Immersion Pulmonary Edema and Comorbidities: Case Series and Updated Review

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CLINICAL SCIENCES

ABSTRACT

PEACHER, D. F., S. D. MARTINA, C. E. OTTENI, T. E. WESTER, J. F. POTTER, and R. E. MOON. Immersion Pulmonary Edema and Comorbidities: Case Series and Updated Review. *Med. Sci. Sports Exerc.*, Vol. 47, No. 6, pp. 1128–1134, 2015. **Purpose**: Immersion pulmonary edema (IPE) occurs in swimmers (especially triathletes) and scuba divers. Its pathophysiology and risk factors are incompletely understood. This study was designed to establish the prevalence of preexisting comorbidities in individuals who experience IPE. **Methods**: From 2008 to May 2010, individuals who had experienced IPE were identified via recruitment for a physiological study. Past medical history and subject characteristics were compared with those available in the current body of literature. **Results**: At Duke University Medical Center, Durham, NC, 36 subjects were identified (mean age = 50.11 ± 10.8 yr), of whom 72.2% had one or more significant medical conditions at the time of IPE incident (e.g., hypertension, cardiac dysrhythmias or structural abnormality or dysfunction, asthma, diabetes mellitus, overweight or obesity, obstructive sleep apnea, hypothyroidism). Forty-five articles were included, containing 292 cases of IPE, of which 24.0% had identifiable cardiopulmonary risk factors. Within the recreational population, cases with identifiable risk factors comprised 44.9%. Mean age was 47.8 ± 11.3 yr in recreational divers/swimmers and 23.3 ± 6.4 yr in military divers/swimmers. **Conclusions**: Cardiopulmonary disease may be a common predisposing factor in IPE in the recreational swimming/diving population, whereas pulmonary hypertension due to extreme exertion may be more important in military cases. Individuals with past history of IPE in our case series had a greater proportion of comorbidities compared to published cases. The role of underlying cardiopulmonary dysfunction may be underestimated, especially in older swimmers and divers. We conclude that an episode of IPE should prompt the evaluation of cardiac and pulmonary function. **Key Words:** EXERCISE, SWIMMING, DIVING, LEFT VENTRICULAR HYPERTROPHY, MYOCARDIAL ISCHEMIA

Immersion pulmonary edema (IPE; also termed swimming-induced pulmonary edema) occurs in swimmers and divers who are often otherwise healthy. Deaths after IPE have been reported in recreational divers (23). Since the syndrome was first described by Wilmshurst et al. (65) in 1981, there have been numerous cases reported; however, its pathophysiology and risk factors are still incompletely understood. The prevailing hypothesis is that IPE results from stress failure of pulmonary capillaries due to increased hydrostatic pressure owing to a combination of factors (40): increased intrathoracic blood volume, increased cardiac output, and, possibly, exaggerated pulmonary vasoconstriction (arterial or venous) in susceptible individuals. It has also been suggested that susceptible individuals may have a smaller-than-normal pulmonary lymphatic network and hence reduced absorptive capacity for excess lung water (10).

During immersion, there is central redistribution of blood, resulting in increased preload and higher pulmonary vascular pressures (64). This effect is augmented in cold water, where there is active venoconstriction (64). This would tend to increase the likelihood of pulmonary edema, especially in the setting of left ventricular (LV) systolic or diastolic dysfunction, valvular disease, or hypertension (67). High airway resistance could also predispose to pulmonary edema owing to the associated greater pleural pressure variation and augmented inspiratory increase in pulmonary vascular volume and increased cardiac preload (57). Obesity may also be a risk factor because of its link with obstructive sleep apnea, which, in turn, is associated with diabetes, metabolic syndrome, and pulmonary hypertension (58).

A typical case of IPE begins during diving or swimming and is characterized by an acute onset of dyspnea, cough, expectoration of frothy sputum, radiographic findings of pulmonary

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Submitted for publication July 2014.
Accepted for publication September 2014.
Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.acsm-msse.org).

0195-9131/15/4706-1128/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/MSS.0000000000000524

edema, and rapid resolution for the subsequent 24–48 h. Normal underlying cardiac and pulmonary functions are commonly reported (57). IPE occurs more commonly in cold water (37). Koehle et al. (37) found an average age of 36.1 ± 14.2 yr (mean ± SD) for all IPE cases and 47.1 ± 11 yr among scuba divers, and only 10 (17.9%) of 56 patients had preexisting medical conditions.

As the number of reported IPE incidents grows, however, patterns in susceptibility are starting to emerge. For example, scuba divers who develop IPE tend to be an older population than military swimmers and thus may be more likely to have preexisting medical conditions or yet-undiagnosed cardiac or pulmonary dysfunction (22). Heavy exertion is more likely to be a characteristic of combat and endurance swimmers than of scuba divers.

We hypothesized that the prevalence of risk factors for IPE as determined by survey has been underestimated. We review the demographic characteristics and risk factors in case reports and case series of IPE through January 2014 and report our experience on cases obtained by recruitment.

METHODS

Subjects. After institutional review, subjects with a history of IPE were identified via recruitment for a physiological study from 2008 to May 2010. Methods of recruitment included the Divers Alert Network (DAN) Alert Diver publication, the Professional Association of Diving Instructors (PADI), National Institutes of Health Clinical Trials registry (ClinicalTrials.gov), a consumer health information Web site (Healthline.com), and referral by physicians. Subjects answered by e-mail or phone correspondence a list of questions regarding diving or swimming circumstances and medical history. Most medical and IPE history was obtained by self-report; however, medical records were reviewed when provided. Subjects from this group are referred to in this article as the "Duke Group."

Literature review. PubMed/MEDLINE and EMBASE were searched using terms "swimming" or "diving" or "scuba" and "pulmonary edema." ISIS Web of Science was used to search for articles that cited selected IPE articles. Case reports and series were included if they met these criteria and exhibited manifestations reported by Adir et al. (1) or radiographically identified pulmonary edema. Non–English-language case reports were not excluded. An effort was made not to double-count cases that were likely to have been included in more than one case report or series (25,45,68). One additional article was also included, in which the case described a syndrome consistent with IPE but was not labeled as IPE or swimming-induced pulmonary edema (20). Articles related to breath-hold (apnea) diving were excluded owing to the distinct mechanism for pulmonary edema in apnea diving that does not occur during scuba diving or surface swimming (lung squeeze) (38). If published series did not specify whether more than one risk factor was present in a given individual, there was the potential for double counting. For these series, when assessing the number of individuals with risk factors, we used only the single risk factor with the largest number of instances. Cases from the literature were classified as military or recreational.

We defined predisposing conditions as risk factors as either overt cardiopulmonary or peripheral vascular disease (PVD) or risk factors for such conditions. These conditions were diagnosed before the incident or during the subsequent evaluation immediately after the incident.

Descriptive statistics are reported as mean ± SD. Comparison of age between recreational group and military group was carried out using two-tailed unpaired *t*-test. Comparison of prevalence of risk factors between the Duke series and the compiled published cases was performed using χ^2. Statistical tests were performed using JMP Pro version 11.0.0 (SAS, Cary, NC) software.

RESULTS

Duke experience. A total of 41 individuals contacted us after incidents consistent with IPE. Sex and age at the time of incident were obtained on all subjects except for three. Past medical history was available for 36 of subjects (15 by medical records and 21 by self-report). A total of five individuals were excluded from the analysis because of insufficient medical history or age/sex data. Ten subjects and their medical records were evaluated in person.

Mean ± SD age at the time of IPE incident for subjects was 50.11 ± 10.8 yr (median = 49.0 yr, range = 25–71 yr). Compared to the literature, the age of the Duke cases is similar to the recreational group, with a difference of 2.3 yr [95% confidence interval (CI) = −6.55 to 1.95, $P = 0.29$]. Of the 36 subjects with available medical history data, 9 (25%) did not have significant past medical history (defined for the purposes of this review as hypertension, pulmonary disease, overweight/obesity, diabetes, sleep apnea, hypothyroidism, or cardiac abnormalities of structure, function, rhythm, or conduction).

The most common medical conditions were overweight/obesity ($n = 12$ with BMI > 25 kg·m^{-2}, of whom 4 had BMI > 30 kg·m^{-2}) and hypertension ($n = 7$). For cardiac abnormalities, there were four cases of dysrhythmias [paroxysmal atrial fibrillation ($n = 1$), symptomatic sinus bradycardia ($n = 1$), "irregular heartbeat with history of syncope" ($n = 1$)] or conduction abnormalities [left bundle branch block ($n = 1$)], two cases of valvular abnormalities [mitral valve prolapse, ($n = 1$) "leaky valve" ($n = 1$)], two cases of abnormal cardiac function [nonischemic cardiomyopathy ($n = 1$), depressed LV ejection fraction ($n = 1$)], one case of repaired patent foramen ovale, one case of repaired atrial septal defect, one case of repaired patent ductus arteriosus in infancy, and one case of pulmonary hypertension. There were five cases of pulmonary disease: three cases of asthma [childhood asthma ($n = 2$), active asthma ($n = 1$)] and one case each of obstructive lung disease and reactive airways disease. There were three cases of hypothyroidism, two cases of diabetes mellitus, and one case of polycystic ovary syndrome. Two subjects had obstructive sleep apnea. Twelve subjects had more than one concurrent medical

TABLE 1. Preexisting risk factors at the time of immersion pulmonary edema incident from literature review.

Asthma (9,57)	4
Cardiomegaly (34)	2
Chronic atrial fibrillation (21,57)	2
Coronary artery disease (14, 23, 27)	3
Diabetes (6,27)	4
Exercise-induced cough (30)	1
Hyperlipidemia (17,27,34,61)	22
Hypertension (14,15,27,32,34,57,61,67)	25
Left ventricular hypertrophy (6,29,34)	9
Peripheral vascular disease (14)	1
Sleep apnea (27,34,61)	6

condition. Of note, at the time of the IPE incident, 3 of the 36 subjects had elevated blood levels of cardiac biomarkers. Two of the 36 subjects had discontinued oral diuretic medication shortly before their IPE incident.

Representative cases from Duke survey. ***Case A.*** A 58-yr-old male recreational diver with medical history significant for obstructive sleep apnea, hyperlipidemia, and obesity (BMI = 32 $kg \cdot m^{-2}$) was making his second dive of the day, to 30 feet of sea water (fsw), in heavy current. He experienced a sudden onset of dyspnea during the dive that worsened with time. Decompression was shortened owing to dyspnea, and on surfacing, he was noted to be coughing and expectorating bloody sputum. He was evacuated by helicopter to a nearby emergency department. He was hemodynamically stable and hypoxic (SaO_2 94%, PaO_2 69 mm Hg on 2 $L \cdot min^{-1}$ O_2). A chest radiograph showed a prominence of central pulmonary vessels suggestive of pulmonary venous hypertension. Notably, cardiac biomarkers troponin, creatine kinase MB, and creatine kinase were elevated. The patient was monitored overnight in the hospital, with serial cardiac biomarkers. As the patient was clinically improving and serum troponin levels began to decrease after 12 h, the patient was discharged for outpatient cardiology follow-up. Subsequent exercise myocardial perfusion scan revealed no evidence of ischemic heart disease, and resting echocardiogram revealed normal systolic and diastolic function and no valvular abnormality.

Case B. A 49-yr-old female with hypertension, severe obstructive sleep apnea, iron deficiency anemia, hyperlipidemia, anxiety, and obesity (BMI = 41 $kg \cdot m^{-2}$) had completed a single 23-min dive to 25 fsw on air in 11°C–15°C water. During the surface swim to return to the boat, she developed a sudden onset of dyspnea and productive cough. She was transported to a local emergency department, where chest radiograph revealed mild pulmonary edema, while EKG and laboratory studies were otherwise normal. Symptoms resolved with supplemental O_2 and intravenous diuresis.

Literature review. Forty-five articles met the criteria for inclusion with a total of 292 cases (see Table, Supplementary Digital Content 1, list of articles reviewed, http://links.lww.com/MSS/A544). There were 156 recreational divers or swimmers (53.4%) and 136 military swimmers or divers (46.6%). Of the recreational group, 67 subjects were female and 89 subjects were male. Except for one, all cases in the military group were male.

For cases reviewed for this article, average age for recreational swimmers and divers in whom ages were reported individually was (n = 106) was 47.8 ± 11.3 yr. Wilmshurst et al. (67) had reported a similar mean age (45.6 ± 2.6 yr) in their series of 11 cases, as had Gempp et al. (27) in their series of 54 cases (mean age 46 ± 13 yr). In military swimmers and divers (n = 136), all reported cases of IPE occurred in those between 18 and 47 yr old. In the two largest series (n = 91), all cases were 18–19 yr old (1,56). Average age for 11 military divers as reported by Gempp et al. (26) was 29 ± 5 yr. Mahon et al. (43) reported a series in which the age range was 22–28 yr (n = 3). Mean age in combat swimmers and divers was 23.3 ± 6.4 yr in the remainder of cases reviewed (n = 20) (36,40,41,54,55,62,69). Comparison of age between recreational and military groups in the cases in which individual ages were reported reveals significantly higher age by 24.5 yr (95% CI = 19.4–29.7) in the recreational group ($P < 0.0001$).

Of the entire cohort of 292 cases, 70 (24.9%) were reported to have significant preexisting risk factors (Table 1). Of these 70 cases, there was evidence of cardiac dysfunction in 50 cases (71.4%) defined as valvular disease, LV dysfunction, or clinical or laboratory evidence of cardiac dysfunction or injury (Table 2). When considered within the recreational subgroup only (n = 156), this higher risk group comprises nearly half (44.9%) of cases. Of the 70 with preexisting risk factors, hypertension (35.7%) (14,15,27,32,34,57,61,67), hyperlipidemia (14.3%) (17,27,34,61), and LV hypertrophy (LVH; 8.6%) (6,29,34) were the most common comorbidities. Other conditions included coronary artery disease (14,23,27), cardiomegaly (34), chronic atrial fibrillation (21,57), diabetes (6,27), exercise-induced cough (30), asthma (9,57), PVD (14), and sleep apnea (27,34,61) (Table 1). On further follow-up by Wilmshurst (67), Carter and Koehle (9), and Gempp et al. (27), additional subjects were noted to develop diagnoses of Raynaud syndrome (n = 1) (67), hypertension (n = 8) (9,27,67), and atrial fibrillation (n = 1) (67). (These latter diagnoses were not included in Table 1.) Of the

TABLE 2. Evidence of cardiac dysfunction reported in immersion pulmonary edema cases in literature review.

Valvular disease	
Mitral valve prolapse (34)	1
Mitral regurgitation (6,34)	4
Ruptured chordae tendineae (34)	1
Tricuspid regurgitation (34)	1
Bicuspid aortic valve with trivial insufficiency (29)	1
Left ventricular dysfunction	
Ventricular hypokinesis (5,11,14,20,27,35,60)	21
Alcoholic cardiomyopathy (35)	1
Takotsubo cardiomyopathy (5,11,14,20)	4
Hypotension (11,14,16,17,20,31,32,35,66)	8
Cardiac arrest (14,16,23,34)	6
Dysrhythmia (extrasystoles) (34)	1
Jugular venous distension (29,34)	2
EKG ischemic changes (5,6,11,14,16,20,27,34,35,66)	21
Elevated cardiac biomarkers (troponin, creatinine kinase) (5–7,11,14,16,18,20,27,34,60)	28
Elevated pulmonary artery pressure (echocardiographic) (60)	1

Because more than one condition may exist in the same patient, these numbers do not add up to the number of patients.

50 IPE cases found to have evidence of cardiac dysfunction, valvular disease was found in 6 cases (6,29,34) and LV dysfunction was found in 26 cases (5,11,14,20,27,35,60) (Table 2). The mean age of those with risk factors for coronary artery disease (hypertension, hypercholesterolemia, or smoking history) was 53.0 ± 6.8 yr versus 45.7 ± 12.0 yr in those without risk factors ($P = 0.0001$).

The percentage of cases with possible predisposing factors in the Duke experience (72.2%) was significantly greater than both the literature as a whole (24%, $P = 1.9 \times 10^{-9}$) and the subset of civilian cases (44.9%, $P = 0.0031$; Fig. 1).

Twenty-two subjects (14.1%) in the recreational group and 27 subjects (19.9%) in the military group, 16.8% overall, had previous episodes suggestive of IPE or recurrent episodes on follow-up (1,9,14,21,23,28,32,35,52,59,61,62). Death was reported in six cases (14,16,23,34). No significant past medical history was reported in the remaining cases (7,11,16,18–20, 22,25,31,33,48,53,59,63).

DISCUSSION

IPE is typically thought to affect relatively young, healthy swimmers and divers. A review by Koehle et al. (37) in 2003 reported of a mean ± SD age of 36.1 ± 14.2 yr for all IPE patients and 47.1 ± 11 yr among scuba divers. For cases reviewed for this article, mean age differed between populations, falling in the fifth decade of life (40–49 yr) in the recreational diving/swimming groups and the third decade of life (20–29 yr) in the military diving/swimming group. Our mixed population of 36 recreational and commercial divers/swimmers had an average age similar to the recreational group reported in the literature.

In the literature, 70 (24.9%) of 292 reported cases occurred in patients with preexisting cardiopulmonary disease or risk factors for cardiopulmonary disease, warranting the conclusion that IPE is generally a condition affecting healthy individuals. Indeed, these figures are higher than those found by Koehle et al. (37), in which 17.2% of cases had any preexisting medical conditions. When considered within their subgroups (recreational vs military), however, the proportion of IPE cases with preexisting comorbidities is 44.9% in the recreational population (total $n = 156$) compared to 0% in the military population (total $n = 136$).

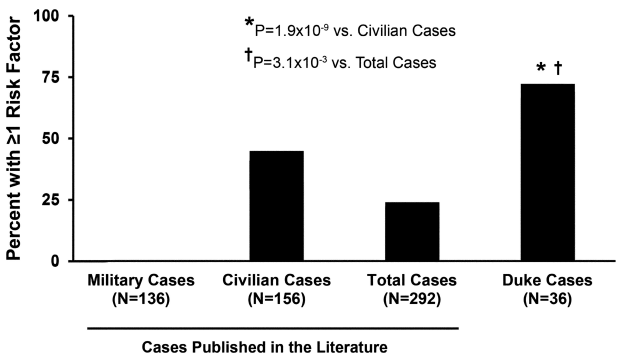

FIGURE 1—Percentage of patients with one or more risk factors for IPE. The greater fraction in the unselected Duke cohort significantly exceeds that of the population reported in the literature. None of the military cohort had any identifiable risk factor.

In our case series, we saw a proportion more similar to the recreational population: 72.2% of subjects had significant preexisting medical conditions, which is significantly greater than the 44.9% reported for civilian cases in the literature ($P = 0.0031$), consistent with our hypothesis of underreporting.

Cases A and B are illustrative examples of recreational divers who did not have known history of cardiovascular disease but did have significant risk factors (e.g., obesity and hyperlipidemia). In addition, obstructive sleep apnea would be a risk factor for developing systemic and pulmonary hypertension.

It is possible that divers with cardiac risk factors are at a higher risk for IPE owing to subclinical cardiopulmonary dysfunction that is exacerbated by immersion and exertion during diving or swimming. Those with underlying valvular disease or LV dysfunction may manifest as IPE in these settings. Ventricular distension by central redistribution of blood and increased afterload due to peripheral vasoconstriction in cold water could exacerbate or unmask valvular dysfunction (e.g., worsen mitral regurgitation by distorting the mitral valve annulus), which would increase the likelihood of developing pulmonary edema. Those without diagnosed heart disease may still be susceptible to demand ischemia or stress cardiomyopathy (Takotsubo). The possible contribution of demand ischemia to the pathophysiology of IPE is supported by evidence of myocardial dysfunction (defined by elevation in troponin T with EKG or echocardiographic abnormalities consistent with ischemia) present on admission, which later normalized (27). Curiously, stress testing at the time of hospital admission for IPE or on follow-up was negative in all cases ($n = 13$), and coronary angiography in six of those cases showed no significant disease. These findings suggest that the physiological changes that occur with immersed exercise are significant to the pathophysiology of IPE. Central redistribution of preload during immersion increases blood volume in the heart and LV wall stress, in turn increasing myocardial oxygen consumption (42). Myocardial dysfunction seen in some IPE cases could be due to a combination of increased myocardial oxygen consumption (e.g., due to increased HR due to exercise, increased ventricular wall stress) and decreased myocardial oxygen supply (e.g., due to hypoxia, coronary vasospasm, or microvascular vasoconstriction) (27). In the presence of ventricular hypertrophy, the combination of increased subendocardial pressure, higher muscle mass, and reduced diastolic time due to tachycardia and aortic stiffening could induce myocardial ischemia even with normal coronaries (50).

There is a well-recognized correlation between IPE and hypertension (and conceivably LVH) (44,67). A recent investigation of IPE cases found a high prevalence of hypertension, with 27.8% in all IPE cases and 60% in IPE cases with reversible myocardial dysfunction (27). Hypertension was the most common medical condition among IPE cases reviewed for this article (25 of 156 recreational swimmers and divers, representing 16.0%).

It is conceivable that myocardial ischemia in some cases of IPE represents a form of silent "syndrome X," which is classically described as angina with normal coronary angiography. It has been proposed that syndrome X is caused by myocardial ischemia due to microvascular dysfunction (8) or reduced coronary reserve as a result of hypertension and LVH (49,50). Although the diagnosis of syndrome X classically requires angina, it has been speculated that myocardial ischemia in some patients with syndrome X could occur without chest pain (8).

Whatever the underlying cause, demand ischemia can increase diastolic LV stiffness (3), which, in the face of increased preload, could predispose to pulmonary edema due to increased LV end-diastolic pressure and thus pulmonary venous pressure. Relevant to the association between IPE and cold water, impaired diastolic function has also been demonstrated in response to the cold pressor test, particularly in older individuals (46). Impaired diastolic function due to LVH is also highly prevalent in those with chronic systemic hypertension, obesity, and sleep apnea.

One possible contributing factor to the discrepancies in age and comorbidities seen between this article and previously published articles may be publication bias. A healthy, extremely fit, 19-yr-old military trainee who develops pulmonary edema during swimming is certainly remarkable, whereas a sedentary 65-yr-old recreational diver with risk factors for coronary artery disease who develops pulmonary edema during diving would be less surprising to most clinicians. Cases like the latter are less likely to be reported, which may lead to an overall skewed characterization of the disease process and its risk factors. In addition, there seems to be a striking difference with regard to comorbidities between the recreational and the military cases of IPE, with essentially all of the comorbidities reported in the recreational diving/swimming population. Whereas cardiopulmonary dysfunction may play a significant role in IPE in recreational divers/swimmers, other factors could be more important in the military population. Pulmonary artery and pulmonary artery wedge pressures (PAP and PAWP) both increase monotonically with increasing cardiac output (47). Owing to the central redistribution of blood, both PAP and PAWP increase further during immersed exercise (2,12,13,24,39,51), particularly in cold water (64), thus providing a mechanism for pulmonary edema in healthy individuals engaged in extreme exercise, such as in military recruits during swim training. Elevated PAP and PAWP could be accentuated by impairment of diastolic filling associated with LVH occurring as a result of strength training (4), which is unlikely to be detected during a standard prerecruitment examination). It is further likely that the contribution of cardiopulmonary disease to IPE in the recreational population has been underreported and underestimated.

LIMITATIONS OF THE STUDY

First, in one subset of patients in the series published by Gempp et al. (27), it was not possible to determine whether some individuals had more than one risk factor. For that series, we assumed the minimum possible number (six with hypertension). Second, not all cases were exhaustively investigated or reported. In such instances, some risk factors (e.g., LVH) may not have been detected. Thus, the total percentage of civilian cases with factors predisposing to IPE may have been underestimated. We would also like to emphasize that while EKG changes and troponin elevation are suggestive of myocardial damage, the fact that most cases recovered satisfactorily without any evidence of coronary artery disease reveals the limitations of these parameters as markers of heart disease. Abnormalities could occur as a result of secondary hypoxemia due to pulmonary edema. Finally, we included as possible risk factors any reported abnormality suggesting lung or heart disease, some of which may have been incidental. Nevertheless, in the cases where we were provided details of medical conditions (Duke series), the percentage of cases with risk factors was significantly higher than in the published cases. This supports our hypothesis that underlying risk factors are more common than supposed by reading the literature. Excluding military cases, it is appropriate to examine each individual with a history of IPE to exclude treatable causes.

In the reported cases, approximately 17% experienced a recurrence. This undoubtedly represents an underestimate because some individuals probably avoid the circumstances in which their IPE occurred.

CONCLUSIONS

IPE has been reported in healthy divers and swimmers without cardiac or pulmonary dysfunction; however, our data indicate a high prevalence of comorbidity in civilian cases of IPE. The latter type of cases may be underreported in the literature. Military cases of IPE seem to lack the same association with cardiopulmonary comorbidities, and other pathophysiological factors may be more important in that population.

We conclude that a search for predisposing conditions is warranted in all individuals with a history of IPE who have not previously been medically screened (as in military trainees). Specifically, the evaluation should include a search for hypertension, valvular heart disease, diabetes, lung disease, and, in individuals with risk factors, silent coronary artery disease. If obstructive sleep apnea is suspected, it may be useful to search for its possible secondary effects. IPE recurrence rate reported in the literature of 16.8% suggests that factors that increase susceptibility to IPE may be persistent, although the multifactorial nature of IPE may make it difficult to predict recurrence. Swimmers and divers who have experienced IPE should be advised of the potential for recurrence.

This study was supported by NAVSEA Contracts N61331-03-C-0015 and N0463A-10-C-0005 and Divers Alert Network. The authors declare that they have no conflicts of interest.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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