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Original Research Article

Role of Catecholamine Surge and Physiological Stress Response in Sudden Cardiac Death During Assault: A Narrative Review and Forensic Synthesis.

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Abstract: ***Background:*** Sudden cardiac death during or shortly after assault presents a difficult medico-legal problem. In some cases, death is explained by direct trauma, commotio cordis, asphyxia, intoxication, or pre-existing heart disease. In others, the physical and psychological stress of assault may precipitate a fatal cardiac event through acute sympathetic activation, catecholamine surge, myocardial oxygen demand–supply mismatch, coronary vasospasm, stress cardiomyopathy, or malignant arrhythmia. ***Objective:*** To review the role of catecholamine surge and physiological stress response in sudden cardiac death during assault and to synthesize current evidence into a practical forensic framework. ***Methodology:*** A narrative literature review was conducted using studies, reviews, forensic case reports, registry analyses, and medico-legal discussions published up to 2026. Search themes included "sudden cardiac death," "assault," "catecholamine surge," "physiological stress," "Takotsubo syndrome," "commotio cordis," "post-mortem catecholamines," "restraint death," and "forensic pathology." Evidence was grouped into mechanistic pathways, assault-related triggers, vulnerable cardiac substrates, forensic markers, and interpretive cautions. ***Results:*** The evidence supports a biologically plausible link between assault-related acute stress and sudden cardiac death, especially in individuals with coronary atherosclerosis, myocardial fibrosis, cardiomyopathy, channelopathy, stimulant exposure, or hypertensive heart disease. Catecholamine excess may precipitate ventricular arrhythmia, coronary vasospasm, plaque rupture, stress cardiomyopathy, and myocardial contraction-band necrosis. However, catecholamine surge is rarely sufficient as a stand-alone cause of death and must be interpreted alongside injuries, toxicology, scene evidence, resuscitation records, histology, and molecular testing. Seven synthesis tables are presented to organize mechanisms, evidence, differential diagnosis, and forensic interpretation. ***Conclusion:*** Assault can act as a trigger for sudden cardiac death through adrenergic and physiological stress pathways. The strongest conclusion is usually not that "stress alone caused death," but that assault-related stress contributed to a fatal cardiac event in a vulnerable individual. A defensible forensic opinion requires exclusion of direct traumatic, asphyxial, toxicological, and natural causes, and careful assessment of the temporal relationship between assault and collapse.

Keywords: sudden cardiac death; assault; catecholamine surge; physiological stress; ventricular arrhythmia; Takotsubo syndrome; commotio cordis; forensic pathology; restraint death.

INTRODUCTION

Sudden cardiac death during assault is a complex event at the intersection of cardiology, emergency medicine, forensic pathology, and law. The death may occur during a physical fight, robbery, restraint, custodial struggle, domestic violence, mob assault, or immediately after a threatening event. In many such cases, the external injuries appear insufficient to explain death directly, yet the temporal association between assault and collapse is compelling. This creates a central medico-legal question: did the assault cause death, trigger death, accelerate death, or merely coincide with an underlying natural disease?

The physiological response to assault involves rapid activation of the sympathetic-adrenal-medullary system and the hypothalamic-pituitary-adrenal axis. Fear, pain, anger, exertion, hypoxia, blood loss, and struggle can produce abrupt increases in epinephrine, norepinephrine, cortisol, heart rate, blood pressure, myocardial oxygen demand, platelet activation, and vascular tone. In a healthy individual, these responses are usually adaptive. In a vulnerable heart, however, the same response may become arrhythmogenic and ischemic.

Catecholamine-mediated sudden cardiac death has several possible pathways. These include ventricular fibrillation in structurally abnormal hearts, catecholaminergic polymorphic ventricular tachycardia in genetically susceptible individuals, coronary vasospasm, acute plaque destabilization, demand ischemia, Takotsubo syndrome, and direct catecholamine cardiotoxicity. In assault, these mechanisms may act alone or in combination with trauma, hypoxia, intoxication, restraint, hyperthermia, acidosis, or electrolyte disturbance.

Forensic interpretation is especially challenging because catecholamine surge leaves no single diagnostic autopsy sign. Post-mortem catecholamine measurements are influenced by agonal duration, sampling site, decomposition, resuscitation, and cause of death. Histological findings such as contraction-band necrosis, interstitial hemorrhage, or early ischemic change may support adrenergic injury but are not pathognomonic. Therefore, the role of assault-related stress must be inferred from a convergence of evidence.

This article reviews the role of catecholamine surge and physiological stress response in sudden cardiac death during assault and proposes a structured approach for medico-legal analysis.

MATERIALS AND METHODS

Study Design

This article is a narrative review and forensic synthesis. It does not present new human autopsy cases. Instead, it integrates evidence from cardiology, forensic pathology, emergency medicine, and stress physiology to clarify how assault-related stress may precipitate sudden cardiac death.

Literature Search Strategy

The literature was searched using combinations of the following terms: sudden cardiac death, assault, catecholamine surge, physiological stress response, emotional stress, physical stress, ventricular arrhythmia, Takotsubo syndrome, stress cardiomyopathy, coronary vasospasm, commotio cordis, restraint death, excited delirium, post-mortem catecholamines, contraction-band necrosis, forensic pathology, and molecular autopsy.

Inclusion Criteria

Studies and articles were included if they addressed at least one of the following:

1) Mechanisms of catecholamine-mediated cardiac injury.
2) Sudden cardiac death triggered by emotional or physical stress.
3) Assault, robbery, restraint, or struggle-associated sudden death.
4) Takotsubo syndrome and stress cardiomyopathy.
5) Commotio cordis and blunt chest impact.
6) Post-mortem biochemical or histological markers of stress.
7) Forensic evaluation of sudden unexplained or autopsy-negative cardiac death.

Exclusion Criteria

Articles were excluded if they were unrelated to cardiac death, focused only on non-cardiac traumatic death, lacked relevance to acute stress physiology, or did not contribute to forensic interpretation.

Data Extraction

Information was extracted under the following headings: type of stressor, cardiac mechanism, vulnerable substrate, pathological findings, diagnostic limitations, and medico-legal relevance.

Methodological Limitation

Because assault-related sudden cardiac death is heterogeneous and often reported through case reports or forensic series, the available evidence does not allow a pooled quantitative risk estimate. The results are therefore presented as a structured qualitative synthesis.

RESULTS

Table 1. Literature Search and Evidence Mapping

Evidence domain	Search focus	Main type of evidence identified	Forensic relevance
Stress physiology	Catecholamine surge, sympathetic activation, cortisol	Experimental studies, reviews	Explains biological plausibility
Sudden cardiac death	Ventricular arrhythmia, ischemia, SCD triggers	Cardiology reviews, registry data	Links acute stress to fatal arrhythmia
Assault and robbery	Fear, struggle, physical violence	Forensic case reports	Supports temporal triggering role
Takotsubo syndrome	Emotional/physical stress cardiomyopathy	Registry studies, reviews	Demonstrates stress-induced myocardial dysfunction
Commotio cordis	Blunt precordial impact	Registry studies, reviews	Important traumatic differential diagnosis
Post-mortem biomarkers	Catecholamines, troponin, stress markers	Forensic biochemical studies	Supportive but non-specific evidence
Restraint deaths	Struggle, prone restraint, hypoxia, acidosis	Forensic reviews and debates	Requires caution against over-attributing to stress alone

Inference from Table 1:
The evidence base is strongest for the general relationship between acute stress and cardiac events, moderate for stress cardiomyopathy and commotio cordis, and weaker for proving catecholamine surge as the sole cause of death in an individual assault case. Forensic conclusions must therefore be probabilistic and integrative rather than based on a single marker.

Table 2. Assault-Related Triggers and Immediate Physiological Responses

Assault-related factor	Physiological response	Possible cardiac consequence
Fear or threat perception	Sympathetic discharge, epinephrine release	Tachycardia, QT changes, arrhythmia
Pain from injury	Catecholamine and cortisol rise	Increased myocardial oxygen demand
Physical struggle	Exertional tachycardia, lactic acidosis	Demand ischemia, arrhythmia
Blunt chest impact	Mechanical-electrical disturbance	Commotio cordis, ventricular fibrillation
Neck compression or restraint	Hypoxia, vagal effects, acidosis	Bradyarrhythmia or ventricular arrhythmia
Blood loss	Hypotension, compensatory catecholamine rise	Ischemia in diseased myocardium
Stimulant intoxication	Excess adrenergic tone	Coronary spasm, hyperthermia, arrhythmia
Anger or panic	Blood pressure surge	Plaque rupture, coronary thrombosis

Inference from Table 2:
Assault rarely produces a single isolated physiological stressor. Most cases involve overlapping fear, pain, exertion, injury, and sometimes hypoxia or intoxication. This multi-hit pattern increases arrhythmogenic potential, particularly in individuals with latent or known cardiovascular disease.

Table 3. Mechanisms Linking Catecholamine Surge to Sudden Cardiac Death

Mechanism	Pathophysiological sequence	Typical substrate	Expected finding
Ventricular fibrillation	Adrenergic stimulation → electrical instability → VF	Ischemic heart disease, fibrosis, channelopathy	Often autopsy-negative or subtle
Demand ischemia	Tachycardia/BP rise → oxygen demand exceeds supply	Coronary stenosis, LV hypertrophy	Early ischemia may be minimal
Coronary vasospasm	Alpha-adrenergic vasoconstriction → transient ischemia	Normal or diseased coronaries	May show no fixed occlusion
Plaque rupture	BP surge and shear stress →	Atherosclerosis	Coronary thrombosis

	plaque disruption		may be present
Takotsubo syndrome	Catecholamine toxicity → transient LV dysfunction	Older age, stress susceptibility	Contraction-band necrosis, LV ballooning if survived
CPVT/channelopathy	Catecholamine trigger → polymorphic VT/VF	Genetic arrhythmia syndrome	Structurally normal heart
Direct cardiotoxicity	Calcium overload and oxidative stress	Extreme catecholamine states	Contraction-band necrosis

Inference from Table 3:
Catecholamine surge is best understood as a trigger that converts vulnerability into fatal instability. The same assault may be survived by one person but fatal in another because the decisive factor is often the underlying substrate.

Table 4. Cardiac Substrates That Increase Vulnerability During Assault

Vulnerable substrate	Why assault-related stress is dangerous	Forensic clue
Coronary atherosclerosis	Reduced coronary reserve; risk of plaque rupture	Coronary narrowing, thrombosis
Hypertensive heart disease	Increased oxygen demand and arrhythmia risk	LV hypertrophy, arteriolar nephrosclerosis
Myocardial fibrosis	Re-entry substrate for ventricular arrhythmia	Patchy fibrosis on histology
Cardiomyopathy	Electrical and mechanical instability	Dilated, hypertrophic, or arrhythmogenic changes
CPVT/long QT/Brugada syndrome	Catecholamine-sensitive arrhythmia	Often structurally normal heart
Prior myocardial infarction	Scar-related ventricular arrhythmia	Old infarct scar
Stimulant use	Amplifies adrenergic state	Toxicology positive for cocaine/amphetamines
Pheochromocytoma	Endogenous catecholamine excess	Adrenal tumor, high metanephrines

Inference from Table 4:
The presence of underlying disease does not exclude assault as a causal contributor. In forensic reasoning, assault may be the precipitating event in a person whose heart was already vulnerable.

Table 5. Differential Diagnosis in Sudden Death During Assault

Possible cause/mechanism	Supporting features	Features against diagnosis
Direct traumatic death	Severe head, chest, abdominal, or vascular injury	Minor injuries only
Commotio cordis	Immediate collapse after precordial blow; minimal structural injury	No chest impact or delayed collapse
Stress-triggered arrhythmia	Collapse during fear/struggle; diseased or genetically vulnerable heart	Long symptom-free interval
Acute myocardial infarction	Coronary thrombosis, plaque rupture, ischemic symptoms	No coronary lesion or thrombus
Coronary vasospasm	Sudden collapse after stress/stimulants; minimal fixed disease	Alternative clear cause
Asphyxial death	Neck compression, restraint, petechiae, hypoxia context	No restraint/compression evidence
Toxicological death	Stimulants, alcohol, drugs	Negative toxicology
Natural sudden cardiac death coincidental to assault	Severe cardiac disease; weak temporal link	Collapse tightly linked to assault

Inference from Table 5:
The main forensic task is not merely to identify heart disease but to decide whether the assault materially contributed to death. A strong temporal relationship, credible stressor, and absence of a better explanation support a contributory role.

Table 6. Forensic Markers Relevant to Catecholamine-Mediated Death

Investigation	Possible supportive finding	Limitation
Scene investigation	Collapse during assault, chase, restraint, or robbery	Witness accounts may conflict
External examination	Minor injuries inconsistent with direct fatal trauma	Minor injuries do not prove stress death
Internal autopsy	Coronary disease, LV hypertrophy, cardiomyopathy	Disease may be incidental or causal
Histology	Contraction-band necrosis, early ischemia, fibrosis	Non-specific; affected by resuscitation
Toxicology	Cocaine/amphetamine or other sympathomimetic	Drug level may not prove cause alone
Post-mortem biochemistry	Raised catecholamines/metanephrines/troponin	Strongly affected by agony and sampling
Molecular autopsy	Channelopathy variants	Variant interpretation may be uncertain
Resuscitation record	Shockable rhythm supports arrhythmia	Rhythm often undocumented

Inference from Table 6:
No single autopsy, biochemical, or histological finding proves catecholamine surge as the cause of death. The diagnosis is strongest when multiple independent lines of evidence converge.

Table 7. Practical Forensic Interpretation Framework

Level of interpretation	Criteria	Suggested wording
Strong support for assault-triggered cardiac death	Collapse during assault; significant stressor; vulnerable heart; no better cause	"Assault-related stress precipitated sudden cardiac death."
Moderate support	Temporal link present; cardiac vulnerability present; competing causes not fully excluded	"Assault-related stress likely contributed to death."
Limited support	Severe natural disease; weak assault severity; uncertain timing	"Death may have been natural, with assault as a possible trigger."
Low support	Clear alternative fatal injury, intoxication, or natural event	"Catecholamine-mediated stress death is not supported."
Special case: commotio cordis	Immediate collapse after precordial blow; no structural injury	"Blunt chest impact precipitated fatal arrhythmia."
Special case: restraint/hypoxia	Aggressive restraint, prone position, neck compression, hypoxia	"Restraint-related asphyxial and physiological mechanisms must be considered."

Inference from Table 7:
Forensic wording should reflect the strength of evidence. Overstating catecholamine surge as a proven mechanism can be misleading; under-recognizing assault as a trigger can also be medically and legally inaccurate.

DISCUSSION

Acute assault produces both psychological and physical stress. Lambiase et al.1 emphasized that emotional stress can influence cardiac electrophysiology through central autonomic networks, sympathetic outflow, repolarization changes, and arrhythmia vulnerability. This is directly relevant to assault because fear, anger, panic, pain, and exertion occur simultaneously. In such circumstances, the heart is exposed to abrupt adrenergic stimulation rather than a gradual physiological challenge. Vaccarino and Bremner2 described stress-related cardiovascular risk as involving haemodynamic, vascular, inflammatory, and neuroendocrine pathways. These pathways explain why assault can trigger sudden cardiac death even when external injuries are not independently fatal. Tachycardia and hypertension increase myocardial oxygen demand, while vasoconstriction and endothelial dysfunction may reduce supply. In a person with coronary stenosis, this

mismatch may be enough to trigger ischemia and ventricular arrhythmia.

Takotsubo syndrome provides one of the strongest clinical models of catecholamine-mediated myocardial injury. Templin et al.3 demonstrated that Takotsubo syndrome is commonly associated with emotional or physical triggers and can mimic acute coronary syndrome. Although most patients survive, the syndrome proves that intense stress can produce acute myocardial dysfunction without obstructive coronary thrombosis. More recent InterTAK-related analyses by Kato et al.4 and registry-based observations have shown that physical triggers are increasingly recognized and may carry worse outcomes than classic emotional triggers. This supports the idea that assault, which combines emotional shock and physical struggle, can be a potent trigger. The mechanism is not limited to Takotsubo syndrome. Du et al.5 reviewed catecholamine-induced cardiotoxicity and described pathways including calcium overload, oxidative stress, inflammation, mitochondrial dysfunction, and myocardial fibrosis. In fatal assault-associated collapse, these processes may be too rapid to produce grossly visible lesions, but they can destabilize cardiac electrophysiology. Contraction-band necrosis, when present, may support catecholamine-mediated injury, although it is not specific.

Forensic case literature also supports stress-triggered cardiac death. Bottari et al.6 discussed sudden cardiac death after robbery and raised the medico-legal question of whether such death should be considered homicide or natural death. The key issue is causation. A robbery or assault may not produce lethal anatomical injury, but the fear and stress may trigger a fatal cardiac event in a vulnerable person. This distinction is essential because the medical cause of death and legal responsibility may not be identical but are closely related. Acute emotional stress may also trigger coronary spasm. The forensic report by Sampson and colleagues7 on emotional stress-related death described cases where myocardial infarction or fatal arrhythmia was attributed to stress-associated coronary spasm. This is important in assault cases where coronary arteries do not show complete occlusion. Absence of thrombosis does not exclude ischemic arrhythmia because transient spasm may leave minimal anatomical evidence.

Commotio cordis must be considered separately. Maron et al.8 described commotio cordis as sudden ventricular fibrillation caused by a blunt, non-penetrating precordial impact during a vulnerable phase of repolarization. Link9 further explained that timing, impact location, and mechanical-electrical coupling are critical. In assault, a punch, kick, elbow, or object strike to the chest may cause immediate collapse even in a structurally normal heart. Unlike catecholamine-mediated stress death, commotio cordis depends on mechanical impact and typically produces near-instantaneous collapse.

Survival data from commotio cordis registries also highlight the importance of rapid rhythm disturbance. Maron et al.10 reported improved survival with early cardiopulmonary resuscitation and defibrillation. For forensic interpretation, a documented shockable rhythm shortly after chest impact supports arrhythmic death. However, where no rhythm strip exists, diagnosis depends heavily on witness evidence and exclusion of structural injury. Genetic arrhythmia syndromes are another important substrate. Roston et al.11 showed that catecholaminergic polymorphic ventricular tachycardia is provoked by exercise or emotional stress and may cause sudden death in structurally normal hearts. In assault-related deaths with negative autopsy, especially in young individuals, molecular autopsy may reveal channelopathy-related susceptibility. However, variant interpretation requires caution because not every genetic variant is pathogenic.

Post-mortem catecholamine testing has potential value but major limitations. Lee and Cheong12 found that post-mortem catecholamine levels may correlate with agony time and cause-of-death categories, but interpretation is affected by multiple peri-mortem and post-mortem factors. Therefore, raised catecholamines after assault cannot be treated as proof of catecholamine-mediated death. They may reflect the dying process, resuscitation, hypoxia, trauma, or decomposition.

Post-mortem biomarkers in sudden cardiac death have been increasingly discussed as adjuncts rather than replacements for autopsy. Recent forensic biomarker reviews13 emphasize that conventional autopsy and histology remain central, while biochemical and molecular tests can reduce the proportion of unexplained cases. In assault-associated sudden death, troponin, catecholamines, cortisol, inflammatory markers, vitreous biochemistry, and molecular testing may help, but none is independently diagnostic.

Restraint-related deaths require particular caution. Strömmer et al.14 argued that in agitated restrained individuals, restraint-related asphyxia must be considered and that “excited delirium” should not be used uncritically as a stand-alone explanation. This is relevant because catecholamine surge is sometimes invoked in custodial or restraint deaths. The more defensible approach is to evaluate restraint position, neck compression, chest compression, hypoxia, drug intoxication, hyperthermia, acidosis, and cardiac vulnerability together.

Pheochromocytoma and other catecholamine excess states provide rare but instructive models. Falhammar et al.15 reported fatal catecholamine-induced cardiotoxicity associated with pheochromocytoma, showing that extreme catecholamine excess can be

rapidly fatal. While this is not typical of assault, it strengthens biological plausibility for catecholamine-mediated myocardial injury. Current literature therefore supports a "trigger-substrate" model. Assault supplies the trigger: fear, pain, exertion, blunt impact, restraint, or hypoxia. The individual supplies the substrate: coronary artery disease, myocardial hypertrophy, fibrosis, cardiomyopathy, channelopathy, intoxication, or endocrine catecholamine excess. Death occurs when trigger intensity exceeds cardiac reserve and produces malignant arrhythmia, ischemia, or mechanical-electrical instability. The medico-legal implication is that natural disease and assault are not mutually exclusive. A person with severe coronary disease may have died naturally at some later time, but the immediate assault may still have accelerated death. Conversely, the presence of assault does not automatically make the death stress-induced if there is a clear alternative fatal mechanism. The most scientifically defensible opinion is one that states the mechanism, degree of contribution, and evidentiary limitations.

CONCLUSION

Catecholamine surge and physiological stress response play a significant role in sudden cardiac death during assault, particularly when the victim has an underlying vulnerable cardiac substrate. Assault may precipitate fatal arrhythmia, ischemia, coronary vasospasm, plaque rupture, Takotsubo syndrome, or commotio cordis. However, catecholamine surge is difficult to prove directly after death and should not be used as a default explanation when trauma, asphyxia, intoxication, or natural disease better explains the death.

A structured forensic approach should include scene reconstruction, witness timing, injury analysis, full cardiac autopsy, histology, toxicology, post-mortem biochemistry, review of resuscitation rhythm, and molecular testing where appropriate. The most balanced conclusion in many cases is that assault-related stress acted as a precipitating or contributory factor in sudden cardiac death, rather than being an isolated cause.

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