PATENT FORAMEN OVALE (PFO)

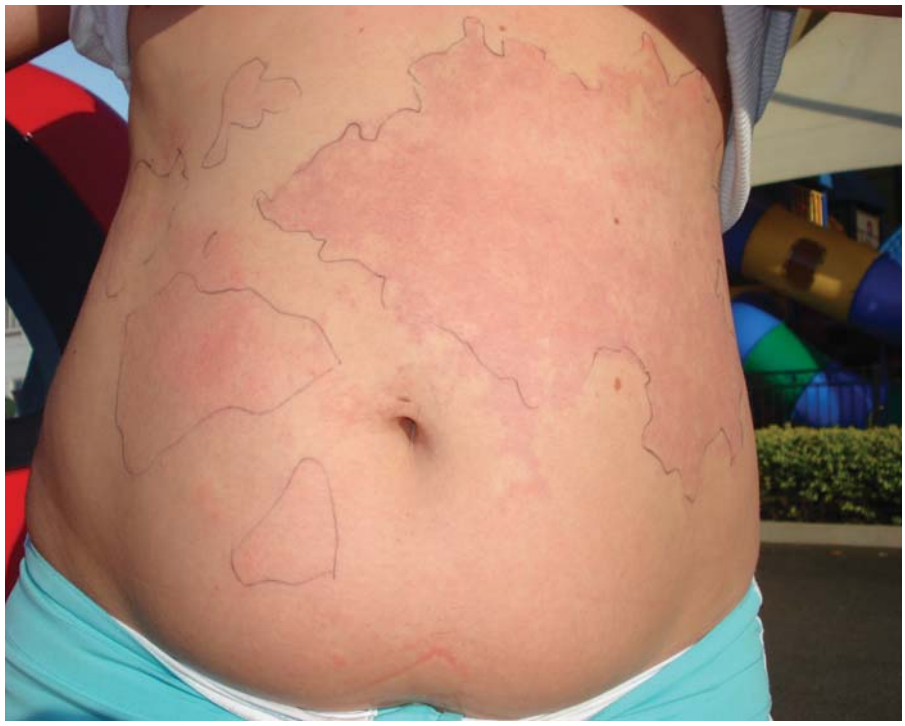
A diver reports her experiences

If you pursue a career in diving you'll find that over the years you notch up quite a few courses, including the mandatory first aid and oxygen updates. So why is it that so many divers don't recognise the fundamental symptoms of a dive injury?

For years I'd been hearing tales of what an incredible dive the SS Yongala wreck is (off the coast of north Queensland) but I'd never made it there. Then in January 2008, Santa Claus was extra kind and organised my dream dive trip for me and my buddy Nick. A double-dive on the wreck followed by a few days R&R in Cairns was the perfect start to a new year.

After a long hot day travelling, the night before the dive was textbook good behaviour. No alcohol, plenty of water and an early night in preparation for the next day's early start. Likewise, on the morning of the dives, we managed a light breakfast despite the excitement, and plenty of water on the way out to the site. It was a perfect Queensland day, with loads of sunshine and calm blue seas.

Both Nick and I had been instructing for a few years and are by no means inexperienced divers. So we planned our dives for the morning, and equipped with our computers and our own gear we did our first dive. We dived to a maximum depth of 28 m, for 47 minutes with an average depth of 18 m and a full safety stop, complete with silly photos on the line. After an hour's surface interval (including plenty more water) we jumped back in for our second dive. Although the maximum depth was 22 m, we spent most of the dive at the top of the wreck at 15 m and again did an extended safety stop.



Extensive decompression-related rash over Aleks' abdomen

While both dives were in every way remarkable with what we saw on the wreck (if you haven't dived the Yongala you need to remedy that situation!), they weren't at all remarkable in terms of our profiles. We stayed within the no-decompression limits of the most conservative computer, ascended very slowly up the line and did our safety stops. All in all – a perfect dive profile and well within our training.

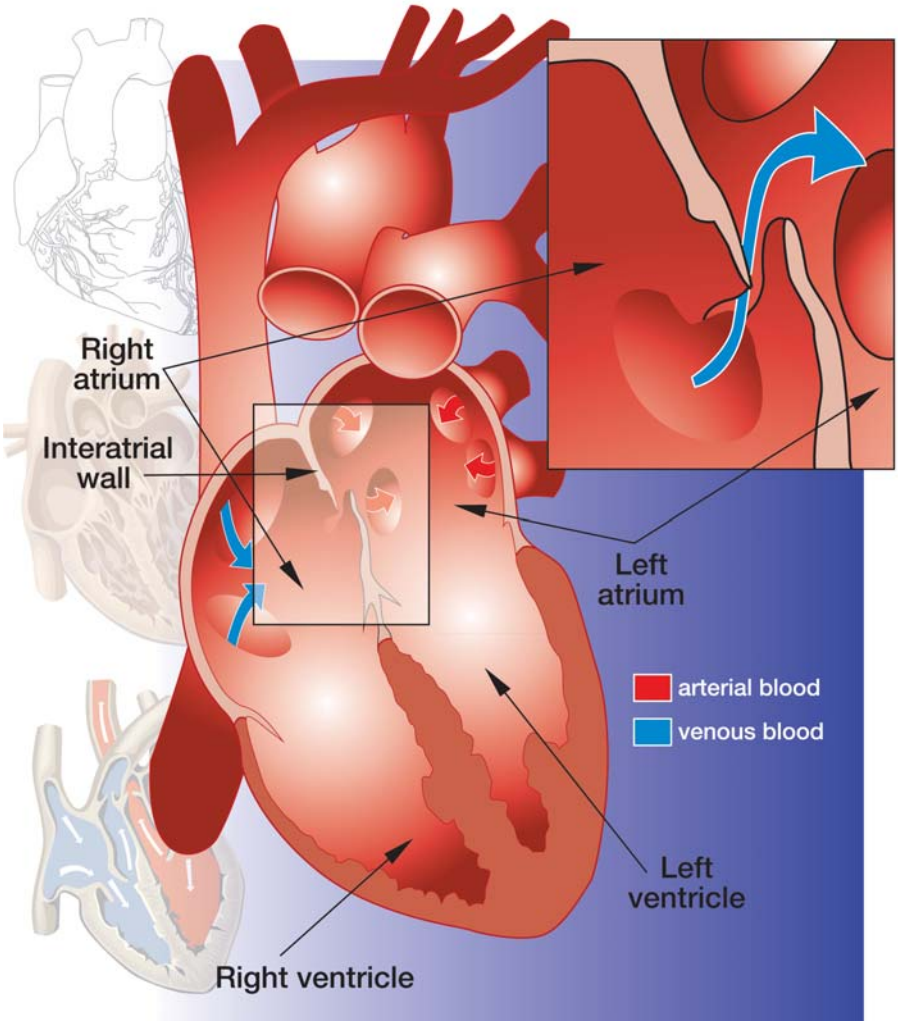
Case Report by
ALEKS WETZLICH

Climbing back on board the boat after a bit of a swim into the current, we began the journey back to shore. It was during this ride that my first symptoms appeared, though I didn't recognise them at the time. As it was pretty warm, I'd undone my wetsuit to the waist and I started to feel an itchy, achy, pinching sensation across my abdomen. After a while, the skin seemed to be a bit pink – almost welted. I presumed that I'd hurt myself climbing back onto the boat or that it could have been an over tight wetsuit (it was a suit that I'd worn when I was instructing full time, and I'd since put on… ahem… a couple of kilos).

By the time we got back to shore, I was in enough pain that Nick had to wash down my gear for me. When he'd finished and came to check up on me, the pinkish welts were beginning to turn more purple and look like bruises. We checked in with the dive crew but they also had no idea of what it could be. No one was particularly worried and consensus was that it was probably a wetsuit squeeze – where a pocket of air gets trapped inside your suit and as you descend that air reduces in volume creating a suction effect if the pressure can't be equalised.

Nick and I had a small lunch with the crew and got on the road to head up to Cairns. As we headed north the skin on my stomach began to look worse, becoming a darker purple with a 'marbled' or 'mottled' appearance. By this stage we were both unsettled by what we saw, so we drew lines around the markings to see if they were getting bigger. They weren't changing in size, however they *were* migrating across the skin on my abdomen!

After a four hour drive, we arrived in Cairns where we met up with some friends (a DM and another instructor) who also took a look but by this stage the pain was subsiding a bit so I decided to see how it was the next day. In the morning, the discolouration had mostly gone and I barely gave it another thought… for the next 12 months.



In 2009, Nick was studying for some technical diving courses, and came across some materials that seemed to describe my symptoms that day over a year earlier: cutis marmorata, a type of skin bend. We got on the internet and started researching and it was soon very clear that, much to my surprise, I'd had a form of cutaneous (skin) decompression illness (DCI). As the penny dropped, I don't know what shocked me more – that I'd had no idea for over a year or that 10 dive professionals of different backgrounds and levels of experience had seen and heard about my symptoms at the time and not one of us suspected DCI. Although there are some brief mentions of various skin DCI contained in the PADI and SSI Instructor materials, there isn't a great deal of information on how to identify this type of DCI. More specifically, there are no photos that would help us identify what the 'red rashes' or 'mottled bruising' indicates.

Nick contacted the DAN office in Australia and sent through an email with my dive profiles, signs and symptoms and our suspected diagnosis along with photos. John Lippmann replied to us confirming the likelihood of a skin DCI. He also suggested that this particular type of DCI was most commonly associated with a patent foramen ovale (or PFO), a hole in the septum wall between the two upper chambers of the heart that you have from birth. This came as quite a surprise considering how many dives I'd done in the past with absolutely no problems.

I saw a general practitioner and 2 cardiologists before I was finally referred to the Hyperbaric Unit at Prince of Wales hospital Sydney where, in conjunction with the cardiology department, I was tested and sure enough found to have a PFO. While the PFO itself was unlikely to cause me many problems in my normal life, in terms of diving I was advised that instructing, repetitive dives and deep dives were ill-advised

due to the increased risk of DCI while the PFO remained open. In order to close it, I'd have to go through a relatively simple and short procedure and six months of medication - none of it without risk. I couldn't imagine not being able to dive how I wanted, but I was 30 years old and the idea of any tinkering with my heart was more than a little scary.

To conclude my story, yes I had the PFO closed. A year later not only am I fit to dive, but have passed my medical and can teach again. I no longer have an increased risk of getting DCI, though I still have the same chance as everyone else, however much I might feel like bionic woman. And the moral of this tale? I don't know if it was dehydration, exertion, my dive profile or that tight wetsuit that caused the DCS that day. Whatever the reason for it, I'm glad that my delayed diagnosis had no residual effect on my diving and hope that my experience might encourage you to do a little research that might help you to recognise cutaneous DCI should you ever come across it.

Many thanks to John Lippmann for his help with a very accurate diagnosis and always taking the time to reply, Dr Michael Bennett at POW Hyperbaric for being the first to know what to do with a diver asking to be tested for a PFO, and Dr Roger Allan at POW for getting me appointments when there were none available, and of course for patching me up.

Eds Note: DAN and the hyperbaric centres that we deal with are encountering an increasing number of divers with skin rashes after diving. It is not clear why this is occurring but, personally, I suspect it may be partly due to the entended dive times, multiple dives and short surface intervals enabled by dive computers.

Many of these divers are tested for, and found to have, a PFO. Having a PFO increases the risk of DCI, although, given that DCI is an uncommon event, the risk remains very small.

Some divers elect to have a closure, and these procedures have become safer and more successful over recent years, although they are not without risk.

Many divers dive safely with a PFO but it depends on the type of diving undertaken, and to some extent, luck!

PFO: YOUR QUESTIONS ANSWERED

By Douglas Ebersole, M.D.

Q: What is a PFO?

A: A patent foramen ovale (PFO) is an incompletely sealed wall between the right and the left atria (see illustration on p17). Prior to birth, this wall develops from the septum primum, which grows upward, and septum secundum, which grows downward. These septa overlap, providing a 'trap door' (the foramen ovale), which allows oxygenated blood from the mother's placenta entering the right atrium to pass to the left atrium. At birth, the lungs expand and the pressure in the left atrium 'slams shut' the foramen ovale. Shortly after birth, the 'door' fuses in most people, but in about 27 percent of people it fails to fuse completely and results in a PFO.

The prevalence of PFO in divers with DCI is two times greater than in the rest of the population. In divers with neurological DCI, it is four times greater. The risk of DCI seems to increase with the size of the PFO. Based on these statistics, it is assumed that divers with PFO are at greater relative risk of DCI than divers without PFO; however, the only prospective study designed to measure directly the relative risk for DCI in divers with PFO is ongoing.

Q: How is a PFO diagnosed?

A: The diagnosis is made by injecting a small amount of air into a vein and observing its passage through the heart by echocardiography. An echocardiogram is performed by placing the ultrasound probe on the chest. Transthoracic echocardiography (TTE) is easy and noninvasive, but it detects a PFO in only about 10-18 percent of the population, around half of those who may have it. Transoesophageal echocardiography (TOE), where the probe is passed into the oesophagus after local anaesthesia and intravenous sedation, detects PFOs in 18 to 33 percent of the population. Even if TOE is more sensitive than TTE, there are many 'false-negative' results with both techniques, and it is important to realise that a well-conducted TTE with a proper technique may be more reliable than a TOE.

Q: What is transcatheter PFO closure?

A: Transcatheter PFO closure is the implantation of an occluder device across the PFO through a catheter inserted via the femoral vein in the groin. The occluders come in various shapes and forms, but most act like a double umbrella that opens on each side of the atrial wall and seals it. With time, tissue grows over and completely covers the occluder surface. The implantation procedure is performed in patients under local anaesthesia and intravenous sedation while the patient remains conscious. It takes less than one hour and can be performed on an outpatient or one-night-stay basis. Most patients can return to normal activity in two days, but they must take anti-coagulation medication and blood thinners for three to six months. Other restrictions include: no elective dental care (such as cleanings) for three months, no contact sports for three months, and no heavy lifting for one week. After implantation, divers must abstain from the sport for three to six months.

Q: What are the outcomes of transcatheter PFO closure?

A: Note: Due to the lack of data in divers, we present outcome data in patients undergoing PFO closure for the prevention of stroke. These patients have underlying medical conditions that may contribute to adverse outcomes.

Efficacy

- Complete closure of shunt: 95 percent
- Incomplete closure: 4 to 5 percent
- No improvement of shunt: 1 percent

Complications

- Overall mortality: 0.093 percent
- Rescue operation for device adverse events: 0.83 percent
- Serious complications
  - death, stroke, infection, bleeding, blood vessel injury: 0.2 percent
  - device movement or dislodgement: 0.25 percent
  - clot forming on device: 0.3 percent
  - major periprocedural complications: 1.2 percent.
  - minor midterm complications: 2.4 percent