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Original Article

Anaesthesia Section

Comparative Study of Atropine Combined with Sodium Nitroprusside Pretreatment to Prevent Trigemino Cardiac Reflex after Trigeminal Ganglion Compression

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ABSTRACT

Introduction: Manipulation of percutaneous compression of the trigeminal ganglion (PCTG) for trigeminal neuralgia (TN) can lead to significant haemodynamic changes, which were termed trigemino cardiac reflex (TCR). Nevertheless, many studies indicated that atropine pretreatment can reduce the incidence of bradycardia and cardiac arrest, but do not take precautions against abrupt rise of blood pressure.

Aim: The purpose of our study was to compare control group {patients receiving Sodium Nitro-Prusside (SNP) pretreatment before PCTG} with study groups (patients receiving different doses of atropine combined with SNP pretreatment before PCTG) in cardiovascular parameters {Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Heart Rate (HR)} at 5 periods during Total Intravenous Anaesthesia (TIVA).

Materials and Methods: In total, 120 patients, who underwent PTCG, were enrolled and randomly assigned into control group {group A (SNP pretreatment before PCTG, n=29)} and study groups {group B (0.002mg /kg atropine combined with SNP pretreatment before PCTG, n=30), C (0.004mg/kg atropine pretreatment before PCTG, n=31) and D (0.006mg/kg atropine combined with SNP pretreatment before PCTG, n=30)}, the relationship between haemodynamic changes and using atropine pretreatment or not was compared. Cardiovascular parameters were measured at five periods: preoperative (T0); before puncture (T1); during compression (T2); 1 min after the compression ended (T3); and 1 min after the procedure ended (T4). Multivariate analysis of variance (MANOVA) and Pearson's χ^2 test were used, and a value of $p < 0.05$ was considered statistically significant.

Results: Compared with the group A, means of SBP and DBP in the study groups (group B, C and D) were not observed significant differences at all time points ($p>0.05$), the mean values of HR showed significant differences, when compared to group C and group D at T2 and T3 ($p<0.001$). Meanwhile, means of SBP, DBP and HR comparison in the same group were observed between T1 and T2, to the group A, B and D, means of HR ($p>0.05$ vs. T1) indicated significant differences, however, there was no significant difference in group C ($p>0.05$). Furthermore, the incidence of post-compression tachycardia was observed in each group.

Conclusion: By comparison, it seemed that 0.004mg/kg atropine pretreatment before PCTG was more reasonable for preventing significant haemodynamic changes.

Keywords: Haemodynamic changes, Percutaneous compression, Trigeminal neuralgia

INTRODUCTION

Percutaneous Compression of the Trigeminal Ganglion (PCTG) as treatment for trigeminal neuralgia (TN) was first described by Mullan and Lichtor [1]. Some drastic haemodynamic changes have been observed during the manipulation of PCTG for TN [1-5], which were related to trigemino cardiac reflex (TCR) [6]. Previous study indicated that using Sodium Nitro-Prusside (SNP) before puncture was an effective method to control abrupt rise of blood pressure, meanwhile, it seemed that PCTG technique returned the bradycardia and cardiac arrest to normal automatically in short time without the need for additional anti-cholinergic medication to patients with normal regulation ability, this study demonstrated that SNP can prevent marked blood pressure elevation, but not prevent bradycardia and cardiac arrest simultaneously [7]. Admittedly, it was still necessary to maintain the preoperative stability of Heart Rate (HR). Although anti-cholinergic medication was a choice for preventing bradycardia and cardiac arrest, few studies had focused on that anti-cholinergic drugs caused cardiac arrhythmias simultaneously, such as postcompression tachycardia [8-11]. Consequently, we planned to find a more suitable dose of anti-cholinergic medication to prevent bradycardia and cardiac arrest without less refractory cardiac arrhythmias on basis of using SNP.

MATERIALS AND METHODS

The study was approved by the Ethical Committee of Liaoning Provincial People's Hospital on 10 August 2013. After institutional review board approval and patients' written informed consent, 120 patients were diagnosed with TN and underwent PCTG, the exact number was 122, because two patients with temporary cardiac arrest were in control group (group A), they were excluded. Among all patients (Chang-Ming Wang took part in operations, which were included in the study, others were excluded) who underwent PCTG between August 2013 and April 2014, no patient showed neurological abnormalities, before the start of anaesthesia.

Characteristics of patients included in our study are summarized in [Table/Fig-1], scored as American Society of Anaesthesiologists (ASA) I–II, aged 39–84 years and weighed 40–98 kg. A uniform protocol of anaesthesia had been established for these patients. The patients were randomly assigned to four groups: control group {group A (SNP pretreatment before PCTG, n=29)} and study groups {group B (0.002mg/kg atropine combined with SNP pretreatment before PCTG, n=30}, C (0.004mg/kg atropine pretreatment before PCTG, n=31) and D (0.006mg/kg atropine combined with SNP pretreatment before PCTG, n=30)}. The surgical procedures for PCTG were performed under general anaesthesia with a laryngeal

mask, which adopted the procedure as first described by Mullan and Lichtor in 1983 [1]. C-arm intensifier fluoroscope and stereotactic navigator system was used for localization. The point of entry into the skin was 2.5 cm external to the angle of the mouth. A 14 gauge catheter being redirected under fluoroscopic guidance until the foramen ovale was entered. The balloon was inflated with Omnipaque water-soluble and balloon placement was not correct until pear shape, pear-like shape and hourglass or dumbbell shape were achieved, which indicated the correct balloon position and surgical success, following ganglion compression for 2 min. The balloon pressure was controlled according to the experience of the operator and the individual condition of patient with a dosage of contrast material varied from 0.3 to 1.0 ml. After compression, the balloon was deflated, the balloon and the needle were withdrawn together. Firm pressure was applied to the puncture site to prevent hematoma.

All patients were premedicated to provide sedation (intramuscular injection of 0.5mg atropine and 0.1 mg/kg diazepam) 30min before surgery. Operative and anaesthesia procedures were performed in the operating theater after a brief period of general anaesthesia had been induced with Laryngeal Mask Airway (LMA) and monitored by noninvasive blood pressure measurement, oximetry and electrocardiological recordings. Sedation and analgesia consisted of the combination of remifentanil hydrochloride, propofol and succinylcholine chloride. Anaesthesia was induced with propofol 2.0mg.kg-1 over 20s followed immediately by remifentanil 1.0μg. kg-1, intravenous succinylcholine 0.5-1.0mg/kg was given 20s after completing the bolus administration of remifentanil. As soon as patients lost consciousness (unconsciousness was measured by eyelash reflex), ventilation was attempted via a mask. Then an appropriately sized laryngeal mask was inserted by using index finger insertion method within 60s. Anaesthesia was maintained with remifentanil (0.15μg.kg-1.min-1), propofol(50μg.kg-1. min-1) and succinylcholine (30μg.kg-1.min-1) continuous pump infusion, which were withdrawn at the end of compression. When patients recovered consciousness, muscle tension, autonomous respiration and swallowing reflex returned to normal, the LMA was extubated, patients were returned to the wards after observed 5-10 min. Preoperative atropine and pacemaker were prepared. All patients' SNP pretreatment was initiated at a rate of 0.5μg/kg/ min according to blood pressure, to regulate SNP speed.

SBP, DBP and HR were measured at five periods: preoperative (T0), before puncture (T1), during compression (T2), 1 min after the compression ended (T3) and 1 min after the procedure ended (T4).

STATISTICAL ANALYSIS

Data were presented as mean ± SD (mean of standard deviations). Variables were tested about normal distribution with Kolmogorov-Smirnov test and Q-Q plots. The parametric test of variance analysis for repeated measures cardiovascular parameters in the above-mentioned subgroups of patients was assessed by Multivariate Repeated Measures ANOVA. Demographic data were compared using Pearson's χ^2 test. The $p<0.05$ was considered to be statistically significant.

RESULTS

The patient characteristics (sex, age and weight) of the 120 patients included in our study are summarized in [Table/Fig-1], There were no significant differences in the patient characteristics of different subgroups. All patients who had idiopathic TN were diagnosed with the same standard by a neurosurgeon (professor Yi Ma), and were refractory to, or intolerant of, carbamazepine.

The means of cardiovascular parameters (SBP, DBP and HR) measured at five periods is shown in [Table/Fig-2]. Compared with the group A, means of SBP and DBP in the study groups (group B,C and D) were not observed significant differences at all time points (p>0.05 vs.group A), the mean values of HR showed significant differences at T2 and T3 in group C (T2:78.2 ±12.8bpm, 50.9±6.7bpm; T3:96.6±16.6bpm, 81.9±16.0bpm, p<0.001vs. group A) and group D (T2:87.5 ±17.4bpm, 50.9±6.7bpm; T3: 111.7±18.2bpm, 81.9±16.0bpm, p<0.001vs.group A). Meanwhile, means of SBP, DBP and HR comparison in the same group were observed between T1 and T2, means of HR (group A:50.9±6.7bpm, 72.8±11.8bpm; group B:52.8±10.1bpm; group C: 87.5±15.4bpm, 70.5±12.8bpm, p<0.001 vs. T1) in the group A, B and D indicated significant differences, however, there was no significant difference in group D (p>0.05vs.T1).

Moreover, the incidence of post-compression tachycardia was observed in each group. As shown in [Table/Fig-3], the percentage of less than 100 beats/min of HR was 72.8%, 73.3%, 61.3% and 36.7% (HR<100beats/ min) in group A, B, C and D respectively. Incidence of post-compression tachycardia was 17.2%, 16.7%, 16.1% and 10.0% (100beats/min≤HR<110beats/min), 0.0%, 0.0%, 12.9% and 16.7% (110beats/min≤HR<beats/min), 0.0%, 0.0%, 9.7% and 36.7% (HR ≥120 beats/ min) in group A, B, C and D separately.

	Group A	Group B	Group C	Group D	p
Sex: male (n [%])	10 (34.5)	12(40.0)	10(32.3)	9 (30.0)	0.864
Mean age (years)	63.7±12.8	64.8±9.1	65.1±8.6	62.1±9.6	0.161
Mean weight (kg)	63.5±9.1	66.3±11.2	62.9±9.0	66.9±13.0	0.404

[Table/Fig-1]: Control group (group A) and study groups (group B, C and D) were paired in their general characteristics (n=120).
The Pearson's χ^2 test, general characteristics were no different between the groups.

	Group A	Group B	Group C	Group D
T0 SBP	158.5(SD=24.8)	166.1(SD=25.8)	156.3(SD=22.4)	160.5(SD=32.8)
T1 SBP	120.6(SD=26.6)	124.4(SD=25.5)	115.4(SD=27.0)	115.9(SD=23.8)
T2 SBP	131.2(SD=29.1)	135.0(SD=27.5)	127.9(SD=19.8)	127.9(SD=36.4)
T3 SBP	128.2 (SD=21.6)	126.9(SD=20.2)	131.3(SD=15.3)	132.5(SD=15.1)
T4 SBP	139.0(SD=17.2)	146.4(SD=14.0)	143.9(SD=18.4)	141.6(SD=21.3)
T0DBP	88.2(SD=11.9)	91.1(SD=10.3)	87.9(SD=9.8)	87.9(SD=12.0)
T1 DBP	70.8(SD=14.5)	70.7(SD=15.8)	69.6(SD=15.6)	70.1(SD=13.7)
T2 DBP	77.2(SD=18.5)	78.3 (SD=16.8)	81.6(SD=12.8)	76.8(SD=22.8)
T3 DBP	75.9(SD=13.4)	80.7(SD=13.6)	80.3(SD=12.0)	79.7(SD=10.9)
T4 DBP	82.1(SD=12.1)	87.1(SD=11.8)	85.9(SD=10.1)	87.1(SD=11.2)
T0HR	81.5(SD=11.8)	75.8(SD=11.7)	79.1(SD=12.5)	78.6(SD=15.9)
T1HR	72.8(SD=11.8)	68.7(SD=9.8)	72.2(SD=17.7)	70.5(SD=12.8)
T2 HR	50.9(SD=6.7)††	52.2 (SD=10.1)††	78.2(SD=12.8)##	87.5(SD=15.4)##††
T3 HR	81.9(SD=16.0)	88.5(SD=11.8)	96.6(SD=14.6)##	111.7(SD=18.2)##
T4 HR	84.2(SD=12.7)	79.1(SD=7.9)	82.5(SD=14.0)	84.3(SD=18.6)

[Table/Fig-2]: Means of SBP (mmHg), DBP (mmHg) and HR (bpm) of patients were at different times in group A (n=29), group B (n=30), group C (n=31) and group D (n=30).
Multivariate analysis of variance (MANOVA).
†There was a statistical difference between the groups at the evaluation during the procedure.
There was a statistical difference between T2 and T1 in four groups.
HR indicates heart rate; DBP, diastolic blood pressure; SBP, systolic blood pressure.
p < 0.001 study groups (group B, C and D) vs. control group (group A).
†† p < 0.001 T2 vs. T1 in the same group.

HR (beats/min)	Group A (n=29, %)	Group B (n=30, %)	Group C (n=31, %)	Group D (n=30, %)
<100	24 (72.8%)	25 (73.3%)	19 (61.3%)	11 (36.7%)
110> ≥100	5 (17.2%)	5 (16.7%)	5 (16.1%)	3 (10.0%)
120> ≥110	0 (0.0%)	0 (0.0%)	4 (12.9%)	5 (16.7%)
≥120	0 (0.0%)	0 (0.0%)	3 (9.7%)	11 (36.7%)

[Table/Fig-3]: Control (group A) and study (group B, C and D) groups were paired at T3 in the range of HR change (n=120).
HR indicates heart rate

DISCUSSION

PCTG is an alternative technique for the treatment of TN [1,12,13]. PCTG relieves pain and brings significant haemodynamic changes (bradycardia, cardiac arrest, hypotension or hypertension and tachycardia) to patients during the procedure simultaneously [14]. The significant haemodynamic changes are related to TCR [15-17].

Whatever measures were taken to prevent TCR, we must balance the curative effect and the potential harmful side effects. Brown and Preul firstly made a detailed description of the occurrence of trigeminal depressor response (now known as TCR) in balloon compression techniques [8], initially they suggested using temporary peacemaker, rather than local anaesthesia, atropine, ketamine and others [18,19]. However, anti-cholinergic medication, such as atropine, still was widely used clinically to prevent PCTG-related TCR [20,21]. It is clear from a pharmocological point of view that anti-cholinergic medication cannot completely prevent the TCR. Many studies had demonstrated that anti-cholinergic medication can reduce the incidence of bradycardia and cardiac arrest [1,22,23]. Precaution of TCR due to the risk of ventricular tachycardia and arrhythmias from anti-cholinergic medication [9,10], although the heart rate-increasing effect of atropine is well known. TCR can occur with the premedication of atropine in few patients in study, we considered that atropine was probably not given at the proper timing of administration and with the proper dosage of atropine. We believed that it was still very important for researchers at the proper timing of administration and with the proper dosage of drugs to prevent the potential harmful side effects. To our knowledge, few studies have focused on the prospective studies about TCR [11], so far there is no study on different doses of atropine how to affect TCR to PCTG and the tachycardia and arrhythmias after PCTG, which caught our concerns.

Some studies elaborated that TCR was a physiological, but not a pathophysiological entity [7,24], it was that TCR possibly be considered a protective reflex, which meant that TCR was a self-protect auto-regulation. Although our previous result showed that PCTG technique returned the HR to normal automatically in short time without the need for additional anti-cholinergic medication [7]. Admittedly, it was still necessary for patients with poor self-regulation to maintain the change in the reasonable range of HR. Our study indicated that transient severe bradycardia and a simultaneous rise in systemic blood pressure were observed immediately after PCTG, which accorded with what Chen et al., reported [9,25]. On the basis of our previous clinical study, we found that it was an effective method to control abrupt rise of blood pressure with SNP [7]. Hanamoto H et al., reported that SNP-induced hypotension was often associated with reflex tachycardia [26], however, our study showed that it was relatively small impact on HR with application of SNP in short time, because bradycardia and cardiac arrest still appeared during this procedure. Herein, we only wanted to seek a relatively reasonable dosage of atropine combined with SNP to prevent bradycardia, cardiac arrest and marked blood pressure elevation. During the clinical study, we observed that the incidence of postcompression tachycardia gradually increased with the increase in dosage of atropine on the basis of SNP, which is reasonable for dose-response. Transient cardiac arrest of two cases happened in the group without application of atropine, however, there were no significant differences in SBP and DBP among groups during the procedure. This illustrated that postcompression tachycardia did not lead to significant fluctuation of SBP and DBP, which was due to SNP. Meanwhile, transient cardiac arrest can indeed be prevented with atropine. It meant that the reasonable dosage of atropine combined with SNP was used at the appropriate point of time which can maintain the stable haemodynamics.

Till now, mechanism related to TCR is still not fully elucidated, although it does not seem plausible to make recommendation for the use of combination therapy (atropine and SNP), symptomatic treatment may be only an effective choice in maintaining the stable haemodynamic changes related to TCR before seeking a method of the etiological treatment. It must be admitted that there were some limitations in the study. For example, all patients received intramuscular atropine as a premedication, however, here it seemed that intramuscular atropine 30min before surgery did not affect PCTG-related TCR in control group. In addition, another control group (using atropine without SNP before PCTG) should be added. Nevertheless, we must consider that postcompression tachycardia might lead to sudden fluctuation of blood pressure without SNP before PCTG, which cannot be accepted by operators and anaesthesiologists. Because it will increase the risk of cheek hematoma and bear the risk of cerebrovascular accidents, particularly for those who are the elderly patients. Sweet et al., had emphasized that abrupt arterial blood pressure can be one factor of inducing intracranial haemorrhage occurring in patients [27]. Herein, we agree with the standpoint that a definite cause-effect relationship is best investigated through an animal study, not in human subject [5]. Besides, we cannot tell which one is more harmful for health between transient bradycardia, even cardiac arrest and postcompression tachycardia. To our knowledge, only few cases die of postoperative hematoma and infection, we do not know whether postoperative hematoma is related to abrupt arterial blood pressure, postcompression tachycardia or others. Consequently, we only attempted to reduce the incidence of transient severe bradycardia, a simultaneous rise in systemic blood pressure and postcompression tachycardia simultaneously through external drug intervention.

CONCLUSION

We must acknowledge that a substantial knowledge about trigeminal ganglion and TCR is insufficient and the current work demonstrates that it works completely differently than the peripheral TCR. Our study indicates that atropine combined with SNP pretreatment at the appropriate point of time can prevent the significant fluctuation of blood pressure and reduce the incidence of transient severe bradycardia. However, the incidence of postcompression tachycardia gradually increases with the increase in dosage of atropine combined with SNP. By comparison, it seems that 0.004mg/kg atropine combined with SNP pretreatment before PCTG is more reasonable for preventing TCR related to significant haemodynamic changes.

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