The condition is a heart defect called patent foramen ovale, or PFO. This mouthful of Latin means a patent or evident, foramen or small opening that is oval. This oval flaplike opening is in the interatrial septum, the tissue between the atria, two of the four chambers of the heart. Everyone has this open hole in the heart before birth when it is needed so fetal circulation can bypass the lungs. A baby’s first breath should cause pressure in the left atrium to increase, so that the flap covering this opening closes and it normally eventually seals. Yet this valve flap stays loose in some persons, causing an atrial septal defect or interatrial shunt called PFO.

Estimates of PFO among adults vary from 10 percent to 30 percent. The variation can be attributed to differences in the sensitivity of various diagnostic techniques, because some PFOs may be forced open only under extreme stress. The incidence of PFO generally is clinically insignificant, since the pressure in the left side of the heart is generally greater than in the right, keeping the flap closed. Diagnostic testing for PFO involves monitoring blood flow during several repetitions of actions likely to force open the hole. A PFO may be rather large—up to 0.18 of a square inch / 120 square millimeters—or consist of a number of small openings. A PFO may not always be open, but a sudden strain and its rebound effects could cause a previ-
Right Lung

To

Lung

Left Lung

To

Pulmonary Veins
(from Lungs)

Aorta

Pulmonary Artery

Normal Heart

Bubbles enter the right atrium 1, pass to the right ventricle 2, and then travel to the lungs through the pulmonary artery 3.

Heart with Patent Foramen Ovale

Bubbles travel to the lungs as before, but they also push through the foramen ovale 4. Bubbles now in the left atrium 5, pass to the left ventricle 6, and travel through the aorta 7 to various body tissues and possibly even the brain.

PFO and DCS

During normal decompression, nitrogen is outgassed from peripheral tissues into the bloodstream. The lungs filter out gas bubbles in blood returning through the veins before they can reach the brain. However, if bubbles are able to leak from the right side of the heart to the left and thereby enter into the arterial circulation, they can lodge in the brain, causing dangerous neurological decompression sickness “hits.”

Given the high prevalence of PFO and the low incidence of DCS, it’s easy to conclude that it’s not generally a problem. Yet PFO may be the culprit when DCS hits early and hard, as in neurological symptoms and in undeserved hits taken by those who have violated no known diving rules. According to the British SubAqua Club, between 30 percent and 50 percent of all cases of DCS involve undeserved hits. Evidence suggests that size matters in PFOs.

P. Germonpre’s group at the Centre for Hyperbaric Oxygen Therapy at the Military Hospital, Brussels, Belgium, compared 37 divers who suffered neurological DCS with a matched control group of divers who never had DCS. The group used transesophageal contrast echocardiography to estimate PFO size. They concluded that PFO plays a significant role in unexplained cerebral DCS, but not of spinal DCS. Their study, published in 1998, reported that divers with DCS with lesions localized in the high cervical spinal cord, inner ear, or cerebellum or cerebrum of the brain had a significantly higher prevalence of PFO than did divers with DCS in the lower spinal cord. In unexplained DCS, this difference was significant only among those with large PFOs.

P.T. Wilmshurst’s group at the Department of Cardiology, St. Thomas’s Hospital, London, studied 61 divers with decompression sickness, 47 percent of whom had received undeserved hits. Most of these divers had PFO. In addition, divers whose DCS symptoms began more than 30 minutes after surfacing or who had joint pain only, actually had fewer right-to-left shunts, or PFOs, than did a control group of divers without DCS: 17 percent versus 24 percent. Yet,
PFO may be the culprit when DCS hits early and hard, as in neurological symptoms, and in undeserved hits taken by those who have violated no known diving rules.

Divers whose neurological symptoms began within 30 minutes of surfacing were 65 percent more likely to have PFO. In addition, they found that “rashes soon after surfacing were related to shunts [PFOs] but late rashes were not.”

**Neurological Damage**

J. Reul upset the diving community in 1995 with a report published in the British medical journal Lancet that stated that divers were more likely to have brain lesions than would a normal control group.

Wilmshurst’s study further found that the prevalence of multiple brain lesions in divers paralleled the prevalence of PFO, and he theorized that divers with PFO would have a higher risk of developing brain lesions. Follow-up research in a study led by M. Knauth in 1997 supported the view that PFO places divers at much greater risk for brain lesions.

Knauth’s group found fewer brain lesions in divers than did Reul’s earlier study. More than half of Reul’s divers had at least one brain lesion, while only 13 percent of Knauth’s diving group had brain lesions. The greater number of lesions in Reul’s group may be the result of differences in measurement technology and definition of lesion. Regardless, those who do extensive decompression diving, especially in conditions involving physical exertion during outgassing, might want to know whether they have a large PFO.

**Detecting PFO in Divers**

Three methods are used to diagnose PFO. All procedures use 0.2 to 0.3 fluid ounces / 6 to 8 milliliters of a contrast solution of agitated saline that contains air bubbles. This bubbly solution is injected into a vein during normal respiration and in conjunction with some repetitive action such as a cough or performing a Valsalva maneuver, which should open the PFO flap. Blood flow following injections is compared during the flap-opening maneuver versus the resting condition.

Three-dimensional transesophageal echocardiography, or TEE, is the most sensitive of measurement methods, but the most uncomfortably invasive since a probe is placed in the back of the throat, which must be anesthetized for optimal imaging of the interatrial septum. Bubble contrast transthoracic echocardiography, or TTE, uses electronic imaging measure across the chest; while transcranial Doppler, or TCD, the least sensitive of the three, uses a sonographic imaging from the of the right middle cerebral artery in the head.

E.K. Kerut’s group at Louisiana State University Medical Center, New Orleans, compared the three diagnostic methods in 26 divers with neurological DCS symptoms versus 30 normal control subjects. No difference was seen between the bent divers and the control subjects at rest. However, when a Valsalva maneuver was performed, the differences in detecting PFO became apparent. Surprisingly, TCD sonography, the least sensitive method, was found to be the best predictor of neurological DCS risk in divers.

Kerut’s researchers concluded that “it is...
likely that transcranial Doppler identified only larger right to left shunts [PFOs],” so this less sensitive method is actually a more effective tool for detecting meaningful PFO in divers, because only large PFOs seem to be implicated in decompression sickness. The smaller leaks detected by the other methods do not seem to be meaningful in DCS risk. This research appears to confirm the folk wisdom that cautions against being overly sensitive when dealing with a broken heart.

**Mending Broken Hearts**

The good news is that not only can PFO be repaired, but that the treatment is becoming safer as well. Instead of invasive cardiopulmonary bypass surgery, repairs are made by inserting a plug in the hole. The Amplatzer septal occluder plug made by the AGA Medical Corp., Golden Valley, Minn., currently is the treatment of choice. K.P. Walsh’s group reported in the *American Journal of Cardiology* in 1999 that AGA’s device had a higher rate of closing PFOs and took less fluoroscopy time. AGA’s device took only 13 minutes of X-rays, compared with the 24 minutes needed to insert the Sideris adjustable buttoned device, a plug made by Custom Medical Devices, Athens, Greece. There may be some doubt about this comparison, however, even though the same team did all of the surgeries. The Sideris plugs were implanted between September 1993 and February 1996, and the Amplatzer devices between December 1996 and March 1998. Increased experience by the surgical team might account for the difference.

F. Berger’s researchers at the Klinik fur Angeborene Herzfehler, Deutsches Herzzentrum Berlin, Germany, reported the results of implanting Amplatzer devices in 1999. Of the 200 patients studied, 98.1 percent had complete closure, and a functionally “trivial” amount of shunting in the remainder throughout 1,898 patient-months, or a follow up of 9.5 months per patient.

Walsh’s group at Alder Hey Children’s Hospital, Liverpool, England, reported in 1999 on the Amplatzer devices used to successfully repair PFOs in seven divers, aged 18 to 60, who had experienced neurological DCS. Follow-up contract echocardiography showed that leakage was fully eliminated in six of the divers, while one still leaked a few bubbles. All patients have received medical clearance to resume diving.

PFO repairs can be made even safer, according to a report published last February by P. Ewert’s researchers at Beitlung Fur Angeborene Herzfehler, Deutsches Herzzentrum Berlin, Germany. Walsh’s group found that implanting Amplatzer devices required an average of 14 minutes of X-ray exposure. In many cases, this exposure can be avoided, according to Ewert. Echocardiography provided sufficient guidance to position implants in 19 of 22 cases, with no increase in surgery time. However, using sound waves to position the devices required significantly more sedation of the patient.

**Safety Suggestions**

Divers with histories of undeserved hits or divers who tend to develop a skin rash during the earlier stages of outgassing are at greater risk. Divers, especially those with PFO, are cautioned by C. Balestra’s researchers to avoid strenuous leg, arm or abdominal exercise after decompression dives. Such activities could cause a rise and fall in chest cavity pressure sufficient to cause a rebound of blood loading to the right atrium of the heart. This rebound could increase the possibility of PFO leaks that could cause paradoxional nitrogen gas bubbles. In 1998, Balestra’s group reported research done at the Laboratory of General Biology in the Universite Libre de Bruxelles, Belgium, comparing various activities that could significantly change chest cavity pressure. Those most likely to open a PFO included breath-hold knee bends, a 172 percent increase; coughing, 133 percent; and maximal Valsalva maneuver, 136 percent. However, a gentle Valsalva maneuver, as used for normal ear-clearing, did not produce a significant post-release shift in blood flow.

Balestra’s group notes that PFOs have been found in divers struck with undeserved hits after over 1,000 safe dives, and hypothesizes that exertion in older divers could cause a previously closed, but only lightly fused, PFO may become largely patent over time. They said that in some persons, the shape of the inter-atrial septum “might not permit a right-to-left shunt in normal circumstances but allow a massive shunt if the diving pressure is sufficiently high.” They added that “several older and more experienced divers have been struck by repeated episodes of unexplained decompression illness after having sometimes performed more than 1,000 dives without any problems. In all of these divers, on TEE, a large PFO was detected.”

Balestra’s group concluded that divers with PFO should not perform any forced Valsalva maneuvers that cause a real rise in chest cavity pressure immediately after ascent because silent bubbles can be present in the central venous blood for two hours after a deep dive. They also advised that outgassing divers should not perform sustained isometric exercise or abdominal strains, such as defeating, lifting diving tanks or orally inflating a buoyancy device at the surface. Another important implication for diving instructors is that students should be taught to avoid performing forceful Valsalva maneuvers. Instead, using Frenzel or Toynbee maneuvers, which employ only jaw and throat muscles, should be given special attention in classes. Although post-dive Valsalva maneuvers would be more likely to occur in the “head” of the dive boat, rather than to equalize pressure in the head of the diver. Forceful ear clearing while descending again after a one-hour surface interval could place a diver at risk as well.

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