Effects of Dexmedetomidine Pretreatment, Posttreatment, and Whole-Course Pumping on Myocardial Damage during Cardiac Valve Replacement

Shenqiang Gao, MD, Guifen Ma, MM, Lina Zhou, MM, Shanhui Guan, MM and Jinjun Zhang, MB

Summary

To compare the effects of dexmedetomidine (DEX) pretreatment, posttreatment, and whole-course pumping on myocardial protection during cardiac valve replacement.

One hundred and twenty patients undergoing cardiac valve replacement were randomly divided into the follow groups: DEX pretreatment (D1 group), DEX posttreatment (D2 group), DEX whole-course pumping (D3 group), and Control (C group). The concentrations of cardiac troponin I (cTnI), malondialdehyde (MDA), tumor necrosis factor alpha (TNF-α), rate of spontaneous heart rebound after aortic opening, time to heart rebound, incidence of arrhythmia, and use of sufentanil and vasoactive drugs were recorded.

Compared with group C, the concentrations of cTnI, MDA, and TNF-α in the D1, D2, and D3 groups were lower, especially in the latter. The time to heart rebound was prolonged in all three groups ($P < 0.05$). The rate of automatic rebound was increased ($P < 0.05$) while the incidence of arrhythmia was decreased ($P < 0.05$) in all groups compared with group C. Group D3 had the highest rate of automatic rebound and the lowest incidence of arrhythmia. Compared with groups C and D2, the use of sufentanil and dopamine was lower in groups D1 and D3 ($P < 0.05$), especially in the latter.

During cardiac valve replacement, DEX pretreatment, posttreatment, and whole-course pumping could have myocardial protective effects. The latter showed better effects.

Key words: Administration method, Myocardial injury, Inflammatory response

During cardiac valve replacement under direct vision, blocking and opening of the aorta together with stopping and resuming of the heart pumping will inevitably cause myocardial ischemia-reperfusion injury. Meanwhile, during cardiopulmonary bypass (CPB), the blood is in direct contact with oxygenators and silicone tubes, which leads to the release of inflammatory factors and further myocardial damage.1,2) Moreover, the operation of cardiac surgery will stimulate the body with a strong stress response. During the operation, the release of catecholamine often causes hemodynamic instability and an imbalance in the myocardial oxygen supply and demand, which also aggravates the myocardial damage.3)

Dexmedetomidine (DEX) is a highly selective α2 adrenergic receptor agonist that is mainly used in clinical practice for sedation, analgesia, and inhibition of the sympathetic response.4) A large number of retrospective studies have suggested that the use of DEX throughout the general anesthesia period of cardiac surgery can reduce the incidence of postoperative complications and in-hospital mortality, during 30 days and 1 year after surgery, and improve the recovery of patients.5,6) Other studies have suggested that both DEX pretreatment and post-treatment can inhibit the inflammatory response and oxidative stress, so as to reduce the myocardial damage during cardiac valve replacement under CPB.7) However, some researchers also found in a model of myocardial ischemia-reperfusion injury in vitro that the area of myocardial infarction can increase when DEX is used immediately after myocardial ischemia.8) Therefore, the timing of DEX administration in cardiac surgery needs further investigation.

Methods

Cases selection and grouping: This randomized, controlled, double-blind study was approved by the Tai’an City Central Hospital and informed consent was obtained from all patients. A total of 120 patients who underwent valve replacement in Tai’an City Central Hospital from September 2018 to December 2019 were selected. The inclusion criteria were as follows: age, 18-70 years; American Society of Anesthesiologists class II-III; New York Heart Association class II-III for heart failure; no history from the 'Department of Anesthesia, Tai’an City Central Hospital, Tai’an, China and 'Taishan Sanatorium and Hospital of Shandong Province, Tai’an, China.

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of hypertension, coronary heart disease, diabetes, and other basic diseases; no arrhythmia or heart failure before the operation; no serious liver or kidney dysfunction; and no history of neurological, psychiatric, endocrine, and immune system diseases. The exclusion criteria were as follows: extracorporeal circulation machine was re-used during the operation; the machine was used for more than 3 hours; and another operation after the first operation. The patients were randomly divided into the following four groups: DEX pretreatment group (D1 group)—0.5 μg/kg DEX was given before anesthesia induction, followed by 0.5 μg/(kg/hour) pumping injection before aortic occlusion; DEX posttreatment group (D2 group)—after aortic occlusion, DEX was given to the aorta with the same dose as that in the D1 group; DEX whole-course-pumping group (D3 group)—DEX with the same dose as that in the D1 group was given before anesthesia induction to the end of operation; control group (Group C)—pumped with normal saline of the same volume as that in group D3 (Figure 1). In this study, each group underwent aortic valve replacement surgery, mitral valve replacement surgery, as well as both valve replacement surgery, with a balanced distribution of the different types of surgical among the four groups. The types of valves used were mechanical valves in patients under 60 years of age and biological valves in patients over 60 years; these were also evenly distributed.

**Anesthesia method:** All patients received intramuscular injection of 0.1 mg/kg of morphine and 0.006 mg/kg of scopolamine 30 minutes before anesthesia. During anesthesia induction, 0.05 mg/kg of midazolam, 1 μg/kg of sufentanil, 0.3 mg/kg of etomidate, and 0.2 mg/kg of cisatracurium were administered slowly in this order. After 3 minutes, tracheal intubation was carried out. The inhaled oxygen concentration was 60%, and the end-expiratory carbon dioxide partial pressure was maintained at 35-45 mmHg. The patients in each group were given DEX according to their group. Anesthesia was maintained with 4-10 mg/(kg/hour) of propofol, 1-2 μg/(kg/hour) of sufentanil, 0.1 mg/(kg/hour) of cisatracurium, and the bispectral index was maintained at 40-60. Intraoperative fluid therapy was performed with lactate Ringer’s solution and hydroxyethyl starch 130/0.4, and vasoactive drugs such as dopamine, adrenaline, and nitroglycerin were used as appropriate to maintain a mean arterial pressure (MAP) of 60-90 mmHg, heart rate of 60-100 times/minute, and central venous pressure of 6-12 cmH2O during the non-CPB period. If necessary, a suspension of red blood cells was infused to maintain an HCT of at least 27%.

**Extracorporeal circulation:** A SORIN C5 extracorporeal circulation machine and a SORIN/INSPIRE membrane oxygenator were used for extracorporeal circulation. The priming solution was made of lactated Ringer’s solution and hydroxyethyl starch 130/0.4. The crystalline-to-gel ratio was 1:2 and the perfusion flow was 2.2-2.6 L/(m2/minute). MAP was maintained at 50-80 mmHg, the nasopharyngeal temperature was 29 °C, and the Hb was 70-90 mg/dL. When the temperature of the nasopharynx was above 34 °C, the ascending aorta was opened. The time of auxiliary circulation was about 1/3 of the time of aortic occlusion. When the temperature of the anus was above 35 °C, the hemodynamics were stable, and the electrolyte and acid-base balance were within normal ranges, the perfusion flow was slowly stopped.

**General indicators:** General indicators, including age, sex, body mass index, cardiac function grade, aortic occlusion time, and CPB time were recorded.

**Indicators of myocardial damage, oxidative stress, and inflammatory response:** A total of 3 mL of radial artery blood was drawn before the injection of experimental drugs (T0, 5 minutes after entering the room, before the injection of the experimental drugs), before aortic occlusion (T1), at the end of the operation (T2), and 12 (T3) and 24 hours after the operation (T4). The concentrations of cardiac troponin I (cTnI), MDA, and TNF-α were measured using two-site enzyme immunoassay, thiobarbituric acid, and enzyme-linked immunosorbent assay, respectively.

**Indicators of heart rebound and other indicators:** The rate of automatic heart rebound, ventricular fibrillation, and the incidence of arrhythmia were recorded. The dosages of sufentanil, dopamine, adrenaline and nitroglycerin were recorded. The incidence of severe hypertension (MAP > 110 mmHg), hypotension (MAP < 50 mmHg), and bradycardia (heart rate < 50 times/minute) were recorded.

**Statistical analysis:** SPSS 22.0 statistical software was used for data processing and analysis. The measurement data were expressed as mean ± SD. Comparisons between groups were conducted using one-way ANOVA. The chi-
square test was used for the comparison of count data. *P < 0.05 indicates statistical significance.

Results

General indicators: A total of 120 patients were recruited, with 30 for each group. There was no significant difference in the general indicators, including age, sex, body mass index, cardiac function grade, aortic occlusion time, and CPB time between the groups (Table I).

Indicators of myocardial damage, oxidative stress, and inflammatory response: Compared with T0 and T1, the concentrations of cTnI, MDA, and TNF-α in all groups increased from T2 to T4 (P < 0.05), as shown in Table II and Figure 2. Compared with group C, the concentrations of cTnI, MDA, and TNF-α in the D1, D2, and D3 groups were lower from T2 to T4 (P < 0.05). Compared with the D1 and D2 groups, the concentrations of cTnI, MDA, and TNF-α in the D3 group were lower at T2, T3, and T4 (P < 0.05). The concentrations of cTnI, MDA, and TNF-α at each time point were not significantly different between the D1 and D2 groups (Table II).

Indicators of heart rebound: Compared with group C, the time to heart rebound in the D1, D2, and D3 groups was prolonged (P < 0.05), while the rate of automatic rebound was increased (P < 0.05). The incidence of ventricular fibrillation and other arrhythmias in the D1, D2, and D3 groups was lower after aortic opening (P < 0.05) compared with group C (Table III). Compared with the D1 and D2 groups, the D3 group had an increased rate of automatic rebound (P < 0.05) and decreased incidence of ventricular fibrillation and other arrhythmias after aortic opening (P < 0.05), as shown in Table III. There was no significant difference in the incidence of severe bradycardia, hypotension, and hypertension among the groups (Table III).

Medication conditions: Compared with groups C and D2, the dosage of sufentanil, dopamine, adrenaline, and nitroglycerin in the D1 and D3 groups was lower (P < 0.05), as shown in Table IV. In addition, the dosage of sufentanil, dopamine, adrenaline, and nitroglycerin in the D3 group was lower compared with the D1 group (P < 0.05).

Discussion

In this study, we found that compared with the control group, the concentrations of cTnI, MDA, and TNF-α in groups D1, D2, and D3 were lower, especially in group D3. The time to heart rebound was prolonged and the rate of automatic rebound was increased, while the incidence of arrhythmia was decreased in all three groups. Furthermore, group D3 had the highest rate of automatic rebound and the lowest incidence of arrhythmia.

During cardiac valve replacement under extracorporeal circulation, due to the existence of surgical stress re-

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**Table I.** General Indicators, Including Age, Sex, Body Mass Index, Cardiac Function Grade, Aortic Occlusion Time, and CPB Time, in Each Group

<table>
<thead>
<tr>
<th>Group</th>
<th>M/F</th>
<th>Age (years)</th>
<th>BMI (kg/m²)</th>
<th>Cardiac function grade (II/III)</th>
<th>Aortic occlusion time (minutes)</th>
<th>CPB time (minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D1</td>
<td>14/16*</td>
<td>63.2 ± 6.8*</td>
<td>24.4 ± 3.1*</td>
<td>12/18*</td>
<td>71.2 ± 19.3*</td>
<td>108.8 ± 22.4*</td>
</tr>
<tr>
<td>D2</td>
<td>17/13*</td>
<td>61.4 ± 7.7*</td>
<td>23.6 ± 4.1*</td>
<td>14/16*</td>
<td>69.6 ± 21.6*</td>
<td>102.3 ± 27.1*</td>
</tr>
<tr>
<td>D3</td>
<td>16/14*</td>
<td>59.8 ± 10.3*</td>
<td>25.1 ± 2.3*</td>
<td>15/15*</td>
<td>70.8 ± 20.5*</td>
<td>106.5 ± 24.6*</td>
</tr>
<tr>
<td>C</td>
<td>15/15</td>
<td>62.5 ± 8.6</td>
<td>23.7 ± 3.7</td>
<td>15/17</td>
<td>68.8 ± 22.3</td>
<td>105.1 ± 25.3</td>
</tr>
</tbody>
</table>

M indicates male; F, female; and BMI, body mass index. *P < 0.05 versus group C.

**Table II.** Comparison of cTnI, MDA, and TNF-α Concentrations at Each Time Point

<table>
<thead>
<tr>
<th>Indicators/Group</th>
<th>T0</th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>cTnI (ng/mL)</td>
<td>C</td>
<td>0.18 ± 0.11</td>
<td>0.23 ± 0.12</td>
<td>1.08 ± 0.21*</td>
<td>3.22 ± 0.25*</td>
</tr>
<tr>
<td></td>
<td>D1</td>
<td>0.21 ± 0.14</td>
<td>0.25 ± 0.14</td>
<td>0.91 ± 0.19**</td>
<td>2.85 ± 0.18**</td>
</tr>
<tr>
<td></td>
<td>D2</td>
<td>0.19 ± 0.09</td>
<td>0.22 ± 0.16</td>
<td>0.88 ± 0.20**</td>
<td>2.93 ± 0.21**</td>
</tr>
<tr>
<td></td>
<td>D3</td>
<td>0.20 ± 0.13</td>
<td>0.26 ± 0.15</td>
<td>0.72 ± 0.15**</td>
<td>2.41 ± 0.16**</td>
</tr>
<tr>
<td>MDA (nmol/mL)</td>
<td>C</td>
<td>6.53 ± 1.12</td>
<td>6.94 ± 1.41</td>
<td>15.38 ± 2.13*</td>
<td>26.75 ± 3.05*</td>
</tr>
<tr>
<td></td>
<td>D1</td>
<td>6.24 ± 1.05</td>
<td>6.81 ± 1.22</td>
<td>13.21 ± 1.82**</td>
<td>23.47 ± 2.88**</td>
</tr>
<tr>
<td></td>
<td>D2</td>
<td>5.98 ± 1.21</td>
<td>6.34 ± 1.32</td>
<td>12.89 ± 2.08**</td>
<td>22.86 ± 2.76**</td>
</tr>
<tr>
<td></td>
<td>D3</td>
<td>6.11 ± 0.08</td>
<td>6.41 ± 1.15</td>
<td>10.11 ± 1.75**</td>
<td>19.58 ± 1.95**</td>
</tr>
<tr>
<td>TNF-α (pg/mL)</td>
<td>C</td>
<td>18.55 ± 3.14</td>
<td>19.47 ± 3.10</td>
<td>61.54 ± 12.37*</td>
<td>115.23 ± 15.22*</td>
</tr>
<tr>
<td></td>
<td>D1</td>
<td>19.23 ± 3.46</td>
<td>20.11 ± 2.95</td>
<td>50.45 ± 13.68**</td>
<td>105.38 ± 16.76**</td>
</tr>
<tr>
<td></td>
<td>D2</td>
<td>17.88 ± 2.96</td>
<td>18.72 ± 3.11</td>
<td>48.33 ± 14.15**</td>
<td>102.14 ± 14.35**</td>
</tr>
<tr>
<td></td>
<td>D3</td>
<td>18.21 ± 3.20</td>
<td>19.31 ± 2.99</td>
<td>39.78 ± 13.45**</td>
<td>92.31 ± 17.12**</td>
</tr>
</tbody>
</table>

*P < 0.05 versus T0; **P < 0.05 versus C group; &P < 0.05 versus group D2. T0: 5 minutes after entering the room and before the injection of experimental drugs; T1: before aortic occlusion; T2: at the end of the operation; T3: 12 hours after the operation; T4: 24 hours after the operation. cTnI indicates cardiac troponin I; MDA, malondialdehyde; and TNF-α, tumor necrosis factor alpha.
Figure 2. Line graph of nTnI, MDA, and TNF-α over the time course.

Table III. Indicators of Heart Rebound in Each Group

<table>
<thead>
<tr>
<th>Group</th>
<th>Heart rebound time (seconds) mean (SD)</th>
<th>Automatic rebound n (%)</th>
<th>Arrhythmias n (%)</th>
<th>Hypertension n (%)</th>
<th>Hypotension n (%)</th>
<th>Bradycardia n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D1</td>
<td>138.5 ± 12.7*</td>
<td>16 (53.3) *</td>
<td>16 (53.3) *</td>
<td>8 (26.7) *</td>
<td>13 (43.3) *</td>
<td>5 (16.7) *</td>
</tr>
<tr>
<td>D2</td>
<td>140.2 ± 10.8*</td>
<td>15 (50.0) *</td>
<td>17 (56.7) *</td>
<td>11 (36.7) *</td>
<td>9 (30.0) *</td>
<td>4 (13.3) *</td>
</tr>
<tr>
<td>D3</td>
<td>143.6 ± 8.5*</td>
<td>24 (80.0) **</td>
<td>8 (26.7) **</td>
<td>7 (23.3) *</td>
<td>14 (46.7) *</td>
<td>6 (20.0) *</td>
</tr>
<tr>
<td>C</td>
<td>102.4 ± 15.2</td>
<td>7 (23.3)</td>
<td>26 (86.7)</td>
<td>12 (40.0)</td>
<td>10 (33.3)</td>
<td>3 (10.0)</td>
</tr>
</tbody>
</table>

*P < 0.05 versus group C; *P > 0.05 versus group C; **P < 0.05 versus groups D1, D2, and C.

Table IV. Medication Conditions (Including the Dosage of Sufentanil, Dopamine, Adrenaline, and Nitroglycerin) in Each Group

<table>
<thead>
<tr>
<th>Group</th>
<th>Sufentanil μg/(kg/hour)</th>
<th>Dopamine μg/(kg/minute)</th>
<th>Adrenaline μg/(kg/minute)</th>
<th>Nitroglycerin μg/(kg/minute)</th>
</tr>
</thead>
<tbody>
<tr>
<td>D1</td>
<td>1.12 ± 0.25*</td>
<td>5.96 ± 1.34*</td>
<td>0.05 ± 0.03</td>
<td>0.57 ± 0.14</td>
</tr>
<tr>
<td>D2</td>
<td>1.43 ± 0.48</td>
<td>7.03 ± 1.15</td>
<td>0.08 ± 0.02</td>
<td>0.79 ± 0.11</td>
</tr>
<tr>
<td>D3</td>
<td>0.91 ± 0.31**</td>
<td>4.90 ± 0.91**</td>
<td>0.04 ± 0.03**</td>
<td>0.45 ± 0.09**</td>
</tr>
<tr>
<td>C</td>
<td>1.51 ± 0.52</td>
<td>6.81 ± 1.22</td>
<td>0.07 ± 0.01</td>
<td>0.82 ± 0.13</td>
</tr>
</tbody>
</table>

*P < 0.05 versus D2 and C groups; **P < 0.05 versus D1 group.

spontaneous ischemia-reperfusion, imbalance between oxygen supply and demand, and other factors, myocardial damage is inevitable to a certain extent, which can be clinically manifested as difficulty of heart rebound, increased frequency of defibrillation, and increased incidence of arrhythmia after aortic opening.11,12) Studies have shown DEX use in cardiac surgery can improve the rate of automatic rebound and reduce the incidence of postoperative tachyarrhythmia.13,14) In this study, we also found that DEX pretreatment, posttreatment, and whole-course pumping could improve the rate of cardiac automatic rebound to various degrees and reduce the occurrence of ventricular fibrillation, ventricular tachycardia, atrial fibrillation, and supraventricular tachycardia after aortic opening. In addition, the whole-course-pumping group had the most obvious effects. This suggests that the use of DEX played a role in myocardial protection during cardiac valve replacement under extracorporeal circulation. How-
ever, it should be noted that the rebound time of all three
groups was prolonged, which is likely related to the inhi-
bition of the sinoatrial and atrioventricular nodes by
DEX.13)

The pathogenesis of myocardial damage is complex
and diverse, in which oxidative stress and the inflamma-
tory response play a very important role.14-16) MDA is
the final product of lipid peroxidation, and its concentra-
tion in vivo can reflect the degree of lipid peroxidation
caused by oxygen free radicals.20) TNF-α is an important inflam-
matory cytokine that plays a key role in the inflammatory
response cascade.21) cTnI has been widely recognized as a
marker of myocardial damage.21,22) In this study, we found
that the concentrations of MDA, TNF-α, and cTnI in all
groups were higher than at the end of the operation and at
12 and 24 hours after the operation, indicating that oxida-
tive stress and the inflammatory reaction might lead to
myocardial damage during cardiac valve replacement un-
der extracorporeal circulation. However, DEX pretreat-
ment, posttreatment, and whole-course pump injection de-
creased the concentrations of MDA, TNF-α, and cTnI, in-
dicating that DEX could contribute to myocardial protec-
tion by reducing oxidative stress and the inflammatory re-
response, among which the whole-course pump injection
had the best effects.

It was found that 0.5-1 µg/kg of DEX pretreatment
followed by 0.2-0.7 µg/(kg/hour) maintenance could make
the hemodynamics more stable, reduce the arrhythmia,
and reduce the dosage of sufentanil, nitroglycerin, and no-
radrenaline, with an increased risk of hypotension and
bradycardia.5,23) In this study, we also found that compared
with the control and posttreatment groups, the amount of
dopamine, adrenaline, and nitroglycerin in the DEX pre-
treatment and whole-process pumping groups was reduced
during the operation, which reflected more stable hemody-
namics. DEX was used for the longest time in the whole-
course-pumping group, whereas the lowest amount of
sufentanil was also used in this group. There was no sig-
nificant difference in the frequency of occurrence of se-
vere hypertension, hypotension, and bradycardia among
the groups, which might be related to the prolongation of
infusion time and the selection of a relatively low dose.

This study has several limitations. First, due to the
limited resources, we did not include follow-up data on
long-term clinical outcomes. Research is needed to further
explore the association between DEX and long-term out-
comes after surgery for cardiac valve replacement in the
future. Second, the sample size was relatively small. Fur-
ther evidence with a larger sample size is warranted to
verify the findings presented here.

Conclusion

In surgery for cardiac valve replacement, DEX pre-
treatment, posttreatment, and whole-course pumping could
produce certain myocardial protection by inhibiting oxida-
tive stress and the inflammatory response, and the whole-
course pumping method had better effects.

Disclosure

Conflicts of interest: There are no potential conflicts of
interest to disclose.

Ethics approval and consent to participate: The study
was approved by the Tai’an City Central Hospital [NO:
2019-06-04], and informed consent was signed by all pa-
tients.

Author Contributions: SQG was responsible for the
study concept and design, statistical analysis, and manu-
script review; GFM was responsible for the definition of
intellectual content, literature research, and manuscript ed-
ting; LNZ was responsible for the experimental studies,
and data analysis; SHG was responsible for the guarantor
of integrity of the entire study, clinical studies, and manu-
script preparation; JJJ was responsible for data acquisi-
tion. All authors read and approved the final manuscript.

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