the increases in the disease burden attributable to overweight, obesity, and physical inactivity.

After decades of efforts to call attention to the disease burden attributable to cardiovascular risk factors,[5,6] these findings represent an important step toward their complete and critical description. This epidemiological evidence should be expected to direct the debates on the new challenges for maintaining and promoting cardiovascular health in the coming years, as well as specific actions that enable the application of multidisciplinary approaches to the prevention and management of the risk factors and their associated comorbidities. Given the complexity of this issue and the fact that the interactions among the determinants of health vary from one context to another, progress in the attempts to control cardiovascular risk factors will require sustained efforts on a regional, national, and international scale.

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CONFLICTS OF INTEREST

The opinions expressed in this letter are the responsibility of the authors and, thus, do not necessarily reflect the point of view of the organizations in which they work. The authors declare that they have no conflicts of interest.

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REFERENCES

1. Catalá López F, Álvarez Martín E, Gènova Maleras R, Morant Ginestar C. Relación en España entre la investigación sanitaria financiada por el Sistema Nacional de Salud y la carga de enfermedad en la comunidad. Rev Esp Salud Pública. 2009;83:137–51.
2. Gènova-Maleras R, Álvarez-Martín E, Morant-Ginestar C, Fernández de Larrea N, Catalá-López F. Measuring the burden of disease and injury in Spain using disability-adjusted life years: an updated and policy-oriented overview. Public Health. 2012;126:1024–31.
3. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380:2224–60.
4. Global Burden of Disease Study. Global Burden of Disease Study 2010 - Western Europe Results by Risk Factor 1990-2010. Seattle: Institute for Health Metrics and Evaluation (IHME); 2012.
5. Romero T, Romero CX. Prevención cardiovascular estancada: tendencias alarmantes y barreras socioeconómicas persistentes. Rev Esp Cardiol. 2010;63:1340–8.
6. Mazón-Ramos P. Riesgo cardiovascular en el siglo XXI. Cómo detectarlo en prevención primaria. Cómo controlarlo en prevención secundaria. Rev Esp Cardiol. 2012;65 Supl. 2:3–9.

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Prevention of Opioid Withdrawal Syndrome After Pediatric Heart Transplantation: Usefulness of Dexmedetomidine

Prevención del síndrome de abstinencia en el postoperatorio de trasplante cardiaco: utilidad de la dexmedetomidina

To the Editor,

Opioids and benzodiazepines are the sedative and analgesic drugs of choice for pediatric patients in cardiac intensive care units. Long-term use of these drugs is associated with the development of withdrawal syndrome. In pediatric patients this is difficult to diagnose due to a wide range of nonspecific symptoms and the scarcity of validated diagnostic scales. In pediatrics, incidence of withdrawal syndrome is 35% to 57%; the greater the accumulated dose and length of treatment, the more frequently it occurs.[1] Accumulated doses of phentanyl of >1.6 mg/kg or >5 days of infusions are associated with developing withdrawal syndrome; with doses of >2.5 mg/kg or >9 days of infusions, incidence of up to 100% has been described.[2]

In pediatric heart transplantation, due to the scarcity of donors the waiting list times increase, extracorporeal circulatory support becomes necessary, cardiac intensive care units stay lengthens, and the probability of developing withdrawal syndrome increases.[3] Dexmedetomidine, an α_2-adrenergic agonist, is a sedative and analgesic with possibly beneficial effects in controlling withdrawal syndrome.[4] As both a sedative and analgesic agent that does not cause depression of the respiratory center, it has gained widespread acceptance for use in pediatric cardiac intensive care units in the USA. Numerous publications report its efficacy and safety.[5] However, evidence of its use in preventing withdrawal syndrome, particularly in the cardiac posttransplantation period, is scarce.[4]

We describe our experience with dexmedetomidine in managing withdrawal syndrome and supporting opioid discontinuation in 2 pediatric heart transplant recipients.

Case 1. Infant aged 11 months transplanted for dilated cardiomyopathy due to myocarditis, who had required 7 days of extracorporeal membrane oxygenation support and ventricular assist device implantation during the 20 days preceding transplantation. Sedation and analgesia were administered in a continuous infusion of opioids, benzodiazepine, and propofol. The patient experienced withdrawal syndrome and morphine dosage could not be reduced despite having started the standard management protocol. The accumulated opioid dose was 1.39 mg/kg in 33 days. We decided to start treatment with dexmedetomidine in continuous infusion with an initial dose of 0.75 μg/kg/h and maximum of 1 μg/kg/h, enabling us to rapidly reduce the opioid without withdrawal syndrome reappearing (Figure A). The patient remained hemodynamically stable after

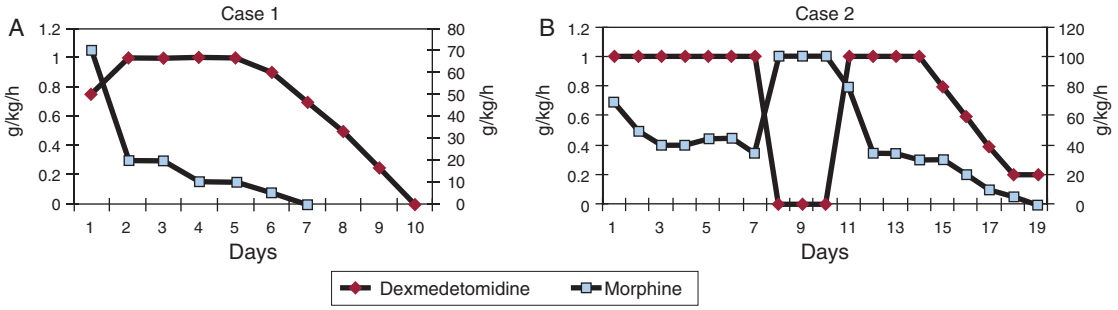

Figure. Opioid-dexmedetomidine transition. A: Case 1. B: Case 2.

Table
Clinical Course of Hemodynamic Parameters After Initiating a Dexmedetomidine Regimen

Vital signs	Patient 1		Patient 2	
	Start	After 1 h	Start	After 1 h
SBP, mmHg	100	91	97	93
DBP, mmHg	65	62	60	59
MBP, mmHg	82	76	68	64
Heart rate, bpm	130	125	155	155
Respiratory rate, rpm	30	25	31	48
Central venous pressure, mmHg	10	8	22	20

DBP, diastolic blood pressure; MBP, mean blood pressure; SBP, systolic blood pressure.

starting the dexmedetomidine regimen and no side effects of its use were observed (Table).

Case 2. A 5-year-old boy was transplanted for noncompaction cardiomyopathy; he had previously been hospitalized for 11 months with ventricular assist device, requiring 5 days postoperative extracorporeal membrane oxygenation. Sedation and analgesia were administered in a continuous infusion of opioids, benzodiazepine, and propofol. He experienced withdrawal syndrome and morphine dosage could not be reduced. The accumulated opioid dose was 1.21 mg/kg in 16 days. We decided to start a dexmedetomidine regimen with continuous infusion at 1 μg/kg/h. After initiating treatment, the morphine dosage was tapered over 6 days without withdrawal syndrome reappearing. At that point, humoral rejection was diagnosed and the patient needed a change of sedative and analgesic to undergo diagnostic tests and venous access cannulation. The dexmedetomidine regimen was suspended for 4 days; morphine was increased and propofol added. Later, dexmedetomidine administration was restarted, propofol suspended, and morphine tapered with cessation at 7 days (Figure B). The patient remained hemodynamically stable and no dexmedetomidine-derived side effects were observed (Table).

In both patients, the use of dexmedetomidine to prevent opioid withdrawal syndrome in the cardiac posttransplantation period was beneficial, with good hemodynamic tolerance and no related adverse effects.

Similarly, Finkel et al. described the beneficial effect of dexmedetomidine in discontinuing opioid treatment in 2 pediatric patients during the cardiac posttransplantation period.[6] In the denervated heart, the pharmacodynamics of the medication depend on the site of action. Drugs that directly affect receptors in the donor heart will be effective, whereas those that act centrally or via autonomic reflexes will not produce the desired response.[3] In our patients, dexmedetomidine was beneficial because of two mechanisms: first, it blocked the catecholaminergic response associated with withdrawal syndrome, reflected in the absence of hypertension and tachycardia; second, cardiac denervation prevented bradycardia, the principle adverse effect of dexmedetomidine. With regard to the latter, during the heart transplantation or pediatric heart surgery postoperative period most patients have pacemaker leads implanted, which enable us to increase their heart rate if necessary. These findings confirm the beneficial effects of dexmedetomidine not only to as an adjuvant to sedation but also to prevent postoperative withdrawal syndrome after heart surgery, and particularly heart transplantation. More thorough prospective studies are needed to establish protocols for its use.

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REFERENCES

1. Ista E, Van Dijk M, Gamel C, Tibboel D, de Hoog M. Withdrawal symptoms in children after long-term administration of sedatives and/or analgesics: a literature review. "Assessment remains troublesome". Intens Care Med. 2007;33:1396–406.

2. Tobias JD. Tolerance, withdrawal, and physical dependency after long-term sedation and analgesia of children in the pediatric intensive care unit. Crit Care Med. 2000;28:2122–32.
3. Schure AY, Kussman BD. Pediatric heart transplantation: demographics, outcomes, and anesthetic implications. Paediatr Anaesth. 2011;21:594–603.
4. Oschman A, McCabe T, Kuhn RJ. Dexmedetomidine for opioid and benzodiazepine withdrawal in pediatric patients. Am J Health Syst Pharm. 2011;68:1233–8.
5. Tobias JD, Gupta P, Naguib A, Yates AR. Dexmedetomidine: applications for the pediatric patient with congenital heart disease. Pediatr Cardiol. 2011;32:1075–87.
6. Finkel JC, Johnson YJ, Quezado ZMN. The use of dexmedetomidine to facilitate acute discontinuation of opioids after cardiac transplantation in children. Crit Care Med. 2005;33:2110–2.

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Electrical Storm Secondary to Acute Myocardial Infarction and Heart Failure Treated With Left Stellate Ganglion Block

Tormenta arrítmica secundaria a infarto agudo de miocardio e insuficiencia cardiaca tratada mediante bloqueo de ganglio estrellado izquierdo

To the Editor,

We report the case of a 52-year-old man, ex-smoker, with no other cardiovascular risk factors, who was admitted for heart failure of 2 weeks' duration. Chest x-ray showed cardiomegaly and bilateral alveolar edema. Electrocardiogram showed sinus tachycardia with left bundle branch block. The initial blood test suggested evolving myocardial infarction.

The patient received inotropic therapy, intravenous diuretic drugs, and noninvasive mechanical ventilation. Echocardiogram showed left chamber dilatation and severe ventricular dysfunction with anterior akinesia. Cardiac catheterization revealed nonrevascularizable severe 3-vessel coronary artery disease.

He was admitted to the coronary care unit, where he made slow but favorable progress. On the 10th day of admission, he underwent polymorphic ventricular tachycardia that was treated by defibrillation. No ischemic, electrolytic, or metabolic triggers or QT prolongation were identified that could explain the arrhythmia (Figure). Intravenous amiodarone was started, and within 12 h there were 13 episodes of ventricular tachycardia that were treated by defibrillation, intra-aortic balloon pump, endotracheal intubation, and mechanical ventilation. Beta blockers and lidocaine infusion were administered intravenously. Within 12 h there were multiple episodes of nonsustained ventricular tachycardia and 10 persistent episodes that were treated by defibrillation. Ablation was rejected due to extreme clinical instability.

Local anesthesia was applied to the left stellate ganglion as an additional measure of sympathetic block. Percutaneous puncture was performed using the anterior approach at the level of C6. Initially, 10 mL of 0.25% bupivacaine was injected, but the effectiveness of the block could not be assessed via the presence of Horner syndrome because the patient was under sedation and analgesia. There was an immediate response followed by the disappearance of arrhythmic events within the next 6 h. Subsequently, the persistent episodes reappeared, which were considered to be related to the diminishing effect of the local anesthetic used to create the sympathetic block. To address this, a new echocardiography-guided puncture was performed and the catheter inserted using a pediatric epidural needle to deliver a continuous infusion of 0.2% ropivacaine at 8 mL/h. Within the next 24 h the patient had 4 sustained ventricular tachycardias, representing an 82% reduction in events. Additional intravenous beta blockers were administered, antiarrhythmia drugs were maintained, and the infusion of ropivacaine was increased to 10 mL/h, with complete cessation of sustained arrhythmias.

After 4 days, the infusion of ropivacaine was withdrawn, without reappearance of the ventricular tachycardia. Hemodynamic stability was achieved, which permitted the removal of the intra-aortic balloon pump and extubation. Oral amiodarone and beta blockers were begun. Hospital stay was prolonged due to nosocomial ventilator-associated pneumonia and ischemia in the right lower limb, which was the insertion site of the intra-aortic balloon pump. A cardiac resynchronization therapy-implantable cardioverter-defibrillator was implanted on the 40th day and the patient was discharged home after 60 days of hospitalization. Ablation was rejected at admission due to the poor state of the patient, as well as the risks inherent to the procedure, its variable efficacy, and its dependence on the experience of the center. At 8 months follow-up, the patient had not had another arrhythmic event.

We define electrical storm as more than 3 episodes of ventricular tachycardia or ventricular fibrillation within 24 h. Treatment involves aggressive therapies, such as intra-aortic balloon pump, sedation, mechanical ventilation, and, occasionally, ablation of the arrhythmia substrate.

Sympathetic hyperactivity favors the onset and maintenance of ventricular arrhythmias.[1] In the 1970s, sympathetic block in the management of ventricular arrhythmias was proposed for the treatment of congenital long QT syndrome resistant to treatment with beta blockers.[2] In 1983, Lombardi et al.[3] showed that increased sympathetic tone in the setting of coronary ischemia reduces the ventricular fibrillation threshold, and thus sympathetic block in this setting would reduce adverse ventricular events.

Nademanee et al.[4] described the addition of left stellate ganglion block to sedation and antiarrhythmic treatment in patients with acute myocardial infarction without cardiogenic shock or acute pulmonary edema. They found that in 49 patients with acute myocardial infarction, a sympathetic block procedure that included the left stellate ganglion was associated with lower mortality, which was maintained at 1 year of follow-up. Mahajan et al.[5] reported the use of the left stellate ganglion block in acute ischemic heart failure in an isolated case. Bourke et al.[6] subsequently described a highly selected group of 14 retrospectively identified patients with very frequent or constant ventricular arrhythmia who underwent sympathetic block. Sympathetic block was performed using thoracic epidural anesthesia or video-assisted thorascopic left cardiac sympathetic denervation. The authors observed a significant reduction in the number of arrhythmia episodes.[6]

This case shows that sympathetic block achieved by the combined use of beta blockers and the infusion of local anesthetic via catheter to the stellate ganglion is a therapeutic alternative in electrical storm. This should be the subject of further studies with larger cohorts of patients.