The use of dexmedetomidine in paediatric intensive care

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Sir,

In recent years, there has been increased interest in dexmedetomidine (DEX), in both anaesthesiology and intensive care [1]. This alpha-2 receptor agonist has been implicated as the drug of choice for sedation of children after cardiac surgery [2], which is undoubtedly associated with its lack of depressive effects, moderate sedative and analgesic action and beneficial antiarrhythmic effects.

We would like to share our experiences with this drug, which has been used in the Paediatric Intensive Care Unit since 2013 after the approval by the hospital therapeutic committee. Until June 2014, dexmedetomidine (Dexdor, Orion Pharma, Espoo, Finland) was used 38 times in 33 children with respiratory failure, as an infusion in an initial dose of 0.5–1.4 µg kg−1 h−1. The drug was administered to discontinue the supply of earlier agents (mainly opioids) during weaning from a ventilator or to change the mode of sedation. During treatment, the following parameters were recorded: heart rate, respiration rate, SpO2, arterial pressure (invasive or non-invasive method). Moreover, diuresis and parameters of acid-base balance were monitored. Changes in parameters exceeding 20% of the baseline value were considered adverse. The data are presented in Table 1.

The drug was administered as follows: postoperatively in 12 children; during pneumonia treatment — 6 children; after injuries — 2 children; sepsis — 2 children; bronchopulmonary dysplasia — 2 children; and, comprising 1 case each, bronchiolitis, airway burns, congenital heart defect, cardiomyopathy, pulmonary hypertension, toxic epidermal necrolysis (Lyell’s syndrome), spinal muscular atrophy, encephalopathy and intracranial haemorrhage. Although in 8 cases, DEX was the only sedative used, in other cases, it was an element of multi-drug sedation: midazolam was used in 22 children, morphine in 15, propofol in 14, ketamine in 4, magnesium sulphate in 2, along with fentanyl, clonazepam, fenobarbital and clonidine, comprising 1 case each. After the inclusion of DEX, morphine was discontinued in 11 cases and mechanical ventilation was successfully completed in 9 children. In total, during DEX infusion, ventilation was discontinued and trachea extubated in 24 children (63%) (following completion of earlier infusions of opioids and other sedatives). In 3 children, DEX was discontinued due to no beneficial effects. In 2 children, a decrease in the heart rate by 20% of the baseline value was observed. Bradycardia subsided after a dose reduction or withdrawal of the drug. One patient died during DEX treatment, an event which was unrelated to its use, the cause of death being acute myeloid leukemia and intracranial haemorrhage. One child mistakenly received too high a dose of DEX (8.6 then 4.3 µg kg−1 h−1), for 11 days in total. Nonetheless, no cardiovascular disorders or other adverse effects, including withdrawal symptoms, were observed.

Initially, DEX was recommended for sedation lasting maximum 24 h; currently, its longer use has also been reported, even for many weeks in the doses of 1.7–3.5 µg kg−1 h−1 [3–5]. However, such a long dosage period leads to the development of withdrawal symptoms.

Besides the intravenous route of DEX administration, its nasal use in children was also found to be useful. DEX effects on the cardiovascular system result from the stimulation of alpha-receptors in the central and peripheral nervous system. This can cause bradycardia (despite normal sinus rhythm and QT intervals), persisting for up to 90 min after withdrawal [7], which can be prevented with ketamine [8]. On the other hand, therapeutic effects of DEX in the treatment of supraventricular tachycardia have been reported [9]. DEX administered in boluses can also temporarily increase pulmonary systemic resistance; large doses (3 µg kg−1 h−1) are likely to increase arterial pressure, while small doses usually induce hypotension, limited by the slow way of administration. The maximum recommended dose of a DEX bolus administered within 5 sec, which does not induce circulatory depression in half of patients studied, is 0.49 µg kg−1 [10]. DEX has potential neuroprotective effects [11], especially on the hypoxia-damaged brain. The use of DEX during paediatric angiography prevents renal damage caused by contrast medium, resulting from limited increases in the levels of vasoconstricting substances, endothelin-1 and rennin [12].

Our observations reveal that DEX can be a valuable supplement of sedation with other agents, when their action is insufficient; more importantly, it can replace opioids, when they are no longer needed and there is a risk of a patient’s adverse reactions to their withdrawal. Since DEX does not

Table 1. Data regarding patients and dexmedetomidine infusion (means ± SD)

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<tr>
<th>Age (months)</th>
<th>Body weight (kg)</th>
<th>Day of ventilation on DEX inclusion</th>
<th>Initial dose (µg kg−1 h−1)</th>
<th>Time of DEX use (days)</th>
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<tbody>
<tr>
<td>50.7 ± 5</td>
<td>20.7 ± 21.4</td>
<td>12.4 ± 31</td>
<td>1.0 ± 1.1</td>
<td>4.4 ± 2.6</td>
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induce respiratory depression, we imply its special usefulness while weaning a patient from a ventilator. However, in order to determine the exact role of DEX in paediatric care, further prospective studies are required.

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References:

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