nomena amongst sports divers (about 300 episodes of neurological DCS in Australia annually from an active diving population of more than 400,000) and yet between 15 and 30% (60,000 - 120,000) of these divers can be expected to have a patent foramen-ovale[17]. The technique used to detect a foramen-ovale is not inexpensive, will only identify between 30% and 50% of those divers who do have a patent foramen ovale[1,17], and is not without risk. For example, microbubbles are injected into the circulation to help to identify any foramen[1,2], and yet if the results obtained in experiments in rabbits just completed in our laboratories can be extrapolated to humans then such microbubbles can have a significant effect on both cerebral blood flow and brain function. A decision to screen all divers should await further data. There is no doubt however that the 2 current issues of most interest in the pathophysiology of DCS is the role of a patent foramen-ovale and AGE[1-4] and the importance of complement protein activity in animal models of DCS[18].

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PATENT FORAMEN OVALE

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This paper was originally written in response to an enquiry from the Training Department of the National Association of Underwater Instructors (NAUI) which was concerned about the incidence of patent foramen ovale amongst trainee divers and the possibility of these people having a diving accident attributable to the defect. In addition the paper has formed the basis of a report to the Australian Sports Medicine Federation, many members of

which do medical examinations for divers, before being offered to the SPUMS Journal.

There has been some concern over recent months in diving circles about the relation between cerebral decompression sickness, cerebral gas embolus and the presence of a patent foramen ovale.

In the foetal circulation, the foramen ovale enables oxygenated blood from the placenta to cross from the right to the left side of the heart thus by-passing the lungs. At birth, with the onset of normal breathing, pressure changes in the heart close the flap value arrangement of the foramen and the shunt ceases. Obviously in a small percentage, this shunt persists and, depending upon its size, can be readily detected and, if necessary, treated by surgical means.

There is a significant proportion of the population in which the foramen ovale persists but is functionally closed and is asymptomatic. However, if there are significant intrathoracic and intracardiac pressure changes then the foramen may reopen and a right to left shunt occur. In an autopsy study of 965 normal hearts, Hagen[1] reported that the incidence of patent foramen ovale is 15-30% in the population so that of the 400,000 divers in Australia 60-120,000 of these can be expected to have a foramen ovale.

In 1988 Lechat[2,] in the New England Journal of Medicine, and Webster[3,] in The Lancet, reported on the association between stroke and the presence of a patent foramen ovale. Webster showed that a patent foramen ovale was present in 30% at rest but this increased to 50% during a Valsalva manoeuvre in the stroke patients. This was compared with normal controls who had an incidence of 7.5% at rest which increased to 15% during a Valsalva manoeuvre. Lechat's figures were very similar. Wilmshurst[4,] in the British Medical Journal, reported a case of cerebral gas embolism in a diver with a previously undiagnosed foramen ovale. This patient dived to 38 metres for 15 minutes and ascended at 15 m/minute, a dive close to the no decompression limits of the RNPL/BASC tables and, within two minutes of surfacing, developed profound cerebral symptoms.

In an article by Moon[5] reported in the Lancet, 30 patients with a history of decompression sickness were examined for the presence of a patent foramen ovale. 37% had a right to left shunt at rest compared with 5% in healthy volunteers. In a subset of 18 patients who had serious signs and symptoms, 61% had evidence of intra cardiac shunting.

It has been well shown that venous gas emboli occur in most divers diving within the no decompression limits and these bubbles result from the release of inert gas from tissues during decompression. Normally these bubbles are filtered out in the lungs and remain asymptomatic. However the presence of a patent foramen ovale may permit the passage of these bubbles into the arterial circulation. These gas bubbles may also 'seed' susceptible tissues and precipitate symptoms of severe decompression sickness.

This evidence then suggests that diagnosis of patent foramen ovale is essential during the medical examination. However Webster[3] showed that clinical examination by a cardiologist, radiology and electrocardiography was unrewarding. Definitive diagnosis of a patent foramen ovale is made by bubble contrast two dimensional echocardiography, a technique available only in some specialist cardiac laboratories which requires injection of small bubbles into the circulation and their detection in the heart by a type of sonar apparatus. This technique has a significant morbidity and not inconsiderable cost to the patient. It cannot be recommended as a routine screening procedure on all prospective divers, especially as the incidence of neurological decompression sickness is still relatively uncommon. Should clinical evidence of a septal defect be detected at a diving medical examination whether or not there is evidence of intracardiac shunt the prospective diver should be advised of the risks associated with the condition and be referred to a cardiologist.

There are factors other than just the Valsalva manoeuvre which can precipitate a right to left shunt across the atrial septum. Arborelius[6] reported that immersion in water may raise the right atrial pressure by 12 mm Hg. Similar pressure changes may be obtained by tilting the patient's head down, by elevating the legs, by wearing a tight wetsuit or by coughing. In the diver therefore these could have potentially disastrous consequences.

First aid for the diving casualty who is suspected of having cerebral gas embolism is then a matter of weighing up advantages and disadvantages. It has been traditional to recommend a head down tilt of 30° with the patient lying on the left hand side. There is good evidence from Gorman[7] and others that gas bubbles will move against the blood flow towards the most elevated part so that the head down position will tend to preserve the cerebral circulation from bubble overload. It is suggested that the left lateral position serves to keep bubbles away from both the pulmonary outflow tract and the interatrial septum. However, if the patient requires external cardiac massage or expired air resuscitation then he needs to be supine. In order to limit, as much as possible, the rise in right atrial pressure the head down tilt should be restricted to 10-15°. Other first aid measures include maintenance of the airway, 100% oxygen, intra venous fluids and urgent recompression. Gorman[8] has reported several incidents of re-embolisation after apparent recovery when the patient was placed head up. It is therefore essential that the patient remain head down until recompressed.

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SEVEN GREEN DIVERS HANGING FROM A LINE

A case reported to Project Stickybeak

Douglas Walker

There was nothing to indicate that this was to be anything other than an uneventful dive. The divers were taking an Advanced Diver Course under the care of an instructor and in the dive boat there remained two men with coxswain qualifications. There was 15 m (50 feet) visibility underwater, the dive was to be to 27 metres for 20 minutes, and there was to be a decompression stop at 5 metres, this being a routine precaution even when the proposed dive was within no-decompression limits. The "stop" was to be taken on a line suspended from a 20 litre plastic buoy with the group remaining together at all times. To prevent uncontrolled fluctuation of diver depth during the decompression stop due to the surge at the surface, the divers were advised to be slightly negatively weighted rather than aiming for neutral buoyancy at the surface.

The dive progressed as planned and the group of eight then clustered on the shot line at 5 metres for the 5 minutes planned. It was only after the instructor had twice needed to equalise his ears in the four minutes they had been hanging on the line that a suspicion rose in his mind that this was somewhat unusual so he checked his depth gauge. It read 15 metres. He looked up and saw that the buoy had been pulled underwater by their weight: it was now visibly indented by the pressure and was consequently now providing even less buoyancy lift to the eight divers. Though he at once started trying to indicate to his pupils that they had to let go of the line this took time, and after obeying this order they lost the buoyancy it had provided so sank deeper until some, if not all, returned to the sea bed.

One of the divers, A, now found that he was low on air and started breathing from the octopus regulator of diver B after indicating his need. The instructor saw that they were connected only by the air hose and put them into the correct, and safer, hands-on contact position before they started their ascent. He then saw another pupil swimming over the sea bed unable to reach the inflator hose and apparently lacking sufficient buoyancy to start ascending. After ensuring the vest's inflation he ascended with this diver and surfaced uneventfully.

On surfacing the instructor found that all was not well as divers A and B had surfaced in distress and were requiring an urgent resuscitation management. At this time there was still one diver not surfaced but fortunately this diver soon returned after completing the planned, but interrupted, 5 minute stop at 5 metres. Witnesses had seen A and B surface then float quietly face up and unresponsive. When the boat reached them one was able to make some response but the other was ash-grey faced and blood was seen in his face mask. They were quickly taken from the water and into the dive boat where A was noted as cold, breathless, and ash-grey faced, with chest pain and tingling fingers, and he was immediately placed in the Trendelenberg position and oxygen commenced. He had these symptoms when the boat reached harbour and he was airlifted with his buddy to a hospital having a hyperbaric unit. However as he appeared to have recovered by the time he reached the hospital and his buddy was similarly well they were both allowed to return home after a short period of observation.

These divers had apparently made an out-of-air ascent from an unstated depth (possibly 5 metres) because the octopus had soon exhausted the buddy's remaining air. They were observed to "pop to the surface". Diver B admitted that "he had held his breath" as he ascended. At the surface he was breathless and felt anxious but far less effected than was diver A. He had retained his weight belt. His scuba diving experience is unknown but diver A had been diving for one year.

When he got home diver A felt very tired and this tiredness was still present the next day after a good sleep so he went back to the recompression unit and on this occasion it was agreed to recompress him. This completely removed his feeling of fatigue and it did not recur.