RELATION BETWEEN INTERATRIAL SHUNTS AND DECOMPRESSION SICKNESS IN DIVERS

PT Wilmshurst, JC Byrne, MM Webb-Peploe

Department of Cardiology, St. Thomas' Hospital, London, SE1 7EH, U.K.

The prevalence of interatrial shunts was determined in a blind controlled study of 97 divers who had had decompression sickness and 109 who had not. The prevalence of shunts was 24% in normal divers. The prevalences were significantly higher in those who had neurological symptoms within 30 minutes of surfacing (66%), skin bends (86%) and cardiorespiratory symptoms (58%). These symptoms followed provocative dives on 36, 24 and 33% of occasions respectively. The prevalences of shunts were similar to that in the control group in divers who had neurological symptoms occurring more than 30 minutes after surfacing (26%) and those who had limb bends (15%). These symptoms followed provocative dives significantly more often than other symptoms (76 and 87% respectively). When early neurological symptoms occurred after unprovocative dives in divers who did not have a shunt, the diver usually had lung disease or was a smoker. Repeated episodes of decompression sickness were usually associated with repeated provocative dives when divers had no shunt (n = 4), but usually followed unprovocative dives in those who had shunts (n = 10). The data suggests that interatrial shunts are implicated in the aetiology of some forms of decompression sickness. Analysis of the dive profiles in these cases suggests that venous bubbles may be involved. These episodes may result from paradoxical embolism of these bubbles.

Introduction

During and after ascent, a diver is at risk of decompression sickness, characterised by mild symptoms (rash or joint pain) or severe injury (cardiorespiratory or neurological). The precise cause of each of these clinical syndromes is unclear, but in some cases of severe decompression sickness the effects are similar to those observed following arterial air embolism resulting from pulmonary barotrauma. Since venous gas is known to be liberated during many decompressions (1), it is possible that right to left shunting of venous gas into the systemic arterial circulation could be responsible for some cases of decompression sickness.

Intracardiac shunts (patent foramen ovale or small atrial septal defects) are common in the population (2,4,5). We and others have demonstrated an association between interatrial shunts and decompression sickness in uncontrolled, open studies (6,9,11). We therefore investigated this relation in a controlled and blind manner in divers. Some earlier data from this study has been presented elsewhere (7,10). Because our previous open observations (9,11) suggested an association between interatrial shunts and neurological decompression sickness soon after surfacing our primary aim was to investigate this relation. For this purpose, 'early neurological

symptoms' was defined in advance as 'symptoms starting within 30 minutes of surfacing'. No attempt was made to exclude divers with possible pulmonary barotrauma and resulting cerebral air embolism, since this diagnosis is often based on time of onset of symptoms. In addition, we investigated the relation between interatrial shunts, other clinical manifestations of decompression sickness and other factors affecting decompression.

Subjects and Methods

Between June 1987 and May 1990, 99 divers who had been diagnosed as suffering from decompression sickness or cerebral gas embolism were seen by us at St. Thomas' Hospital. Ninety-seven took part in this study. One who we previously stated (10) had refused investigation subsequently changed her mind and is included. The study group does not include those divers investigated as part of a separate validation study at the Diving Diseases Research Centre in Plymouth (8). The individuals studied were subgrouped according to their manifestations of decompression sickness using predetermined criteria. The subgroups were not mutually exclusive:

Group Ia - 50 divers, who on 58 occasions, had neurological symptoms which started within 30 minutes of surfacing. Cardiorespiratory symptoms were associated on 9 occasions.

Group Ib - 35 divers, who on 38 occasions, had neurological symptoms which started more than 30 minutes after surfacing. Cardiorespiratory symptoms were associated on 3 occasions.

Group Ic - 20 divers had joint pain on 23 occasions. Fourteen episodes were associated with neurological symptoms (6 early, 8 late onset).

Group Id - 14 divers had cutaneous decompression sickness on over 29 occasions. Four individuals had multiple episodes. Two individuals each had more than 6 episodes but the precise number of episodes in those cases is uncertain. Neurological symptoms occurred in 9 of the divers who had cutaneous decompression sickness on the same or a separate occasion.

Group Ie - The 12 divers with cardiorespiratory symptoms were also considered as a separate group.

Group II - 109 amateur divers who had never had decompression sickness acted as controls.

The methods used have been described previously (10) but are summarised. The series of dives preceding an episode of decompression sickness was considered to start after the last dive-free interval of 48 hours. Each series of dives was analysed for risk factors for decompression sickness - namely, missed decompression stops (according to the decompression table or computer used by the diver), uncontrolled and rapid ascents, ascents to altitude after a dive, dives to deeper than 50m (the UK legal limit for commercial air diving), repetitive deep diving (to depths of 40m or greater at intervals of less than 24 hours), frequent diving (more than 3 dives daily to deeper than 10m) and decompression sickness in a diving companion exposed to the same pressure profile. A series of dives containing one or more risk factors was considered provocative.

Contrast echocardiography was performed and interpreted blind to history by a technician and doctor using a set protocol (8,10). Images were obtained using a Hewlett Packard HP 77020AC/AR and contrast microbubbles were produced in 5-6ml 0.9% sterile saline by the two-syringe and three-way tap method (4). The contrast medium was injected into a left antecubital

vein and if no shunt was demonstrated without manoeuvres, a modified Valsalva manoeuvre with sudden release was performed. A further five injections with modified Valsalva and two injections with coughing were performed unless right to left shunting was demonstrated earlier. Any shunting seen during these procedures was considered a positive test.

Statistical analysis was by the chi-squared test with Yates' correction for continuity and by the unpaired t test. Values of $p < 0.05$ were regarded as significant. Results are expressed as mean ± standard deviation.

Subjects gave written consent to the study which had the approval of the hospital ethical committee.

Results

The prevalence of interatrial shunts (Table 1) was significantly higher in divers who had had decompression sickness than in those who had not. This difference was the result of a high prevalence of shunt in divers with early neurological, cardiorespiratory and cutaneous manifestations, but not divers with late neurological or limb bends.

Table 1 Prevalence of interatrial shunts in the groups

	Group I (Total)	Ia Early CNS symptoms	Ib Late CNS symptoms	Ic Limb DCS	Id Skin DCS	Ie Cardio-respiratory symptoms	II Normal Divers
No. of divers	97	50	35	20	14	12	109
No. with a shunt	47	33	9	3	12	7	26
% with a shunt	48*	66*	26§	15§▪	86*	58*	24§▪

* Significantly different from Group II
§ Significantly different from Groups Ia and Id
▪ Significantly different from Group Ie

The dive series preceding early neurological symptoms was a single dive on 40% of occasions. Single dives tended to be implicated less frequently in late neurological symptoms (21%, $p < 0.1$) and limb bends (13%, $p < 0.05$).

Late neurological and limb decompression sickness usually followed a provocative dive series, characterised by dive-related risk factors, but this was not the case with early neurological, cutaneous or cardiorespiratory symptoms (Table 2).

Fourteen divers had decompression sickness on more than one occasion. Four divers without interatrial shunts had symptoms on nine occasions. Ten divers with shunts had symptoms on 37 occasions. Provocative dive series were more frequent in those without shunts (8/9) than in those with shunts (7/37, $p < 0.001$).

In divers who had late neurological symptoms, the prevalence of dive-related risk factors in those with shunts was lower than the prevalence in those without shunts (50 v 86%) but the difference was not significant. A similar finding was observed in divers with limb bends (shunt 67%, no shunt 90%).

Table 2 Prevalence of risk factors in dives causing symptoms

	Group Ia Early CNS symptoms	Ib Late CNS symptoms	Ic Limb DCS	Id Skin DCS	Ie Cardiorespiratory symptoms
No. of episodes	58	38	23	>29	12
No. with dive-related risk factors	21	29	20	7	4
% with risk factors	36	76*	87*	24	33

* Significantly different from Groups Ia, Id and Ie.

Table 3 Early neurological symptoms and dive-related risk factors

	Interatrial shunts	Lung disease	No pathology
No. of episodes	40	4	14
No. with dive-related risk factors	11	0	10
% with risk factors	28	0	71*

* Significantly different from other groups.

Analysis of divers with early neurological symptoms revealed different results (Table 3). In those with shunts, 28% of episodes of early neurological symptoms followed provocative dives. Four divers had lung disease and each had symptoms after an unprovocative dive. The clinical diagnosis was arterial gas embolism due to pulmonary barotrauma in each case. The 14 remaining episodes occurred in divers without abnormal pathology (interatrial shunt or obvious pulmonary pathology). 10/14 episodes were preceded by provocative dive series (single dives in 5, repetitive dives in 5) with very major deviations from accepted safe decompression procedures, including uncontrolled ascents and/or missed decompression stops in each. In the remaining four cases, symptoms followed

an unprovocative dive (without significant preceding nitrogen load), to a significantly shallower depth (Table 4). 3/4 were smokers. The prevalence of smokers was higher than in other groups (although not significantly so, but numbers were small).

Cardiorespiratory symptoms occurred in association with neurological symptoms in 12 divers. One diver with lung disease had symptoms soon after surfacing from an unprovocative 21m dive. Seven divers with shunts had symptoms soon after dives to depth 33.1 ± 6.2m. 4/7 were unprovocative. Four divers without abnormal pathology had cardiovascular symptoms after provocative dives to significantly greater depth (43.8 ± 5.8m, $p < 0.05$).

Two individuals without interatrial shunts had skin bends. Both occurred after deep repetitive diving and rashes were observed 1-4 hours after the last dive. Shunts were present in 12/14 divers with skin bends. In these cutaneous decompression sickness was usually observed within 30 minutes of surfacing and always within 2 hours, but persisted for some hours. Pruritis was usually the first manifestation. Rarely severe pruritis occurred without a rash. Rashes were usually erythematous and often punctate, but when severe 'mottling' or 'marbling' occurred. The rashes occurred in divers wearing dry suits, wet suits and no thermal insulation.

Table 4 Early neurological symptoms, smoking and depths of dives

	Pathology Present		No Pathology	
	Interatrial shunts	Lung disease	Risk factors present	Risk factors absent
No. of episodes	40	4	10	4
No. in smokers	8	1	2	3
% in smokers	20	25	20	75
Depth of last dive in series (m)	30.7 ± 6.8	28.3 ± 4.9	36.3 ± 11.1	19.0 ± 4.8*

* Significantly different from other groups.

Discussion

The data suggests that there are a number of mechanisms producing symptoms of decompression sickness. Although there is overlap between the groups it seems that neurological symptoms arise by one of three mechanisms. Neurological symptoms starting more than 30 minutes after surfacing are not associated with an increased prevalence of interatrial shunts but are associated with risk factors in the dive, such as missed stops, rapid ascents, deep and repetitive diving. (Decompression sickness in a diving companion was not a separate risk factor, since pairs of divers were never affected after unprovocative dives.)

Neurological symptoms soon after surfacing were also sometimes associated with provocative dives without detectable pathological abnormalities (n = 10). Indeed, some of the most major deviations from generally accepted safe

diving practices occurred in these individuals. However, the majority of divers with early neurological symptoms had abnormal pathology, either interatrial shunts (n = 33) or lung pathology (n = 4), rather than provocative dive profiles as their risk factor. Four individuals had neither abnormal pathology nor provocative dives. Each had symptoms after a relatively shallow dive and it is likely that as with those with lung disease, these individuals suffered pulmonary barotrauma and arterial gas embolism. It may be relevant that 3/4 were smokers.

Clearly pulmonary barotrauma can occur after a dive to any depth including those which are shallow. Provocative dives causing decompression sickness are more likely to be deep. It is interesting that early neurological symptoms in divers with interatrial shunts were of intermediate depth. The most likely explanation for these findings is that paradoxical embolism of venous gas bubbles across the shunt embolise tissues and undergo peripheral amplification when they enter fast tissues with a high nitrogen content (e.g. nervous tissue)(3). Only depth-time profiles capable of loading fast tissues with nitrogen and ascent rates sufficiently rapid to liberate venous bubbles whilst leaving embolised tissues loaded with nitrogen (to allow bubble amplification) are likely to be provocative in these individuals. Therefore, very shallow dives are unlikely to be implicated. In addition, factors increasing shunting may be necessary in some individuals for the dive to produce symptoms. Not every dive with a particular profile would produce symptoms.

Cardiorespiratory symptoms occurred in some individuals with shunts (n = 7) and one with lung disease. The others had no abnormal pathology but had performed highly provocative dives (n = 4). Although, such symptoms in those who performed provocative dives could be due to massive pulmonary artery gas embolism, this could not be so in the individual who had lung disease and pulmonary barotrauma. It is also unlikely to account for the symptoms after unprovocative dives in those with shunts. A more likely explanation for these symptoms is that they result from coronary embolism by arterial gas. When such symptoms occur in individuals with neither shunts nor lung disease, it is an index of severity of provocative decompression.

Skin bends also usually occurred in individuals with shunts, after unprovocative dives and sometimes in association with early neurological symptoms. The two exceptions followed provocative dives. Skin symptoms also appear to be related to arterialised gas in most cases. Limb bends were not associated with an increased prevalence of interatrial shunts, which suggests that their aetiology may be unrelated to arterial gas embolism.

References

1. Daniels S, Davis JM, Paon WDM, Smith EB. Recent experiments using ultrasonic imaging to monitor bubble formation in divers. In: Bachrach AJ, Matzen MM, eds. Underwater physiology VIII. Bethesda: Undersea Medical Society 249-57, 1984.

2. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. Mayo Clin Proc 59: 17-20, 1984.

3. Hennessy TR. On the site of origin, evolution and effects of

decompression microbubbles. In: Brubakk AO, Hemmingsen BB, Sundnes G, eds. Supersaturation and bubble formation in fluids and organisms. Trondheim: Royal Norwegian Society of Sciences and Letters 291-340, 1989.
4. Lechat PH, Mas JL, Lascault G, et al. Prevalence of patent foramen ovale in patients with stroke. N Eng J Med 318: 1148-52, 1988.
5. Lynch JJ, Schuchard GH, Gross CM, Wann LS. Prevalence of right-to-left atrial shunting in a healthy population: detection by Valsalva maneuver contrast echocardiography. Am J Cardiol 53: 1478-80, 1984.
6. Moon RE, Camporesi EM, Kisslo JA. Patent foramen ovale and decompression sickness in divers. Lancet i: 513-4, 1989.
7. Wilmshurst P. Analysis of decompression accidents in amateur divers. Prog Underwater Sci 15: 31-7, 1990.
8. Wilmshurst P. Interatrial shunts and decompression sickness in divers. Lancet i: 915, 1990.
9. Wilmshurst PT, Byrne JC, Webb-Peploe MM. Neurological decompression sickness. Lancet i: 731, 1989.
10. Wilmshurst PT, Byrne JC, Webb-Peploe MM. Relation between interatrial shunts and decompression sickness in divers. Lancet ii: 1302-6, 1989.
11. Wilmshurst PT, Ellis BG, Jenkins BS. Paradoxical gas embolism in a scuba diver with an atrial septal defect. Br Med J 293: 1277, 1986.