural cardiac disease	An independently sufficient disease or injury limits attribution of death solely to a reflex
Ancillary investigation	Were cardiac histology, toxicology, imaging, neuropathology, and device/scene data obtained?	Targeted dissection, cardiac and brainstem histology, toxicology, and imaging where relevant; negative findings interpreted within test limitations	Incomplete autopsy; no toxicology; no focused neck/brainstem examination; poor scene reconstruction
Opinion formulation	Does the wording distinguish cause, mechanism, and manner of death?	Qualified wording: "consistent with" or "probable cardioinhibitory reflex contributing to…"	Reflex listed as a stand-alone cause without the initiating injury/disease or without evidentiary qualification





window were discussed separately and did not alter the included-study totals. The principal interpretive contribution is the explicit separation of observed injury or physiology, biological plausibility, and competing mechanisms, while preserving uncertainty where the terminal event cannot be established.

CONCLUSION

Fatal vagal and related cardioinhibitory reflexes are physiologically credible but remain difficult to establish after death. The strongest support combines an immediate, anatomically appropriate trigger with contemporaneous rhythm documentation or focal neural injury and a comprehensive assessment of alternative causes. An independently sufficient explanation, including complete airway obstruction, major trauma, vascular rupture, delayed pulmonary edema, or structural cardiac disease, should not be displaced by an unproven reflex hypothesis. Forensic conclusions should state the degree of confidence in reflex involvement, distinguish a possible contribution from an exclusive fatal mechanism, and separately identify the initiating cause, terminal mechanism, and legal manner of death.

DECLARATIONS

Consent for Publication

Not applicable. This systematic review synthesizes previously published reports and contains no newly collected patient data or identifiable patient images.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

Mohammad Tegar Indrayana: Conceptualization, Methodology, Writing – Original Draft.
Bima Diokta Alparisi: Conceptualization, Methodology, Supervision, Writing – Review & Editing.
Regina Trisya Arenja: Investigation, Data Curation, Formal Analysis.
Najmi Khairussyifa: Investigation, Data Curation, Validation.
Patricia Dean Ully Marbun: Investigation, Data Curation, Writing – Original Draft.
Shalsabila Ghaasani Amanda: Investigation, Data Curation, Visualization.

Conflict of Interest

The authors declare that they have no financial or non-financial conflicts of interest relevant to this work.

Declaration on the Use of Artificial Intelligence

During the revision of this manuscript, the authors used AI to support English-language editing, editorial restructuring, consistency checks, and the preparation of revised text, figure captions, and schematic diagrams. The AI tool was not used to generate patient data, conduct study selection, or extract data. The authors retain full responsibility for the accuracy of all statements, references, interpretations, and figures and have thoroughly reviewed and approved the final manuscript before submission.

REFERENCES

1. Mehra R, Tjurmina OA, Ajijola OA, Arora R, Bolser DC, Chapleau MW, et al. Research opportunities in autonomic neural mechanisms of cardiopulmonary regulation: a report from the national heart, lung, and blood institute and the national institutes of health office of the director workshop. JACC Basic Transl Sci. 2022;7(3):265–93. doi: 10.1016/j.jacbts.2021.11.003.
2. Japundzic-Zigon N, Sarenac O, Lozic M, Vasic M, Tasic T, Bajic D, et al. Sudden death: neurogenic causes, prediction and prevention. Eur J Prev Cardiol. 2018;25(1):29–39. doi: 10.1177/2047487317736827.
3. Yekben MG, Erbas O. Sudden death: causes, epidemiology, and associations in cardiology. Demiroglu Sci Univ Florence Nightingale J Transplant. 2023;8(1-2):41–7. doi: 10.5606/dsufnjt.2023.17.
4. Capilupi MJ, Kerath SM, Becker LB. Vagus nerve stimulation and the cardiovascular system. Cold Spring Harb Perspect Med. 2020;10(2):a034173. doi: 10.1101/cshperspect.a034173.
5. Prescott SL, Liberles SD. Internal senses of the vagus nerve. Neuron. 2022;110(4):579–99. doi: 10.1016/j.neuron.2021.12.020.
6. Baquiran M, Bordoni B. Anatomy, head and neck: anterior vagus nerve [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK547696/. PMID: 31613476.
7. Camara R, Griessenauer CJ. Anatomy of the vagus nerve. Nerves and Nerve Injuries. 2015;1:385–97. doi: 10.1016/B978-0-12-410390-0.00028-7.
8. Berthoud HR, Neuhuber WL. Functional and chemical anatomy of the afferent vagal system. Auton Neurosci. 2000;85(1–3):1–17. doi: 10.1016/S1566-0702(00)00215-0.
9. Fitchett A, Mastitskaya S, Aristovich K. Selective neuromodulation of the vagus nerve. Front Neurosci. 2021;15:685872. doi: 10.3389/fnins.2021.685872.
10. Yuan H, Silberstein SD. Vagus nerve and vagus nerve stimulation, a comprehensive review: part I. Headache. 2016;56(1):71–8. doi: 10.1111/head.12647.





11. Habecker BA. Identifying vagal sensory neurons driving the Bezold–Jarisch reflex. Trends Neurosci. 2024;47(2):90–1. doi: 10.1016/j.tins.2023.12.005.

12. Lovelace JW, Ma J, Yadav S, Chhabria K, Shen H, Pang Z, et al. Vagal sensory neurons mediate the Bezold–Jarisch reflex and induce syncope. Nature. 2023;623(7986):387–96. doi: 10.1038/s41586-023-06680-7.

13. Potenza S, Tavone AM, Scipione C, Arcudi G, Marella GL, Tor R. Death by stimulation of the left vagus nerve in a case of neck grasping in a healthy young man. Med Leg Update. 2021;21(2):1122–5. doi: 10.37506/mlu.v21i2.2836.

14. Barranco R, Molinelli A, Ventura F. An unusual murder: vasovagal death from atypical stabbing to the neck. Am J Forensic Med Pathol. 2018;39(4):367–9. doi: 10.1097/PAF.0000000000000429.

15. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021;372:n71. doi: 10.1136/bmj.n71.

16. JBI. Checklist for case reports [Internet]. 2020 [cited 2026 Oct 20]. Available from: https://jbi.global/sites/default/files/2020-08/Checklist_for_Case_Reports.pdf.

17. Mattu PS, Orde MM. Chewing gum in the larynx: foreign body aspiration or iatrogenic artifact? challenges in determining the cause of death in a road traffic accident victim with resuscitation intervention. Cureus. 2024;16(8):e67085. doi: 10.7759/cureus.67085.

18. Kumar S, Das PK, Jha BN, Chaudhary DK, Sinha S. Post-obstructive pulmonary oedema in delayed death by hanging: a case report. Cureus. 2023;15(3):e36420. doi: 10.7759/cureus.36420.

19. Hickey TBM, Mathews RZ, Pickup MJ. Pediatric push pin aspiration: clinical, radiologic, and pathologic correlation — case report. Egypt J Forensic Sci. 2022;12(1):36. doi: 10.1186/s41935-022-00295-1.

20. Tuchtan L, Delteil C, Godio-Raboutet Y, Kolopp M, Leonetti G, Thollon L, et al. Sudden death after facial impacts: is the brainstem involved? Morphologie. 2022;106(354):217–23. doi: 10.1016/j.morpho.2021.07.004.

21. Vadysinghe AN, Nayakarathne TB, Banda I, Perera RAEI. Choking together with aspiration of gastric contents: rare form of maternal death. Egypt J Forensic Sci. 2022;12:58. doi: 10.1186/s41935-022-00318-x.

22. Benevento M, Mandarelli G, Ferorelli D, Trotta S, Bottari G, Caterino C, et al. Complex suicide by drowning and self-strangulation: an atypical "holy" way to die. Forensic Sci Int Rep. 2021;3:100190. doi: 10.1016/j.fsir.2021.100190.

23. Aoki Y, Kodera S, Watanabe T, Miyauchi Y, Kanda J, Ooe K. Autopsy findings in takotsubo cardiomyopathy with special reference to the autonomic nervous system. Int J Cardiol. 2016;203:236–7. doi: 10.1016/j.ijcard.2015.10.121.

24. Liang Y, Tong F, Zhang L, Li W, Zhou Y. Sudden death due to rupture of the right internal carotid artery in neurofibromatosis type 1: a case report. Leg Med (Tokyo). 2016;21:33–7. doi: 10.1016/j.legalmed.2016.05.004.

25. Omokanye H, Alabi S, Ige OA, Afolabi O, Omotoso W, Iyanda NO, et al. Vagal reflex stimulation complicating retrieval of an unusual foreign body from the laryngotracheal lumen: case report. Egypt J Anaesth. 2019;32(2):237–40. doi: 10.1016/j.egja.2015.11.003.

26. Nouma Y, Ammar WB, Bardaa S, Hammami Z, Maatoug S. Accidental hanging among children and adults: a report of two cases and review of the literature. Egypt J Forensic Sci. 2016;6(3):310–4. doi: 10.1016/j.ejfs.2015.07.001.

27. Watanabe M, Unuma K, Fujii Y, Noritake K, Uemura K. An autopsy case of vagus nerve stimulation following acupuncture. Leg Med (Tokyo). 2015;17(2):120–2. doi: 10.1016/j.legalmed.2014.11.001.

28. Sonwani NS, Thakur K, Ateriya N, Kumar A, Kohli A. Cafe coronary deaths: medicolegal observations from a tertiary care center. Cureus. 2025;17(11):e98188. doi: 10.7759/cureus.98188.

29. Wang B, Wang X, Wang C, Chen Z, Han Q, Feng Y, et al. Vagus nerve-induced cardiac arrest during percutaneous nephrolithotomy: a clinical challenge. BMC Anesthesiol. 2026;26(1):125. doi: 10.1186/s12871-025-03592-z.

30. Lathrop SL, Wiest PW, Andrews SW, Elifritz J, Price JP, Mlady GW, et al. Can computed tomography replace or supplement autopsy? J Forensic Sci. 2023;68(2):524–35. doi: 10.1111/1556-4029.15217.

31. Hueck U, Muggenthaler H, Hubig M, Heinrich A, Guttler F, Wagner R, et al. Forensic postmortem computed tomography in suspected unnatural adult deaths. Eur J Radiol. 2020;132:109297. doi: 10.1016/j.ejrad.2020.109297.