

Patent foramen ovale (PFO) has been associated with an increased risk of decompression sickness (DCS) in divers. Pathophysiologically this has been ascribed to paradoxical embolization of nitrogen bubbles from venous blood to systemic circulation, resulting in obstruction of peripheral capillaries and ischemic injury. However, the role of PFO has been largely debated and experimental and prospective clinical data has been missing. It is of note, that this hypothesis is not only of theoretical importance. The proof of PFO as a causative factor of DCS and, importantly, of unpredictable events (unprovoked DCS) could affect millions of divers worldwide through improved therapy and prevention.

In our research we aimed to describe the pathophysiological role of PFO in decompression sickness and to determine whether the prevention of arterialization of post-dive venous gas emboli (VGE) would decrease the incidence of unprovoked DCS in divers. We have screened 489 scuba divers for the presence of PFO by means of transcranial color-coded Doppler ultrasonography. In a retrospective analysis we found that the incidence of unprovoked decompression sickness was 7% among these divers and that PFO was the only risk factor.

Subsequently, we have studied the occurrence of VGE and arterial gas emboli (AGE) in divers with large PFOs or after catheter-based PFO closure in a series of experimental studies in hyperbaric chamber. We found that Bühlmann regimen dives (recommended for recreational divers in Europe) were associated with lower occurrence of VGE than US Navy decompression regimen dives. In another study we demonstrated that after two provocative dive profiles, that generated significant number of VGE, catheter-based PFO closure led to elimination of post-dive AGE and DCS symptoms. In a following study conservative dive profiles (limited depth-time nitrogen exposure and prolonged decompression) led to decreased occurrence of

VGE, but not to complete elimination of AGE. When compared with catheter-based PFO closure, conservative dive profiles were less effective in AGE reduction after a single simulated dive.

Based on our results, we suggest that the presence of a PFO is associated with increased post-dive occurrence of AGE and an increased risk of DCS in divers. Our original findings are that i) PFO was the only risk factor for unprovoked DCS, ii) catheter-based PFO closure and conservative dive profiles reduced post-dive AGE occurrence and might thus prevent DCS in divers with a PFO. However, the clinical efficacy of DCS prevention using these measures needs to be confirmed in further experimental and, importantly, in long-term clinical follow-up studies.