Because clinicians require objectively demonstrable neurological deficits to confirm a diagnosis, the recognition of embolic events in the nervous system is generally restricted to the effects of ischemic necrosis produced by arterial occlusion. However, magnetic resonance imaging (MRI) has shown that lesser degrees of damage associated with small emboli are common, especially in the mid brain, and are usually clinically silent. They are frequently associated with atheromatous embolism in the elderly, but microembolic debris, such as fat, is common in the systemic venous return of healthy people and generally trapped in the microcirculation of the lung being removed by phagocytosis. However, pulmonary filtration may fail and microemboli may also pass through an atrial septal defect in so-called 'paradoxical' embolism. Studies of bubbles formed on decompression in diving have demonstrated the importance of pulmonary filtration in the protection of the nervous system and that filtration is size dependant, as small bubbles may escape entrapment. Fluid and even small solid emboli, arresting in or passing through the cerebral circulation, do not cause infarction, but disturb the blood-brain barrier inducing what has been termed the 'perivenous syndrome'. The nutrition of areas of the white matter of both the cerebral medulla and the spinal cord depends on long draining veins which have been shown to have surrounding capillary free zones. Because of the high oxygen extraction in the microcirculation of the gray matter of the central nervous system, the venous blood has low oxygen content. When this is reduced further by embolic events, tissue oxygenation may fall to critically low levels, leading to blood-brain barrier dysfunction, inflammation, demyelination and eventually, axonal damage. These are the hallmarks of the early lesions of multiple sclerosis where MR spectroscopy has also shown the presence of lactic acid. Significant elevation of the venous oxygen tension requires oxygen to be provided under hyperbaric conditions. Arterial tension is typically increased ten-fold breathing oxygen at 2 atmospheres absolute (ATA), but this results in only a 1.5-fold increase in the cerebral venous oxygen tension. The treatment of decompression sickness, and both animal and clinical studies, have confirmed the value of oxygen provided under hyperbaric conditions in the restoration and preservation of neurological function in the 'perivenous' syndrome.

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