Whiplash injury

Whiplash injury may deregulate the biological clock

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Several risk factors may predispose to circadian rhythm disturbances

n this issue Bloch *et al*¹ (*pp* 1178–80) describe a patient who developed a free running circadian rhythm after a whiplash injury. The concisely documented case report strongly suggests that a whiplash injury may result in a treatable circadian rhythm disorder. This is important for neurologists and other caregivers involved in patients with a chronic whiplash syndrome (CWS).

The demonstration of a disturbed circadian rhythm (by clinical history, and supported by actigraphy and salivary melatonin) can help CWS patients, people in their environment, and insurance companies to understand one of the possible reasons why sleep, concentration, and memory may be disturbed after a whiplash trauma. A disturbed circadian rhythmicity is a well known cause of these symptoms.²

The possibility of a circadian rhythm disorder following a whiplash injury was demonstrated not until the early nineties of the last century by Patten *et al.*³ They described a 13 year old boy who developed a delayed sleep phase syndrome after a whiplash injury. From that time on several other studies confirmed their findings.⁴

The reason why a whiplash injury can disturb circadian rhythmicity is not well

understood. Nagtegaal *et al*⁵ suggested that a whiplash injury might damage the cervical part of the neural connections between retina and pineal gland. Consequently the endogenous melatonin rhythm delays, resulting in a delayed sleep phase syndrome.

Bloch et al¹ found an aneurysm that might have damaged the nucleus suprachiasmaticus-the location of the biological clock. If this is true, then neurologists should see circadian rhythm disturbances more frequently in patients with aneurysms or tumours in the vicinity of the nucleus suprachiasmaticus. It seems unlikely that the aneurysm should have deprived the biological clock from all light-dark information, which plays a key role in the development of free running circadian rhythms. The aetiology of the aneurysm is uncertain. The lack of clear clinical symptoms of the aneurysm makes a posttraumatic origin unlikely, so probably the aneurysm pre-existed. In that case the alternative explanation of the authors for the origin of the circadian rhythm disorder is more reliable. Namely the patient could have a predisposition to a short circadian period, which she did not manifest before the accident because of her regular lifestyle. The phase of convalesce at

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home could have unmasked the short circadian period.

Several risk factors such as clock gene polymorphisms, and irregular lifestyle may predispose to circadian rhythm disturbances. The presence/absence of some of these risk factors may explain why the same whiplash injury causes circadian rhythm problems in one patient and not in the other.

Non-pharmacological treatment with social "zeitgebers" can improve circadian rhythms, as the case report shows. Well timed bright light, chronotherapy, and melatonin³ are other possibilities to shift circadian rhythms into the desired direction.

It can be expected that more studies will be published in the future showing the importance of biological clock functions in cervical and brain injuries. They will encourage placebo controlled trials comparing the effectiveness of different treatments.

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