Long-Term Neurologic Damage and Brain Lesions in Recreational Divers

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Abstract
Whether recreational diving can cause brain lesions and/or neuropsychological impairment has been the subject of several studies, but an unequivocal consensus opinion has not yet been established. This article reviews some of the research on structural brain changes and neuropsychological testing in divers and focuses on recreational diving.

Key Words: Recreational diving · Brain damage

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Neurologische Spätschäden und Hirnläsionen bei Sporttauchern

Zusammenfassung
Ob Sporttauchen Hirnläsionen und/oder neuropsychologisch messbare Minderungen der Hirnleistungsfähigkeit verursachen kann, ist Gegenstand diverser Studien gewesen, eine eindeutige Klärung hat bisher jedoch noch nicht stattgefunden. Dieser Beitrag gibt einen Überblick über Studien zu den genannten Fragestellungen und fokussiert auf den Sporttaucherbereich.

Schlüsselwörter: Tauchen · Hirnschäden

Introduction

Does diving make you go daft? This question is sometimes posed anxiously, sometimes provocatively and even boldly and has been drifting through the recreational diving community for many years, providing a headline in newspapers with a wide variation in the level of seriousness. This article will introduce and discuss selected literature on the topic. The focus will be on recreational diving and chronic damage.

Various working groups have found evidence that long-term damage can occur in divers in the central nervous system. This evidence has been produced from either clinical neurologic and neuropsychological testing or from tests performed with the aid of modern imaging procedures, particularly magnetic resonance imaging (MRI).

Damage to the central nervous system can occur as a result of dive *accidents*, which can lead to severe and permanent loss of function or even death. Warren et al. [1] examined 14 divers with Decompression Sickness II (DCS II): based on the symptoms and the clinical examination, cerebral damage was suspected in four divers. In the MRI examination of the brain, acute pathologic changes were established in three of the four divers, and

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the localization correlated well with the clinical symptoms. That acute damage to the brain can occur as a result of a diving accident is undisputed.

Among the studies dealing with chronic brain damage, there are some that examine whether an increased number of neurologic or neuropsychological symptoms (e.g., memory impairment or inability to concentrate) occur among divers. Another approach is to examine divers' brains with MRI. The advantage of MRI is that it is very sensitive to evidence of changes in the brain, though the disadvantage is that you do not know the significance of these brain lesions for the affected diver. It would be ideal if there were a longitudinal study involving repeated neurologic, neuropsychological and MR brain exams of a large group of recreational divers over a longer period of time. The best thing would be to examine dive novices over perhaps a decade. However, such a study would be very time-consuming, expensive and correspondingly very difficult to carry out.

Studies on Neuro(psycho)logical Long-Term Damage and Structural Changes to the Brain in Divers

But now let us get to the point: which studies involve neurologic long-term damage and/or brain changes in divers? The following studies represent a selection.

The Norwegian Professional Diver Study

A Norwegian working group [2] examined 156 professional divers and 100 nondiving volunteers. The examination protocol included a history and clinical neurologic and electrophysiological tests (electroencephalogram [EEG]) as well as MRI of the brain. The authors definitely detected more abnormal EEG findings in the group of professional divers in the form of focal slow waves and sharp potentials mostly located in the temporal region. Abnormal EEGs correlated significantly with the exposure to saturation diving and the prevalence of decompression sickness. These abnormal EEG findings were seen in 18% of divers. The conduction times of acoustic signals in the brain stem were also prolonged in the dive group. These changes suggested chronic damage to the central nervous system but were not reflected in the MRI of the brain. However, the MRI part of the study had serious methodological flaws.

The crucial information from the Norwegian study on professional divers is that there is some evidence of chronic brain damage in professional divers.

Milwaukee Study on Compressed-Air Workers

In this study [3], 19 compressed air workers who worked on tunnel construction projects in and around Milwaukee in hyperbaric air were examined by brain MRI and compared with eleven control persons who belonged to the same group of workers but had not been exposed to compressed air. In the compressed-air workers' group, significantly more brain lesions were found than in the control group (152 vs. 22 brain lesions). The brain lesions in the compressed-air workers' group were very unevenly distributed. While at least 20 lesions were found in seven compressed-air workers (37%), the number of lesions for the twelve other compressed-air workers did not differ significantly from the number of lesions in the control group.

As compressed-air workers and recreational divers both breathe compressed air, the results of this study can possibly be extrapolated to divers, even though the conditions for the compressed-air work do not correspond exactly to those of diving because of the very much longer exposure times.

The Milwaukee study of compressed-air workers supplied the first evidence that there was a subcollective of workers exposed to compressed air who had a higher risk of suffering brain lesions identifiable by MRI as a result of hyperbaric exposure. The authors do not offer any explanation for this.

The Aachen Diver Study

The Aachen working group [4] was the first to examine a large collective of recreational divers for the presence of brain lesions. 52 recreational divers and 50 nondiving control persons were matched with regard to gender, age and risk factors and examined by brain MRI. A statistically significantly higher number of brain lesions was observed among the recreational divers. While 86 brain lesions were found among 52 divers, there were only 14 such lesions among the 50 control persons. Looking at the "dive group" separately, we notice that 68 brain lesions (79%) were found in 14 divers (27%; Table 1), while for the rest of the divers the number of brain lesions was not significantly greater than that of the control group.

In the Aachen diver study, the significant difference in the frequency of brain lesions was also based on a subgroup of about 25–30% of divers who show by far the greatest majority of brain lesions in the dive group. The authors do not offer a pathophysiologically plausible mechanism for the origin of the brain lesions in gen-

eral or their limitation to a diver subgroup in particular. Indeed, it is presumed that inert gas bubbles play a role, but these would have to occur intraarterially to be able to explain the brain lesions.

The Aachen diver study caused furore. The authors found significantly more brain lesions in recreational divers compared to nondivers. Among the divers a subgroup of about 25–30% appeared to be particularly affected. An explanation or a mechanism of origin was not offered.

Excursus: Patent Foramen Ovale (PFO)

All mammals – including humans – have a patent foramen ovale while they are in the womb, which usually closes after birth. However, in land mammals, in some individuals there remains a usually insignificant connection between the right and left atrium, the so-called persistent (or "patent") foramen ovale (PFO). The frequency of a PFO in an adult human population amounts to approximately 10–30% [5].

Although *venous* inert gas bubbles frequently occur during and after a dive [6], they cannot normally pass through the filter of the lungs [7] and therefore do not reach the arterial system. A de novo intra*arterial* origin for inert gas bubbles can be practically ruled out under the conditions of slow decompression that apply to recreational diving [8]. If there is a PFO (or another right left shunt), the venous inert gas bubbles can, however, bypass the filter of the lungs and reach the arterial system.

The Heidelberg-Mannheim Diver Study

The basic concept of the first Heidelberg-Mannheim diver study [9] was that divers who demonstrate MRI evidence of brain lesions are possibly those who have PFO. The suspected pathomechanism was the migration of venous inert gas bubbles via a PFO into the arterial system, leading to arterial embolisms which then caused brain lesions (Figure 1).

A brain MRI was performed in 87 divers to detect brain lesions. All divers also had a contrast-enhanced transcranial Doppler sonography to check for the presence of a PFO. A PFO was found in 25 divers, and in 13 cases this was classified as "large".

The MRI found 41 lesions in eleven divers. Seven of the divers without PFO only had a single brain lesion, while four divers with a PFO showed 34 lesions.

The frequency of multiple lesions was significantly higher in the group of divers with PFO than in the rest of the divers, especially if the PFO was large. Figure 2 shows an example of an affected diver.

Table 1. In the Aachen diver study the difference in frequency of brain lesions was caused by a subgroup amounting to 27% of the divers that comprised almost 80% of the brain lesions (modified from [4]).

Brain lesions (n)	Divers (n)	Controls (n)
0	25	40
1	8	7
2	5	4
3	5	3
4	2	0
5	0	0
6	4	0
> 6	3	0

The results of the first Heidelberg-Mannheim diver study suggest that a large PFO represents a risk factor for the occurrence of brain lesions in divers and that the probable pathomechanism is arterialization of venous inert gas bubbles. On the other hand, even the majority of divers with a large PFO did not have any brain lesions. A large PFO is therefore only one risk factor. It does not automatically lead to brain lesions.

The Swiss Recreational Divers Study

The most important other study that has been published on this topic is from the Swiss working group led by Schwerzmann [10]. The study design was similar to the first Heidelberg-Mannheim diver study, but also included a nondiving control group.

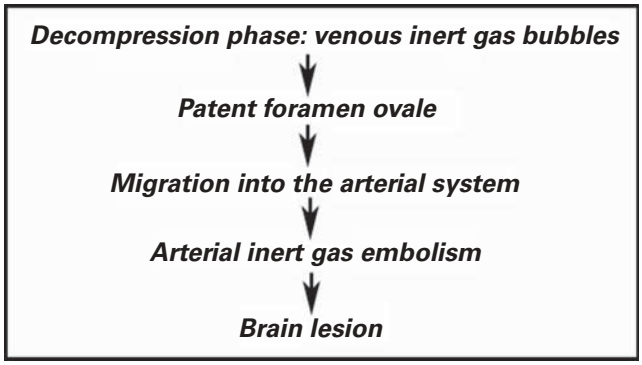

Figure 1. Suspected pathomechanism for the origin of brain lesions in recreational divers. Venous inert gas bubbles which frequently occur during and after decompression from relatively shallow depths can cross over into the arterial system via a patent foramen ovale or another right-left shunt. Brain lesions could be caused by arterial gas embolisms.

Schwerzmann et al. found twice as many brain lesions in divers with a PFO as in divers without a PFO. They also found significantly more brain lesions in (non PFO) divers than in the nondiving control group (Figure 3).

The twice-as-high number of brain lesions found by Schwerzmann et al. among divers with a PFO is well compatible with the results of the first Heidelberg-Mannheim diver study.

The higher number of lesions also found among all divers compared to the nondiving control group indicates that apart from the arterialization of inert gas bubbles via a PFO there are probably other mechanisms whereby divers can develop brain lesions, e.g., an "overflow" of the lung filter [11] or tears in the lungs with subsequent arterial gas embolism.

The Swiss recreational divers study found significantly more brain lesions in recreational divers than in nondivers. Divers with PFO were particularly affected by this – which matches well with the results of the first Heidelberg-Mannheim diver study and supports the hypothesis of paradoxical inert gas embolisms as (one) mechanism for the origin of brain lesions. The fact that the number of brain lesions was also increased in the group of divers without PFO suggests that there are also other mechanisms for the origin of brain lesions in divers besides PFO.

The Geneva "Memory Dive" Study

In this study [12], in 215 recreational divers the cerebral blood flow was measured using a SPECT examination and neuropsychological testing was performed. Both were correlated with the divers' diving activity. The authors found a negative influence of dive depth (> 40 m) and dive environment (> 80% cold-water dives) on global cerebral blood flow. Cognitive performance (predominantly speed and flexibility) was reduced in divers with a high frequency of dives (> 100 dives per year) or a high number of dives deeper than 40 m. A correlation of the results with MRI examinations ("brain lesions") and PFO ("paradoxical inert gas embolisms") would have been desirable but was not part of the study.

The Geneva "memory dive" study examined the long-term neurologic consequences for recreational divers for the first time. There was some evidence that diving under "extreme conditions" has a negative effect on global cerebral blood flow and certain neuropsychological tests. The authors found "risk factors" to be a high dive frequency (> 100 dives per year), dive depth (number of dives deeper than 40 m), and dive environment (> 80% cold water dives).

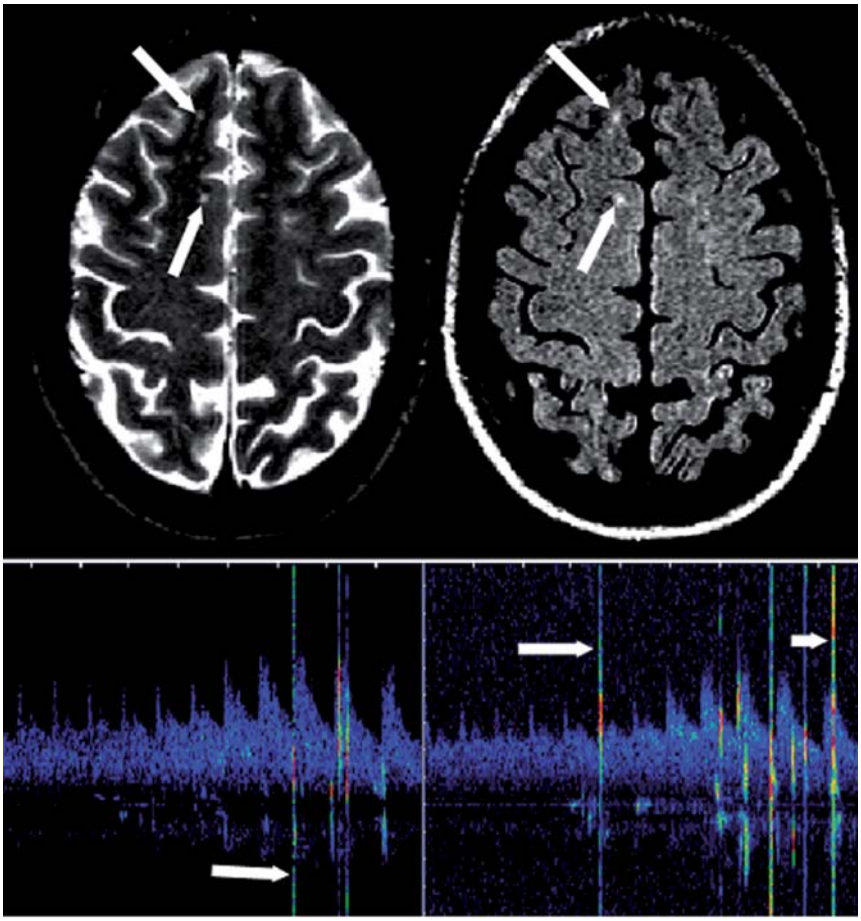

Figure 2. 42-year-old diver with 853 dives. The MRI (upper part of the figure) shows brain lesions in the subcortical white matter (arrows). In total, twelve brain lesions were found. The transcranial Doppler ultrasound (lower part of the figure) shows multiple high-intensity transient signals (arrows) in the flow signal of the middle cerebral artery: this is evidence of a large right-left shunt.

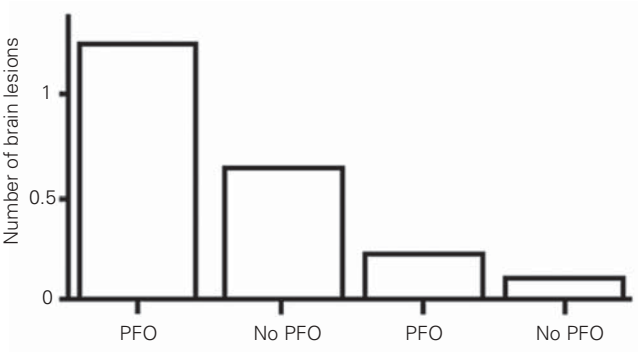

Figure 3. All divers showed more brain lesions than nondiving controls. Within the dive group, the divers with patent foramen ovale (PFO) had twice as many lesions as divers without PFO (modified from [10]).

Discussion

It is not easy: these studies of long-term damage in the field of recreational diving answer many questions, but leave just as many unanswered, as well as raising new questions.

Nevertheless, I will try to extract a few key messages.

(1) In the cerebral MRI there was evidence of an increased number of "brain lesions" or "white spots" or "areas of hyperintensity" in recreational divers compared to nondiving control groups. This message appears to apply to divers as a whole, but particularly to recreational divers with a large PFO.

(2) The higher frequency of brain lesions in divers with a large PFO implies that the arterialization of venous inert gas bubbles plays an important role in the origin of brain lesions. This mechanism is probably also the cause for the association of certain forms of decompression sickness with a PFO that has been found by other working groups [13].

(3) Affected divers should try to avoid or reduce the occurrence and migration of venous inert gas bubbles. The dive computer or dive tables should not be "pushed to the limits". It is probably advisable to avoid decompression dives in general. If inert gas bubbles might already be present in the venous blood, Valsalva maneuvers should be avoided (i.e., no "yo-yo" dives), as these can lead to increased migration of venous inert gas bubbles into the arterial system. The period before which flying may be allowed after a dive should preferably be generously extended for divers with a large PFO.

The advice given in this paragraph is plausible, but not scientifically proven.

(4) It must be emphasized that even divers without PFO do not have a license for unsafe diving: in the Schwerzmann study, even the group of divers without PFO showed a (slightly) increased number of brain lesions compared with nondiving controls. This underlines the fact that while a PFO represents a risk factor for brain lesions in divers, there are probably also other mechanisms whereby inert gas bubbles may become arterialized, e.g., by an "overflow" of the lung filter or by arterial gas embolism.

(5) Until now, there have not been any studies to show that brain lesions identified by MRI are connected with neuropsychological impairment. However, such evidence is difficult to obtain, as high numbers of cases are needed to create evidence for a slight reduction in neuropsychological performance.

Even though it has not been proven that brain lesions lead to neuropsychological impairment, common sense dictates that brain lesions should not be taken too casually.

(6) In the Geneva study, a reduction in cerebral blood flow and neuropsychological performance was found in recreational divers. These changes were associated with a high dive frequency (> 100 dives per year), dive depth (> 40 m), and dive environment (cold-water dives).

Testing for a correlation of the changes with brain lesions and a PFO was not subject of the study, although this would have been desirable.

Conclusion

Long-term neurologic damage in the form of neuropsychological performance or brain lesions should neither be dramatized nor played down. Brain lesions have so far not been linked with a reduction in neuropsychological performance, and the neuropsychological impairment in divers in the Geneva study occurred in relation to quite extreme diving behavior.

On the other hand, recreational diving is a leisure activity and the brain is the organ to which we owe our mental capacity and our personality. As in other areas of life, the same should apply to diving: moderation in all things.

In any case, there is so far no evidence of long-term neurologic damage in the form of reduction of cerebral capacity with moderate recreational diving.

Conflict of Interest Statement

I certify that there is no actual or potential conflict of interest in relation to this article.

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