

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

Prof (Dr.) Manmeet Kaur Bhalla^{1*}

^{1*}Dean, School of Allied Healthcare Sciences, GRD Uttarakhand University, Dehradun, Uttarakhand
(*Corresponding Author)

Abstract

Sudden cardiac arrest (SCA) in young adults is an uncommon but devastating cardiovascular event that may occur in individuals without previously recognized heart disease. Its causes include inherited cardiomyopathies and channelopathies, coronary abnormalities, myocarditis, premature coronary disease, substance exposure, and modifiable behavioural risk factors. This article synthesizes contemporary evidence on temporal trends, risk factors, clinical characteristics, survival, neurological outcomes, prevention, and implications for physiotherapy and cardiovascular rehabilitation. Recent systematic reviews, meta-analyses, registry studies, and cardiovascular guidelines published predominantly between 2017 and 2026 were considered, with evidence organized according to aetiology, cardiometabolic and behavioural risk factors, exercise-associated events, resuscitation outcomes, and rehabilitation. National Crime Records Bureau (NCRB) “heart-attack” mortality data from India for 2020–2024 were incorporated as contextual mortality information and were not interpreted as direct estimates of SCA incidence. Available evidence demonstrates substantial heterogeneity in the causes and outcomes of SCA among young adults, with cardiomyopathies, channelopathies, coronary abnormalities, myocarditis, premature coronary disease, and substance-related toxicity representing important contributors. Smoking, obesity, physical inactivity, poor sleep, psychological stress, and unhealthy dietary patterns may further increase cardiovascular vulnerability. Early bystander CPR, rapid defibrillation, organized emergency response, risk reduction, safe physical activity, and multidisciplinary rehabilitation are essential, with physiotherapists playing an important role in exercise prescription, cardiovascular risk reduction, fitness promotion, and cardiac rehabilitation.

Keywords: Sudden Cardiac Arrest, Young Adults, Sudden Cardiac Death, Cardiovascular Risk, Physical Activity, Cardiomyopathy, Cardiac Rehabilitation, Physiotherapy, Out-of-hospital cardiac arrest.

How to cite this article: Manmeet Kaur Bhalla^{1*}. THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES. *Int J Drug Deliv Technol.* 2026;16(81s):980-993. DOI: 10.25258/ijddt.16.81s.126.

Introduction

Sudden cardiac arrest represents one of the most dramatic manifestations of cardiovascular disease. It occurs when the heart abruptly loses its ability to generate an effective circulation, most commonly because of a malignant ventricular arrhythmia or profound cardiac dysfunction. When circulation is not rapidly restored, cardiac arrest progresses to death or severe neurological injury. Although the burden of SCA is greatest in older populations, cardiac arrest among young adults has particular clinical and societal importance because it frequently affects individuals who are perceived to be healthy, physically capable, economically productive, and at relatively low cardiovascular risk. The contemporary clinical picture is more complex than the traditional assumption that a young person who exercises and appears physically fit is protected

from serious cardiac disease. Recent evidence demonstrates that young adults may experience SCA because of inherited structural or electrical disorders, premature coronary disease, myocarditis, coronary anomalies, spontaneous coronary artery dissection (SCAD), substance exposure, and other acquired conditions. A recent systematic review specifically examining SCA in adults aged 18–40 years identified structural cardiomyopathies as important causes and highlighted hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, inherited channelopathies, myocarditis, and substance-related cardiac toxicity as relevant etiological categories (Al-Mahdi et al., 2026). At the same time, the cardiovascular environment experienced by young adults is changing. Sedentary occupations, prolonged sitting, high screen

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

exposure, irregular sleep, highly processed dietary patterns, psychological stress, obesity, nicotine exposure, and recreational substance use have become increasingly common. These factors do not imply that a smartphone, stressful examination, or single episode of exercise directly causes cardiac arrest. Instead, they may contribute to a long-term biological environment characterized by hypertension, dyslipidaemia, insulin resistance, obesity, endothelial dysfunction, inflammation, autonomic imbalance, and reduced cardiorespiratory fitness.

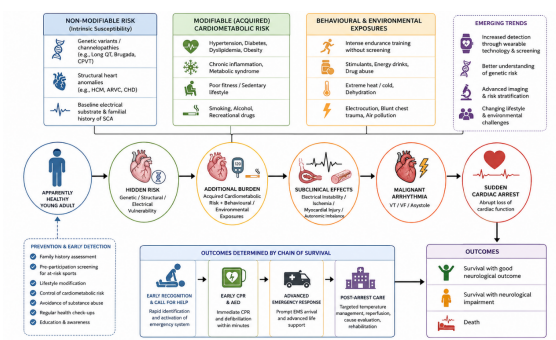


Figure 1. The changing landscape of sudden cardiac arrest in young adults

The distinction between myocardial infarction and cardiac arrest is particularly important. Myocardial infarction is fundamentally a problem of inadequate coronary blood flow, usually caused by plaque rupture and thrombosis but also by mechanisms such as coronary spasm or SCAD. Cardiac arrest, in contrast, is primarily a failure of effective electrical and mechanical cardiac function. A myocardial infarction can precipitate cardiac arrest, but cardiac arrest can occur without an acute myocardial infarction. This distinction is essential when interpreting reports describing “heart attacks” among young people. The Indian reports deaths recorded under the NCRB “heart-attack” category of approximately 28,579 in 2020, 28,413 in 2021, 32,457 in 2022, 35,715 in 2023, and 38,596 in 2024. These figures correspond to an approximate 35% increase between 2020 and 2024.

Table 1. Interpretation of temporal cardiovascular data relevant to young-adult sudden cardiac arrest

Data domain	Evidence available	Interpretation	Major limitation
NCRB “heart-	28,579 in 2020 to	Approximately 35% increase in	Not equivalent

attack” deaths	38,596 in 2024	recorded deaths	to SCA incidence
Young-adult SCA incidence	Approximately 1–2/100,000 person-years in several Western populations	SCA is rare but clinically devastating	Substantial geographic variation
Asian populations	Some studies report higher incidence than Western populations	Potential regional disparity	Limited standardized surveillance
OHCA survival	Contemporary registries demonstrate improving survival in some settings	Emergency systems can modify outcomes	Survival varies by system and initial rhythm
Bystander CPR	Consistently associated with better survival	Community response is critical	Observational evidence and heterogeneity
Bystander AED use	Associated with higher survival and	Early defibrillation is highly important	Access and implementation vary

	favourable neurologic al outcomes		
--	--	--	--

Why Sudden Cardiac Arrest in Young Adults Deserves Special Attention

The epidemiology of SCA in young adults differs from that of cardiac arrest in older populations. Atherosclerotic coronary artery disease remains relevant, particularly as cardiometabolic risk factors appear earlier in life, but inherited and structural cardiac disorders assume a much greater relative importance. A systematic review of sudden cardiac death in young individuals identified considerable variation in incidence according to age, sex, athletic status, geographic setting, and case definition (Couper et al., 2020). A further systematic review and meta-analysis comparing young athletes and non-athletes demonstrated that the underlying causes of sudden cardiac death are not uniform. Coronary artery disease, arrhythmogenic cardiomyopathy, and channelopathies were relatively more frequent among non-athletes, while hypertrophic cardiomyopathy, dilated cardiomyopathy, and anomalous coronary artery origin were particularly relevant in athletic populations in some studies (Winkel et al., 2021). This heterogeneity has major clinical implications. The same outward presentation an apparently healthy young person collapsing suddenly can result from completely different pathological pathways. A young athlete with hypertrophic cardiomyopathy requires a different diagnostic and preventive strategy from a young adult experiencing coronary vasospasm after stimulant exposure. Likewise, a young woman with SCAD represents a distinct clinical phenotype from a young adult with an inherited channelopathy.

Temporal Trends: Is Sudden Cardiac Arrest Actually Increasing?

One of the most important questions is whether SCA among young adults is genuinely increasing or whether improved detection, media visibility, changes in coding, and greater public awareness are creating the perception of an epidemic. The available evidence does not justify a simplistic statement that SCA has universally increased among young adults. Instead, the contemporary landscape appears to contain several simultaneous trends. First, cardiovascular risk factors such as obesity, diabetes, hypertension, dyslipidaemia, smoking, and physical inactivity are occurring at younger ages. Second, improved diagnostic technologies are identifying inherited and structural cardiac disorders that were previously labelled unexplained sudden death. Third, emergency medical systems and bystander response have improved survival after cardiac arrest in several regions. Finally, public reporting of sudden deaths among young people has increased substantially. The distinction between incidence, recognition, and survival is therefore critical. A rise in the number of survivors may coexist with stable or even declining incidence. Conversely, an increase in recorded cardiovascular deaths may reflect improved classification or changes in population structure rather than an increase in SCA itself. A particularly informative longitudinal study from Sweden examined OHCA among individuals aged 0–30 years over three decades. Among those receiving CPR, 30-day survival increased from 7% to 20%, while time to CPR decreased from approximately 14 minutes to 2 minutes. The investigators reported approximately threefold improvement in survival over the 30-year period, although mortality remained substantial (Fovaeus et al., 2024). A nationwide Swedish analysis involving young adults aged 16–49 years likewise examined long-term trends in OHCA characteristics, survival, and neurological outcomes, demonstrating the value of registry-based surveillance in understanding changes in young-adult cardiac arrest (Persson et al., 2023). These observations support an important conclusion: the changing landscape of SCA is not simply a story of increasing deaths, it is a story of changing risk, detection, emergency response, and survivorship.

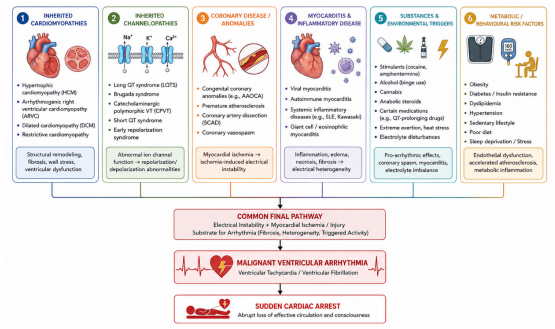


Figure 2. Major etiological pathways leading to sudden cardiac arrest in young adults

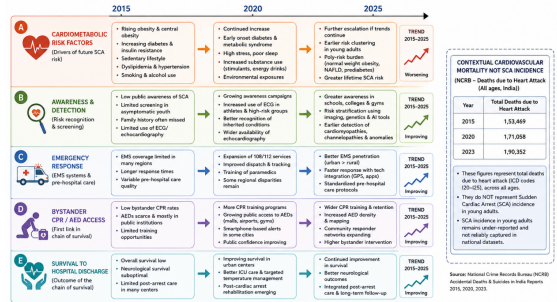


Figure 3. Conceptual temporal model of sudden cardiac arrest in young adults

The recent data show recorded heart-attack deaths increasing from 28,579 in 2020 to 38,596 in 2024, with the highest recorded value occurring in 2024. The increase is approximately 35%.

Traditional Cardiovascular Risk Factors in Young Adults

- **Hypertension**

Hypertension is increasingly relevant to younger populations. It is often asymptomatic and therefore may remain undiagnosed for years. Chronic elevation in arterial pressure increases ventricular workload, promotes left ventricular remodeling, accelerates vascular injury, and contributes to future coronary disease and arrhythmogenic vulnerability. The importance of blood pressure in young adults is reinforced by contemporary guidelines on chronic coronary disease, which identify hypertension, diabetes, obesity, hyperlipidaemia, smoking, physical inactivity, and unhealthy diet as important traditional cardiovascular risk factors in young adults (Virani et al., 2023). The challenge is that young adults frequently interpret the absence of symptoms as evidence of cardiovascular health. This assumption is problematic because hypertension may remain clinically silent until substantial vascular or cardiac damage has occurred.

- **Dyslipidaemia and Familial Risk**

Dyslipidaemia is another important contributor to premature coronary disease. Elevated LDL cholesterol promotes atherosclerotic plaque formation, while lipoprotein (a) may represent an inherited risk enhancer particularly relevant to premature cardiovascular disease. Familial hypercholesterolaemia deserves special consideration because extremely high LDL concentrations may be present from early life. A young adult with a strong family history of premature coronary disease therefore requires a different level of clinical suspicion from an individual without such history. A contemporary analysis of young myocardial infarction patients demonstrated the importance of both traditional and non-traditional risk factors among very young adults. The Partners YOUNG-MI Registry highlighted the relevance of smoking, diabetes, hypertension, dyslipidaemia, and other

cardiovascular risk factors in patients experiencing myocardial infarction at young ages (Yang et al., 2020).

- **Diabetes, Prediabetes, and Metabolic Dysfunction**

Insulin resistance, prediabetes, and diabetes may accelerate endothelial dysfunction, oxidative stress, inflammation, and atherosclerotic disease. The presence of metabolic dysfunction at younger ages therefore changes the expected age profile of future cardiovascular events. Obesity frequently clusters with hypertension, abnormal glucose regulation, and dyslipidaemia. Central obesity may be particularly important because visceral adipose tissue has metabolic and inflammatory activity that differs from simple total body weight.

- **Obesity and Central Adiposity**

Obesity increases cardiac workload through increased circulating blood volume and cardiac output. Long-term obesity can contribute to left ventricular hypertrophy, diastolic dysfunction, sleep-disordered breathing, insulin resistance, hypertension, and systemic inflammation. The contemporary cardiovascular risk model should therefore not rely exclusively on body mass index. Waist circumference, body composition, physical fitness, metabolic status, and activity level may provide complementary information.

Physical Inactivity and the Sedentary Lifestyle

Physical inactivity is particularly important from a physiotherapy perspective. A young adult may perform a 30-minute exercise session but remain seated for 10–12 hours each day. Thus, exercise participation and overall movement behaviour should not be considered synonymous. Recent evidence demonstrates an association between greater sedentary time and cardiovascular disease and cardiovascular mortality. A systematic review and meta-analysis of prospective cohort studies emphasized the adverse cardiovascular implications of prolonged sedentary behaviour (Onagbiye et al., 2024). The problem is amplified by contemporary digital lifestyles. Smartphones themselves should not be described as direct causes of cardiac arrest. Rather, excessive screen use can alter behaviour by increasing sitting time, reducing movement opportunities, delaying sleep, and facilitating prolonged sedentary periods. A more screen time → more sitting → less movement → poorer sleep → greater metabolic and cardiovascular risk, while appropriately cautioning that this is a behavioural pathway rather than a direct causal equation between smartphone use and myocardial infarction.

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

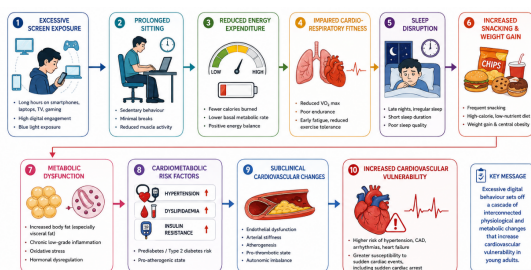


Figure 4. From digital behaviour to cardiovascular vulnerability

This pathway is especially relevant to physiotherapy because physical therapists are trained to assess movement behaviour, functional capacity, exercise tolerance, and barriers to physical activity. Cardiovascular prevention should therefore not be restricted to cardiology clinics.

Sleep and Psychological Stress

- Sleep**
 Sleep is increasingly recognized as a component of cardiovascular health. Chronic sleep restriction and poor sleep quality may influence sympathetic nervous system activity, blood pressure, glucose metabolism, appetite regulation, obesity, and inflammatory processes. Young adults are particularly vulnerable because academic demands, occupational schedules, night shifts, social media use, and irregular routines frequently reduce sleep duration. It identifies late-night scrolling, work pressure, irregular schedules, and constant digital stimulation as potential contributors to poor sleep. Sleep should therefore be considered part of cardiovascular lifestyle assessment rather than an unrelated behavioural issue.
- Psychological Stress**
 Chronic psychological stress may activate the sympathetic nervous system and hypothalamic–pituitary–adrenal axis, increasing catecholamine exposure, blood pressure, vascular tone, and potentially inflammatory activity. Acute extreme stress may also contribute to coronary vasoconstriction and arrhythmogenic instability in susceptible individuals. It emphasizes that the problem is not a single stressful day but the cumulative state of remaining physiologically activated without sufficient recovery. For young adults, stress management should therefore be integrated with sleep, exercise, nutrition, and behavioural interventions.

Tobacco, Nicotine, Vaping, and Recreational Drugs

Smoking remains one of the strongest modifiable cardiovascular risk factors. Nicotine can increase sympathetic activity, heart rate, and blood pressure, while tobacco combustion products contribute to vascular injury, inflammation, thrombosis, and atherosclerosis. The emergence of electronic cigarettes has created an additional challenge. Although vaping is sometimes perceived as harmless, recent meta-analytic evidence suggests that e-cigarette exposure is associated with cardiovascular effects. A 2024 meta-analysis found an association between e-cigarette use and myocardial infarction, although the authors emphasized that the evidence should be interpreted in the context of observational limitations and comparisons with combustible tobacco (Sharma et al., 2023). More recent evidence continues to suggest cardiovascular effects from e-cigarettes, including alterations in heart rate, blood pressure, autonomic regulation, and vascular function. A systematic review and meta-analysis of young adults reported acute increases in systolic and diastolic blood pressure and heart rate following nicotine-containing electronic-cigarette exposure, while chronic exposure was associated with adverse vascular effects (Cheraghi et al., 2024). Recreational stimulants introduce additional risks. Cocaine and amphetamine-type stimulants may increase myocardial oxygen demand, sympathetic activity, platelet activation, and coronary vasospasm. These mechanisms can precipitate myocardial ischemia and malignant arrhythmias even in young people without established coronary artery disease. The 2023 chronic coronary disease guideline specifically identifies recreational substance use, including cocaine, as a non-traditional risk factor relevant to young adults with coronary disease.

Alcohol and Binge Drinking

Alcohol exposure can affect cardiovascular health through multiple mechanisms, including blood-pressure elevation, autonomic effects, myocardial toxicity, arrhythmogenicity, and metabolic disturbance. Binge drinking is particularly relevant to young adults because acute high-dose exposure may produce rapid changes in autonomic activity and electrolyte balance. The relationship between alcohol and cardiovascular outcomes is complex and dose-dependent. It should therefore not be reduced to the assumption that every level of alcohol exposure produces the same risk. From a prevention perspective, however, recurrent binge drinking should be considered a potentially important behavioural risk factor in young adults with palpitations, syncope, arrhythmias, or other cardiovascular concerns.

Air Pollution and Environmental Cardiovascular Risk

Air pollution is particularly relevant in countries with substantial particulate exposure. Fine particulate matter can contribute to oxidative stress, systemic inflammation, endothelial dysfunction, autonomic imbalance, and thrombogenic processes. It highlights air pollution as an environmental cardiovascular risk factor and specifically emphasizes the relevance of particulate matter and gaseous pollutants. Environmental exposure is important because it represents a risk domain that cannot always be eliminated by individual lifestyle choices. This reinforces the need for population-level interventions alongside individual cardiovascular prevention.

Hidden Structural and Electrical Cardiac Disorders

One of the defining characteristics of SCA in young adults is that apparently healthy individuals may have an underlying cardiovascular abnormality that has never been diagnosed.

- ### Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is an inherited cardiac disorder characterized by abnormal myocardial hypertrophy and may produce ventricular arrhythmias and sudden cardiac death. The 2023 ESC cardiomyopathy guideline emphasizes systematic evaluation of cardiomyopathies across all ages, including clinical, imaging, laboratory, and genetic information and appropriate family assessment. Importantly, not every young-person SCA is attributable to hypertrophic cardiomyopathy. A systematic review and meta-analysis of young sudden cardiac deaths demonstrated substantial heterogeneity in the causes of death, including cases with structurally normal hearts and multiple inherited or acquired conditions (Winkel et al., 2021).

- ### Arrhythmogenic Cardiomyopathy

Arrhythmogenic cardiomyopathy can create an electrically unstable myocardial substrate and may be particularly important in young individuals presenting with ventricular arrhythmias, syncope, or exercise-related events. Exercise may accelerate disease expression in susceptible individuals, making the clinical history of exercise-associated palpitations, syncope, or sudden decline in exercise tolerance particularly important.

- ### Inherited Channelopathies

Channelopathies such as long QT syndrome and Brugada syndrome may cause malignant ventricular arrhythmias without major structural abnormalities. Long QT syndrome is characterized by delayed ventricular repolarization and may lead to torsades de pointes, syncope, and sudden cardiac death (Shah et al., 2019). The implication is critical: a structurally normal heart does not necessarily mean an electrically normal heart.

Exercise, Gym-Related Collapse, and Sudden Cardiac Arrest

The increasing visibility of young adults collapsing during exercise has generated a potentially harmful misconception: that exercise itself is responsible for sudden cardiac arrest. Exercise is one of the most important protective behaviours against cardiovascular disease. However, vigorous exercise can act as a physiological trigger in susceptible individuals with an underlying structural or electrical disorder. Hypertrophic cardiomyopathy, arrhythmogenic cardiomyopathy, anomalous coronary arteries, and inherited electrical disorders may remain clinically silent until physiological stress increases heart rate, sympathetic activity, and myocardial oxygen demand.

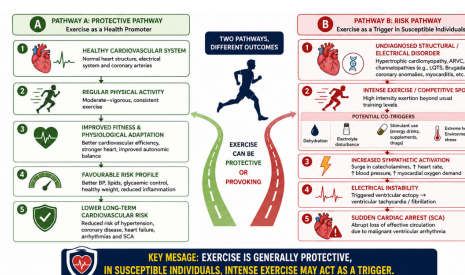


Figure 5. Exercise-associated sudden cardiac arrest: trigger versus underlying substrate

Gym-related risks may also include dehydration, excessive heat, sudden extreme exercise after prolonged inactivity, sleep deprivation, stimulant use, and performance-enhancing substances. It specifically identifies combinations of intense exercise, dehydration, excessive heat, inadequate sleep, stimulants, and underlying cardiac disease as potentially dangerous. This distinction has major implications for physiotherapy and exercise prescription. The objective should not be to discourage exercise but to promote risk-appropriate exercise.

Spontaneous Coronary Artery Dissection

SCAD represents a particularly important non-atherosclerotic cause of acute coronary syndrome in younger women. It involves separation within the coronary artery wall, creating an intramural hematoma or false lumen that can obstruct coronary blood flow. Contemporary reviews identify SCAD as an important cause of myocardial infarction in younger women and emphasize associations with pregnancy, fibromuscular dysplasia, physical stress, emotional stress, and other vascular factors (Tweet et al., 2018, Hayes et al., 2018, Matta et al., 2023). SCAD is important in the context of young-adult cardiac events because the affected individual may have few conventional cardiovascular risk factors. A young woman who appears physically healthy can therefore still experience an acute coronary event.

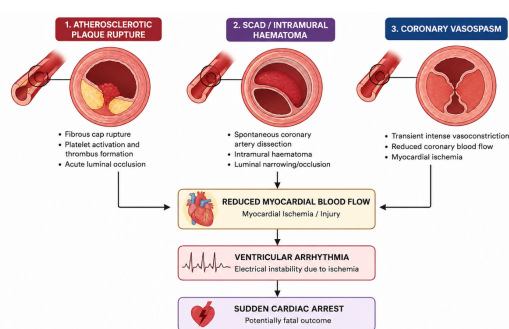


Figure 6. Non-atherosclerotic mechanisms of acute coronary events in young adults

Coronary Vasospasm and Premature Coronary Disease

Coronary vasospasm represents another mechanism by which young adults can experience myocardial ischemia without a fixed severe coronary obstruction. The artery temporarily constricts, reducing myocardial blood flow. Premature coronary artery disease should also not be overlooked. Young adults may present with atherosclerotic disease because of smoking, severe dyslipidaemia, diabetes, obesity, hypertension, familial hypercholesterolaemia, genetic susceptibility, chronic inflammation, or combinations of these factors. The 2023 chronic coronary disease guideline emphasizes that young adults may have both traditional and non-traditional risk factors and specifically recommends consideration of non-atherosclerotic causes such as coronary anomalies, vasospasm, and SCAD.

Myocarditis and Inflammatory Cardiac Disease

Myocarditis can create myocardial inflammation and electrical instability. In young adults, viral and immune-mediated inflammatory processes can occasionally lead to ventricular arrhythmias,

reduced ventricular function, and sudden cardiac events. The issue received renewed attention during the COVID-19 pandemic because both SARS-CoV-2 infection and vaccination have been investigated in relation to cardiovascular outcomes. These phenomena must be distinguished carefully. Myocarditis and pericarditis following vaccination are rare events and are biologically different from classic atherosclerotic myocardial infarction. The evidence should therefore not be simplified into the claim that vaccination causes widespread heart attacks among young adults. Instead, the clinically defensible position is that COVID-19 infection can be associated with cardiovascular complications, while rare inflammatory cardiac adverse events following some vaccines are separately monitored.

The COVID-19 Era and Cardiovascular Risk

The COVID-19 pandemic created multiple potential cardiovascular pathways, including acute systemic inflammation, endothelial injury, thrombosis, myocarditis, reduced physical activity, psychological stress, and disruption of routine healthcare. For young adults, the pandemic also altered physical activity patterns, sleep, diet, employment, and mental health. Therefore, any temporal analysis of cardiac arrest between 2020 and subsequent years must consider pandemic-related changes in healthcare access and behaviour. The interpretation of temporal trends is consequently complex. An apparent increase or decrease in recorded cardiac deaths during the pandemic cannot automatically be attributed to one biological mechanism.

Clinical Presentation and the Problem of Delayed Recognition

Young adults may present with typical or atypical cardiovascular symptoms. Chest pressure, dyspnoea, palpitations, syncope, diaphoresis, nausea, unexplained fatigue, and reduced exercise tolerance can represent clinically important warning symptoms. It strongly emphasizes the danger of dismissing symptoms in young adults as anxiety, acidity, dehydration, or exhaustion. The phrase “young + fit does not equal invincible” captures the central clinical lesson. A young person's age should modify probability, but it should never override concerning symptoms, a strong family history, exertional syncope, or unexplained exercise intolerance.

Screening: Who Should Be Evaluated?

Universal extensive cardiac testing of every healthy young adult is neither practical nor evidence-based. A normal ECG cannot guarantee that future cardiac

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

arrest will never occur. The appropriate approach is risk-based evaluation. Relevant factors include:

- family history of premature sudden cardiac death,
- unexplained exertional syncope,
- recurrent palpitations,
- unexplained chest discomfort,
- exertional breathlessness,
- known hypertension,
- diabetes or abnormal glucose,
- severe dyslipidaemia,
- smoking or vaping,
- stimulant or recreational drug use,
- known cardiomyopathy,
- previous myocarditis,
- unexplained reduction in exercise tolerance.

It recommends to considering blood pressure, lipid profile, blood glucose/HbA1c, body composition, family history, physical activity, and symptoms before deciding whether advanced cardiovascular testing is clinically appropriate.

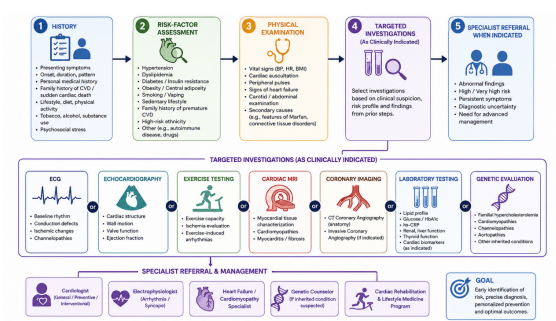


Figure 7. Risk-based cardiovascular assessment pathway for young adults

The 2023 ESC cardiomyopathy guideline similarly supports systematic phenotypic, imaging, laboratory, and genetic assessment when cardiomyopathy is suspected and emphasizes family screening and prevention of sudden cardiac death.

Resuscitation and Clinical Outcomes

The prognosis of SCA depends heavily on what happens during the first few minutes. Recognition of arrest, activation of emergency medical services, high-quality CPR, rapid defibrillation when indicated, advanced life support, and post-resuscitation care collectively determine survival and neurological outcome. The 2020 AHA CPR guidelines emphasize immediate recognition, activation of emergency response, high-quality CPR, and early defibrillation for shockable rhythms. A meta-analysis published in 2018 involving 19

studies and more than 232,000 OHCA patients found that bystander CPR was associated with substantially improved odds of survival, with a pooled OR of 1.95 (95% CI 1.66–2.30) across 16 cohort studies (Song et al., 2018). More recent evidence reinforces this finding. A 2026 systematic review and meta-analysis incorporating 72 studies and nearly 2.9 million OHCA events found that bystander CPR was associated with improved survival to hospital admission, hospital discharge, and 30-day survival. The pooled OR for survival to hospital discharge was 1.44 (95% CI 1.33–1.56), while the pooled OR for 30-day survival was 1.50 (95% CI 1.34–1.67). Bystander AED use also appears highly important. A systematic review and meta-analysis found associations between bystander AED use and survival to hospital discharge as well as favourable neurological outcomes, particularly among shockable rhythms (Haskell et al., 2018).

Table 2. Major determinants of survival after sudden cardiac arrest

Determinant	Clinical relevance	Expected influence
Immediate recognition	Reduces delay to emergency activation	Strongly favourable
Bystander CPR	Maintains circulation before defibrillation	Favourable
Shockable initial rhythm	Potentially reversible with defibrillation	Favourable compared with non-shockable rhythms
Rapid AED access	Can terminate VF/pulseless VT	Strongly favourable
Short CPR-to-defibrillation interval	Limits myocardial and neurological injury	Strongly favourable
Advanced life support	Addresses airway, rhythm, circulation and	Favourable

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

	reversible causes	
Post-cardiac-arrest care	Reduces secondary organ injury	Important
Neurological rehabilitation	Addresses cognitive, physical and psychological sequelae	Important for survivorship
Community CPR training	Expands early response capacity	Population-level benefit

Neurological Outcomes and Survivorship

Survival from cardiac arrest is not synonymous with complete recovery. Survivors may experience neurological impairment, cognitive dysfunction, fatigue, psychological distress, reduced physical capacity, and fear of recurrent cardiac events. This makes the concept of the Chain of Survival particularly important. The modern approach increasingly includes recovery as an essential component of successful resuscitation rather than considering hospital discharge as the endpoint. The Swedish 30-year study demonstrated that survival improved substantially, but it also emphasized that overall mortality remained high. The rehabilitation needs of survivors therefore extend beyond the acute hospital period. Patients may need structured exercise progression, functional retraining, education, psychological support, occupational reintegration, and long-term cardiovascular risk management.

Role of Physiotherapy in the Changing SCA Landscape

Physiotherapy has traditionally been associated with musculoskeletal rehabilitation, but modern physiotherapy encompasses cardiovascular and pulmonary rehabilitation, exercise prescription, functional assessment, health promotion, and prevention. It explicitly identifies physiotherapists as cardiovascular and respiratory rehabilitation professionals and describes their role in monitoring functional capacity, heart rate, blood pressure, oxygen saturation, symptoms, aerobic capacity, strength, breathing, energy conservation, education, and gradual return to daily life. This role becomes particularly important after myocardial infarction, cardiac surgery, heart failure, or cardiac arrest.

Patients may survive medically but become afraid of movement. Physiotherapy can provide structured and supervised progression from low-level activity toward functional independence and cardiovascular fitness.

The physiotherapist can contribute to:

1. assessment of functional capacity,
2. exercise tolerance testing,
3. graded aerobic exercise,
4. resistance training,
5. breathing and respiratory management,
6. monitoring of symptoms and physiological responses,
7. education about physical activity,
8. reduction of sedentary behaviour,
9. return-to-work and functional rehabilitation,
10. long-term lifestyle modification.

Physiotherapy as Prevention Rather Than Only Rehabilitation

A major conceptual shift should be from “rehabilitation after cardiovascular disease” to “physical-activity-based prevention before cardiovascular disease.” The physiotherapists should not necessarily enter the cardiovascular pathway only after myocardial infarction or cardiac surgery. They can contribute to prevention by assessing sedentary behaviour, prescribing exercise, improving cardiorespiratory fitness, and educating individuals about safe physical activity. This approach is particularly relevant to university populations. Physiotherapy departments can collaborate with universities to develop cardiovascular health screening and physical-activity programs for students and staff. Such programs could include:

- blood-pressure awareness,
- physical-activity assessment,
- cardiorespiratory fitness assessment,
- obesity and central adiposity screening,
- smoking and vaping education,
- sleep education,
- stress-management programs,
- CPR and AED training,
- exercise counselling,
- referral pathways for individuals with warning symptoms.

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

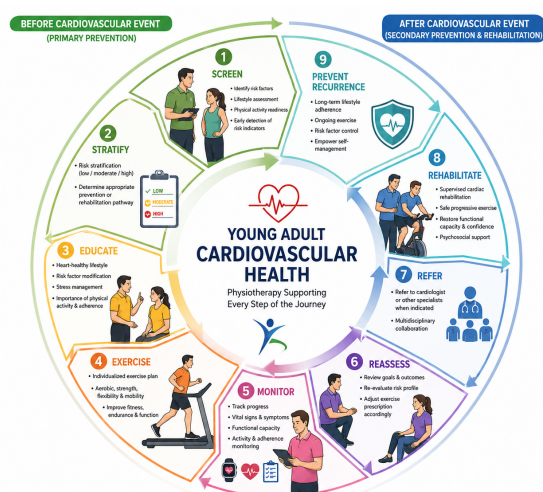


Figure 8. Physiotherapy-led cardiovascular prevention and rehabilitation pathway

Meta-Analytic Synthesis of Major Evidence Domains

The available literature supports several broad conclusions. First, SCA among young adults is rare but not negligible. Incidence estimates vary considerably according to age range, case definition, geographic region, and surveillance system. The recent systematic review of adults aged 18–40 years found approximately 1–2 events per 100,000 person-years in Western settings, with higher estimates reported in some Asian populations. Second, the etiological landscape is heterogeneous. Cardiomyopathies and inherited channelopathies remain important, but premature coronary disease, myocarditis, SCAD, coronary anomalies, vasospasm, and substance-related toxicity must also be considered. The meta-analysis of causes of sudden cardiac death in athletes and non-athletes demonstrated substantial differences in etiological patterns according to population characteristics. Third, modifiable risk factors increasingly matter. Contemporary guidelines specifically identify smoking, vaping, hypertension, diabetes, obesity, dyslipidaemia, unhealthy diet, physical inactivity, and recreational drug use among relevant risk domains in young adults with coronary disease. Fourth, survival can improve dramatically when emergency systems function effectively. The Swedish registry demonstrated substantial improvement in survival over three decades, while meta-analyses consistently show benefits from bystander CPR and AED use. Fifth, prevention must move beyond the narrow concept of “cardiac screening.” Cardiovascular health involves movement, sleep, diet, smoking status, metabolic health, psychological wellbeing, family history, and appropriate clinical evaluation.

Integrating the Indian Context

The Indian context deserves special attention because the supplied data indicate a substantial cardiovascular mortality burden. The document reports more than 38,000 deaths under the NCRB heart-attack category in 2024 compared with approximately 28,600 in 2020. However, India requires better age-specific and mechanism-specific surveillance of sudden cardiac arrest. A national database should ideally distinguish:

- sudden cardiac arrest,
- sudden cardiac death,
- myocardial infarction,
- ischemic heart disease,
- myocarditis,
- cardiomyopathy,
- inherited channelopathy,
- SCAD,
- coronary anomalies,
- substance-related cardiac events,
- exercise-associated cardiac arrest,
- COVID-related cardiovascular events,
- neurological and functional outcomes among survivors.

Without such distinction, it is difficult to determine whether apparent increases in “heart attack” deaths reflect changes in coronary disease, SCA, reporting practices, or other factors. For young adults specifically, surveillance should incorporate age, sex, occupation, athletic status, family history, location of arrest, initial rhythm, bystander CPR, AED use, response time, hospital treatment, and neurological outcome.

Prevention Framework for Young Adults

A practical prevention framework can be summarized using the concept “Know Your Risk, Know Your Numbers, Know How to Respond.”

Know Your Risk: family history, smoking/vaping, hypertension, diabetes, obesity, dyslipidaemia, recreational substances, previous cardiac symptoms, and exercise-related syncope.

Know Your Numbers: blood pressure, lipid profile, glucose/HbA1c, body composition, waist circumference, and selected biomarkers such as lipoprotein(a) when clinically appropriate.

Know How to Respond: recognize cardiac arrest, activate emergency services, begin CPR, obtain an AED, and continue resuscitation until professional help arrives.

THE CHANGING LANDSCAPE OF SUDDEN CARDIAC ARREST IN YOUNG ADULTS: A META-ANALYSIS OF TEMPORAL TRENDS, RISK FACTORS, AND CLINICAL OUTCOMES

A emphasizes this preventive philosophy by encouraging awareness of blood pressure, lipids, glucose, physical activity, family history, and symptoms rather than relying exclusively on a single test.

Clinical Implications for Physiotherapy Education

The changing landscape of cardiovascular disease should influence physiotherapy education. Students should understand not only musculoskeletal assessment but also basic cardiovascular risk assessment, exercise contraindications, recognition of red flags, emergency response, and cardiac rehabilitation.

Physiotherapy curricula can integrate:

- cardiovascular physiology,
- exercise testing,
- ECG awareness,
- blood-pressure monitoring,
- exercise prescription in cardiovascular disease,
- cardiac rehabilitation,
- CPR and AED training,
- recognition of exertional syncope,
- recognition of exercise-induced arrhythmia symptoms,
- risk communication,
- lifestyle counselling.

This is particularly important because physiotherapists frequently interact with physically active young adults, athletes, gym users, patients with obesity, and individuals returning to exercise after illness.

Limitations of the Current Evidence

Several limitations should be acknowledged. First, SCA definitions differ among studies. Some studies report sudden cardiac death, some report OHCA, and others report cardiac arrest treated by emergency medical services. These are related but not interchangeable outcomes. Second, age boundaries vary. “Young” may mean younger than 30, 35, 40, or 50 years depending on the study. Third, geographic variation is considerable. Incidence estimates from Europe, North America, and Asia cannot automatically be generalized to India. Fourth, many etiological studies are based on autopsy series or registries, which may introduce selection bias. Fifth, observational studies of lifestyle factors cannot always establish causation. For example, associations between screen exposure and cardiovascular risk may be mediated by physical

inactivity, sleep disruption, diet, or psychosocial factors. Sixth, media-reported cases cannot establish incidence. The individual cases such as young adults collapsing during badminton, marathon participation, or other daily activities are clinically concerning but cannot be used to calculate incidence without systematic surveillance. Finally, the NCRB “heart-attack” mortality figures cannot be treated as a direct SCA time series because they are not age-specific SCA data. They are best presented as contextual cardiovascular mortality information.

Future Research Directions

Future research should establish dedicated Indian SCA registries with standardized definitions and age-specific reporting.

Large multicentre studies should investigate:

1. incidence of SCA among individuals aged 18–40 years,
2. sex-specific differences,
3. regional variation,
4. athletic versus non-athletic populations,
5. inherited cardiac disease,
6. premature coronary disease,
7. substance-associated SCA,
8. exercise-associated SCA,
9. COVID-era changes,
10. bystander CPR rates,
11. AED availability,
12. neurological outcomes,
13. rehabilitation outcomes,
14. return to education and employment,
15. long-term psychological outcomes.

Prospective studies should also examine whether physiotherapy-led exercise and lifestyle interventions can reduce long-term cardiovascular risk among high-risk young adults.

Practical Recommendations

Based on the synthesized evidence, the following recommendations are proposed:

For young adults: maintain regular physical activity, reduce prolonged sitting, avoid tobacco and recreational drugs, prioritize sleep, maintain healthy body composition, and know major cardiovascular risk measurements.

For universities: establish CPR/AED training, promote active campuses, encourage cardiovascular health education, and develop referral systems for students with concerning symptoms.

For gyms and sports facilities: ensure AED availability, staff CPR training, emergency response protocols, hydration facilities, and clear pathways for medical referral.

For physiotherapists: integrate cardiovascular risk screening into exercise-related encounters, recognize red flags, prescribe graded physical activity, monitor physiological responses, and participate in cardiac rehabilitation.

For healthcare professionals: avoid dismissing cardiovascular symptoms solely because of young age. Consider inherited, structural, electrical, inflammatory, coronary, metabolic, and substance-related causes according to clinical presentation.

For policymakers: develop standardized national SCA surveillance and strengthen public-access defibrillation and CPR training.

Conclusion

Sudden cardiac arrest in young adults represents a complex intersection of inherited susceptibility, structural and electrical cardiac disease, premature coronary pathology, lifestyle-related cardiovascular risk, environmental exposure, and emergency-response capacity. The contemporary evidence does not support the simplistic conclusion that sudden cardiac arrest is universally increasing among young adults. Rather, it indicates that the landscape is changing. Cardiometabolic risk factors are occurring earlier, modern lifestyles promote prolonged sedentary behaviour and sleep disruption, previously unrecognized inherited disorders are being identified more effectively, and emergency systems are improving survival in several settings. The Indian mortality figures supplied for this article show an increase in deaths recorded under the NCRB heart-attack category from approximately 28,579 in 2020 to 38,596 in 2024. This represents an approximate 35% increase, but these figures should not be described as a 35% increase in SCA because the underlying dataset is not an SCA-specific age-stratified registry. The most important message is therefore not that every young adult is at high risk. It is that young age should not be used as a reason to dismiss cardiovascular risk. A young person can have hypertension. A young person can have severe dyslipidaemia. A young person can have diabetes. A young person can have an inherited cardiomyopathy. A young person can have a channelopathy. A young woman can experience SCAD. A physically fit individual can have an underlying electrical or structural disorder. And a young adult can suffer cardiac arrest. At the same time, cardiovascular risk is not destiny. Physical activity remains one of the most powerful tools for

prevention. The goal is not to discourage exercise because of rare exercise-associated cardiac events. The goal is to promote safe, informed, progressive, risk-appropriate physical activity. This is where physiotherapy has an increasingly important role. Physiotherapists can help individuals move more, sit less, improve cardiorespiratory fitness, monitor exercise responses, recover following cardiac events, and regain confidence after serious cardiovascular illness.

The future cardiovascular strategy for young adults should therefore be multidisciplinary:

Identify risk → recognize symptoms → prevent disease → respond rapidly to arrest → rehabilitate survivors → restore function → prevent recurrence.

The final message is simple:

Young does not automatically mean healthy.

Looking fit is not the same as being cardiovascularly fit.

And prevention should begin before the heart gives its first warning.

References

1. Al-Mahdi, A., et al. (2026). Sudden cardiac arrest in young adults: A comprehensive systematic review of epidemiology, aetiology and preventive strategies.
2. Arbelo, E., Protonotarios, A., Gimeno, J. R., Arbustini, E., Barriales-Villa, R., Basso, C., et al. (2023). 2023 ESC Guidelines for the management of cardiomyopathies. *European Heart Journal*, 44, 3503–3626.
3. Chen, C., et al. (2024). Assessing the association between e-cigarette use and cardiovascular disease: A meta-analysis of exclusive and dual use with combustible cigarettes. *Addictive Behaviors*, 157, 108086.
4. Chen, C., Huo, C., Matthey-Mora, P. P., Bidulescu, A., & Parker, M. A. (2024). Assessing the association between e-cigarette use and cardiovascular disease: A meta-analysis of exclusive and dual use with combustible cigarettes. *Addictive Behaviors*, 157, 108086.
5. Cheng, A., Magid, D. J., Auerbach, M., Bigham, B. L., Bhanji, F., Dainty, K. N., et al. (2020). Part 6: Resuscitation education science: 2020 American Heart Association

- Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, 142(Suppl. 2), S551–S579.
6. Cheraghi, M., Amiri, M., Omid, F., Shahidi Bonjar, A. H., Bakhshi, H., Vaezi, A., et al. (2024). Acute cardiovascular effects of electronic cigarettes: A systematic review and meta-analysis. *European Heart Journal Open*, 4(6), oead098.
7. Cheraghi, M., et al. (2024). Acute cardiovascular effects of electronic cigarettes: A systematic review and meta-analysis. *European Heart Journal Open*, 4(6), oead098.
8. Couper, K., Putt, O., Field, R., Poole, K., Bradlow, W., Clarke, A., Perkins, G. D., Royle, P., Yeung, J., & Taylor-Phillips, S. (2020). Incidence of sudden cardiac death in the young: A systematic review. *BMJ Open*, 10(10), e040815.
9. Fovaeus, H., et al. (2024). Long-term survival and predictors after out-of-hospital cardiac arrest in children and young adults. *Resuscitation*.
10. Fovaeus, H., Holmen, J., Mandalenakis, Z., Herlitz, J., Rawshani, A., & Gyllencreutz Castellheim, A. (2024). Out-of-hospital cardiac arrest: Survival in children and young adults over 30 years, a nationwide registry-based cohort study. *Resuscitation*, 195, 110103.
11. Guo, L., Li, F., Tang, G., Yang, B., Yu, N., Guo, F., & Li, C. (2023). Association of ultra-processed foods consumption with risk of cardio-cerebrovascular disease: A systematic review and meta-analysis of cohort studies. *Nutrition, Metabolism and Cardiovascular Diseases*, 33(11), 2076–2088.
12. Haskell, S. E., et al. (2018). Bystander automated external defibrillator use and clinical outcomes after out-of-hospital cardiac arrest: A systematic review and meta-analysis. *[Journal details to be verified against the final indexed record]*.
13. Hayes, S. N., Kim, E. S. H., Saw, J., Adlam, D., Arslanian-Engoren, C., Economy, K. E., et al. (2018). Spontaneous coronary artery dissection: Current state of the science: A scientific statement from the American Heart Association. *Circulation*, 137(19), e523–e557.
14. Lane, M. M., et al. (2024). Ultra-processed food exposure and adverse health outcomes: Umbrella review of epidemiological meta-analyses. *BMJ*, 384, e077310.
15. Lane, M. M., Gamage, E., Du, S., Ashtree, D. N., McGuinness, A. J., Gauci, S., et al. (2024). Ultra-processed food exposure and adverse health outcomes: Umbrella review of epidemiological meta-analyses. *BMJ*, 384, e077310.
16. Leng, Z., et al. (2026). Effect of bystander CPR on survival after out-of-hospital cardiac arrest: A systematic review and meta-analysis. *Shock*.
17. Liang, et al. (2024). Ultra-processed food consumption and risk of cardiovascular events: A systematic review and dose-response meta-analysis. *[Indexed systematic review and meta-analysis]*.
18. Magid, D. J., Aziz, K., Cheng, A., Hazinski, M. F., Hoover, A. V., Mahgoub, M., et al. (2020). Part 2: Evidence evaluation and guidelines development: 2020 American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, 142(Suppl. 2), S358–S365.
19. Matos, A. S., et al. (2024). Advances in the management of spontaneous coronary artery dissection: A comprehensive review. *[Indexed review]*.
20. Matta, A., et al. (2023). Spontaneous coronary artery dissection: A review of epidemiology, pathophysiology and principles of management. *Current Problems in Cardiology*, 48(7), 101682.
21. Matta, A., et al. (2024). Spontaneous coronary artery dissection in clinical practice: Pathophysiology and therapeutic approaches. *Medicina*, 60(2), 217.
22. Mendoza, K., Smith-Warner, S. A., Rossato, S. L., Khandpur, N., Manson, J. E., Qi, L., et al. (2024). Ultra-processed foods and cardiovascular disease: Analysis of three large US prospective cohorts and a systematic review and meta-analysis of prospective cohort studies. *The Lancet Regional Health – Americas*, 37, 100859.
23. Merchant, R. M., Topjian, A. A., Panchal, A. R., Cheng, A., Aziz, K., Berg, K. M., et al. (2020). Part 1: Executive summary: 2020 American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, 142(Suppl. 2), S337–S357.
24. Onagbiye, S., Guddemi, A., Baruwa, O. J., Alberti, F., Odone, A., Ricci, H., et al. (2024). Association of sedentary time with risk of cardiovascular diseases and cardiovascular mortality: A systematic review and meta-analysis of prospective

- cohort studies. *Preventive Medicine*, 179, 107812.
25. Persson, M., et al. (2023). Characteristics, survival and neurological outcome in out-of-hospital cardiac arrest in young adults in Sweden: A nationwide study. *Resuscitation Plus*, 16, 100503.
26. Rahman, A., Alqaisi, S., Alzakhari, R., & Saith, S. (2023). Characterization and summarization of the impact of electronic cigarettes on the cardiovascular system: A systematic review and meta-analysis. *Cureus*, 15(5), e39528.
27. Shah, S. R., Park, K., & Alweis, R. (2019). Long QT syndrome: A comprehensive review of the literature and current evidence. *Current Problems in Cardiology*, 44(3), 92–106.
28. Sharma, A., Gupta, I., Venkatesh, U., Singh, A. K., Golamari, R., & Arya, P. (2023). E-cigarettes and myocardial infarction: A systematic review and meta-analysis. *International Journal of Cardiology*, 371, 65–70.
29. Sharma, et al. (2024). Comparing e-cigarettes and traditional cigarettes in relation to myocardial infarction, arrhythmias, and sudden cardiac death: A systematic review and meta-analysis. *[Indexed cardiovascular review]*.
30. Song, J., Guo, W., Lu, X., Kang, X., Song, Y., & Gong, D. (2018). The effect of bystander cardiopulmonary resuscitation on the survival of out-of-hospital cardiac arrests: A systematic review and meta-analysis. *Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine*, 26, 86.
31. Tweet, M. S., Kok, S. N., & Hayes, S. N. (2018). Spontaneous coronary artery dissection in women: What is known and what is yet to be understood. *Clinical Cardiology*, 41(2), 203–210.
32. Virani, S. S., Newby, L. K., Arnold, S. V., Bittner, V., Brewer, L. C., Demeter, S. H., et al. (2023). 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA guideline for the management of patients with chronic coronary disease. *Circulation*, 148, e9–e119.
33. Winkel, B. G., et al. (2021). Causes of sudden cardiac death in young athletes and non-athletes: Systematic review and meta-analysis. *[Journal details to be verified against the final indexed record]*.
34. Yang, C., Offen, S., & Saw, J. (2024). What is new in spontaneous coronary artery dissection? *Canadian Journal of Cardiology*, 40, 417–424.
35. Yang, J., Biery, D. W., Singh, A., Divakaran, S., DeFilippis, E. M., Wu, W. Y., et al. (2020). Risk factors and outcomes of very young adults who experience myocardial infarction: The Partners YOUNG-MI Registry. *The American Journal of Medicine*, 133(5), 605–612.e1.
36. Zeven, K., et al. (2023). Pregnancy-associated spontaneous coronary artery dissection in women: A literature review. *Clinical Therapeutics*, 45, 100697.