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How does spinal cord injury lead to obstructive sleep apnoea?

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Spinal cord injury (SCI) to the cervical region almost always results in impaired ventilation and cough. A less widely appreciated, respiratory-related impact is the considerable increase in sleep disordered breathing (SDB) as compared to the general population.

The apnoeas and hypopneas associated with SDB can generally be classified as 'central' or 'obstructive'. A central event reflects a lack of respiratory effort, whereas an obstructive event indicates a respiratory effort against a narrowed or occluded airway. The growing literature on this topic indicates that obstructive sleep apnoea (OSA) is the predominant form of SDB after SCI (Fuller *et al.* 2013; Chiodo *et al.* 2016). In a cross-sectional survey, Berlowitz *et al.* (2012) observed that >90% of persons with neurologically complete cervical SCI had OSA. Central sleep apnoea also occurs after SCI, particular in tetraplegia (Chiodo *et al.* 2016).

Why does SCI lead to an increase in airway obstructions during sleep? This is not a straightforward question, particularly since the ability to activate the pharyngeal muscles which regulate upper airway patency is typically not impaired after SCI. Many explanations have been offered, and it is likely that the increased incidence of OSA does not reflect a single mechanism, but rather reflects a complex interaction between multiple variables. Factors including unopposed parasympathetic neural activity and increased nasal resistance (Gainche *et al.* 2016), increased soft palate volume (O'Donoghue *et al.* 2018), medications, and neuroplastic changes in the respiratory control system (Fuller *et al.* 2013) may all contribute. The underlying mechanisms may also dynamically change during progression from acute to chronic SCI.

A new report in this issue of *The Journal of Physiology* from Wijesuriya and colleagues sheds new light on this complex physiological question (Wijesuriya *et al.* 2018). The electryomographic activity of an important pharyngeal dilator muscle – the genioglossus (GG) – was studied in two groups of individuals with OSA. One group had chronic SCI, and the other did not. When a negative pressure pulse (i.e. a collapsing force) was applied to the pharyngeal airway, approximately one-half of the SCI cohort were unable to reflexly activate the GG. In contrast, 8 of 9 able-bodied control subjects demonstrated GG activation to this stimulus. In those SCI subjects who were able to activate the GG, the response occurred with a markedly delayed latency as compared with the control group. Lastly, a subset of the SCI group showed only reflex *inhibition* of GG activity – a response that was never observed in the control group. Collectively, these new results show that SCI is associated with fundamental changes in the way that the GG muscle responds to airway collapsing pressures. These changes are not beneficial compensatory responses, but rather appear to predispose the airway to collapse. The data support the hypothesis that altered and/or impaired GG reflexes are a significant component of the complex physiological sequela that leads to OSA after SCI.

Like all good science, this new report raises further questions, and sets the stage for interrogation of the underlying physiological mechanism(s). Establishing the neuroanatomical substrate for the impaired GG reflex and determining the cellular/molecular mechanisms will likely require the use of animal models. Lastly, the importance of this new work should be emphasized. OSA is directly associated with reduced quality of life after SCI, and likely decreases the overall health status in this population (Berlowitz *et al.* 2012). Adherence to standard OSA treatment is low in persons with SCI (O'Donoghue *et al.* 2018), and a deeper understanding of the mechanisms leading to increased incidence of OSA should lead to improved treatment options.

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Additional information

Competing interests

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