Does decompression sickness lead to brain injuries?

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Introduction: When neurological damage occurs in divers the suspected primary cause is vascular gas bubbles. Entrapment of these bubbles may lead to cellular injury, cerebral oedema and increased permeability of the blood-brain barrier (BBB). Furthermore, studies of North Sea saturation divers have shown that divers report problems with concentration and memory more frequently than controls, and this can possibly be explained by CNS injuries. In this study, we have investigated effects of compression and decompression in a diving chamber on the rat brain using several MRI-protocols, including DTI, manganese-enhanced MRI (MEMRI) and dynamic enhanced MRI (DC-MRI), at several time points after decompression.

Materials and Methods: Rats (n=9) were compressed at a rate of 200 kPa/min to a pressure of 700 kPa, maintained for 45 min and then decompressed to the surface (100 kPa) at a rate of 50 kPa/min, breathing air throughout the procedure. Control rats (n=5) were kept at 100 kPa, breathing air for a similar time period. MRI was performed 1 hour, 7 days and 14 days after decompression on a 7T Bruker Biospec (Bruker Biospin, Germany) with a 72 mm volume coil for transmit and an actively decoupled rat head surface (100 kPa) at a rate of 50 kPa/min, breathing air throughout the procedure. Control rats (n=5) were kept at 100 kPa, breathing air for a similar time period. MRI was performed 1 hour, 7 days and 14 days after decompression on a 7T Bruker Biospec (Bruker Biospin, Germany) with a 72 mm volume coil for transmit and an actively decoupled rat head.

Discussion and Conclusion: Extensive MR protocols at three time points after decompression of rats have been used to look for effects on the brain caused by decompression. Two rats died immediately after decompression, and one additional rat died after the first MRI (rat with abnormal dynamic response, fig. 1g), which indicates the severity of the decompression protocol. However, we did not detect any structural differences in the brain between decompressed rats and controls using MRI and histology. Dynamic contrast enhanced MRI demonstrated increased relative signal intensity and area under the dce-curve (AUC) in decompressed rats compared to controls (p=0.002 and 0.015, fig. 1c, d, h, i). Dec- curves were in general similar in both groups (fig. 1e, f), but one of the decompressed rats displayed an abnormal dynamic response and controls (fig. 1g).

In conclusion, severe decompression does not seem to cause any long term structural or cellular injury to the brain tissue, but may cause temporary changes in brain perfusion and integrity of the blood-brain barrier.