Summary

Higher pressure under water and loss of gravity have special physiological effects on the human body and also influence the cardiovascular system. Cardiovascular disease is in addition to other medical problems a risk for diving. A careful examination is necessary for safe diving. In a cardiovascular evaluation there are three main questions: is there an illness or pathology which may cause either sudden cardiac death, syncope or impaired physical mobility.

Key word: diving; cardiovascular disease

Pathophysiology

There are some interesting and important cardiovascular effects on the human body when under the surface of water [1, 2].

The pressure in water depends on depth. Under water the diver’s legs are usually deeper than the rest of the body. Because of the higher pressure around the legs the blood pooled in the lower limbs is displaced into the vena cava, to the chambers of the right heart and the pulmonary arteries. The second mechanism causing increased central blood volume is loss of gravity. This redistribution of blood increases intrathoracic volume by up to 700 ml. Cardiac output, and thus the work of the heart, is increased by up to 30%. In a healthy diver the consequence is diving diuresis. An increase in central blood volume can cause decompensation in divers with heart disease.

The diving reflex is triggered by sensory stimulation of the trigeminal nerve and causes bradycardia down to 60% of the pre-dive heart rate and triggers intense selective peripheral visceral reflexory vasoconstriction in order to maintain blood pressure and perfusion of the tissues more sensitive to hypoxia. In a healthy diver this reflex is without consequences. But a person who is susceptible to cardioinhibitive dysregulation (vaso-vagal) could easily faint.

Another effect of submersion on the cardiovascular system is peripheral vasoconstriction: the colder the water the more intense the increase in vasoconstriction. Increased afterload can lead to angina pectoris in patients with coronary artery disease (CAD) or acute decompensation in patients with heart failure.

In healthy individuals peripheral vasoconstriction is, in addition to pulmonary factors, one of the main reasons for the rare, formerly termed “cold-induced pulmonary oedema in divers and swimmers”. It even occurs in warm water.

Risk/fitness to dive

The crucial question, however, is whether underwater diving is dangerous and what kills [3]. Data show 3 fatalities in 100 000 practitioners per year. This is a low number compared to mountaineering, with 533 fatalities in 100 000 climbers per year. Although diving is not a dangerous sport, illness is one of the risk factors. The study “Project Stickybeak” analysed 100 consecutive fatal diving accidents in Australia and New Zealand from 1980 to 1990 [4]. During the fatal dive 43% had serious medical problems and, evaluated with hindsight, 25% of the divers were unfit to dive. In the DAN (Divers Alert Network) registry 11% of all accidents are related to acute myocardial infarction and in only 23% of these cases was coronary artery disease known beforehand.

Diving medicine suffers from a lack of evidence-based knowledge. Performing prospective studies in the field of recreational diving to obtain reliable data is very difficult. Most criteria of fitness to dive are based on the experience of experts.

Periodic medical evaluation of fitness to dive has proven, by analysis of fatal diving accidents, essential...
for safe diving. Cardiovascular examination is a very important part of fitness to dive. Fitness to dive can also be compromised by medical problems affecting the respiratory system (e.g., obstructive pulmonary disease), the ear (e.g., structural problems affecting pressure balance), allergies (e.g., asthma), the endocrine system (e.g., diabetes), the nervous system (e.g., seizure disorders), the gastrointestinal system and psychiatry (see also table 1).

In cardiovascular evaluation of fitness to dive there are three main questions to be answered. Is there an illness or pathology which may cause either sudden cardiac death (SCD), syncope or a restriction of physical fitness resulting in a panic reaction?

The incidence of SCD in young athletes seems to be higher than estimated (1:100,000) in the past. New data from 2011 show an incidence of 1:43770 per year in men and 1:76646 in women.

Physical fitness ultimately depends on the function of the heart and circulation. Diving is not normally strenuous, being classified mild to medium. But a sudden unexpected current or an emergency situation needing a rescue operation needs sports standard physical fitness. Lack of capability to swim 500 meters carries an elevated risk of a diving accident. The American Society for Sports Medicine classifies the workload in recreational diving with a capacity of 5–10 METs. For recreational divers there are no definite recommendations. From a pragmatic point of view for sports standard physical fitness around 120% of expected workload is advisable. For professional divers 13 METs are considered adequate.

**Specific conditions**

**Coronary artery disease (CAD)**

In the “Project Stickybeak” study mentioned above the 45–50 and 50–55 age groups show a higher prevalence of fatal diving accidents than other groups (fig. 1) [4]. This is not due to a larger number of participants in diving sport in this age group as it is in the 20- to 35-year age groups. CAD is mainly responsible for this increased mortality. Diving with CAD involves the potential hazard of angina during effort, myocardial infarction and SCD. A major problem is silent ischaemia. Over 75% of fatal diving accidents related to myocardial infarction were previously asymptomatic. In the evaluation of fitness to dive, especially in the over-40 age group, we must bear CAD in mind (history and atherosclerotic risk calculation). Known CAD needs at least a yearly in-depth cardiovascular evaluation. The diver must present sports level fitness and no signs of ischaemia. After revascularisation (PCI or CABG) unfitness to dive is set at 6 months, after which the criteria are the same as in known CAD. Some authors even recommend refraining from diving for 12 months.

A CT-scan after thoracic surgery and also after CABG is requested by a few experts in diving medicine since the presence of any air pocket is deemed to preclude diving.

**Heart failure**

A diver with heart failure (left ventricular ejection fraction <50% or indication for medical therapy) is unfit irrespective of any symptoms. During diving an increase in central blood volume is caused first by displacement of pooled blood in the legs, because of the higher pressure around the legs, and second by loss of gravity (see also chapter “Pathophysiology”). A sudden increase in central blood volume can cause immediate decompensation of heart failure and therefore carry a high risk for safe diving.

**Valvular heart disease**

Severe valvular heart disease (stenosis or incompetence) carries a too high risk for diving (restriction of physical fitness, syncope or SCD). Moderate valvular heart disease
needs close cardiovascular evaluation to ensure haemodynamic relevance or that symptoms are not missed. After surgical correction (valve replacement or reconstruction) fitness to dive in a patient with a properly functioning valve depends on symptoms and physical performance. Anticoagulation is not a contraindication. The risk of accidents is very low in diving, even lower than in many other recreational sports.

**Arrhythmias**

In divers with arrhythmias fitness to dive depends on the underlying heart disease, or on the restriction of physical fitness during the tachycardia or bradycardia – e.g., lone atrial fibrillation without symptoms is no problem; otherwise AV-nodal reentry tachycardia with restriction of physical performance disqualifies from diving.

**Congenital heart disease/PFO**

In congenital heart disease a particular problem is to be noted: right-to-left shunt. Even when diving according to decompression rules nitrogen micro-bubbles will probably enter the veins. Normally they are filtered in the lungs. With a right-to-left shunt these bubbles may cross over to the arterial side and produce gas emboli in the arterial system. Decompression illness with neurological symptoms (DCI type II) will often be the consequence.

An atrial septal defect (ASD) is characterised by a left-to-right shunt with volume overload of the right heart chambers. However, during every heart cycle an ASD has in the early systole a short phase with right-to-left shunt and micro-bubbles could cross. An ASD is therefore a contraindication for underwater diving.

What of patent foramen ovale (PFO)? PFO has an incidence of 27% (higher in the young and lower in old age). It is not a hole like an ASD, but paradoxical embolism is possible. Nitrogen bubble shunting is therefore a risk. The problem is not the PFO; the bubbles are the problem.

In 1986, Moon et al. demonstrated in 30 divers with a DCI type II that the incidence of PFO in these divers was above average (37% overall and 61% in the severe cases). The risk of DCI is higher when associated with a PFO. In 1995, Reul et al. showed in an MRI study significantly more focal cerebral hyperintensities in divers compared with matched controls, but the controls had “diver’s spots” as well. These hyperintensities were classified as ischaemic brain lesions. The aetiology of the “diver’s spots” in the controls and the reason for them remained unexplained. A correlation analysis with the existence of a PFO was not done. The first study analysing the correlation between diving accidents and focal cerebral intensities in MRI was made by Schwerzmann et al. The incidence of DCIs in divers with PFO was 4.5 times higher. The “diver’s spots” in cerebral MRI were significantly more frequent compared to the non-diving controls, but there was no significant difference between divers with and without PFO.

In 2003, Balestra et al. analysed the distribution of these hyperintensities in the brain with fractal dimensions and compared the pattern with the angiography of the cerebral vessels, ischaemic brain lesions and multiple sclerosis lesions [5]. As expected, a significant correlation was found between the ischaemic MRI brain lesions and the angiography of the cerebral vessels. There was also a correlation between the distribution of the “diver’s spots” in MRI and the lesions in multiple sclerosis, which are not vascular-associated. However, no correlation was found between the “diver’s spots” and the angiography of the cerebral vessels. It can thus be concluded that the “diver’s spots” are not of ischaemic origin, in which case they are unlikely to originate from arterial nitrogen bubbles (gas embolisms). The origin and morphological correlation of the “diver’s spots” are still unknown.

Before promoting closure of PFO in divers with PFO, a risk evaluation for PFO closure and diving with a PFO is needed. PFO closure is accompanied by 5% peri-interventional complications, 1/1000 peri-/post-interventional severe events (permanent damage) and 7/100 peri-/post-interventional minor events (thrombus on the device, fracture of the device, displacement of the device, perforation of myocardium) [6, 7]. New data show better results with a peri-procedural complication rate of 2.2%, while no in-hospital deaths and none of the procedural complications resulted in permanent sequelae [8]. In the long-term follow-up 5–8% of the patients show post-interventional paroxysmal atrial fibrillation – one-time or recurrent [9]. A residual shunt after device closure is present in 0–50% of patients in older series and in 12% in newer data [8].

The risk of DCI type II (neurological symptoms) is 2.3 per 10 000 dives. The incidence of DCI type II in divers with a PFO is 5.2 per 10 000 dives [10]. This is a 2.25 times higher risk. For divers with a PFO the absolute risk of an accident with neurological symptoms is 1 per 1750 dives. Recreational divers only exceptionally perform more than 1000 dives.

The risk of PFO device closure is low, but the risk of DCI is very low. The device closure is therefore not the first therapy of choice, the low risk of the closure procedure is too high for a leisure activity.

The problem is not the PFO – the bubbles are the problem. Bubbles cause the decompression illness. As in paradoxical thromboembolism with a stroke where anticoagulation should be the primary therapeutic approach, the same strategy applies to divers with PFO, called “low bubble diving procedures” [11, 12]. Decompression procedures in former days were calculated (perfusion-limited nitrogen elimination model) only for arterial blood and several tissues with various different coefficients. Due to the problem of the venous bubbles with shunting over a PFO (or the lung), new de-
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Compression algorithms were developed to reduce the number of venous bubbles. The bubble load in venous blood can be reduced by these new decompression procedures to almost zero. This technique is called the “low bubble diving” decompression model. In addition, provocation of shunting over the PFO should be avoided. Hence Valsalva manoeuvre should not be performed while ascending from the last 10 meters and 2 hours after the dive. Third, avoiding strenuous work during decompression and thereafter will reduce the liberation of gas nuclei from tissues that might grow due to inert gas oversaturation.

In certain circumstances, e.g., a professional rescue diver (coast guard, fire brigade, police, etc.) with a large PFO, device closure must be evaluated. A rescue diver must sometimes ignore the rules of “low bubble diving”. For mixed gas diving (oxygen, nitrogen and helium) no decompression rules have been developed for “low bubble diving”. For these scenarios device closure of a large PFO should be discussed between a diving medicine physician with a well-established routine in this field and an experienced interventionalist.

Screening examinations for PFO are unnecessary due to the very low absolute risk. For divers with known PFO grade II and III “low bubble diving” procedures are recommended to reduce the risk of shunting through a PFO.

Conclusions

Cardiovascular disease is, apart from other medical problems (lungs, ears, central nervous system, endocrine system, etc.), a risk for the diver. Careful examination is indicated for safe diving. In cardiovascular evaluation there are three main questions: is there an illness or pathology which may cause either sudden cardiac death (SCD), syncope or a restriction of physical fitness?

- CAD with ischaemia is a risk factor for safe diving. The incidence of CAD increases after the age of 40. Many divers belong to this age group.

- Heart failure or the necessity of medical heart failure therapy is a contraindication for diving.

- Severe valvar heart disease is a too high a risk for the diver. Surgical correction does not preclude fitness to dive.

- Fitness to dive in arrhythmias depends on the underlying heart disease and symptoms.

- Congenital heart disease with right-to-left-shunt is a contraindication for diving. Diving with a PFO is possible with adaptation of the decompression algorithms called “low bubble diving”. The problem is not the PFO – the bubbles are the problem.

References