The medial medullary syndrome is characterised by the development of ipsilateral weakness of the tongue and contralateral hypeesthesia and hemisensory disturbance [1]. Ipsilateral palatal weakness and facial nerve weakness may also be part of the syndrome. As a form of brainstorm ischaemia paramedian medullary infarction is rare when compared to the more common lateral medullary infarction. We report a case of medial medullary syndrome in a young and fit weightlifter without any obvious risk factors for vascular disease but with the finding of patent foramen ovale on transoesophageal echocardiography. A 37-year-old right-handed man was exercising with weights at the gym when he struck the front of his head off one of the bars. He felt unsteady for a few seconds but quickly recovered to the point where he was lying on a bench and lifting a bench press above him when he suddenly developed a sensation that the room around him was oscillating from side to side. He also developed slurring of speech, a sensation of impaired hearing and numbness and weakness of the right arm. This episode lasted approximately 3 min and seemed to resolve completely. He continued lifting weights but about 2 h later he had a similar episode with a sensation of external rotation, slurred speech and right arm weakness and numbness. From the time of the incident until review in clinic 3 months later he had occasional transient episodes of right arm numbness and noted a persisting problem with movements of his tongue. He denied using anabolic steroids or other performance-enhancing drugs.

He had a past history of cystinuria and had an episode of labyrinthitis at the age of 12. There was a family history of renal calculi in his father. He was an unemployed steel engineer who neither smoked nor drank alcohol.

On examination he was a well-built muscular individual with a blood pressure of 110/70. There were no cardiac murmurs, carotid bruits, nor any stigmata to suggest a hyperlipidaemic syndrome. Neurological examination showed decreased left palatal movement, both voluntarily and to stimulation. The left side of the tongue was wasted and the tongue deviated to the left on protrusion. The remainder of the neurological examination was normal. The clinical diagnosis was that of a medial medullary lesion, probably vascular in origin in view of the sudden onset.

Haemoglobin 17.4 g/dl, packed cell volume 0.53 1, platelets 251 X 109/1 and erythrocyte sedimentation rate 1 mm/h, all normal. Prothrombin time, activated partial thromboplastin time, urea and electrolytes, liver function tests, calcium and phosphate, random blood sugar, autoantibody screen, syphilis serology and urinalysis were normal. Fasting cholesterol and triglycerides were normal. Prothrombotic screen, including protein C, protein S, antithrombin III, and anti-phospholipid antibodies, was negative. Magnetic resonance imaging of the brain was normal. Magnetic resonance angiography showed no abnormality of the cerebral vasculature. Transoesophageal echocardiography using air contrast bubbles showed the presence of a patent foramen ovale with a minor right-to-left atrial shunt. The degree of shunting was not increased by performing the Valsalva manoeuvre.

The onset of symptoms in this man coincided with the lifting of a heavy weight, which we feel is crucial to the pathophysiology of this case. The Valsalva manoeuvre occurring during such acute effort is associated with reversal of flow through the patent foramen ovale, which may place the patient at risk of paradoxical embolism [2–4]. Screening tests for other causes of stroke in young adults were negative.

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