# A 32-year-old man with acute bilateral leg weakness following recreational diving

A 32-year-old previously healthy recreational diver presented to the Ottawa Hospital with bilateral leg weakness and paresthesias. He was an experienced recreational diver. Earlier that day he had dived to a depth of 89 feet for a 20-minute period. He climbed to 15 feet for a 3-minute safety stop. Upon surfacing, the man became aphasic, tremulous and began vomiting. He was taken to a community hospital, where his symptoms resolved after he received oxygen. He was discharged home and subsequently experienced progressive leg weakness over the next hour. The man was brought to a second community hospital by his family, where a spinal cord injury was suspected. He was urgently transferred to our neurology service, about 9 hours after his dive.

The man reported bilateral leg weakness, tingling of the left leg and buttock and an inability to walk. He was a nonsmoker, was not taking any medications and has had 4 prior episodes of possible decompression illness related to diving that consisted of joint and abdominal pains. He had not been diving in the days preceding this dive. Neurological exam revealed bilateral lower extremity weakness, with scant antigravity strength on the left hip flexor. Left ankle flexion and extension were only 2/5. Pain and temperature sensations were intact, but he reported a slight decrease in pinprick sensation below the midthorax. Proprioception and vibration sense were intact. Reflexes were 2+ for upper extremities, 3+ bilaterally at the patella, with sustained clonus at both ankles. There was a cross-adductor reflex and bilateral Babinski's sign. Sacral sensation and rectal tone were preserved.

Blood work and EKG results were normal, as were a brain MRI and chest X-rays. An MRI of the spine revealed a 6 mm T2-enhancing lesion in the central cord at the level of T9 (Fig. 1, yellow arrows). This lesion did not enhance with gadolinium, which is less consistent with an acute demyelinating event or vascular neoplasm. A transthoracic echocardiogram revealed a mobile and possibly aneurysmal interatrial septum (Fig. 2, green arrow), consistent with a patent foramen ovale. This was further supported by a positive venous bubble study, wherein air bubbles visibly traveled from the right atrium to the left atrium and ventricle via a septal defect (Fig. 2, red arrows).

The patient was given a diagnosis of Type II decompression sickness (DS) with spinal cord dysfunction and immediately started hyperbaric oxygen treatment according to the Hyperbaric Unit Protocol.[1] He received 5 more treatments prior to discharge. One month after his last treatment, the patient continued to experience numbness of his left foot, left knee and medial thigh, but had recovered full strength. His left leg was spastic with clonus at the left ankle, but he was able to walk with a slight limp. Sensory exam was normal and abdominal reflexes were absent on the left.

DS and arterial gas emboli are generally described together under the global term decompression illness.[2] Type II DS reflects the involvement of the CNS or cardio-respiratory system. Although the pathophysiology is unclear, spinal cord DS may be caused by nitrogen bubbles deposited in neural tissue and vasculature. The presence of a patent foramen ovale is associated with an increased risk of neurological DS in divers, although the role of screening echocardiography is controversial.[3] Although MRI is often normal, it may reveal increased T2 signalling that does not enhance with contrast.[4] Nevertheless, diagnosis should be based on a history of exposure to a decompressive event, followed by the onset of symptoms. Recognition of the syndrome and rapid transfer to a facility with a decompression chamber is crucial for recovery.

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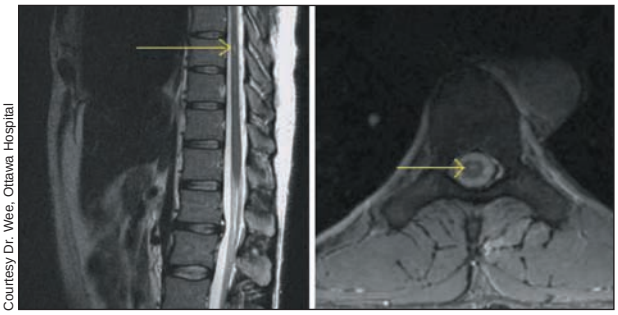
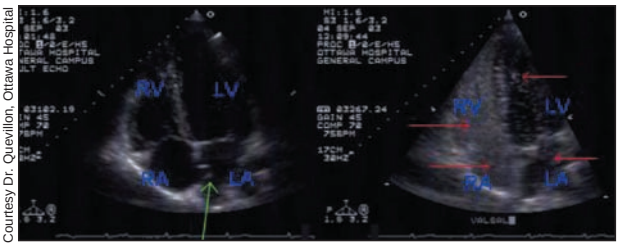


Courtesy Dr. Wee, Ottawa Hospital




Courtesy Dr. Quevillon, Ottawa Hospital

## References

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3. Saary MJ, Gray GW. A review of the relationship between patent foramen ovale and type II decompression sickness. *Aviat Space Environ Med* 2001;72 (12):1113-20.
4. Newton HB. Neurologic complications of scuba diving. *Am Fam Physician* 2001;63(11):2211-8.

To see a video of the echocardiogram from this case, see www.cmaj.ca/cgi/content/full/170/12/1792/DC1