ICU
MANAGEMENT & PRACTICE

Dyspnoea, Psychological Trauma and PTSD

In a recent review, the authors hypothesise that some patients suffer unnecessary psychological sequelae such as depression, anxiety and post-traumatic stress disorder (PTSD) from dyspnoea that persists despite pharmacologic sedation during severe acute respiratory failure. The authors point out that the primary cause of dyspnoea is not the contraction of respiratory muscles. Air hunger arises from an increase in respiratory drive.

Dyspnoea and air hunger can be psychologically traumatising and may not necessarily be relieved by mechanical ventilation, sedation and other routine therapies used for respiratory failure. In fact, some of these therapies may actually worsen the symptoms. Administration of neuromuscular blocking agents (NMBAs) in particular could exacerbate the problem because the patient is then unable to communicate, and the observable signs of dyspnoea are obliterated. Hence, it is important to understand this problem because that is the only way proper treatment protocols can be devised - protocols that can retain the benefits of therapies for refractory respiratory failure but, at the same time, minimise psychological trauma.

Air hunger is the most common form of dyspnoea in non-ICU patients and in ICU patients who are mechanically ventilated. This air hunger can have long-term psychological effects. It is estimated that clinically significant symptoms of PTSD, including nightmares and flashbacks of dyspnoea, occur in around 22% of ICU survivors. These symptoms may also be accompanied by depression, anxiety and other behavioural disorders. Air hunger activates the cortical regions of the brain, comprising the salience network. These regions integrate external stimuli and emotions that are necessary to sustain homeostasis and survival. The salience network is believed to be involved in the development of PTSD.

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Mechanically ventilated patients are routinely sedated so that patient discomfort can be avoided. However, some patients treated with NMBAs often remember aspects of their care despite heavy sedation. They may experience a loss of a sense of reality and a loss of control over their bodies. Benzodiazepines, for example, are frequently used for sedation in the ICU but have a negligible effect in treating discomfort from dyspnoea. In critically ill patients, excessive sedation can thus be harmful and increases the risk of psychological sequelae.

A proper distinction must be made between sedation, pain relief, dyspnoea relief and relief from traumatising stress. Different drugs have different effects. For example, low-dose opiates provide greater relief from dyspnoea and are not linked to the development of post-ICU PTSD. Another example is propofol. It is a very effective amnestic sedative but may still be ineffective at suppressing activation of the amygdala. Hence, patients can still develop maladaptive fear responses to stimuli. Deep sedation with propofol does not diminish pain-related activation in the cerebral cortex. More data is needed to determine the effects of sedatives such propofol, ketamine or dexmedetomidine on post-ICU psychiatric disease.

During the last few months, clinicians have been managing patients with COVID-19. Early studies suggest psychiatric sequelae following coronavirus disease as well.

Overall, there is significant opportunity for research in this area. The pathophysiology of post-ICU mental illness and risk factors should be explored further. Preventing traumatic experiences completely may not be possible but recognising that mental illness post-ICU exists can pave the way towards improved strategies. Ongoing research in this area is critical to improve understanding of psychological trauma in the ICU and beyond.

Source: CHEST

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Published on : Sun, 7 Feb 2021