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Dexmedetomidine

A Viewpoint by Dr Erkan Hassan

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Dexmedetomidine provides an alternative to parenteral clonidine in markets outside the US and a unique modality within the US as a sedative for intensive care unit (ICU) patients. Both agents produce sedation via α_2-adrenoceptor agonism; however, dexmedetomidine has approximately 8 times higher affinity for these receptors than clonidine.

With its haemodynamic properties, which occur as an extension of its pharmacological effects, appropriate patient selection for dexmedetomidine is important. The α_2-agonist activity of dexmedetomidine may produce hypotension and bradycardia in 20 to 25% of patients. Therefore, ICU patients who are hypovolaemic or severely vasoconstricted should not receive dexmedetomidine. Although dexmedetomidine will most likely have a role in critically ill patients with multi-organ failure, it is unclear if it will be effective alone or will need to be given in combination with currently available sedatives in this specific patient population.

The administration of dexmedetomidine also provides unique dosing considerations for the ICU clinician compared with other sedative agents. Bolus doses (in the traditional sense) of dexmedetomidine should not be given. Rather a rapid 10-minute infusion of 6 μg/kg/h followed by a continuous infusion of 0.2 to 0.7 μg/kg/h is the preferred method of administration.

The pharmacological properties of dexmedetomidine will require new clinical considerations in the approach to ICU sedation. It produces rapid and effective sedation in mechanically ventilated postoperative patients; however, a unique feature of this drug is the ability of patients to be easily awakened. Clinicians should not misinterpret this easy awakening as a subtherapeutic effect, but rather recognise it as the optimal clinical endpoint for dexmedetomidine. It is yet to be determined if this characteristic of dexmedetomidine will have cost implications by avoiding prolonged sedation in the ICU.

Dexmedetomidine is the first sedative with analgesic properties that does not produce clinically significant respiratory depression. The clinical implications of these effects are 2-fold. First, analgesic requirements will be reduced (although not eliminated) in patients receiving dexmedetomidine especially as they make the transition from the operating room to the ICU. Secondly, dexmedetomidine does not need to be discontinued prior to extubation. In clinical trials, dexmedetomidine was continued throughout the extubation process and for at least 6 hours after extubation without impairing respiratory drive.

Instead of simply adding another drug to a patient's regimen, we should reformulate the optimal approach to sedation in the ICU to include appropriate patient selection, rational dosing principles and monitoring parameters to ensure the safe and effective use of all sedative drugs. The availability of dexmedetomidine will assist in our continuing quest to improve the comfort of postoperative mechanically ventilated patients while they recover in the ICU. ▲