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Hypertension, Left Ventricular Hypertrophy and Sudden Cardiac Death in Scuba Diving

By Petar J. Denoble, MD

Introduction

Cardiac disease is associated with 20-30% of fatalities amongst recreational divers.[15, 39] In most cases the scenario of death fits the description of sudden cardiac death (SCD)[20, 21] which is also one of the most common causes of death in the general population.[50] The most common risk factors for SCD are atherosclerosis and structural heart changes which increase with age, but most cases occur in apparently healthy people and those classified as low risk.[73] Health requirements for diving used by most agencies exclude subjects with severe heart disease.[45] However, the average age of divers is increasing and thus the prevalence of marginal and undetected cardiovascular diseases may contribute to an increased incidence of scuba diving fatalities in the future.[17] To stratify risks among scuba divers and to establish targets for possible preventive interventions, in the absence of severe cardiovascular disease, other markers are necessary.[27] Left ventricular hypertrophy (LVH), which is an independent risk factor for SCD, may be such a marker. LVH is associated with hypertension but it may be present in apparently healthy people or in subjects with borderline hypertension.[53,68] In this article we will consider causal relationship of hypertension, LVH with SCD, their prevalence in the population studied, prevention in the general population, and possible preventive strategies to reduce SCD in diving.

Definition of Sudden Cardiac Death

Sudden cardiac death describes the unexpected natural death from a cardiac cause within a short time period, generally <1 hour from the onset of symptoms, in a person without any prior condition that would appear fatal.[74] According to The International Classification of Diseases, tenth Revision (ICD-10) this includes death due to any cardiac disease that occurs out of hospital, in an emergency department, or in an individual reported dead on arrival at a hospital. Some definitions also include a death that was unwitnessed and unexpected, unless a specific noncardiac cause of death was confirmed.[13]

In scuba diving, SCD is suspected in victims who in some cases go unconscious after indicating feeling suddenly ill, but in some cases without obvious or protracted struggle.[21] SCD can occur without obvious external triggers[16] or after exertion,[38] while at depth, at the surface or upon exiting the water, or in the absence of evidence of specific diving injuries or other causes of death.[9] Victims may or may not have a history of cardiac disease. Post-mortem evidence of myocardial infarction, excessive atherosclerotic changes, or other structural changes may or may not be present.[20, 22, 54]

Causes and Mechanisms of SCD

Structural or coronary heart disease is by far the most common cause of sudden cardiac arrest.[40, 47] In 60% to 80% of cases, the underlying condition is coronary artery disease but in many cases SCD may be its first clinical manifestation.[18] Nonischemic cardiomyopathy and infiltrative, inflammatory, and acquired valvular diseases account for most other SCD events. A small percentage of SCDs occur without overt heart disease due to inherited primary electric abnormalities long/short QT syndromes,[1] Brugada syndrome, and catecholaminergic ventricular tachycardia. Other causes include metabolic imbalance, hyperkalemia/hypokalemia, hypocalcemia, hypomagnesemia, acidosis,

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drug overdose, concealed structural changes, arrhythmogenic right ventricular cardiomyopathy, myocarditis, coronary spasm, sarcoidosis,[52] and rare non-cardiac causes.

SCD can manifest as ventricular tachycardia (VT), ventricular fibrillation (VF), pulseless electrical activity (PEA), or asystole.[24] The most common mechanism of SCD is ventricular tachycardia, which proceeds to ventricular fibrillation and asystole. The most common recorded rhythm at the time of sudden cardiac arrest is VF 75% to 80% followed by bradyarrhythmias 15–20%, including advanced atrio-ventricular AV block and asystole.[72] In ambulatory patients who had SCD while undergoing Holter recording, 62.4% had VF, 16.5% had bradyarrhythmias, 12.7% had torsades de pointes, and 8.3% had primary VT.[4] Over the last few decades the fraction of SCD due to VF is decreasing while the percentage of pulseless electrical activity PEA or asystole is increasing.[13]

Factors known to trigger or modulate VT/VF include changes in autonomic nervous system activity, metabolic disturbances, myocardial ischemia, electrolyte abnormalities, acute volume and/or pressure overload of the ventricles, ion channel abnormalities, and proarrhythmic actions of cardiac and noncardiac drugs.[26] Exercise may increase risk of SCD, but the relative numbers of SCD associated with exercise are small.[67]

Incidence of SCD and Risk Factors

Depending on the definition of SCD, its estimated incidence in the U.S. varies between 180,000 to 400,000 people. It is increasing with age from 20/100,000 in white men between ages 32 to 36, to 800/100,000 in white men between ages 72-76. The incidence is greater in African Americans of all age groups.[24,35,41] SCD occurs at home,49 in public places, at rest, and during physical activities like swimming,11, 65 jogging,[58, 69] etc.

In scuba diving, the overall incidence of sudden cardiac death is 5/100,000 and increases with age to 29/100,000.[17] In comparison to the incidence in the general population, this seems very small. However, scuba fatality rates were derived from a subpopulation of divers who did in average 25 dives per year adding up to 25 hours or approximately one day of cumulative exposure in comparison to the incidence in the general population calculated for 365 days.

Risk of SCD is increased in subjects with cardiac diseases, especially in post myocardial infarction patients with ejection fraction less than 30%. Patients with established disease usually do not participate in diving and it does not help to list clinical and diagnostic findings associated with their increased risk of SCD. What is of greatest interest for divers is that the largest number of SCD occurs in apparently healthy subjects or those considered at marginal risk[26, 73] and there are not reliable predictors that could be used in this subpopulation besides the common cardiac heart disease risk factors.[24, 41] Additional risk factors that can be valuated non-invasively include left ventricular hypertrophy (LVH),[3, 28] QTc prolongation,[12, 64] diabetes mellitus,[31] elevated resting heart rate,[32] and autonomic nervous dysfunction.[5] These risk markers may prove useful when trying to risk stratify scuba divers.

Left Ventricular Hypertrophy (LVH)

LVH is an independent risk for overall cardiac mortality and sudden cardiac death.[28,63,66] It develops in response to hemodynamic pressure overload, like in hypertension and aortic stenosis, or volume overload like in chronic aortic regurgitation, mitral regurgitation or anemia.[8,42,44,70,71] Pressure overload stimulates addition of sarcomeres, increase in myocytes width resulting in increased wall thickness and an increase in the ratio of wall thickness versus chamber dimensions. This type of LVH is called concentric hypertrophy. Volume overload causes lengthening of myocytes and increase in chamber dimensions while the ratio wall/chamber dimension decreases. This form is called eccentric hypertrophy.[42]

LVH can be established based on measurements of the left ventricular mass (LVM), left ventricular volume, thickness of the left ventricular posterior wall (LVW) or interventricular wall (IVW). LVM and LVW thickness increase with body weight, obesity,[2] valvular heart disease, chronic elevations in systolic blood pressure,[8] history of cardiovascular disease, male gender,[14] smoking, and diabetes.[23] Increases in left ventricular mass is also associated with ageing (due to reduced arterial elasticity), and in the Framingham study was seen in women more than men.[37] However, when indexed by body weight, body height or body surface area, left ventricular wall thickness does not change much throughout life and is similar in men and women.[33] LVW increases with age even in healthy subjects with blood pressures less than 140mmHg/90mmHg (current diagnostic threshold for hypertension), and the rate of increase is proportional to systolic pressure, where those with higher systolic pressures will have a greater lifetime risk and faster progression of LVH.[25]

The prevalence of LVH is associated with the prevalence

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of cardiac disease in the studied population and is influenced by how LVH is defined and measured (note the differences below between the parameters used by Framingham and Trömse). Based on the criteria of left ventricular mass >143gm-1 in men and >102gm-1 in women, the Framingham Heart Study's published LVH prevalence was 19% in men and 24% in women.[37] The Trömse Study included subjects between the ages of 25-85 years and defined LVH in individuals with a left ventricular mass per height > 145.5gm-1 for men and > 125.4gm-1 for women. In this population the prevalence of LVH was 14.9% in males and 9.1% in females.[60]

Whether the prevalence of LVH among recreational scuba divers is equivalent to the general population, as described by either of these studies, is currently unknown.

Mechanisms of Arrhythmogenesis and Diastolic Dysfunction in LVH

The arrhythmogenic propensity associated with LVH is related to the hypertrophy of myocytes and the degree of myocardial fibrosis, i.e. an increase in extracellular matrix ECM of proteins.[7] The ECM separates adjacent myocytes and causes their uncoupling which may slow down electrical propagation, create condition for reentry ventricular tachycardia, and result in suboptimal contraction force.[42] Risk of SCD increases with the degree of LVH.[66] However, any degree of LVH is detrimental for the left ventricular function which deteriorates left ventricular systolic dysfunction (LVSD) and over time may lead to heart failure. This is not true for exercise caused hypertrophy which is related primarily to the increase in myocyte mass not accompanied by interstitial fibrosis.[61]

LVSD and LVH independently increase SCD risk (OR 1.9, 95% CI 1.1 – 3.2 for severe LVSD; OR 1.8, 95% CI 1.1 – 2.9 for LVH). When present together, the risks of LVSD and LVH for SCD add up (OR 3.5, 95% CI 1.7 – 7.2).[55]

LVH has been associated with both ventricular and supraventricular arrhythmias. Patients with electrocardiographic evidence of LVH have a higher prevalence and greater complexity of ventricular premature beats and more serious arrhythmias than patients without LVH or normotensive subjects.[40,43] The frequency and complexity of ventricular premature beats (VPBs) is related to the severity of LVH as well as chamber volume and indices of left ventricular contractility. For every 1 mm increase in wall thickness, there was a two to three fold increase in the occurrence and complexity of VPBs.[59, 63]

In addition to the occurrence of spontaneous ventricular arrhythmia, LVH is associated with an increased ability to induce sustained ventricular tachyarrhythmia, particularly in association with myocardial ischemia, supraventricular tachycardia, and atrial fibrillation.[59,63]

High Blood Pressure (HBP)/ Hypertension

Hypertension is a chronic condition that affects over 50 million people in the USA and increases in prevalence with age.[10] It is an independent risk factor for heart disease and stroke. Hypertension increases pre-load and vascular resistance which forces the heart to work harder to maintain cardiac output and minute volume. It is the most prevalent stimulus for development of LVH. Mechanical stimulation is transduced via redundant signaling pathways including integrin, angiotensin II, calcineurin, among others, into growth of myocytes and disproportionate growth of nonmyocyte cells. Improving our knowledge of these pathways may enable better therapy of hypertension and regression of LVH. Animal studies indicate that, regarding this objective, there are three classes of anti-hypertensive drugs: a) drugs with no effects on LVH and fibrosis (direct vasodilators); b) drugs with clear effects on LVH, i.e. myocytes regression diuretics, α- and β-adrenergic receptor antagonists; and c) agents with proven effects on regression of LVH and fibrosis angiotensin-converting enzyme (ACE inhibitors, AT_1 receptor antagonists, Ca^{2+} - channel-blockers, and centrally acting adrenergic agents).[7]

These findings have been confirmed in human trials which demonstrate that LVH may resolve with treatment. A 2003 meta-analysis showed that angiotensin II receptor blockers (ARBs) were the most efficient class of agents for reducing the left ventricular mass. ARBs decreased the LVM by 13%, followed by calcium-channel blockers at 11%, ACE inhibitors at 10%, diuretics at 8%, and beta-blockers at 6%. In pairwise comparison, ARBs, calcium channel blockers, and ACE inhibitors were all significantly more effective in reducing the left ventricular mass than beta-blockers.[34]

Despite all advances in knowledge about hypertension, damage it causes and treatment options, approximately 30% of adults are still unaware of their hypertension, >40% of individuals with hypertension are not on treatment, and two-thirds of hypertensive patients are not being controlled to BP levels <140/90 mmHg.[56] Data about the prevalence of hypertension among scuba divers and their awareness and compliance with treatment are not available.

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Effects of Diving on Cardiac Functions and Arrhythmogenecity

Diving involves immersion, breathing under pressure, hyperoxia, thermal effects, and psychic stress, all of which affect cardiovascular function. Immersion exposes the body to a pressure gradient which varies depending on the position of the body in the water column. Immersion while vertical causes blood pooling in the thoracic area, increased filling pressure of the heart, increased heart volumes, increase in pulmonary artery pressure, and systemic arterial pressure.[36, 57] Vasoconstriction in cold water and hyperoxia add to these effects regardless of body position and also increases afterload. Adaptations in healthy subjects include increased stroke volume and slower heart rate, with homeostasis established quickly after immersion. Subjects with cardiovascular disease or excessive vasoconstriction in response to cold and hyperoxia, may develop heart failure and immersion pulmonary edema due to increased preload and systemic vasoconstriction.[6,29,62]

Effects of immersion on autonomic control of the cardiovascular system depend on water temperature. During thermo-neutral head-out water immersion, sympathetic activity both cardiac and vascular is decreased and cardiac parasympathetic activity predominates. In contrast, immersion in slightly cold water elicits sympathetic vascular and parasympathetic cardiac hyperactivity.[48, 46] Autonomic dysfunction often presents in older subjects and in diabetics and may increase risk for arrhythmia.[30]

While diving may provide an extensive list of possible triggers of arrhythmias, they are rare in healthy subjects. Subjects with impaired cardiac function, the substrate with the highest risk for SCD, are disqualified from diving. However, subjects with hypertension, LVH, mild cardiac dysfunction, diabetes and mild to extensive atherosclerotic changes may be quite common among divers since there are no screening requirements for asymptomatic divers.

Possible effects of immersion and hyperoxia on cardiac functions in healthy subjects have shown small negative reversible effects[51] but effected divers with risk markers for SCD have not been systematically studied. Possible effects of diving on arrhythmogenic propensity of LVH have not been studied.

Prevention of SCD in Scuba Divers

The existing health requirements for recreational scuba divers are intended to exclude subjects with overt cardiac disease and those at high risk for SCD.[45] Maximal preventive impact will occur when asymptomatic individuals or those with mild disease like hypertension, LVH and early diabetes can undergo reliable screening for SCD markers within reasonable cost/benefit parameters. If the incidence of SCD in diving is similar to the incidence in the general population, general preventive strategies in reducing cardiovascular risks which have proven successful may suffice in scuba diving too.[19] However, if triggers encountered in diving additionally increase the risk of SCD, which may be the case,[17] additional risk stratification is necessary to reduce surplus deaths within the ageing diving community. Specifically, if the propensity of LVH associated with hypertension increases in diving conditions, stricter control of divers with hypertension should be contemplated and proper medications used to revert LVH and reduce SCD risk. This question may be answered by further studies.

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Dr. Petar Denoble, MD, DSc, is a senior director of the medical research department at Divers Alert Network. After graduating from medical school, Petar joined the Navy in the former Yugoslavia and specialized in naval and diving medicine. For 13 years, he was involved with training, supervision, and treatment of divers in open circuit, closed circuit, deep bounce, and saturation diving. He did his doctoral thesis on oxygen consumption in underwater swimming. For the last 20 years, he has been with Divers Alert Network. His work at DAN has involved him in the development of the largest database of exposure and outcomes in recreational diving, the monitoring of diving injuries, and the study, treatment, and prevention of fatal outcomes and long-term consequences of diving accidents.

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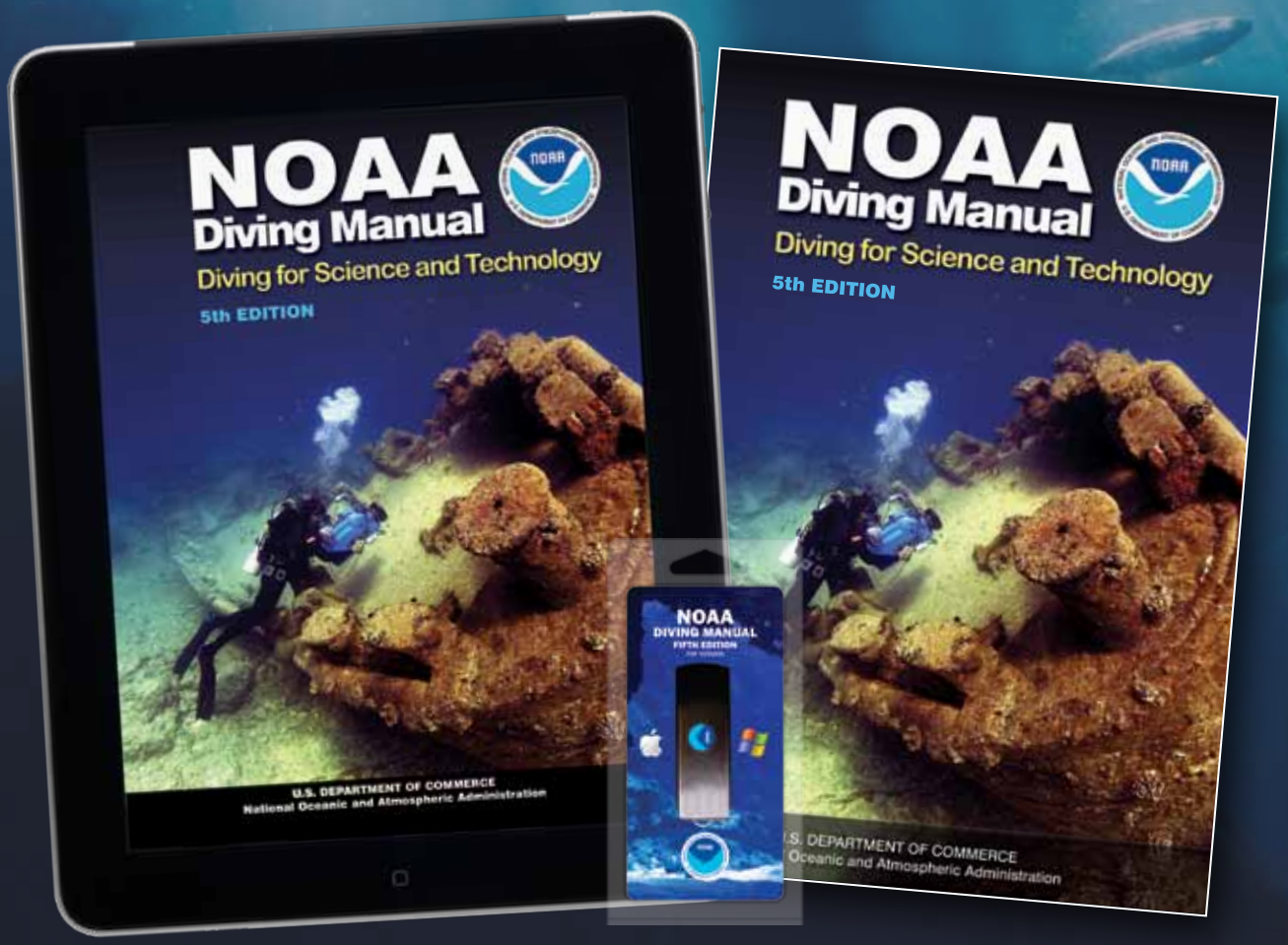

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