decompression sickness in divers. *Lancet* 1989: ii: 1302-1304
3 Cross SJ, Evans SA, Thomson LF, Leed HS, Jennings KP aand Shields TG. Safety of subaqua diving with a patent forament ovale. *Brit Med J* 1992; 304: 481-482
4 Moon RE, Camporesi EM and Kisslo JA. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989: i: 513-514
5 Moon RE, Kissolo JA, Wassey FW, Fawcett TA and Theil DR. Patent foramen ovale (PFO) and decompression illness. *Undersea Biomed Res* 1991; 13 (Suppl): 15
6 *Report on diving accidents and fatalities*. Durham,South Carolina: Divers Alert Network, 1996

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## PATENT FORAMEN OVALE IN UNDERWATER MEDICINE

Paul Langton

**Abstract**

The foramen ovale, between the right and left atria, exists in the foetal heart as a vital physiological communication. Haemodynamic closure occurs in the neonatal period with most people having permanent fusion of the foramen. In up to a third of adults the closure is functional only and a potential right to left atrial communication persists as a patent foramen ovale. Studies in patients with decompression illness after diving suggest a consistent increase in the prevalence of patent foramen ovale, as detected by transthoracic contrast echocardiography. The association is strongest for those patients with early onset of neurological decompression illness, particularly those cases occurring in the absence of other risk factors traditionally associated with decompression illness. However, patent foramen ovale is a common finding in the general population and the absolute risk of decompression illness, even in the presence of a patent foramen ovale, remains very low.

**Key Words**

Decompression illness, heart conditions, investigations.

**Introduction**

There has been considerable interest in the potential contributory role of the foramen ovale in the development of decompression illness (DCI) and arterial gas embolism (AGE) in SCUBA divers. Venous bubble formation is known to occur during hyperbaric gas exposures well within the recommended limits of recreational diving.[1] The relative absence of clinical decompression sickness is thought to be related to the filtering of venous bubbles as they pass through the pulmonary circulation, thus preventing systemic arterial exposure. It is proposed that the presence of a patent foramen ovale (PFO) allows venous bubbles to pass across the interatrial septum into the left heart and then into the arterial circulation, with the potential to cause AGE.

**Background**

The foramen ovale exists as a vital physiological communication between the right and left atria during foetal life. Atrial division (Fig 1) initially occurs with the formation of the septum primum, a crescentic structure grows from the top of the common atria and fuses with the endocardial cushions that demarcate the atrioventricular junction. As it develops some of the central tissue of the septum primum beaks down to create the foramen secundum, maintaining interatrial communication. The septum secundum then grows from the right superior margin of the septum primum to incompletely divide the atria; it remains deficient inferiorly, against the endocardial cushions. The combined atrial septum (primum and secundum) thus forms the foramen ovale and allows oxygenated inferior vena caval blood (returning from the placenta) to be directed across the atrial septum to the left heart and thenceforth to the developing brain (Fig 2). In contrast, deoxygenated (superior vena caval) blood streams preferentially from the right atrium through the right ventricle to the pulmonary circulation and then via the ductus arteriosus back to the placenta. The foramen ovale remains open in the foetus because of the existence of significantly higher pressure in the right atrium as compared with the left.

The physiological changes that occur at birth include a profound lowering of pulmonary vascular resistance secondary to lung aeration, and a fall in right atrial pressure. At the same time systemic pressures increases, with a rise in left atrial pressure and hence the functional closure of the foramen ovale (Fig 3). In most infants this functional closure is followed by fusion of the flap like membrane, forming the fossa ovalis. In about a third of individuals fusion does not occur and a potential inter-atrial shunt persists as a PFO. For shunt flow to occur however the right atrial pressure must exceed that in the left atrium. The phasic nature and right to left flow of a PFO help distinguish this anatomical variant from an atrial septal

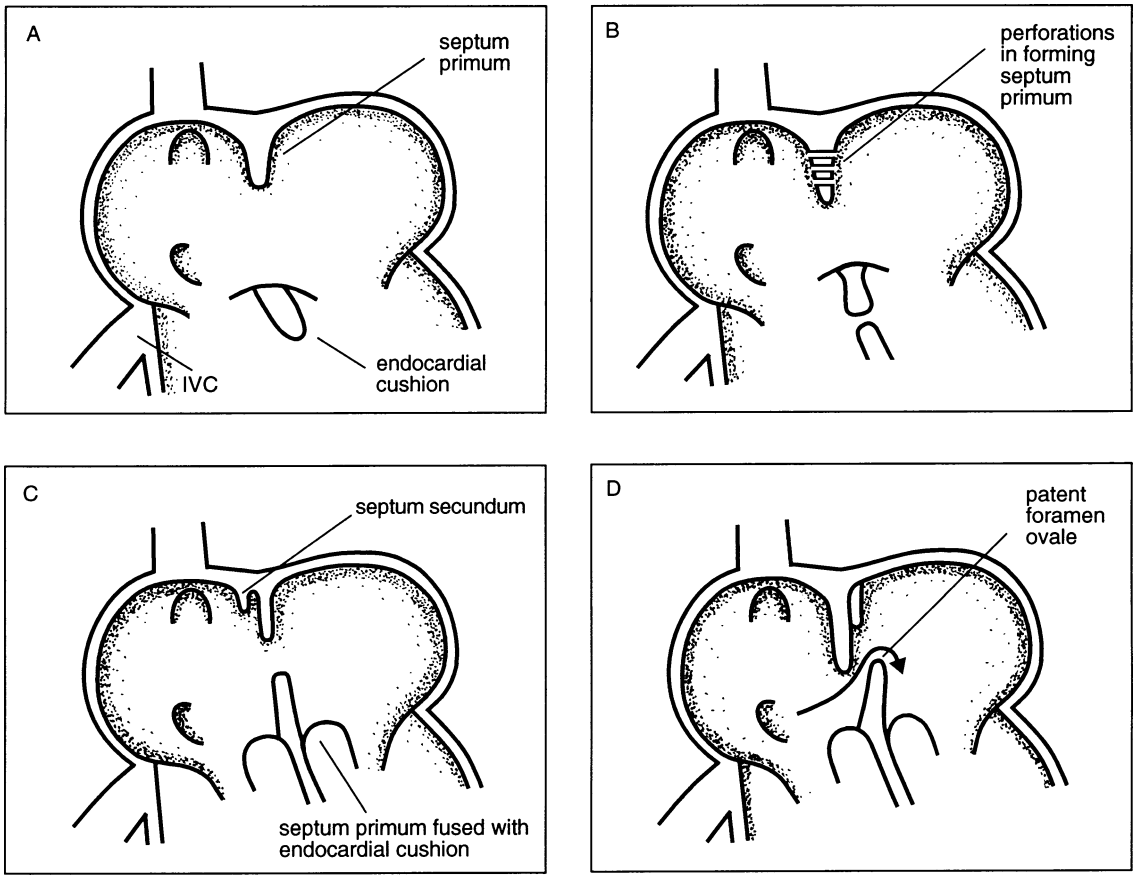


**Figure 1.** Diagrammatic representation of the formation of the interatrial septum in the primitive common atria. Diagram 1D demonstrates flow from the inferior vena cava across the foramen ovale.

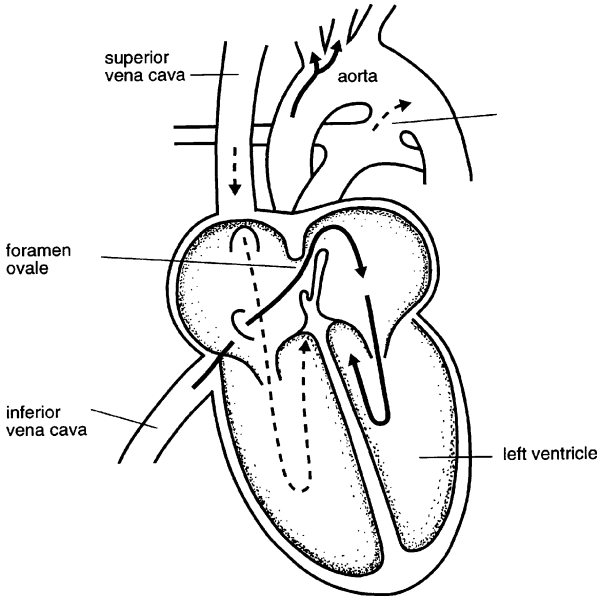


**Figure 2.** In the foetal circulation oxygenated blood from the placenta is directed via the inferior vena cava and foramen ovale to the developing brain (solid lines). Deoxygenated blood (broken lines) from the superior vena cava passes to the pulmonary artery and then crosses the ductus arteriosus to the aorta and is returned to the placenta.

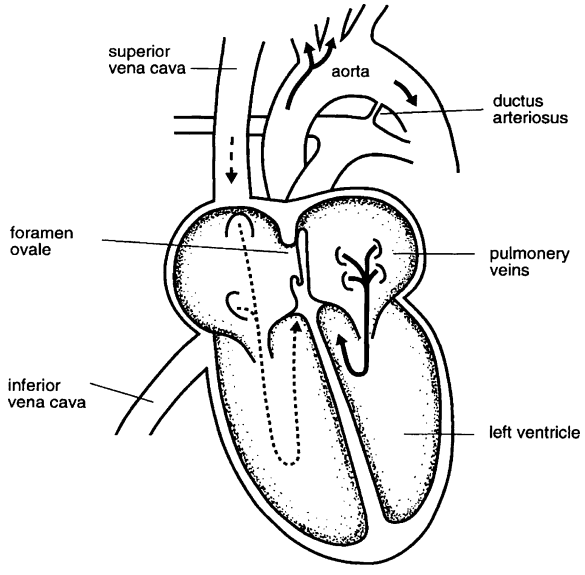


**Figure 3.** In the neonate the lowering of right atrial pressure and increase in left atrial pressure cause the functional closure of the foramen ovale.

defect, where there is a predominantly left to right (bidirectional) shunt across the septum and associated dilation of the right heart and left atrium.

Post mortem studies have attempted to define the frequency of PFO by detection of probe patency from right to left through the fossa ovalis. Hagen et al at the Mayo Clinic have studied 965 "normal" hearts in an autopsy series, using calibrated probes to define the maximum potential size of the PFO.[2] By this method most PFOs were small (mean 4.9 mm), however communications of up to 19 mm were found. The incidence of probe defined PFO was approximately one third in subjects below 30 years of age, falling to approximately one quarter for those above this age. There was a tendency for the average size of PFO to increase with age, suggesting that smaller PFOs may spontaneously close in early adult life.

Anatomical presence of PFO does not imply interatrial shunting. Although mean right atrial pressure is lower than left atrial pressure, there can be transient phasic inversions in this gradient in early atrial systole with the potential for right to left shunting in the absence of any unusual physiological circumstances. Situations that elevate right atrial relative to left atrial pressure would increase the tendency for any right to left shunting,[3] such as breath holding, coughing and the Valsalva manoeuvre (all common during scuba diving). Immersion itself may cause an elevation of right atrial pressure, but has not been shown to affect the interatrial pressure differential.

**Echocardiography**

PFO during life has most commonly been detected by transthoracic echocardiography, and more recently by transoesophageal (TOE) echocardiography, through either the detection of interatrial shunting with colour flow Doppler, or by the observance of ECHO contrast transit from the right to the left heart after injection of contrast agents such as agitated saline In most cases the degree of the shunt is very small and occurs as left atrial pressure rises above the right. Less commonly right to left flow is seen. The colour Doppler technique is limited by the need for good views of the interatrial septum which are most readily obtained from the subcostal approach, but adequate views are often difficult to obtain.

Agitated saline is most commonly used as the contrast agent for the detection of PFOs. It has many microbubbles suspended in solution, which form an effective contrast medium for ultrasonic detection.[4] In practice agitated saline is injected into a large peripheral vein and this bolus can be visualised within several cardiac cycles as it passes through the right heart. The microbubbles are then normally filtered out by the pulmonary microcirculation.[5] In the case of some subjects with a PFO, ECHO contrast material can be seen to pass spontaneously from the right to left heart, usually distinguishable at the atrial level. Detection of right to left shunting can be increased by the performance of provocative manoeuvres that elevate relative right atrial pressure, such as after a cough or in the release phase of the Valsalva manoeuvre, at the time of ECHO contrast injection.[6,7] Left to right shunting is occasionally seen as a negative contrast effect (which has to be distinguished from inferior vena cava (IVC) flow), although this would be more typical of an atrial septal defect. There is considerable variability in the ability of contrast injections to detect shunting from moment to moment and at least 2-3 boluses are usually injected (with up to 6 being reported), in routine practice.

Because of the technical considerations involved in the detection of PFO, routine transthoracic ECHO studies looking for PFO need to be performed by an experienced sonographer using a validated protocol of baseline and repeated contrast ECHO views before and after provocative manoeuvres. With such a protocol the incidence of PFO in a normal control population can be defined, with most studies identifying functional PFOs in about 15 - 31 % of people.[7-9]

Transoesophageal echocardiography detection of PFO has been widely used in both unexplained stroke in younger patients and in all patients with stroke. While there is clear evidence that TOE has greater sensitivity over transthoracic ECHO for the detection of PFO, this applies equally to control subjects and patients with stroke.[10] When comparing the prevalence of PFO in young stroke patients to that of true control subjects, studies show somewhat conflicting results as to whether PFO is more common in the patient group.[10,11] TOE seems able to detect a greater number of (possibly) smaller PFOs their relevance to disease states remains to be proven.

Importantly the detection of PFO by any contrast ECHO relies on the passage of microbubbles into the arterial circulation, in a similar way to the proposed mechanism for DCI (although in DCI the bubble size is likely to be greater). There is a published incidence of predominantly transient neurological side-effects after contrast ECHO (about 1 in 6,000).[12] It would seem prudent to defer investigation of a possible PFO in a patient with DCI until after the episode has completely resolved.

**PFO and decompression sickness**

Several case reports from the early 1980s identified the association of PFO with decompression illness (DCI) after diving.[13,14] Moon's group found that PFO was detectable on transthoracic ECHO in 11 of 30 patients with DCI (37%), and that the subgroup of patients with severe signs and symptoms (weakness, vertigo, cognitive impairment) had PFO in 11 of 18 (61%).[14] Interestingly, all these patients had a PFO evident during spontaneous

breathing. The authors did not study a control group of normal subjects, relying on reports from other studies which may not have been directly comparable. Cross et al. have subsequently attempted to define the incidence of PFO by transthoracic ECHO in a population of 'normal' control divers.[9] They examined 78 divers who had no history of DCI, by contrast ECHO before and after Valsalva manoeuvre. Twenty four divers were found to have PFO, with the incidence of 31 % being similar to the rate of PFO detected in a similar age group (their mean was 34 years) in Hagen's autopsy series.[2] This suggests that previous lower estimates of background rates[7,8] may have underestimated the prevalence of PFO in the diving population, possibly by the study of relatively older patients and/or methodological issues. That the overall incidence of PFOs in Moon's series[14] was similar to rates reported at autopsy and by Cross[9] suggests the primary role of PFO in overall DCI incidence is open to question.

In a second series reported by Wilmshurst et al.[15] 61 patients with DCI were divided into predetermined clinical subgroups. The control subjects were the diving "buddies" of the patients or experienced divers who had never had DCI and were of similar age to the patient group. The incidence of PFO on transthoracic ECHO in this control group was 24% (15/63). The overall incidence in divers with DCI was 41%, however in the subgroup of 29 patients with onset of neurological DCI within 30 minutes of surfacing 19 had PFO (66%, $p<0.001$ cf controls or other patients).

With respect to dive profile associations with DCI, Wilmshurst subdivided all patients into those with and without recognised risk factors for DCI.[15] The patients whose dive profiles would have otherwise been considered "safe" were more likely to have PFO (16/25) than in those who performed dives that would be accepted as having increased risk of DCI (9/36). This finding supports the hypothesis that PFO is probably causally related to these episodes of early onset neurological DCI occurring after otherwise safe dives. However the small numbers and subgroup analysis do limit the validity of this interpretation.

Cross et al. reported 19 cases of neurological DCI and found PFOs on transthoracic ECHO in only 6 of 19 patients (32%).[16] The clinical severity (ie sensory changes only or more severe neurological signs) and time between surfacing and symptom onset were not reported.

Limited prospective data regarding the association exists. In a study by Vik et al. anaesthetised pigs were exposed to air at 5 bar for thirty minutes and then rapidly recompressed. Arterial bubbles were detected by transoesophageal ECHO.[17] Presence of PFO was defined anatomically at subsequent autopsy. The pigs with PFO had a much higher rate of arterial bubble detection (6/6) than the non-PFO group (2/8). This supports the hypothesis that PFO increases the risk of arterial gas embolism and hence presumably the risk of clinical DCI.

**PFO and other neurological syndromes.**

The frequency of unexplained stroke in relatively young patients greatly exceeds that of divers with DCI and the occurrence of such strokes may be related to PFOs. In 1988 two studies reported an increased incidence of transthoracic contrast ECHO detected PFO in young patients (<55 years) with otherwise unexplained stroke (40% vs 10% and 50% vs 15% respectively).[8,18] It is thought that PFO allows the passage of venous derived thromboemboli into the arterial circulation and hence cause neurological events. This is similar to the proposed mechanism for some cases of early onset neurological DCI (passage of gaseous emboli).

In comparison to PFOs detected incidentally, patients with unexplained stroke generally have larger foramina, with a greater degree of (semi-quantitative) right to left contrast shunt, and are more likely to have an associated atrial septal aneurysm with their PFO.[19-23] Any relation of atrial septal aneurysm with DCI is however uncertain, and this may be a confounding factor when trying to compare data derived from stroke patients with those relating to DCI.

With respect to risk of recurrent neurological events, several studies have followed up young patients (<60 years) with a PFO and an otherwise unexplained stroke for two or more years.[23-25] The risk of further events is up to 2 % per annum if there are associated cerebrovascular risks (e.g. atrial fibrillation), or up to 4.5 % when both PFO and an atrial septal aneurysm exist. In the absence of these added factors, the risk of recurrence is very low (<1 %).

**Absolute Risk**

On the limited data outlined above, it would seem that the presence of PFO may confer an increase in the relative risk of some types of DCI. This relative risk needs to be interpreted in light of the overall incidence of DCI. It is estimated that there are over 50,000 divers in the United Kingdom, of whom 12-15,000 would have a PFO.[26] The reported incidence of DCI is around 100 cases per year, of which only approximately 50 represent the early onset of more severe neurological DCI that has been most closely linked to PFO. Looking at the relative incidence of PFO in patients with early severed DCI versus controls (66 % versus 24%),[15] PFO is associated with an excess of about 42% or about 21 cases a year. This represents an increased risk in the order of 1 in 600 for subjects with a PFO. Although the confidence intervals for these estimates would be large, the estimates do provide us with a starting point to put the absolute risk associated with PFO in perspective.

**Management**

Any management of PFO is dependant on the circumstances in which it is detected.

There is no general agreement to support screening for a PFO prior to diver training. If a PFO had been picked up incidentally during an ECHO, the reason why the subject had been having the ECHO in the first place may be more important in assessing future diving risk. It would be reasonable however to explain the potential risk of DCI (in absolute terms) to such a patient. If a subject had an incidental ECHO that did not identify a PFO, unless the technician had been using a protocol to formally look for PFO, one could not assume that PFO was absent.

In a patient who has had early onset of neurological DCI, particularly in the absence of other well recognised risks for DCI, it is reasonable to look for a PFO with a transthoracic contrast ECHO study. It would be prudent to defer this investigation until the episode had completely resolved. A PFO detected may or may not be relevant compared to other risks for DCI. Those with a greater degree of (semi-quantitative) right to left shunting are possibly more important than very small shunts. All patients who have suffered DCI need careful advice about future diving. If a PFO has been demonstrated a detailed discussion of the problem with the patient, including their likely future risk, probably more important than a proscriptive approach banning further diving. Regardless of the presence of a PFO, the diver has to be exposed to a bubble forming dive profile before being at any risk and it may well be possible for them to dive more conservative profiles without forming bubbles. The vast majority of subjects with PFO do not suffer from DCI, despite the fact that many of them will form venous bubbles during recreational diving exposures.[1]

Open heart surgery to close a PFO alone would not be advocated. Cardiac bypass itself exposes the patient to gaseous microemboli, and leaves some scarring in the chest. Transvenous devices can be used to occlude an atrial septal defect, including a PFO.[27] However some trans-septal flow often persists and currently these devices US Food and Drug Administration approval. Their use would be considered experimental.

**Conclusions**

Patent foramen ovale is a common finding in the normal population. On limited data it appears to confer an increased risk for early onset neurological decompression sickness, particularly if there is a large shunt evident. Although PFO may, under conditions that predispose to DCI, make it more likely to be apparent, overwhelming consideration remains the predisposing diving conditions. Detection of suspected PFO requires a protocol of contrast transthoracic ECHO before and after provocative manoeuvres; the possible role of transoesophageal ECHO is undefined. The absolute risk of DCI in a subject with a PFO remains very low. The risk of recurrent DCI in those patients with PFO who continue to dive is uncertain, but is likely to be minimised by adherence to conservative dive protocols.

**References**


1 Dunford R, Wacholz CJ, Irwin J et al. Ultrasonic Doppler bubble incidence following sports dives. *Undersea Biomed Res* 1988; 15(Suppl): 45-46

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

3 Opdyke DF and Brecher GA. Effect of normal and abnormal changes in intrathoracic pressure on effective right and left atrial pressures. *Am J Physiol* 1950; 160: 556-566

4 Barrera JG, Fulkerston PK, Rittgers SE and Nerem RM. The nature of contrast echocardiographic "targets". *Circulation* 1978; 58: 233

5 Meltzer RS, Tickner EG and Popp RL. Why do the lungs clear ultrasonic contrast? *Ultrasound Med Biol* 1980; 6: 263-265

6 Dubourg O, Bourdarias J-P, Farcot J-C et al. Contrast echocardiographic visualisation of cough induced right to left shunt through a patent foramen ovale. *J Am Coll Cardiol* 1984; 4: 587-594

7 Lynch JJ, Schuchard GH, Gross CM and Wann LS. Prevalence of right-to-left atrial shunting in a healthy population; detection by Valsalva manoeuvre contrast echocardiography. *Am J Cardiol* 1984; 53: 1478-1480

8 Webster MW, Chancellor AM, Smith HJ et al. Patent foramen ovale in young stroke patients *Lancet* 1988; ii: 11-12

9 Cross SJ, Evans SA, Thomson LF, Lee HS et al. Safety of subaqua diving with a patent foramen ovale. *Brit Med J* 1992; 304: 481-482

10 Hausmann D, Mugge A, Becht I and Daniel WG. Diagnosis of patent foramen ovale by transoesophageal echocardiography and association with cerebral and peripheral embolic events. *Am J Cardiol* 1992; 70: 668-672

11 Siostrozonek P, Lang W, Zangeneh M et al. Significance of left sided heart disease for the detection of patent foramen ovale by transoesophageal contrast echocardiography. *J Am Coll Cardiol* 1992; 19: 1192-1196

12 Bonner W, Shah PM, Allen H et al. The safety of ultrasonic contrast. Report of the Contrast Committee, American Society for Echocardiography. *J Am Coll Cardiol* 1984 ; 3: 6-13

13 Wilmshurst PT, Ellis BG and Jenkins BS.

Paradoxical embolism in a scuba diver with an atrial septal defect. *Brit Med J* 1986; 293: 1277
14 Moon RE, Camporesi EM and Kisslo JA. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989; 335: 513-514
15 Wilmshurst PT, Byrne JC and Webb-Peploe MM. Relation between interatrial shunts and decompression sickness in divers. *Lancet* 1989; 335: 1302-1306
16 Cross SJ, Thomson LF, Jennings KP and Shields TG. Right-to-left shunt and neurological decompression sickness in divers. (letter) *Lancet* 1990; 336: 568
17 Vik A, Jenssen BM and Brubakk AO. Arterial gas bubbles after decompression in pigs with patent foramen ovale. *Undersea Hyperbaric Med* 1993; 20: 121-131
18 Lechat P, Mas JL, Laschault G et al. Prevalence of patent foramen ovale in patients with stroke. *New Eng J Med* 1988; 318: 1148-1152.
19 Stone DA, Godard J, Corretti MC, Kiiner SJ et al. Patent foramen ovale: association between the degree of shunt by contrast echocardiogram and the risk of future ischaemic neurological events. *Am Heart J* 1996; 131: 158-161
20 Hausmann D, Mugge A and Daniel WG. Identification of patent foramen ovale permitting paradoxical embolism. *J Am Coll Cardiol* 1995; 26: 1030-1038
21 Homma S, di Tullio MR, Sacco RL, Mihaltos D et al. Characteristics of patent foramen ovale associated with cryptogenic stroke. A biplane transesophageal echocardiographic study. *Stroke* 1994; 25:582-586
22 Cabanes L, Mas JL, Cohen A, Amarenco P et al. Atrial septal aneurysm and patent foramen ovale as risk factors for cryptogenic stroke in patients less than 55 years of age. A study using transesophageal echocardiography. *Stroke* 1993; 24: 1865-1873
23 Hanna JP. Sun JP, Furlan AJ, Stewart WJ et al. Patent foramen ovale and brain infarct. Echocardiographic predictors, recurrence and prevention. *Stroke* 1994; 25: 782-786
24 Mas JL and Zuber M. Recurrent cerebrovascular events in patients with patent foramen ovale, atrial septal aneurysm, or both and cryptogenic stroke or transient ischemic attack. *Am Heart J* 1995; 130: 1083-1088
25 Bogousslavsky J, Garazi S, Jeanrenaud X, Aebischer N and van Melle G. Stroke recurrence in patients with patent foramen ovale: the Lausanne Study. *Neurology* 1996; 46: 1301-1305
26 Wilmshurst PT and de Belder MA. Patent foramen ovale in adult life. (editorial) *Brit Heart J* 1994; 71: 209-212
27 Bridges ND, Hellenbrand W, Latson L, Filiano J et al. Transcatheter closure of patent foramen ovale after presumed paradoxical embolism. *Circulation* 1992; 86: 1902-1908

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# SPUMS ANNUAL SCIENTIFIC MEETING 1996

## THE SCOPE OF NON-CONVENTIONAL RECREATIONAL DIVING

R W Bill Hamilton

**Key Words**
Deep diving, mixed gases, oxygen, recreational diving, safety, tables.

### What is "recreational diving"?

Non-conventional recreational diving has to begin with a basic definition of recreational diving, which can be summarised as diving for fun. Recreational diving is well recognised as being scuba diving in the range to 40 msw (msw = metres of sea water; 1 msw ≈ 0.1 bar or 10 kPa), and further it is diving with air as the breathing gas and not involving decompression stops. Realistically, these are not the limits within which all recreational divers operate, but until recently they were the limits to which divers have been trained by the recreational diving training agencies, particularly in the U.S.A. The British Sub-Aqua Club (BSAC) trains divers to 50 msw and allows decompression stops. This zone defined above is also recognised by the U.S. Occupational Safety and Health Administration as being outside commercial diving. Thus instructors can teach diving within these limits without their employers having to comply with the Commercial Diving Standard. In Britain recreational diving instructors at work who breathe non-air mixtures are considered to be commercial divers.

As mentioned and as the name implies, recreational divers are doing it for fun. Implicit in this is that these divers are not employees and they are not at work. Other types of sport diving are "recreational" in not being work, but may involve considerable specialisation and skill. Some of these are cave diving, ice and other types of overhead-