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Editorial

Delayed Central Sleep Apnoea Following Cervical Laminectomy

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Sleep apnea is a clinical symptom in sleep-related breathing disorders that are divided into Obstructive Sleep Apnea (OSA), Central Sleep Apnea (CSA) and mixed apnea by analysis using polysomnography. OSA is defined as a cessation of airflow for at least 10 sec during sleeping. The event is obstructive if during apnea there is effort to breathe. On the other hand, in CSA there is no effort to breathe during sleep, which is a less common clinical problem.

It has been rarely reported in patients with upper cervical lesions caused by rheumatoid arthritis, Arnold-Chiari type 1 malformation, anterior C1-2 osteochondroma and osodontoideum. Such complication has been rarely described following cervical laminectomy.

The occurrence of a delayed sleep apnoea is an extremely rare complication of cervical laminectomy for spondylotic myelopathy. Ventilatory insufficiency has been, of course, described as a complication following cervical vertebral and spinal cord surgery. However, the event of breathing disorders occurring after an operation such as cervical laminectomy has been more rarely described, and additionally, the event of a CSA syndrome occurring after cervical laminectomy is to be considered exceptional.

So far only three papers have been reported in literature concerning such an event. Naim-ur-Rahman in 1994 reported on a case of postoperative CSA following C3-C6 laminectomy, occurring right after surgery and associated with spyncterial incontinence, that spontaneously recovered three weeks after onset [1]. My Group reported in two papers the occurrence of delayed onset (nearly three weeks after surgery) of CSA not associated to any other neurological sign of spinal cord damage (postoperative neurophysiological tests showed instead an improvement compared to preoperative tests) [2,3].

The mechanisms underlying such an event are difficult to be interpreted. The Breathing Anatomy and Physiology Breathing is a rhythmic motor behavior generated and controlled by hindbrain neuronal networks. Neural circuits controlling breathing in mammals are organized within serially arrayed and functionally interacting brainstem compartments extending from the pons to the lower medulla. The core circuit components that constitute the neural machinery for generating respiratory rhythm and shaping inspiratory and expiratory motor patterns are distributed among three adjacent structural compartments in the ventrolateral medulla: the Bötzinger complex (BötC), Pre-Bötzinger complex (pre-BötC) and Rostral Ventral Respiratory Group (rVRG). The respiratory rhythm and inspiratory-expiratory patterns emerge from dynamic interactions between: (i) excitatory neuron populations in the pre-BötC and rVRG active during inspiration that form inspiratory motor output; (ii) inhibitory neuron populations in the pre-BötC that provide inspiratory inhibition within the network; and (iii) inhibitory populations in the BötC active during expiration that generate expiratory inhibition. Nevertheless more recent models describe interacting populations of respiratory neurons spatially distributed within the BötC and pre-BötC and rostral ventrolateral medulla that contain core circuits of the respiratory Central Pattern Generator (CPG). Network interactions within these circuits along with intrinsic rhythmogenic properties of neurons form a hierarchy of multiple rhythm generation mechanisms. The functional expression of these mechanisms is controlled by input drives from other brainstem components, including the retrotrapezoid nucleus and pons, which regulate the dynamic behavior of the core circuitry. The emerging view is that the brainstem respiratory network has rhythmogenic capabilities at multiple levels of circuit organization. This allows flexible, state-dependent expression of different neural pattern-generation mechanisms under various physiological conditions, enabling a wide repertoire of respiratory behaviors. Some models consider control of the respiratory CPG by pulmonary feedback and network reconfiguration during defensive behaviors such as cough. The location and fiber arrangement of the descending respiratory pathways (involuntary respiratory pathway) in the ventral reticulospinal tract is close to the descending micturition pathways within the upper cervical cord [3].

Interestingly CSA syndrome in pathologies involving the Cranio-Vertebral Junction (CVJ), such as axis rheumatoid arthritis, Arnold-Chiari type 1 malformation, anterior C1-2 osteochondroma

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osodontoideum and occipital encephalocele can be referred to a respiratory center dysfunction. More precisely a dysfunction in or adjacent to the pre-Bötzinger complex in lower medulla oblongata might be postulated to determine a loss of normal autonomic response to chemical changes in the blood.

On the other hands spondylotic compression on the spinal cord is definitely anatomically far for the lower medulla (C3-C6) and also surgery at this level is too distant to hypothesize a direct compressive/traumatic mechanism determining a disturbance to the pre-Bötzinger area respiratory centers. Is we critically analyze postoperative MR after posterior decompression, we do not find spinal cord displaced posteriorly thus determining an angular deformation of the lower medulla oblongata that could justify such a mechanism.

In conclusion no definitive mechanism has been recognized so far for delayed post cervical laminectomy. A transient dysfunction of the reticulo-spinal fibers directed to the nucleus of the phrenic nerve can be speculated although neither emi-diaphragm paralysis, nor the prominent nocturnal sleep-related disorder is associated to.

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