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TO WHOM IT MAY CONCERN

**Re : Wayne Michael DOUGLAS, dob : 9-9-66**  
**NHI : PJU6268**

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I have been a Consultant Neurologist for 15 years. I received neurological training in Auckland, New Zealand, and at the Mayo Clinic, Rochester, Minnesota, USA. I have been working full-time as a Consultant Neurologist at Auckland City Hospital since 1995.

I have seen Wayne Douglas on two occasions at Auckland City Hospital: on 12 December 2001 and on 1 October 2003.

Mr Douglas has told me that one day in May 2000 he woke during the night with intense vertigo and unsteadiness on his feet. The vertigo had partially resolved after eight hours but he remained unable to walk without support for a week. He was seen urgently at the Saitama Hospital where an MRI scan of the brain was negative. His symptoms were thought to be due to Vestibular Neuronitis and he was prescribed several medications which he took for one month. For 2.5 years following this he had a fluctuating, low-grade sensation of imbalance in his head.

In June 2000 Mr Douglas consulted Dr [REDACTED]. Dr [REDACTED] apparently told Mr Douglas that he had Sylvian Aqueduct Syndrome. He was prescribed five different medications to be taken concurrently. Mr Douglas then developed a wide range of additional symptoms, some or all consistent with exposure to benzodiazepine medications.

Neurologic examinations by me in 2001 and 2003 have not shown any residual neurological signs.

I would agree with doctors at the [REDACTED] Hospital that Mr Douglas most likely had an episode of Acute Vestibulopathy in May 2000. This condition has multiple synonyms including Vestibular Neuronitis, Acute Vestibular Neuritis, Acute Labyrinthitis and Neurolabyrinthitis. The preferred expression is Acute Unilateral Peripheral Vestibulopathy<sup>1</sup>. It refers to an acute lesion of the vestibular apparatus (semicircular canals and related structures) or vestibular nerve on one or other side. Most clinical neurologists will encounter one or more cases each year, although the patient is sometimes not seen until after the acute symptoms have resolved. The clinical features include intense vertigo, vomiting, and unsteadiness of gait. Neurologic examination in the acute phase shows unidirectional nystagmus with the fast phase away from the affected ear<sup>2</sup>. Some authorities believe that Acute Unilateral Peripheral Vestibulopathy is due to reactivation of Herpes Simplex virus infection<sup>3</sup>. The patient's symptoms usually subside gradually over days to weeks.

A range of neurological conditions (for example, cerebellar infarction, multiple sclerosis) can mimic Acute Unilateral Peripheral Vestibulopathy, but all are excluded or made very unlikely if the brain MRI scan is negative. Methylprednisolone significantly improves the recovery of peripheral vestibular function in patients with Acute Unilateral Peripheral Vestibulopathy<sup>3</sup>. Patients are sometimes also administered intravenous fluids and anti-emetics.

The Sylvian Aqueduct Syndrome is rare and seldom encountered in modern neurological practice. A literature search in February 2008 (Pubmed) using the expression "Sylvian Aqueduct Syndrome" yielded just 23 articles since 1966, with only 3 articles in the past 20 years. It refers to a syndrome

whose features include vertical gaze restriction, abnormal pupillary reaction, upper lid retraction, and convergence-retraction eye movements. Paralysis of convergence and skew deviation may also occur. Sylvian aqueduct syndrome usually occurs in patients with shunted hydrocephalus whose shunts become blocked<sup>4</sup>. Single case reports have also described the syndrome in patients with midbrain infarction<sup>5</sup>, multiple sclerosis<sup>6</sup>, thalamic haemorrhage<sup>7</sup>, tumours in the pineal region<sup>8</sup> and unilateral midbrain lesions<sup>9</sup>. Mr Douglas apparently did not have the above constellation of neurological signs, but more importantly his brain MRI scan did not show hydrocephalus or any other disorder which can produce the Sylvian Aqueduct Syndrome.

I do not know of any reason, theoretical or otherwise, why benzodiazepine medication would have had a role in the treatment of Mr Douglas in 2000. Benzodiazepine drugs do not have useful anti-emetic or anti-vertiginous properties and have no clear role in the treatment of Acute Unilateral Peripheral Vestibulopathy. Further, benzodiazepine drugs would not be effective in treating hydrocephalus or any of the other conditions which may produce the Sylvian Aqueduct Syndrome.

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