Incidence of cardiac arrhythmias and left ventricular hypertrophy in recreational scuba divers

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Abstract
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Introduction: The aims of this study were to investigate the potential impact of age, sex and body mass index (BMI) upon the incidence of arrhythmias pre- and post- diving, and to identify the prevalence of left ventricular hypertrophy (LVH) in older recreational divers.
Methods: Divers aged ≥ 40 years participating in group dive trips had ECG rhythm and echocardiograph recordings before and after diving. Arrhythmias were confirmed by an experienced human reader. LVH was identified by two-dimensional echocardiography. Weighted (0.5 fractional) values were used to account for participation by seven divers in 14 trips.
Results: Seventy-seven divers undertook 84 dive trips and recorded 677 dives. Among divers with no pre-trip arrhythmias (n = 55), we observed that 6.5 (12%) recorded post-trip arrhythmias and the median increase was 1.0 arrhythmia. In divers with pre-trip arrhythmias, 14.5 had a median of 1.0 fewer post-trip arrhythmias, 2.0 had no change and 5.5 had a median of 16.0 greater. Age, but neither sex nor BMI, was associated with change in the number of arrhythmias before and after dive trips (P = 0.02). The relative risk for experiencing a change in the frequency of arrhythmias after a diver trip, was 2.1 for each additional 10 years of age (95% CI 1.1, 4.0). Of the 60 divers with imaging of their heart, five had left ventricular hypertrophy.
Conclusions: We observed a higher than expected prevalence of arrhythmias. Divers with pre-trip arrhythmias tended to be older than divers without pre-trip arrhythmias (P = 0.02). The prevalence of LVH in our cohort was one quarter of that found post-mortem in scuba fatalities.

Introduction

Sudden cardiac death (SCD) is one of the most common causes of scuba fatalities, accounting for 20–30% of all cases.[1–3] SCD is an unexpected natural death from a cardiac cause within one hour of the onset of acute symptoms in a person with no prior acute condition that would appear fatal.[3] The most common suspected mechanism of SCD is acute arrhythmia triggering cardiac arrest, and the incidence of SCD increases with age, both in the general population and in scuba divers.[1,4] SCD is now more commonly suspected in recreational diving fatalities than even just two decades ago, and recreational diving fatalities also appear to be increasing with both age and body mass index (BMI).[5]

SCD is poorly understood, and there may be contributing factors associated with scuba diving. Known risk factors for SCD in the general population include a history of coronary heart disease, male sex, cigarette smoking, hypertension, diabetes mellitus, hypercholesterolaemia, obesity and left ventricular hypertrophy (LVH).[4,6–8] LVH is strongly associated with age, high systolic blood pressure and obesity.[6,9] Prevalence of LVH varies between populations. In Norway, for example, among 126 control subjects with no history of either inflammatory joint disease or cardiovascular disease, seven (6%) had LVH and 27 (21%) concentric left ventricular geometry (concentric LVH or concentric remodelling).[10] In 100 consecutive North American scuba diver autopsy reports, LVH was identified in 31% of the divers, whereas in a similar age-sex control group of autopsies from traffic fatalities in San Diego County that occurred over the same period (2007–11), prevalence of LVH was 20% (P = 0.042).[11]

Compared with movement on land, movement underwater exacts additional demand for oxygen and, consequently, both

stroke volume and heart rate increase.[12,13] This occurs while immersion itself causes blood shift from the cardiovascular periphery to the thoracic cavity, placing further stress on the cardiovascular system.[14] These stresses may alter the incidence of arrhythmias in divers and could subsequently be provoking factors for SCD. An earlier study showed arrhythmias in young scuba divers.[15]

The aims of this study were to investigate the potential impact of age, sex and BMI on the incidence of arrhythmias pre- and post-diving, and identify the prevalence of LVH in older recreational divers.

Methods

Prior to the study commencing, human research ethics approval was obtained from the Divers Alert Network Institutional Review Board. Divers aged ≥ 40 years participating in group dive trips were recruited and signed informed consent was obtained. A medical history questionnaire was completed and divers with medical contraindications for diving were excluded from further participation. The study involved six dive trips over seven years (2013–2019).

Prior to dive trips, each subject's blood pressure was recorded in both arms using either a mechanical manometer and stethoscope or an electronic blood pressure monitor (model BP761N, Omron Healthcare Co. Ltd, Muko, Kyoto, Japan) and averaged. Before and after dive trips, a 12-lead electrocardiogram (ECG) and rhythm recordings for either 300 or 360 seconds, depending on the PC-based system used (PC ECG, Midmark IQecg or IMED Cardiax) and echocardiographic measurements were collected (General Electric Vingmed Ultrasound).

Divers rested in a supine position for a few minutes until heart rates stabilised. Then baseline conventional 10 second 12-lead ECGs plus the 5–6 minute rhythm recordings were conducted. Arrhythmias were identified by the various ECG systems' automated interpretation software and confirmed or corrected by an experienced human reader (JG), then categorised by type and frequencies of arrhythmias counted. Arrhythmias were classified as premature ventricular contraction (PVC), premature atrial contraction (PAC) or 'other' (other premature ectopic beats including the less-common premature junctional contraction (PJC) and some with ambiguous origins). They were scaled to a 300 second standard and summed to give a total number pre- and post-dive trip. The numbers of arrhythmias mentioned further in this article are the total numbers of PVC, PAC or 'other' per five minutes ECG recording time. Left ventricular hypertrophy (LVH) was identified by echocardiography.

The M-mode study was performed under two-dimensional control using commercially available Vivid Q-7 (GE Healthcare, Chicago, USA). End-diastolic and end systolic measurements were taken by an experienced technician with the patient in partial left lateral decubitus according to the American Society of Echocardiography recommendations.[16] Frames with optimal visualisation of interfaces and showing simultaneous visualisation of septum, left ventricular internal diameter (LVID) and posterior wall were used for reading. Measurements were made on the screen using callipers. A long-axis parasternal approach was first examined to check perpendicularity of the ultrasonic beam with respect to the septum. Then, the short-axis approach was used to take left ventricle (LV) diastolic and systolic measurements (the average of three consecutive cycles on the best single reading set was considered). The LV mass (LVM) was calculated using Equation 1.[17]

$$LVM\,(g) = 0.80 \times (1.04 \times [(\text{septal thickness} + \text{LVID diastolic} + \text{posterior wall thickness})^3 - (\text{LVID diastolic})^3]) + 0.6 \quad \text{(Eq. 1)}$$

Left ventricular mass index (LVMI) was calculated by dividing LVM by body surface area (BSA) which was calculated using equations 2 and 3 (where W = weight in kg and H = height in cm).[18]

$$\text{Women BSA} = 0.000975482 \times W^{0.46} \times H^{1.08} \quad \text{(Eq. 2)}$$

$$\text{Men BSA} = 0.000579479 \times W^{0.38} \times H^{1.24} \quad \text{(Eq. 3)}$$

The relative wall thickness (RWT) was calculated using equation 4.[19]

$$RWT = (2 \times \text{posterior wall thickness}) / \text{LVID diastolic} \quad \text{(Eq. 4)}$$

LVH was established based on published LVMI cut-off values.[20] Male subjects with LVMI ≥ 125g·m^{-2} were classified as LVH and female subjects with LVMI ≥ 110 g·m^{-2} were classified as LVH. Geometry of LVH was classified according to published threshold values.[21] Subjects with No LVH and an RWT < 0.43 were classified as normal (N). Subjects with no LVH and an RWT ≥ 0.43 were classified as exhibiting concentric remodelling (CR). Subjects with LVH and an RWT < 0.43 were classified as eccentric hypertrophy (EH). Subjects with LVH and an RWT ≥ 0.43 were classified as concentric hypertrophy (CH).
Table 1 displays these classifications.

Sensus Ultra dive loggers (Reefnet, Mississauga, Canada) were worn by most of the divers (n = 59 of 84 diver-trips, 70%) with a default sampling rate of one record per 10 seconds. These loggers recorded dive duration, water temperature and estimated depth based on recorded water pressure. Water temperature and dive depths were weighted by dive duration to calculate overall means. All dives were made with open-circuit equipment using compressed air or nitrox.

Table 1

Classification table showing left ventricular mass index (LVMI) cut off values used to establish left ventricular hypertrophy (LVH), and relative wall thickness (RWT) cut off values to define left ventricular geometry. CH – concentric hypertrophy; CR – concentric remodeling; EH – eccentric hypertrophy; LVH – left ventricular hypertrophy; LVMI – left ventricular mass index; N – normal

Parameter	Subject sex	Threshold $g \cdot m^{-2}$	Classification	RWT	
				< 0.43	≥ 0.43
LVMI	male	≥ 125	LVH	EH	CH
		< 125	No LVH	N	CR
	female	≥ 110	LVH	EH	CH
		< 110	No LVH	N	CR

Data were stored in MS® Excel and imported into SAS (SAS, Cary, NC) version 9.4 for analysis. Skewness and kurtosis were measured for quantitative variables (e.g., age and BMI), histograms were plotted and normality was tested using the Shapiro-Wilk test. Means and standard deviations are reported for quantitative variables with Gaussian distributions, and medians with interquartile ranges (IQR) for non-parametrically distributed data. Range is reported in place of IQR when $n < 4$.

Differences between the number of pre-dive and post-dive arrhythmias (dA) were classed as less, none, or more. For regression analysis, data from the 14 dive trips made by seven divers who attended two dive trips each were given a weighting of 0.5 and the other 70 single dive trip participants were given a weighting of 1.0. The weighted ternary outcomes (dA`) were tested for association with age, sex and BMI in a weighted multivariate logistic regression model, stratified by dive trip (Trip). The model was optimised by backwards elimination according to the hierarchical principle, with non-significant interactions removed first. Significance was accepted at $P < 0.05$. The initial model is shown in Equation 5.

$$\mathrm{Ln}[P(dA`_i)/[1 - P(dA`_i)]] = \alpha_j + \beta_1 Sex_i + \beta_2 Age_i + \beta_3 BMI_i + \beta_4 Sex_i * Age_i + \beta_5 BMI_i * Age_i + \beta_6 Sex_i * BMI_i + \beta_7 Sex_i * Age_i * BMI_i + \beta_8 Trip_i \quad \text{(Eq. 5)}$$

Where α_i = the intercept for outcome j, $\beta_{1\text{-}8}$ are the respective estimates for each independent variable for each participant i, Sex = male (0) or female (1), Age is in whole years, BMI is in $kg.m^{-2}$ and Trip is the individual group dive trip (1–6). Deviance and Pearson Goodness of Fit Tests were performed to assess if expected outcomes significantly differed from observed outcomes. $P \leq 0.05$ was accepted as significant when deciding whether to reject the null hypothesis that there was no association between an ordinal increase in the number of arrhythmias between pre- and post-dive trip ECG recordings and either age, sex or BMI.

To test if any change in arrhythmias was associated with divers who recorded pre-dive arrhythmias, a binary outcome variable (Change, 0 or 1) was fitted to the optimised model described above. A Wilcoxon signed-rank test was used to assess differences in number of post-trip arrhythmias, among divers who had recorded pre-trip arrhythmias.

Because the study design was a prospective cohort study, not a case-control design, adjusted odds ratios (OR) generated by the logistic regression were converted to adjusted relative risks (RR) using Equation 6 and contingency Table 2. The 95% confidence intervals for the RR were calculated by substituting the respective OR for the 95% confidence interval OR generated by the regression. P_c is the unadjusted risk in the control group (pre-trip, where arrhythmias = no).

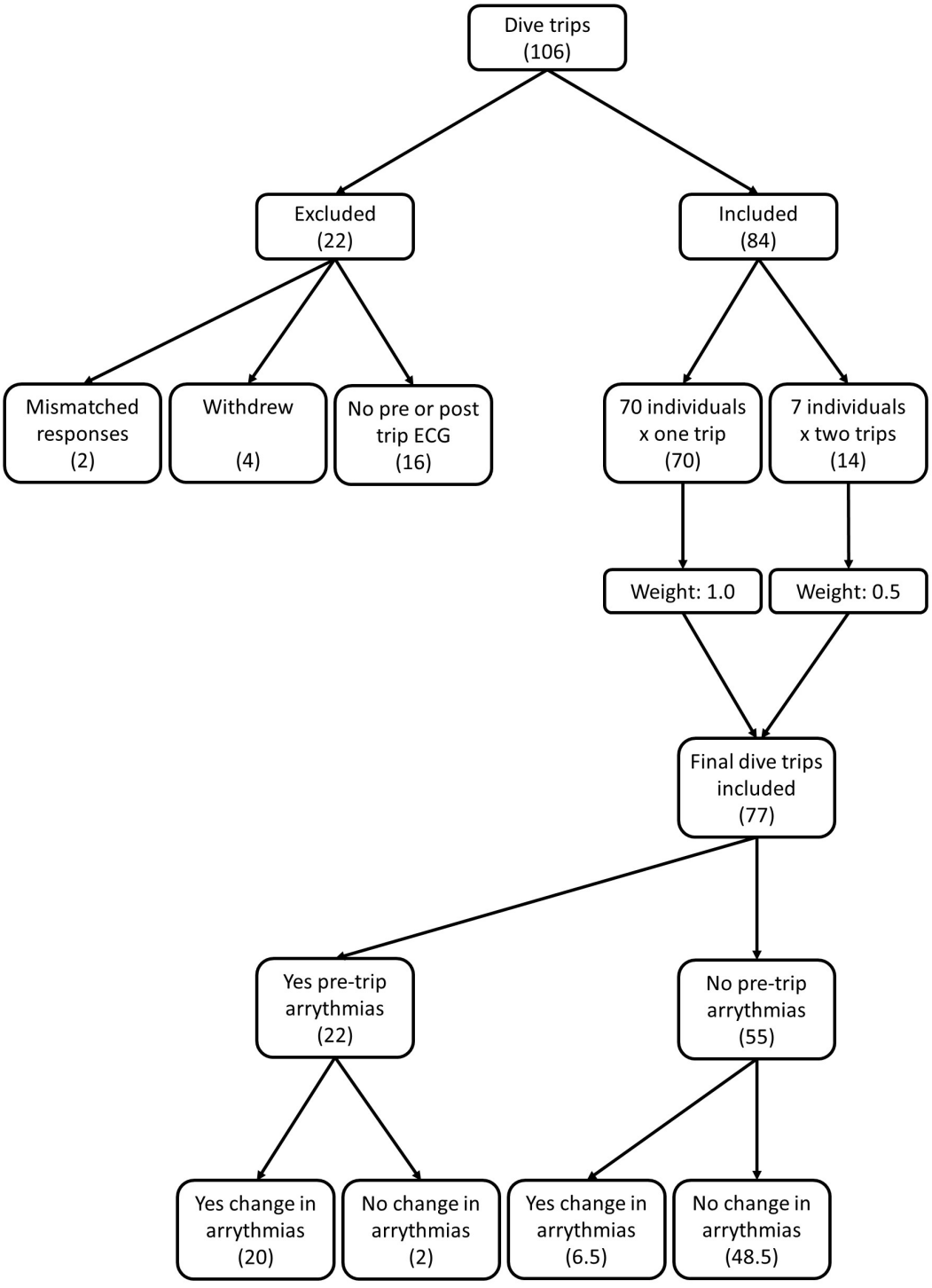
$$RR = \frac{OR}{(1-P_c)+(P_c.OR)} \quad \text{(Eq.6)}$$

Results

There were 106 diver trips recorded by eligible divers. Of those, 22 diver trips were excluded from the analysis, (after four withdrew, one diver gave mismatched responses on two separate trips and 16 had either pre-trip or post-trip ECG recorded, or neither, but not both). The final dataset for analysis consisted of 84 diver trips made by 77 individual divers (seven divers each made two trips). Dive loggers were worn during 59/84 diver trips (70%), recording a total of 677 dives (Figure 1), a mean of 11.5 dives per recorded diver trip (SD 9.2).

Mean age at the start of each trip was 53 (SD 9) years in females (n = 30, 36%) and 59 (9) years in males (n = 54, 64%). Mean body mass index (BMI) was 27 (4) $kg.m^{-2}$ in females and 29 (5) $kg.m^{-2}$ in males. Thirty divers (39%) reported being past smokers, having smoked for between 2–30 years, but only one diver reported being a current smoker. Sixty-seven (87%) self-reported consuming alcoholic drinks. Six divers (8%) reported a family history of heart disease, 26 (34%) had been diagnosed with high cholesterol of whom 16 (21%) were taking medication for it; 25 (32%) had been diagnosed with high blood pressure and 19 (25%) were prescribed blood pressure medication. Three subjects (4%) had a history of previous myocardial infarction and one of those subjects had undergone previous cardiac surgery. Two (3%) subjects reported a history of

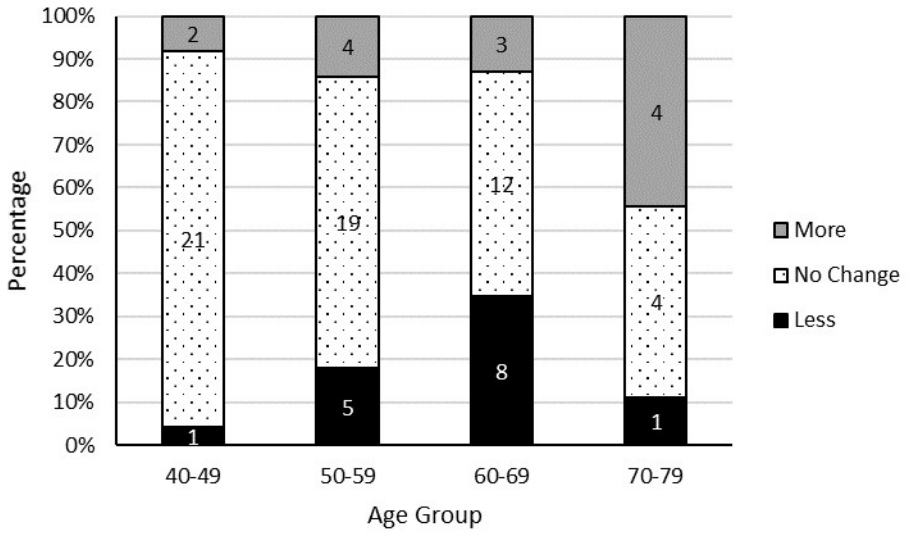
Figure 1
Selection process for inclusion of subjects and data



cardiomyopathy and two subjects (3%) reported exercise induced shortness of breath. A total of 39 (51%) reported currently taking any prescription medication. Mean blood pressure before diving trips was 129/82 mmHg. Mean heart rate prior to diving was 68 beats per minute (bpm) and post-diving it was 72 bpm.

With regards to diving experience, the divers reported a median lifetime experience of 300 dives (IQR 484), diving for a median of 20 years (IQR 29) and having made a median of 12 dives (IQR 27) during the previous six months. The median number of dives made during each trip was 11 (IQR 17), with a median total bottom time of 9 hours (IQR 17). Weighted mean depth was 8.6 metres of seawater

Figure 2
Changes in detected arrhythmias between pre- and post-dive trips, by age group, with *n* labelled



(msw) (SD 4.1), median maximum depth 23 msw (IQR 25) and weighted mean water temperature 21°C (SD 8, range 8–28°C). Median maximum ascent rate during each trip was 9.7 m.min^{-1} (IQR 4.0).

Conventional 10 s 12-lead ECGs from the divers showed similar arrhythmias to the longer rhythm recordings for those divers who presented arrhythmias. The conventional 10 s 12-lead ECGs were otherwise predominantly normal. Only one of the many 12-lead ECGs was automatically interpreted as LVH by machine algorithm, confirmed by the human reader (JG).

Fitting the data to the model shown in Equation 5, the initially optimised model following backwards elimination is shown in Equation 7, (without coefficients).

$$\mathrm{Ln}[P(dA`_i)/[1 - P(dA`_i)]] = \alpha_j + \beta_2 Age_i + \beta_8 Trip_i \quad \text{(Eq. 7)}$$

After adjusting for stratification of the data by dive trip, age (but neither sex nor BMI) was associated with change in the number of arrhythmias recorded before and after dive trips (R^2 = 0.27, P = 0.021). Differences between pre- and post-dive trips in the number of arrhythmias detected over 5 min are shown by age group in Figure 2.

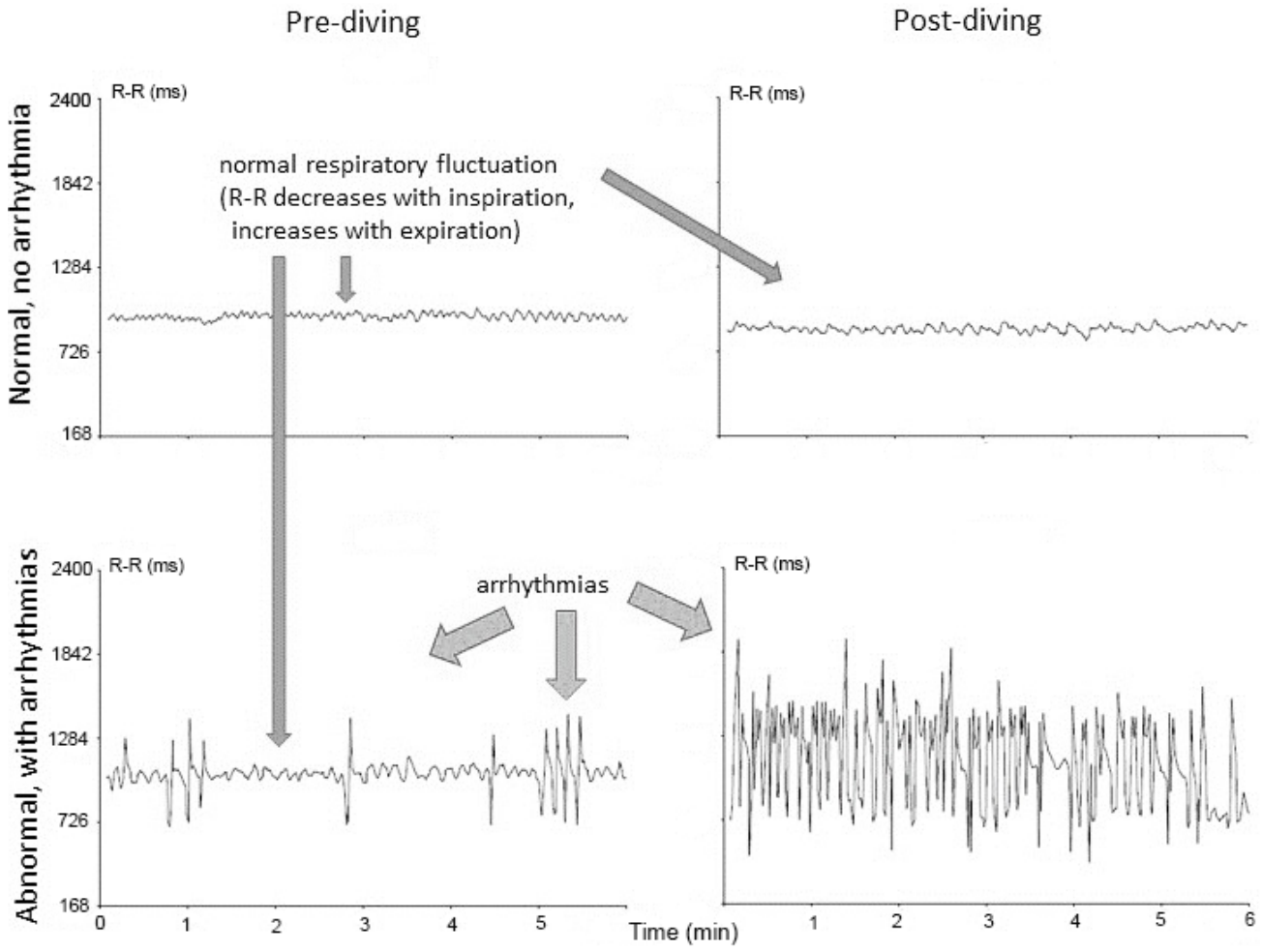
There were 23 diver trips made by 22 divers where the diver recorded arrhythmias before the diving commenced (Table 2). Of the 22 divers with pre-trip arrhythmias, 10 showed single PVCs (median 2, IQR 2) , eight showed single PACs (median 3, IQR 20.5), and nine showed "*other*" (unspecified) arrhythmias. These were the only divers who were able to record fewer arrhythmias after their dive trips, since the others each had no pre-trip arrhythmias, (and one cannot have fewer than zero arrhythmias).

The median decrease in number of arrhythmias (among n = 14.5 divers who recorded fewer post-trip arrhythmias) was 1.0 (IQR 3.0) while the median post-trip increase in arrhythmias among divers (n = 5.5) who had recorded pre-trip arrhythmias was 16.0 (IQR 17.0, P = 0.0003). Figure 3 illustrates exemplar pre- and post- diving beat-to-beat ECG recordings both for a diver without pre-trip arrhythmias and a diver with pre-trip arrhythmias. Among divers with no pre-trip arrhythmias (n = 55), we observed that 6.5 (12%) recorded post-trip arrhythmias and the median increase was 1.0 (IQR 7.0). Of the 55 divers without pre-dive arrhythmias, 6.5 showed arrhythmias post-dive, namely 3 PVCs (median 1, range 26), 2 PACs (median 3, range 4) and 3 'other' (unspecified). Of the 22 divers with arrhythmias pre-dive, 14.5 had less arrhythmias post-dive: 4 PVCs (median 1.5, IQR 9.5); 3 PACs (median 1, range 87); 2 "other" (unspecified); and 8.5 none. In contrast, 5.5 had

Table 2
Contingency table used to convert the adjusted OR into RR, (with weighting for number of trips), showing number of divers and number of dive trips

Divers		**Change in arrhythmias**		
		Yes	**No**	**Total**
Pre-Trip Arrhythmias	Yes	20	2	22
	No	6.5	48.5	55
	Total	26.5	50.5	77
Diver trips		**Change in arrhythmias**		
		Yes	**No**	**Total**
Pre-Trip Arrhythmias	Yes	21	2	23
	No	7	54	61
	Total	28	56	84

Figure 3
Examples of beat-to-beat (R-R) interval (milliseconds [ms]) trends of divers before versus after dive trips, 360 sec (6 min) recordings



more arrhythmias: 2 PVCs (median 29, range 56); 2 PACs (median 30.5, range 47); and 3 'other' (unspecified). In two divers, the number of arrhythmias pre- and post-dive was unchanged (both 'other' (unspecified)).

Of the 77 divers, $n = 14$ (18%) recorded PVCs and of those, six recorded a post-trip decrease in the number of PVCs, seven recorded an increase and one recorded the same number of post-trip PVC over 300 seconds as they did pre-trip. Using a prior diagnosis of high blood pressure as a simple proxy for cardiovascular risk, there was no significant difference in the prevalence of high blood pressure between those with increased arrhythmias (4/24) and those without (8/52).

In the penultimate analyses divers ($n = 48.5$) who had no arrhythmias either before or after dive trips ($n = 56$) were compared with divers ($n = 28.5$) in whom the number of arrhythmias either increased or decreased following dive trips, shown in Table 2. Hosmer and Lemeshow goodness of fit test Chi-square $P = 0.32$, $R^2 = 0.25$ and age was again associated with change in the number of arrhythmias recorded before and after dive trips ($P = 0.016$), (after adjusting for stratification by dive trip). Mean age among the 28.5 divers who recorded a change in the frequency of arrhythmias after their dive trips was 61.8 years (SD 8.2), a mean of 7.3 years older than the divers who experienced no arrhythmias either before or after diving. Compared with divers who recorded no arrhythmias either before or after dive trips, the OR for experiencing a change in the frequency of arrhythmias after a dive trip (either more, or less), was 2.7 for each additional 10 years of age (95% CI 1.2, 5.9), and 2.0 for each additional 7 years of age (95% CI 1.1, 3.5). Using Equation 2 and the values shown in Table 2, these ORs were converted to RR using Pc = (6.5/55). Compared with divers who recorded no arrhythmias either before or after dive trips, the RR for experiencing a change in the frequency of arrhythmias after a dive trip (either more, or less), was 2.1 for each additional 10 years of age (95% CI 1.1, 4.0), and 1.7 for each additional 7 years of age (95% CI 1.1, 2.7).

Finally, two-dimensional echocardiography imaging of the heart was available for 60 of the 77 (78%) divers. Of those, five divers (8%) had left ventricular hypertrophy identified and 36 (60%) had abnormal left ventricular geometry detected ($n = 33$, 55% with concentric remodelling and

n = 3, 5% with concentric hypertrophy). Of the 60 divers with heart imaging, 40 reported no history of high blood pressure and 20 reported a prior diagnosis of high blood pressure. Within those sub-groups, there were 2/40 (5%) and 3/20 (15%) with LVH respectively.

Discussion

In this prospective study, recreational divers who experienced a change in the frequency of recorded arrhythmias after a dive trip, compared with before the trip, were older by a mean of 7 years ($P = 0.021$). Furthermore, the RR for experiencing a change in the frequency of recorded arrhythmias associated with a mean of seven additional years of age was 1.7. The clinical significance of this observation is for now unclear, and warrants further investigation. Divers who recorded at least one pre-trip arrhythmia also appeared to record more freequent post-trip arrhythmias than divers who did not record a pre-trip arrhythmia (6/23 diver trips vs. 7/61 diver trips, median 16.0 more vs. 1.0 more respectively, $P = 0.0003$).

Arrhythmias were observed in 28.5 of 77 divers (37%). This likely represents a higher frequency than would be observed for the general public in this age group under comparable measurement conditions aside from diving. Lindberg et al.[22] found a much lower prevalence (13%) of arrhythmias in a large elderly population (mean age 74 y) in Sweden. A UK study of half a million community-dwelling middle-aged to elderly adults (mean age 58 y) found an even lower 2% prevalence of baseline abnormal rhythms.[23] Differences between study design and population samples may account, at least in part, for these lower prevalences than found in the present study.

The limitations of this study include that age is a confounder for arrhythmias, and the age of those with arrhythmias was older than the age of the divers without arrhythmias. Stratum-specific risk ratios for divers aged < 58 vs. ≥ 58 years might uncover the scale of any potential confounding by age but the sample size in this study is too small for that sub-analysis. Also, among the divers with pre-trip arrhythmias, it is not certain how much of an influence diving had on the observed changes, or if other factors played a greater role (exercise, alcohol consumption, etc). The sampled population was non-random (divers on pre-organised trips), almost entirely Caucasian from the USA, and may not represent recreational divers in this age group in general. There was wide variation among divers including age and other demographic characteristics, physical and medical conditions, diver histories, and recent diving activities. There was also wide variation among dive trips including depths of dives, water temperatures (and types of protective suit), lengths of trips (from one or two to seven days), and whether live-aboard or shore-based (all trips were in salt water). Furthermore, there was variation, evolution, and refinement over the seven-year period of this study in our methodology and protocols, including equipment used, technician experience, and such factors as elapsed time between the last dive of a trip and when post-diving recordings were made.

Some arrhythmias such as atrial fibrillation were suspected to have occurred in divers during these trips but, by chance, were not captured during our relatively brief periods of measurement. Longer (hours) and more frequent (including at night) periods of measurement, such as with Holter recorders or even long-recording heart rate monitors such as those commonly used by runners and cyclists, would greatly enhance the ability to detect arrhythmias which are highly variable and erratic in their occurrence. We recommend longer recordings in future studies.

Abnormal non-respiratory or non-phasic sinus arrhythmia (nrSA), as opposed to normal respiratory arrhythmia, occurred in several of the divers. Non-respiratory SA is an arrhythmia of interest and potential issue of concern for diving. However, it is quite variable; it is not well studied or understood; its frequency and measurable characteristics are affected by heart rate; and the definitive identification of nrSA is not well standardised and can be problematic in some cases. Furthermore, quantification of nrSA also is not standardised and is not compatible with counts of the other arrhythmias. Hence, we excluded nrSA from the present analyses.

A potential confounding factor in our study is exercise-induced arrhythmias, including structural differences of the heart in athletes vs. non-athletes who experience arrhythmias.[24] Our methods did not permit direct assessment of the exercise factor *per se*. However, swimming, breath-hold diving, scuba diving, and even simple face immersion have long been known associated with increased arrhythmias.[15,25,26]

Risks for mortality and morbidity from arrhythmias are highly variable and dependent on numerous factors including the kind of arrhythmia and an individual's associated heart conditions. Calculating the actual risks of PVCs leading to more serious problems including death have been problematic and appear to vary among populations.[27,28] The strengths of this study include the prospective cohort design, though further studies of a more controlled, less exploratory nature are warranted. In particular, the effect of diving upon the incidence of PVCs remains to be quantified. We have developed and refined protocols that could support such studies.

Of additional concern is the pre-hypertension mean blood pressure recorded before diving, that half the cohort were currently taking prescribed medication, and that most of the cohort (86%) reported regularly consuming alcohol. One third had been diagnosed with high blood pressure at some time and one quarter were currently taking medication for this. We also noted three times as many cases of LVH (15%) in the divers with high blood pressure, compared with 5%

LVH among divers with no history of high blood pressure, though the number of LVH overall (n = 5) was too small from which to draw firm inferences. Despite these concerns, these were apparently relatively active divers, having made a median of 12 dives during the previous six months.

Compared with a case-control study, the prevalence of LVH in the present study of active divers was 8%, similar to that observed in a Norwegian control group,[10] but far lower than the 31% detected in 100 consecutive recreational scuba diver autopsies.[11] This supports the concern that LVH may be an important contributor to diving fatalities. It may also prove important to clarify the relationship between LVH and changes in arrhythmias after dive trips. For the moment, the evidence for this potential association is limited and the role of this potential risk factor remains to be confirmed. It should be noted that our LVH data were derived from two-dimensional echocardiography imaging. Although we originally expected the conventional 12-lead ECGs to provide LVH data, that did not occur. Conventional 12-lead ECGs are, in fact, notoriously poor for interpreting LVH.[29,30] Advanced ECG and a new approach that considers left ventricular electrical remodelling have proven much better for detecting LVH and related conditions, and should be considered in future studies.[30]

Insights from this study and future research may help provide recommendations to divers and potential divers for participating in diving, particularly as they age, similar to recent recommendations for non-diving activities.[31]

Conclusions

Among this cohort of active, older recreational divers with pre-existing risk factors for SCD:
a) A higher than expected prevalence of arrhythmias was observed;
b) divers with pre-trip arrhythmias tended to be older than divers without pre-trip arrhythmias;
c) in the unweighted sample, one-in-nine divers (n = 7 out of 61) with no pre-trip arrhythmias recorded post-trip arrhythmias;
d) compared with pre-trip no-arrhythmia divers, divers with pre-trip arrhythmias showed increased post-trip arrhythmias, which was related to age, but not to BMI or sex;
e) compared with pre-trip no-arrhythmia divers, divers with pre-trip arrhythmias were at elevated risk for changes (up or down) in the frequency counts of arrhythmias over 300 seconds post-trip;
f) the prevalence of LVH in this cohort was one quarter of that found in 100 recreational scuba diving autopsies, suggesting the possibility LVH may be associated with increased risk of mortality while scuba diving; and
g) these results provide a step toward making recommendations to older and arrhythmia-prone persons for participating in scuba diving.

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