EFFECTS OF DEXAMETHASONE AND PHENIRAMINE MALEATE ON HEMODYNAMIC AND RESPIRATORY PARAMETERS AFTER CEMENTATION IN CEMENTED PARTIAL HIP PROSTHESIS

ABDULKADIR YEKTAŞ*, FUNDA GÜMÜŞ*, TOLGA TOTOZ*, NURTEN GÜL*, KEREM ERKALP* AND AYŞİN ALAGÖL*

Summary

Purpose: To prevent hemodynamic and respiratory changes that are likely to occur during cementation in partial hip prosthesis by prophylactic use of pheniramine maleate and dexamethasone.

Methods and Materials: The study included 40 patients aged between 60 and 85 years with an American Society of Anesthesiologists (ASA) grade of II-III who underwent partial hip prosthesis. Just after spinal anesthesia, 4 mL normal saline was pushed in patients in Group S, whereas 45.5 mg pheniramine maleate and 8 mg dexamethasone mixture was pushed intravenously in a total volume of 4mL in patients in Group PD.

Results: Amounts of atropine and adrenaline administered after cementation were significantly higher in Group S than in Group PD (P <0.05). There was a significant difference between SpO₂ values before and after cementation in Group S; SpO₂ value was lower after cementation (P <0.05) except for 1. min after cementation. SpO₂ value increased 1 min after cementation (P = 0.031).

Conclusion: Prophylactic use of pheniramine maleate and dexamethasone in partial hip prosthesis led to an increase in SpO₂ value and a decrease in the utilization of adrenaline and atropine after cementation.

Keywords: Methylmethacrylate, partial hip prosthesis, bone cement implantation syndrome, pheniramine maleate, dexamethasone.

Introduction

Partial hip arthroplasty is usually performed in elderly population in whom concomitant diseases may enhance the likelihood of a more progressive BCIS (Bone Cement Implantation Syndrome). BCIS is the most important cause of intraoperative morbidity and mortality in patients undergoing cemented hip arthroplasty, and rarely, hypoxia and confusion may be encountered in the postoperative period. Intraoperative mortality rate is 0.43 % for cemented partial hip prosthesis in patients with or without femur fracture¹. Although the etiology and pathophysiology of BCIS have not been understood clearly, few mechanisms including monomer-mediated model, embolic model, histamine release and hypersensitivity, complement activation, and multimodal model

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have been suggested. In a study, blockade of histamine receptors by clemastine and cimetidine (H₁ and H₂ antagonists) was reported to have protective effects.

Pheniramine maleate is an H₁ receptor antagonist. H₁ receptor antagonists have been successfully used before drug infusion to prevent and for acute treatment of type I hypersensitivity. Dexamethasone is a synthetic glucocorticosteroid. In animal studies, this glucocorticosteroid has been demonstrated to inhibit airway eosinophilia in the presence of antigen by inhibiting interleukin-5 synthesis and to be successfully used for the prophylaxis of type I hypersensitivity reaction.

The aim of the present study is to assess the effect of pheniramine maleate and dexamethasone on incidence of hypotension, bradycardia and hypercarbia associated with BCIS.

**Methods and Materials**

After obtaining approval from the Ethical Committee of Bağcılar Training and Research Hospital and written informed consents of the patients, 40 patients aged between 60 and 85 years with an American Society of Anesthesiologists (ASA) grade of II-III who underwent partial hip prosthesis with cement due to femur neck fracture under spinal anesthesia were included in the study. The present study was designed as a prospective, randomized and double-blinded study.

Data of the patients regarding age, height, weight, gender, concomitant diseases, medications, smoking status, ASA classification and surgery duration of surgery were recorded.

Patients with allergic diseases, those using anti-allergic medications or corticosteroids, who have deep venous thrombosis and lower extremity venous insufficiency, those with cardiac disease likely to cause atrial fibrillation or thromboembolism, or paraplegia-hemiplegia, who were immobile before fracture (i.e., bedridden patients), those with previous cardiac surgery, and those having a contraindication for regional anesthesia were excluded from the study.

The patients did not receive any premedication. The patients were positioned with the hip that would be operated being on top, and an 18 G intravenous canula was placed into a peripherial vein in the dorsal aspect of the hand opposite to the surgery side and 0.9% NaCl was administrated at a rate of 10 mL kg⁻¹ h⁻¹ for first hour and than 5 mL kg⁻¹ h⁻¹. In order to obtain blood sample for blood gas analysis, an intra-arterial catheter was inserted into the radial artery of the arm on the surgery side via a 20-gauge intravenous catheter, and it was washed with heparinized fluid and closed using a three-way tap. The patients were administrated oxygen at a rate of 2 L/min through a free oxygen mask which was fixed hole in any side of the free oxygen mask in which the free end of end-tidal CO₂ (ET-CO₂) line. All patients underwent electrocardiography, non-invasive arterial blood pressure measurement, peripheral pulse oximetry and end-tidal CO₂ monitoring.

All patients underwent spinal anesthesia. Patients in both groups were positioned laterally with the leg that would be operated being on top, and spinal anesthesia was performed via a 27-gauge Quincke-type needle after performing cutaneous-subcutaneous infiltration anesthesia using 2 mL of 2% lidocaine through L₄-L₅ space. After observing cerebrospinal fluid (CSF) outflow, 1 mL (5 mg) isobaric bupivacaine and 25 µg (0.5 mL) fentanyl were administered in a total volume of 1.5 mL. Spinal anesthesia was performed by a specialist in both groups. The patients in whom spinal anesthesia was unsuccessful three times were excluded from the study. Then the patient were randomly divided into the following two groups using sealed envelopes: 1) Group S (n = 20) patients were administrated 4 mL of normal saline, 2) Group PD (n = 20) patients were administrated 45.5 mg pheniramine maleate and 8 mg dexamethasone. Prior to spinal anesthesia, basal values of arterial blood pressure, heart rate, oxygen saturation by pulse oximeter (SpO₂), end-tidal CO₂ (ETCO₂), and blood gases were recorded. Pre-prepared syringes were used; thus, both the anesthesiologist and the patient were blinded to the content.

Cephazoline sodium 1 g was administered iv approximately 30 to 60 min prior to surgery for prophylaxis.

Values of arterial blood pressure, heart rate, SpO₂, end-tidal CO₂, and blood gases were recorded for all patients prior to surgical procedure and at 10-min intervals until the first minute before cementation.
Amount of bleeding, amount of crystalloid administered by IV route, and, if used, doses of atropine and adrenaline were also recorded until the first minute before cementation. Those parameters were recorded at 1-min intervals in the first 5 min after cementation and then every 5 minutes. Measurements were discontinued at the 25th min after cementation. Amount of administered crystalloid throughout the surgical procedure and amount of bleeding were recorded. Arterial blood gases were analyzed before spinal anesthesia, just before cementation, and at the 10th and 25th min after cementation.

A 5 µg adrenalin was pushed via venous route in the following conditions: 1) a decrease in mean blood pressure more than 30% of the initial value until cementation, 2) after cementation, a more than 30% decrease in the blood pressure value measured before cementation. In case of a decrease in heart rate below 50 beats/min, 0.5 mg atropine was injected. Patients in whom amount of bleeding before cementation exceeded 400 mL were excluded from the study (bleeding before cementation exceeded 400 mL in any of the patients), and blood loss was replaced by normal saline.

When the surgical procedure was completed and following postoperative observation, patients with a modified Aldrete score of ≥9 were transferred to the clinic.

Statistical Analysis

Based on a preliminary study performed in 10 patients, we estimated that a sample size of 20 patients in each group would be sufficient with a 5% error and a statistical power of 80% assuming that the mean arterial pressure on the 25th min would be 92 ± 15 mmHg in Group PD and 85 ± 15 mmHg in Group S.

Descriptive statistics of data were expressed as mean ± standard deviation. Normal distribution of variables was tested by Kolmogorov Smirnov test. Comparison of data regarding age, height, weight, gender, type of surgery, duration of surgery, time elapsed from spinal anesthesia to cementation, and ASA grades were compared between the groups by an independent-t test. Data analyses were performed using the Statistical Package for the Social Sciences (SPSS, Inc. Chicago, IL, USA) version 11.5. A P value <0.05 was considered statistically significant.

Results

Distributions of age, weight, height, type of surgery, duration of surgery, duration of cementation, ASA grades and gender in the study groups are presented in Table 1. There were no statistically significant differences between the groups in terms of age, weight, height, type of surgery, duration of surgery, duration of cementation, ASA grade and gender.

Table 1
Distribution of age, weight, height, type of surgery, duration of surgery, duration of cementation, the American Society of Anesthesiologists grades and gender in the study groups. Data are presented as mean ± SD

<table>
<thead>
<tr>
<th></th>
<th>Group S (n = 20)</th>
<th>Group PD (n = 20)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>80.50 ± 7.05</td>
<td>76.85 ± 9.31</td>
<td>0.198</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.75 ± 11.14</td>
<td>70.60 ± 10.45</td>
<td>0.989</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.45 ± 8.40</td>
<td>166.35 ± 5.65</td>
<td>0.462</td>
</tr>
<tr>
<td>Duration of surgery (min)</td>
<td>79.30 ± 27.29</td>
<td>84.55 ± 18.01</td>
<td>0.579</td>
</tr>
<tr>
<td>Duration of cementation (min)</td>
<td>61.60 ± 22.87</td>
<td>64.35 ± 10.55</td>
<td>0.968</td>
</tr>
<tr>
<td>ASA (II/III)</td>
<td>14/6</td>
<td>13/7</td>
<td>0.708</td>
</tr>
<tr>
<td>Gender (F/M)</td>
<td>11/9</td>
<td>12/8</td>
<td>0.061</td>
</tr>
</tbody>
</table>

SD: standard deviation; ASA: the American Society of Anesthesiologists; F: female; M: male

Doses of atropine and adrenaline and the amounts of intravenous fluid and bleeding in the study groups are presented in Table 2. The dose of adrenaline used...
after cementation was significantly higher in Group S than in Group PD (P = 0.022). The dose of atropine used after cementation was significantly higher in Group S than in Group PD (P = 0.014).

Comparison of the study groups in terms of systolic diastolic and mean arterial pressures, heart rate and SpO₂ before and after cementation are presented in Table 3. There were no statistically significant differences between the groups in terms of systolic/diastolic and mean arterial pressure, heart rate and SpO₂. Heart rate increased significantly 2 min after cementation in Group PD when compared to before (p = 0.008) (Table 3).

In Group S, SpO₂ increased 1 min after cementation (P = 0.031), and were significantly lower at the rest of all times when compared to the value before cementation (P = 0.003, P = 0.003, P <0.001, P = 0.003, P = 0.001, P = 0.001, P = 0.001, P = 0.004, respectively) (Table 3).

There were no significant differences between Group S and Group PD in terms of Pₗ (P = 0.168), partial arterial carbon dioxide pressure (PₐCO₂) (P = 0.067), partial arterial oxygen pressure (PₐO₂) (P = 0.056) and end-tidal CO₂ values before cementation and at the 10th (P = 0.067) and 25th (P = 0.152) min after cementation.

In addition, there were no significant differences in both groups in terms of pH, PₐCO₂, PₐO₂ and end-tidal CO₂ values before cementation, and at the 10th min pH (P = 0.102), PₐCO₂ (P = 0.063), PₐO₂ (P = 0.175) and end-tidal CO₂ values (P = 0.448) and 25th min pH (P = 0.193), PₐCO₂ (P = 0.186), PₐO₂ (P = 0.084) and end-tidal CO₂ values (P = 0.054) min after cementation.

One patient in Group S developed cardiopulmonary arrest at the 1st min after cementation and accepted as dead after performing cardiopulmonary resuscitation (CPR) for 45 min. Blood gas values and end-tidal CO₂ values of this case was not consistent with pulmonary embolus.

All patients were transferred to the clinic after observing postoperatively in recovery room for 2 hours and then all patients were discharged. None of the patients developed postoperative hypoxia or confusion.

**Table 2**

| Distribution of amounts of atropine, adrenaline, and intravenous fluid, and the amount of bleeding in the study groups. Numbers are presented as mean ± SD. |
|---------------------------------|---------------------------------|------------------|
| **Group S** (n = 20) | **Group PD** (n = 20) | **P** |
| Adrenaline after cementation (µg) | 4.50 ± 11.34 | 0.75 ± 1.83 | 0.022 |
| Atropine after cementation (mg) | 0.25 ± 0.91 | 0.00 ± 0.00 | 0.014 |
| Intravenous crystalloid before cementation (mL) | 560.50 ± 206.33 | 480.00 ± 176.51 | 0.345 |
| Intravenous crystalloid administered throughout surgical procedure (mL) | 890.00 ± 282.65 | 827.50 ± 181.71 | 0.274 |
| Amount of bleeding before cementation (mL) | 184.25 ± 96.99 | 158.75 ± 77.01 | 0.243 |
| Total amount of bleeding (mL) | 228.75 ± 118.73 | 211.00 ± 77.50 | 0.059 |

SD: standard deviation

**Discussion**

Respiratory and cardiovascular changes during partial hip replacement seriously affect the prognosis of patients. These changes exist in a wide spectrum of disorders ranging from temporary hypoxia to cardiac rhythm disorders and even cardiac arrest12,13.

Several mechanisms have been suggested in the etiology and pathophysiology of BCIS including monomer-mediated model, embolic model, histamine release and hypersensitivity, complement activation, and multimodal model1. A study has shown that implantation of methacrylic bone cement into the femur may increase plasma histamine level by more than 1 ng/mL. Temperate histamine release may cause severe,
Table 3

*Systolic, Diastolic and Mean Arterial Pressure, Heart Rate and Peripheral Oxygen Saturation (Mean ± SD)*

<table>
<thead>
<tr>
<th>Group</th>
<th>Before C</th>
<th>1.min</th>
<th>2.min</th>
<th>3.min</th>
<th>4.min</th>
<th>5.min</th>
<th>10.min</th>
<th>15.min</th>
<th>20.min</th>
<th>25.min</th>
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<tbody>
<tr>
<td>SAP</td>
<td>S</td>
<td>137.4 ± 27.0</td>
<td>127.2 ± 28.3</td>
<td>128.2 ± 24.4</td>
<td>127.2 ± 25.9</td>
<td>130.3 ± 28.3</td>
<td>131.5 ± 25.5</td>
<td>135.3 ± 24.3</td>
<td>130.2 ± 28.4</td>
<td>135.1 ± 24.1</td>
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<tr>
<td></td>
<td>PD</td>
<td>125.6 ± 22.1</td>
<td>124.0 ± 26.0</td>
<td>118.8 ± 27.6</td>
<td>121.1 ± 22.9</td>
<td>122.1 ± 22.7</td>
<td>119.9 ± 34.1</td>
<td>132.8 ± 20.3</td>
<td>134.6 ± 23.5</td>
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<tr>
<td>DAP</td>
<td>S</td>
<td>76.4 ± 21.0</td>
<td>69.9 ± 17.7</td>
<td>73.6 ± 17.4</td>
<td>74.3 ± 21.8</td>
<td>74.3 ± 23.1</td>
<td>74.0 ± 20.1</td>
<td>74.2 ± 21.4</td>
<td>72.9 ± 21.4</td>
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<tr>
<td></td>
<td>PD</td>
<td>71.8 ± 16.5</td>
<td>69.8 ± 15.6</td>
<td>67.0 ± 14.3</td>
<td>67.8 ± 13.6</td>
<td>67.9 ± 15.4</td>
<td>68.0 ± 13.6</td>
<td>70.9 ± 12.4</td>
<td>70.7 ± 11.8</td>
<td>69.1 ± 11.6</td>
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<td>MAP</td>
<td>S</td>
<td>93.8 ± 20.7</td>
<td>83.1 ± 22.1</td>
<td>88.3 ± 20.7</td>
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<td>91.6 ± 27.2</td>
<td>89.5 ± 20.6</td>
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<td>89.9 ± 18.5</td>
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<tr>
<td></td>
<td>PD</td>
<td>85.4 ± 21.8</td>
<td>81.7 ± 16.9</td>
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<td>78.9 ± 17.4</td>
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<td>87.7 ± 15.8</td>
<td>90.8 ± 14.9</td>
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<td>HR</td>
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<td>76.4 ± 13.0</td>
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<td>PD</td>
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<td>SpO2</td>
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<td>97.5 ± 2.8</td>
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<td>92.6 ± 21.9*</td>
<td>92.1 ± 21.9*</td>
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<td>PD</td>
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<td>95.6 ± 2.9</td>
<td>93.4 ± 9.8</td>
<td>95.5 ± 3.3</td>
</tr>
</tbody>
</table>

SAP: Systolic Arterial Pressure; DAP: Diastolic Arterial Pressure; MAP: Mean Arterial Pressure; HR: Heart Rate; SpO2: Peripheral Oxygen Saturation; Group; S: Saline; Group; PD: pheniramine maleate-dexamethasone; Before C: Before cementation.

There is no statistical difference between groups (P >0.05)

* P <0.05 when compared with Before Cementation.
Cardiac problems defined to date have been mostly about wall motion and rhythm disorders, with no remarkable changes in heart rate\textsuperscript{14,15}. We also found no significant difference between heart rates before and after cementation; heart rate at the 2\textsuperscript{nd} min after cementation was higher than before cementation only in Group PD. In Group S, as compared with before cementation, heart rate decreased by 0.40\% at the 2\textsuperscript{nd} min, 1\% at the 3\textsuperscript{rd} min, 2.5\% at the 4\textsuperscript{th} min and 3.1\% at the 5\textsuperscript{th} min, but thereafter returned to the normal values. The dose of atropine administered was significantly higher in Group S than in Group PD. However, as heart rate was recorded with 5-min intervals, such decrements in HR were not reflected in the statistical analysis. In Group PD, an increase in the heart rate was observed by 12.73\% at the 2\textsuperscript{nd} min (P <0.05), 3\% at the 3\textsuperscript{rd} min, 4.62\% at the 4\textsuperscript{th} min and 3.79\% at the 5\textsuperscript{th} min.

Potential causes of hypotension, one of the parameters of Bcis, include histamine release and type I hypersensitivity reaction, or hemodynamic impairment caused by pulmonary air or by fat embolism and reflex mechanisms responsive to this\textsuperscript{16,17}. An experimental study has demonstrated that methylmethacrylate monomers influence intracellular and extracellular calcium mobilization, resulting in direct relaxation in venous and arterial smooth muscles\textsuperscript{18,19}. Nevertheless, this hypothesis has not been verified by in vivo animal experiments and it has been determined that plasma methylmethacrylate concentration after cemented hip arthroplasty is lower than the concentration that is likely to cause pulmonary or cardiovascular effects\textsuperscript{20,21}. A clinical study evaluating plasma concentrations revealed that maximum levels of serum methylmethacrylate was measured 30 s after cementation and this was thought to cause a decrease in arterial blood pressure\textsuperscript{20}. Another study compared non-cemented hip arthroplasty with cemented hip arthroplasty using transesophageal echocardiography and demonstrated a higher embolic load in the cemented hip arthroplasty group\textsuperscript{22}. The authors concluded that, embolism might also cause hypotension due to increase in pulmonary arterial pressure together with decrease in right ventricular function\textsuperscript{23}. In the present study, there were no significant differences between the groups in terms of the amounts of crystalloid and bleeding before cementation and throughout the surgical procedure.

The amount of adrenaline administered in Group S was significantly higher than that administered in Group PD; however, as blood pressure was measured at specific time intervals, such variations in blood pressure had no impact on the statistical outcomes.

A patient in Group S developed hypotensive bradyarrhythmic arrest at the 1\textsuperscript{st} min after cementation, which was not responsive to CPR. There were no significant differences in $P_\text{aO}_2$, pH, $P_\text{aCO}_2$ and end-tidal CO\textsubscript{2} values of this patient before cementation and during CPR performed after cementation; this ruled out the possibility of massive embolism.

In the present study, despite oxygen provided through a free oxygen mask at a rate of 2 L/min in both groups, there was a significant difference between SpO\textsubscript{2} values before and after cementation in Group S. An increase by 0.25\% at the 1\textsuperscript{st} min after cementation and a decrease by 7.1\% at the 2\textsuperscript{nd} min after cementation were observed, and this decrease continued until the end of surgery in the same manner. Various studies have demonstrated hypoxia development during hip and knee arthroplasty\textsuperscript{24,25}. This has been thought to result from pulmonary embolism or pulmonary vasoconstriction due to cement toxicity\textsuperscript{21,26}. In the present study, there was no significant difference between SpO\textsubscript{2} values before and after cementation in Group PD; this suggests that dexamethasone and pheniramine maleate prevented bronchospasm resulted from pulmonary vasoconstriction caused by cement toxicity and pulmonary embolism. Studies performed after observation of intraoperative deaths occurred during cemented arthroplasty have demonstrated the presence of bone marrow embolism, fat embolism and bone embolism and methylmethacrylate particles in the lungs\textsuperscript{27,28,29}. Medulla residue may cause embolism in the lungs, heart or paradoxically in the brain and coronary arteries\textsuperscript{30,31}. This suggests that hypotension occurs due to characteristic hypoxia and right ventricular dysfunction as the consequence of pulmonary embolism\textsuperscript{21}. However, a definite correlation could not be established in the literature between the degree of embolism detected by performing transesophageal

Echocardiography and the severity of hypoxia.

It has been reported that blood cement monomer levels do not reach high concentrations that likely can cause toxicity in humans, in which both mechanisms are considered to play a role. Among anaphylatoxins, C3a and C5a are potent mediators that lead to vasoconstriction and bronchoconstriction. The levels of C3a and C5a have been observed to increase in cemented hemiarthroplasties by complement activation. In human studies, high dose (2g) methylprednisolone have been demonstrated to reduce hypoxia and complement activation. The decreases in anaphylatoxin release and oxygen saturation are reduced by methylprednisolone. However, whether methylmethacrylate particles or embolus material causes complement activation is not clear. Complement levels were not studied in the present study. We thought that complement activation might have been prevented by dexamethasone, due to the less decrease in hemodynamic values—although no statistically difference was found, but clinically important—1 min after cementation and lack of desaturation in Group PD.

In conclusion, the dose of adrenaline and atropine used after cementation was significantly lower and, SpO2 values were stable after cementation in Group PD; on the other hand, in Group S, SpO2 values decreased after cementation, except for 1 min after cementation. SpO2 value increased 1 min after cementation. This suggests that prophylactic administration of pheniramine maleate and dexamethasone may minimize the clinical symptoms of BCIS.
References


The key to

Lock-up

Postoperative Pain

Initial bolus
Inject 1 ampoule Tramal® 100 mg i.v. or i.m. slowly over 2-3 minutes

Ways of administration after initial bolus

Infusion
Inject 2 ampoules Tramal®, each 100 mg, in 500mL of infusion solution. Infusion rate 12-24 mg Tramal® (16-20 mL/hour or 30-60 mL/hour). Subsequent increments of 30 mg with a lock-out time of 5 minutes.

If needed further doses of Tramal® 50 mg up to a total of 200 mg (excluding the initial bolus) within the first 60 min.

PCA
Usual dose is 50 mg or 100 mg 14 hourly up to a total daily dose of 400 mg except in special clinical circumstances which might necessitate daily dose up to 400 mg. Further treatment with Tramal® boluses on demand.

Injection

Follow-up
1-2 capsules every 4-6 hours
50 mg
20-40 drops every 4-6 hours
100 mg
1 suppository every 4-6 hours
slow release
1 tablet every 12 hours

Intra-Operative
Loading Dose
2.5 - 3 mg/kg at wound closure

Post-Anaesthesia Care Unit

An intra-operative loading dose of Tramal® will reduce PONV rates

If intra-operative dose not given then:
BOLUS I.V.*
100 mg over 2-3 mins

*if needed further doses of 50 mg up to a total of 200 mg (incl. the initial bolus) may be given within the first 60 min.

References:
For patients with localized burning, shooting, or stabbing neuropathic pain, Versatis 5% lidocaine medicated plaster works where it hurts.
BRIDION—**for optimal neuromuscular blockade management** and improved recovery

**Predictable and complete reversal**
- 98% of BRIDION patients recovered to a TOF ratio of 0.9 from reappearance of T₂ within 5 minutes²
- 97% of BRIDION patients recovered to a TOF ratio of 0.9 from 1 to 2 PTCs † within 5 minutes¹

**Rapid reversal**
- BRIDION rapidly reversed patients from reappearance of T₂ in 1.4 minutes²
- BRIDION rapidly reversed patients from 1 to 2 PTCs † in 2.7 minutes³

**BRIDION** is indicated for the reversal of neuromuscular blockade induced by rocuronium or vecuronium. In children and adolescents (aged 2-17 years), **BRIDION** is only recommended for routine reversal of moderate rocuronium-induced neuromuscular blockade.¹

**Important safety information**

BRIDION is not recommended in patients with severe renal impairment. Studies in patients with hepatic impairment have not been conducted and, therefore, patients with severe hepatic impairment should be treated with great caution. Cautions should be exercised when administering BRIDION to pregnant women as no clinical data on exposed pregnancies are available.

If neuromuscular blockade is required within 24 hours of BRIDION administration, a non-neuromuscular blocking agent should be used instead of rocuronium or vecuronium. The most commonly reported adverse reactions were dysgeusia (metal or bitter taste) and anesthetic complications (movement, coughing, grimacing, or sucking on the endotracheal tube). In patients treated with BRIDION, a few cases of awareness were reported. The relation to BRIDION was uncertain. In a few individuals, allergic-like reactions (ie, flushing, erythematous rash) following BRIDION were reported. Clinicians should be prepared for the possibility of allergic reactions and take the necessary precautions. In a trial of patients with a history of pulmonary complications, bronchospasm was reported in 2 patients and a causal relationship could not be fully excluded.

Volunteer studies have demonstrated a slight (17%-22%) and transient (<30 minutes) prolongation of the prothrombin time/activated partial thromboplastin time (PT/aPTT) with BRIDION, however, clinical studies have demonstrated no clinically relevant effect on peri- or postoperative bleeding complications with BRIDION alone or in combination with anticoagulants. As BRIDION has demonstrated an in vitro pharmacodynamic interaction with anticoagulants, caution should be exercised in patients on anticoagulation for a pre-existing or concomitant condition. This pharmacodynamic interaction is not clinically relevant for patients receiving routine postoperative prophylactic anticoagulation. Although formal interaction studies have not been conducted, no drug interactions were observed in clinical trials. Preclinical data suggest that clinically significant drug interactions are unlikely with the possible exceptions of toremifene, fusidic acid, and hormonal contraceptives.

¹ Train-of-four
² Post tetanic counts
³ Second twitch

**REFERENCES**

Please see summary of product characteristics for full prescribing information.

**MSD**

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