Treatment of supraventricular tachycardias in patients with non-cardiac surgery by dexmedetomidine during the perioperative period

Yan Xu
the 74th Group Army Hospital of P.L.A
CuiWen Hu
The Affiliated Lianyungang No.2 People's Hospital of Bengbu Medical College
Xuan Guo
the Affiliated No.4 Hospital of Jiangsu University
ZhiHong Hu
the 74th Group Army Hospital of P.L.A
Hui Shi
The Affiliated Lianyungang No.2 People's Hospital of Bengbu Medical College
JunLong zhang (527823224@qq.com)
the Affiliated Lianyungang No.2 People's Hospital of Bengbu Medical College

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Abstract

Background: Supraventricular tachycardias (SVTs) can increase the risk of adverse events in perioperative period. Previous studies have shown that application of dexmedetomidine (DEXm) combined anesthesia during surgery can significantly reduce postoperative cardiovascular and cerebrovascular complications and mortality in patients with cardiac disease. In fact, many anesthetic drugs have cardiac protection effects. However, it is a pity that these findings are not well applied in clinical practice to treat cardiac disease. Therefore, the aim of this study was to explore the therapeutic effect of DEXm on perioperative SVTs in adult patients with non-cardiac surgery.

Methods: Forty-two patients with SVTs, aged between 35 and 61 years, were randomly divided into DEXm group (group D) and midazolam group (group M). The patients undergoing elective surgery in two groups were infused intravenously DEXm 0.5-1μg/kg or midazolam 0.06-0.08mg/kg using a micro-pump for 10 minutes, respectively. The the Observer's Assessment of Alertness/Sedation (OAA/S) score, heart rate (HR), mean arterial pressure (MAP), pulse oxygen saturation (SpO_2) and occurrence of SVTs, heart rate variability (HRV) including normalized low frequency power (LFnorm), normalized high frequency power (HFnorm) and the balance ratio of sympathetic to vagal tone (LF/HF) in two groups were recorded at T0 (before the infusion DEXm or midazolam), T1 (5 minutes after the infusion), T2 (at the end of the infusion), T3 (5 minutes after the end of the infusion), and T4 (10 minutes after the end of the infusion).

Results: The OAA/S score in two groups at T4 was obviously decreased compared with T0. And the OAA/S score in group M was lower than in group D at T4 (P<0.05). Compared to T0, HR and MAP in two groups were obviously decreased, and HR and MAP in group D were apparently lower than group M from T1 to T4 (P<0.05). Three patients developed mild hypotension in group D. However, none of patients developed clinically significant bradycardia, hypotension, and anoxia. There was no significant difference for SpO_2 from T0 to T4 in group D. Compared to T0 or group D, SpO_2 in group M obviously decreased at T2 (P<0.05). In addition, SVTs in all patients were terminated until T4 in group D after DEXm infusion. However, only two patients were finally improved in group M. Compared to T0, HFnorm were elevated, and LFnorm and LF/HF were decreased from T1 to T4, furthermore, the changes in HFnorm, LFnorm and LF/HF had statistical significance (P<0.05) in group D. However, there was no significant difference for HFnorm, LFnorm and LF/HF in group M from T0 to T4.

Conclusions: Perioperative use of dexmedetomidine had a significant therapeutic effect for supraventricular tachycardias without significant adverse effects in adult patients .

Trial Registration: ClinicalTrials.gov Registration Number: NCT04284150 on 26th February 2020

Background

Supraventricular tachycardias (SVTs) is a common arrhythmia in the perioperative period, which is associated with adverse stimulus such as cardiovascular risk factors, emotional tension, hypoxia, CO_2 accumulation, hypokalemia, and pain[1, 2]. Although anesthetic drugs have not been used widespreadly

to prevent cardiovascular event, many anesthetic drugs, such as propofol, sevoflurane, have shown the ability of cardiac protection. Meanwhile, many anesthetic drugs with cardiovascular protection is a common adjuvant method for the treatment of perioperative SVTs [3]. Dexmedetomidine (DEXm) exerts anti-stress, sedative, and analgesic roles by selectively acting on the α-2 receptors of the central and peripheral nervous systems. DEXm is widely used as an adjuvant to general anesthesia [4].

DEXm is approved by FDA in operating room for adjuvant anesthesia and in intensive care unit for sedation in adults. Although DEXm is not approved for the treatment of arrhythmias, a growing number of evidences indicated that it can serve as a potential treatment for arrhythmias. Liu et al. confirmed that DEXm can reduce ventricular rate and improve atrial fibrillation in pediatric patient with cardiac surgery [5]. Ji et al. [6] aslo found that DEXm can be effective in reducing cardiovascular and cerebrovascular complications and mortality in pediatric patients one year after coronary bypass surgery. Although many previous studies of pediatric patients have shown that DEXm can effectively terminate perioperative tachyarrhythmia [7–11], the therapeutic effect of DEXm on SVTs in the adult population is unclear. Furthermore, some literatures reported that patients with SVTs did not respond to adenosine, amiodarone, digoxin and verapamil, and these drugs may cause many serious adverse effects, such as bronchospasm or ventricular fibrillation [12, 13]. Therefore, we explored the therapeutic effect of DEXm on perioperative SVTs in adult patients.

Materials And Methods

The protocol of this study was approved by the medical ethics committee of hospital, and written informed consent was obtained from all subjects participating in the trial. The trial was registered prior to patient enrollment at clinicaltrials.gov (NCT04284150, Principal investigator: JunLong Zhang).

Diagnostic criteria for SVTs

SVTs is defined as abnormal cardiac rhythms with heart rates exceeding 100 beats/min at rest, and electropathologic substrate originating above the Bundle of His [14]. We followed that convention and chose regular SVTs including sinus tachycardia, atrial flutter, atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reciprocating tachycardia (AVRT), atrial tachycardia (AT) in our study [15]. A continuous 12-lead electrocardiogram was used in patients.

Patient monitoring

The following indicators of patients are recorded every 5 minutes, including T0 (before the infusion DEXm or midazolam), T1 (5 minutes after the infusion), T2 (at the end of the infusion), T3 (5 minutes after the end of the infusion), and T4 (10 minutes after the end of the infusion), as shown in Table 1.

Sedation depth was assessed by the Observer's Assessment of Alertness/Sedation (OAA/S) score and documented as a score of 5 (responding readily to name spoken in normal tone); 4 (lethargic response to name spoken in normal tone); 3 (responding only after name is called loudly or repeatedly); 2 (responds

only after mild prodding or shaking); 1 (no response to mild prodding or shaking); 0 (no respond to noxious stimuli) [16].

Table 1
Observation indicators at T0, T1, T2, T3, T4 in two groups

Observation indicators at T0, T1, T2, T3, T4	
occurrence of SVTs	the Observer's Assessment of Alertness/Sedation (OAA/S) score
normalized low frequency power (LFnorm)	heart rate (HR)
normalized high frequency power (HFnorm)	mean arterial pressure (MAP)
the balance ratio of sympathetic to vagal tone (LF/HF)	pulse oxygen saturation (SpO2)

Exclusion criteria

1. 1. Patients who suffered from significant hemodynamic instability and had recent history of complete atrioventricular (AV) block, were thus excluded from the study.
2. 2. Patients suffered from other types of arrhythmia, not SVTs, and coupled with anaphylaxis to α-2 receptors agonist (α2-RA) and benzodiazepines, were excluded from the study.

SVTs management

Forty-two patients with SVTs, aged between 35 and 61 years, American Society of Anesthesiologists physical status □-□, undergoing elective non-cardiac surgery, were randomly divided into group DEXm (D) and group midazolam (M). The patients calm down for 5–10 minutes after getting into the operating room, and were commenced with intravenous infusion DEXm (0.5-1 μg/kg, Jiangsu Hengrui Pharmaceutical Co., Ltd., Lianyungang, China) or midazolam (0.06–0.08 mg/kg, Jiangsu Nhwa Pharmaceutical Co., Ltd., Xvzhou, China) using a micro-pump for 10 minutes. In this study using a double-blind method, DEXm and midazolam were administered by an anesthesiologist, and the therapeutic effect was evaluated by a professional cardiologist. Patients who did not respond to DEXm and midazolam, were subsequently treated with the appropriate and conventional approach.

Evaluation of treatment response

SVTs were terminated and convert to normal sinus rhythm (60–100 bpm), which was used to assess the therapeutic effect of DEXm and midazolam.

Statistical analysis

Statistical analysis were performed with IBM SPSS 25.0 (IBM Corp, Armonk, NY, USA). The data were expressed as mean ± standard deviation. The comparisons between two groups were done with independent sample *t*-test. Intra-group comparisons were performed using the analysis of variance test of

repeatedly measured data. Frequency percentages were analyzed using χ2-test. A *P* value < 0.05 was considered statistically significant.

Results

The general information in two groups of patients

There was no significant difference in the general information (Table 2) and the SVTs profile (data not shown) between two groups. Two patients were excluded because of preoperative use of midazolam and missing data at T2 and T3, respectively.

Table 2
The general information comparison for patients in two groups (*P* > 0.05, n = 20)

	Group D	Group M
Age (yr)	43.4 ± 5.6	44.1 ± 4.9
Sex (female/male)	7/13	9/11
BMI (kg/m^2)	23.1 ± 3.7	22.6 ± 2.5
Affiliated disease		
Abnormal electrocardiogram before operation	3	2
Hypertension	2	2
Diabetes	1	2
Coronary heart disease	1	0
Hypokalemia	1	0

Adverse effects

Three patients developed mild hypotension that responded well to intravascular volume in group D. Three patients in group M developed a decrease of SpO_2 (below 92%), which was quickly improved by mask oxygen inhalation. None of the patients developed any clinical significant hypotension (20% decrease in blood pressure), bradycardia, and anoxia. And they all remained hemodynamic stability .

Table 3
OAA/S scores in the two groups (n = 20)

	T0	T4
group D	5.00 ± 0.00	$3.76 \pm 0.44^{*\#}$
group M	5.00 ± 0.00	$3.07 \pm 0.78^{*}$

$^{*}P < 0.05$ compared with T0. $^{\#}P < 0.05$ compared with group M.

The degree of sedative in two groups of patients

Compared with T0, the OAA/S score in two groups was obviously decreased at T4, and the OAA/S score in group M was lower than group D at T4 (Table 3). The lower the OAA/S score produced the deeper sedation in two groups. The deep sedation caused the depression of respiration, thereby resulting in hypoxia, CO_2 accumulation, increased heart rate and other adverse effects.

The vital signs of patients in two groups

At time T0, there was no difference (P 0.05) for HR, MAP and SpO_2 in two groups. Compared to T0, HR and MAP in two groups are obviously decreased, and HR and MAP in group D is apparently lower than group M from T1 to T4 (P>0.05). Three patients developed mild hypotension in group D. However, none of patients developed clinically significant bradycardia, hypotension, and anoxia. There was no significant difference for SpO_2 from T0 to T4 in group D. Compared with T0 or group D, SpO_2 in group M obviously decrease at T2. Three patients with infusion midazolam even experienced a decrease of SpO2 at T2 (>92%), but which were quickly improved by mask oxygen inhalation (Table 4). These evidences indicated that DEXm improved HR of the patients with SVTs better than midazolam, and can moderately reduce blood pressure. However, its respiration inhibition was much than midazolam.

Table 4
Comparison of vital signs between the two groups (n = 20)

		T0	T1	T2	T3	T4
HR (bpm)	Group D	115.1013.06	93.109.91*#	72.1010.44*#	70.657.86*#	74.406.08*#
	Group M	111.877.59	106.054.62*	104.283.89*	103.623.71*	103.073.25*
MAP (mmHg)	Group D	107.5512.73	100.33 ± 9.96	83.9812.35*#	80.4110.84*#	85.737.50*
	Group M	107.7418.13	99.2713.37*	93.4313.70*	89.5512.60*	90.5811.92*
SpO_2 (%)	Group D	96.82 ± 2.07	96.00 ± 2.26	96.06 ± 1.48	95.41 ± 1.37	96.88 ± 1.32
	Group M	97.93 ± 1.48	96.78 ± 1.17	94.93 ± 3.02*#	95.49 ± 1.20	96.72 ± 2.08

*P < 0.05 compared with T0. #P < 0.05 compared with group M.

The treatment efficiency of SVTs in two groups

Among the twenty SVTs patients in group D, SVTs in thirteen patients were terminated at T1, and the effective rate was 65%; SVTs in eighteen patients were terminated at T2 and T3, and the effective rate was 90%; SVTs in all patients were terminated at T4. However, only two patients with SVTs were improved until T4 in group M, which was distinctly different from group D (P>0.05), as shown in Fig. 1. Figure 2-Figure 5 showed four patients with typical SVTs converted to NRS during 4–18 minutes after infusion DEXm. Notably, no severe adverse events occurred in both groups from T1 to T4. These evidences showed that DEXm had a good treatment efficiency for SVTs.

The heart rate variability (HRV) in two groups

The differences had no statistical significance between two groups for LFnorm, HFnorm and LF/HF at T0 (P 0.05). Compared to T0, HFnorm were elevated, and LFnorm and LF/HF were decreased, furthermore, the changes in HFnorm, LFnorm and LF/HF have statistical significance (P>0.05) in group D from T1 to T4. However, the changes in HFnorm, LFnorm and LF/HF in group M have no statistical significance (P 0.05), as shown in Fig. 6.

HRV is one of the noninvasive quantitative indicators of autonomic nerves regulation of cardiac activity. LFnorm and HFnorm mainly reflects sympathetic activity and vagal activity, respectively, in frequency-domain analysis. LF/HF as balance ratio of sympathetic to vagal tension can more directly reflect the changes of sympathetic and vagal regulation than a single LF or HF. The changes in HFnorm, LFnorm and LF/HFin group D from T0 to T4, which indicated DEXm can regulate cardiac autonomic nervous activity, and enhance the vagal neural activity.

Discussion

Supraventricular tachycardias (SVTs) is the most common cardiac complications, and can frequently cause serious cardiovascular events and hemodynamic instability, leading to increased morbidity and mortality in the perioperative period. SVTs is associated with disorders of autonomic nervous regulation of cardiac activity, and reentrant excited. Dexmedetomidine (DEXm), a highly selective α2-RA, was initially used as sedative and anaesthetic adjuvants. The antiarrhythmic effect of DEXm in pediatric cardiac surgery and intubation reflex was observed in previous literature, suggesting that its antiarrhythmic effect in noncardiac surgery and adult patients needs to be further explored. Here, we designed the study to evaluate therapeutic effect of DEXm on SVTs in adult patients. Furthermore, we took full advantage of DXEm effects, such as bradycardia and the negative chronotropic effects of the heart (e.g. treat tachyarrhythmia). Given that the SVTs had proven to be difficult to control and had resulted in adverse effects, we chose to continuously infuse DEXm. Our results (Table 4 and Fig. 1) has demonstrated that DEXm had a good therapeutic effect and safety profile, and much few adverse effects for the treatment of SVTs. Heart rates started to decrease and thirteen patients with SVTs were remarkably improved only 5 minutes after infusion of DEXm (0.5-1 μg/kg), and SVTs of all patients without recurrence of SVTs in group D were eventually controlled and returned to normal sinus rhythm (NSR) by continuous intravenous infusion of DEXm for 10-minutes, which may be because DEXm can alter the ability to induce the arrhythmia by reducing sympathetic tension. Chrysostomou C et al. [17] also demonstrated that the continuous infusion of DEX during cardiac surgery can decrease incidence of postoperative ventricular and supraventricular tachyarrhythmias in pediatric patients. Meanwhile, none of patients developed clinically significant bradycardia, hypotension, respiratory depression, anoxia, and hemodynamic disorder, except for three patients developing mild hypotension in group D, which is perhaps due to use DEXm alone, and thus a synergistic or additive effect with other sedative and analgesic drugs is impossible. Furthermore, DEXm simultaneously maintain a sufficient level of sedation with a unique property of easy arousal. Previous studies indicated that DEXm can prevent shivering, which effectively reduce body oxygen consumption. Furthermore, the earliest recurrence of SVTs after infusion DEXm was 1 hour, and the duration of its effect increase the amount of arrhythmia-free time and hemodynamic stability [9]. Although DEXm caused mild hypotension, this limitation may be offset because of its unique minimal effect on respiration. Moreover, DEXm has bronchodilatory properties, therefore, it can still be used during asthma attacks. However, only two patients are eventually improved, other patients failed to respond in group M. The sedation degree in group M was deeper than group D (Table 3), and midazolam caused three patients SpO2 drop at T2 (>92%, Table 4), which got better after oxygen inhalation. Earlier studies have shown that intravenous infusion of midazolam 5 mg did not alter the reentrant tachycardia [3]. These data indicated that the sedation is not the mechanism of DEXm for the treatment of SVTs.

Patients suffer from hypokalemia, and/or diabetes, and/or hypertension, and/or coronary heart disease in our study (Table 2). These evidences demonstrated that DEXm had therapeutic effects on SVTs patients coupling with different diseases. It was impressive with a significantly (100%) termination of SVTs using DEXm, which may be associated with types of SVTs we chose. The types of SVTs have narrow-complex regular tachycardias and similar characteristics [18, 19]. Meanwhile, Delwadia S et al. [13] reported DEXm

successfully treat SVTs in a 5 year-old boy with repair of atrial-septal-defect. In especial, DEXm does not cause any significant sinus pause or asystole. It has no negative inotropic or protachyarrhythmic effects, in contrast with many other agents such as β-adrenergic antagonists and amiodarone. Above all, compared with other drugs to treat SVTs, DEXm has a dual purpose: it can be used both as an anti-stress, sedative, analgesic and as an antiarrhythmic drug, which may be more suitable for surgical patient with SVTs.

Table 4 and Fig. 1 have shown that the rhythm or heart rate was significantly decreased, and SVTs were successfully terminated and converted to NSR using DEXm in adults. This is perhaps because DEXm can act on the medullary vasomotor center leading to decrease of catecholamine release from the nerve terminal. In addition, DEXm can decrease approximately 70% of epinephrine and norepinephrine levels in plasma, using the clinical recommended dosage [20]. Previous study reported that DEXm had therapeutic effect on atrial and junctional tachyarrhythmias [7], and can significantly decrease heart rate via regulating autonomic nerve homeostasis [21]. Figure 2-Figure 5 showed four patients with typical SVTs converted to NRS during 4–18 minutes after infusion DEXm. The key physiologic effects of DEXm can significantly affect the cardiac electrophysiology and conduction system, and thus depress sinus and atrioventricular (AV) nodal function [22, 23], as evidenced by significant prolongation of heart rate-adjusted PR and QT interval, AV node block cycle; lengthening of sinus node recovery time and effective refractory period [24]; diminishing atrial excitability [25].

HRV including HF, LF, and LF/HF is considered to be a quantitative indicator of autonomic nerve activity in the heart. HF and LF mainly reflects vagus nerve activity and sympathetic nerve activity, respectively [26]. LFnorm and HFnorm are the normalized values of LF and HF, respectively, consequently, which can more directly reflect the changes of sympathetic and vagus nerve regulation than LF or HF [27]. In our study, Fig. 6 has shown that HFnorm was elevated, and LFnorm and LF/HF were decreased after twenty patients infused DEXm in group D, which revealed that DEXm changed the balance between the sympathetic and parasympathetic tone, and regulated cardiac autonomic nervous system, and powerfully enhanced vagal neural activity, thereby overall rhythm or heart rate control was achieved. Finally, SVTs of all patient were successfully terminated, and converted to NSR. Above all, DEXm exerts antiarrhythmic effects by acting on peripheral and central nervous system. Some studies have shown that paroxysmal SVTs has an imbalance of autonomic nervous system, and tachyarrhythmia is treated by stimulating the vagus nerve and regulating the activity of autonomic nerve [28, 29]. Kamibayashi T et al. [21] by animal-dogs study demonstrated that the antiarrhythmic effects of DEXm are mediated through enhancement of the vagal neural activity. Previous studies have suggested that injury to cardiac parasympathetic nerves resulted in predominance of sympathetic activity during thoracic surgery, which was the primary autonomic mechanism triggering postoperative SVTs. Meanwhile, DEXm containing an imidazole ring can activate imidazoline receptors in the central nervous system, which can prevent adrenaline-induced ventricular tachycardia [30]. Hultin M et al. [31] reported that two patients (1.5 and 3.5 years) with recurrent SVTs were successfully treated by the intranasal administration of DEXm. In a word, DEXm with multiple administrations is safety, appropriate and effective for SVTs and may be an ideal agent for SVTs.

Additionally, use of DEXm can avoid other antiarrhythmic drugs, such as adenosine, verapamil and diltiazem, coupling with cardiac arrest, ventricular dysfunction and cardiovascular collapse [9], which showed DEXm had the important clinical value for surgical patient with SVTs. Therefore, it can be used relatively safely for patients with SVTs in usual clinical doses. However, large doses or rapid infusion DEXm should be used with caution. Because it can potentially have an adverse impact on patients with underlying heart disease, and thus may cause left ventricular dysfunction and hemodynamic instability [32]. Flores-González et al. [33] reported that the infusion duration of DEXm for 10 days and cessation it for 12 hours resulted in 4-year-old girl occurrence of a paroxysmal SVTs.

Conclusion

In conclusion, DEXm may be highly safe and effective treatment choices for terminating perioperative SVTs by regulation the balance of autonomic nervous system and enhancement of vagal neural activity, which reduce the incidence of cardiovascular events. DEXm is a promising agent for SVTs in the future, especially achieving the desired sedation without occurrence of significant respiratory depression. However, the current study is limited by the small number of patients. Our observation period was the time when patients was in the operating room, and we might have missed arrhythmias that occurred afterwards. Therefore, further clinical study is needed to clarify the mechanisms of effects, feasibility of wide clinical application, the dosage and rate of DEXm for the treatment of SVTs.

Abbreviations

SVTs: Supraventricular tachycardias; DEXm: Dexmedetomidine; AVNRT: Atrioventricular nodal reentrant tachycardia; AVRT: Atrioventricular reciprocating tachycardia; AT: atrial tachycardia; OAA/S: The Observer's Assessment of Alertness/Sedation; MAP: Marterial pressure; SpO_2: pulse oxygen saturation; HRV: Heart rate variability; LFnorm: Normalized low frequency power; HFnorm: normalized high frequency power; AV: atrioventricular; α2-RA: α-2 receptors agonist.

Declarations

Availability of data and materials

The datasets used and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

Our study was approved by the medical ethics committee of hospital (The Affiliated Lianyungang No. 2 People's Hospital of Bengbu Medical College, 41Hailian East Road, Lianyungang, China), and written informed consent was obtained from all subjects participating in the trial. The trial was registered prior to patient enrollment at clinicaltrials.gov (NCT04284150, Principal investigator: JunLong Zhang).

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Author's Contributions

XY, HCW, and ZJL designed the study, completed the project, analyzed the data, and drafted the manuscript. GX, HZH, and SH recruitment of patients, assisted with various experiments and helped with analyzing data. XY, and ZJL revised the manuscript. All authors read and approved the final manuscript.

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Author details

[1] The Affiliated Lianyungang No. 2 People's Hospital of Bengbu Medical College,

Lianyungang, China. [2] The 74th Group Army Hospital of P.L.A, Guangdong, China.

[3] Department of Anesthesiology, the Affiliated No. 4 Hospital of Jiangsu University,

Zhenjiang, China.

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Figures

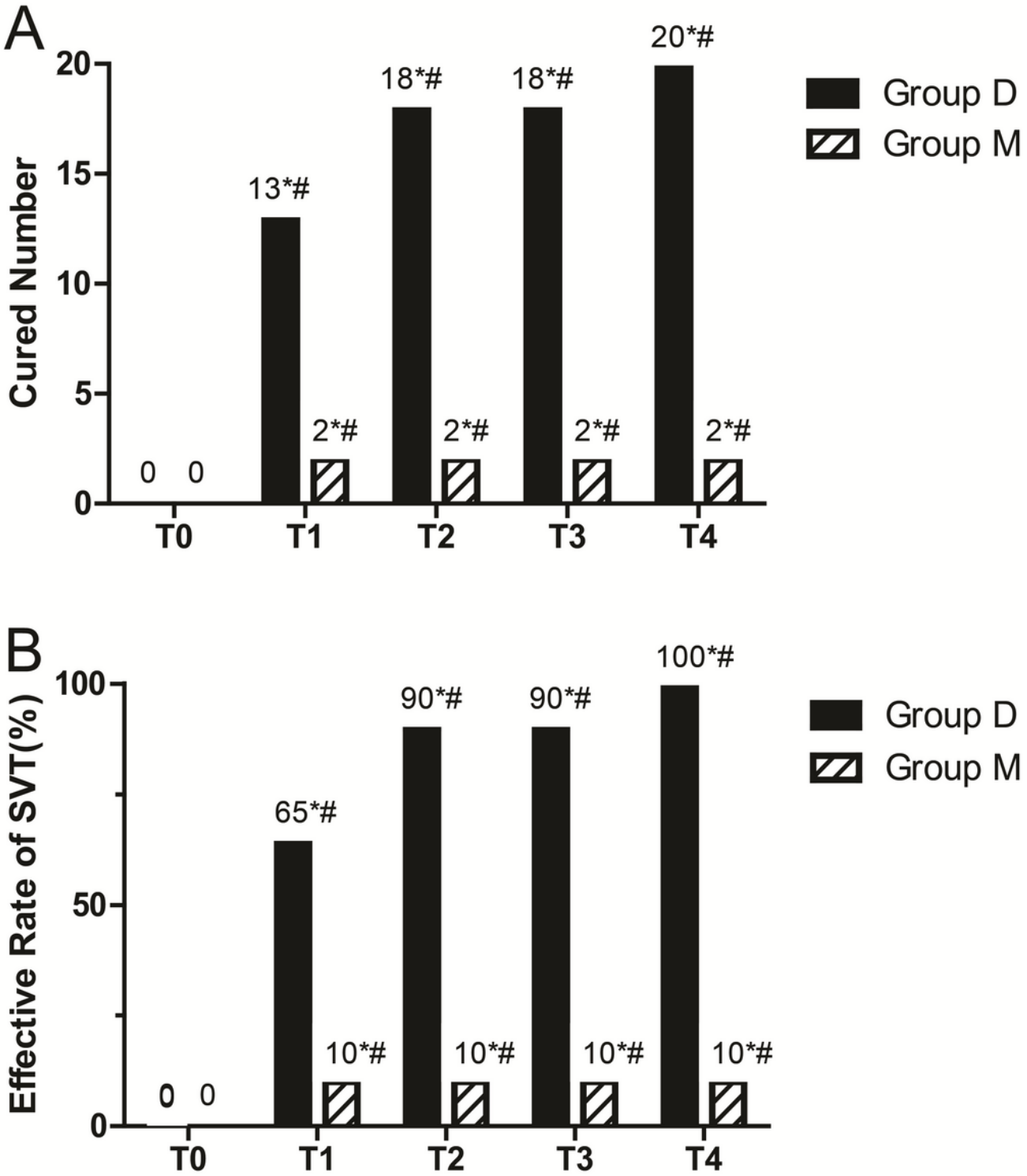


Figure 1

The number of patients with SVTs disappeared (A), and the effective rate (B) from T0 to T4 in both groups. *P<0.05 compared with T0, #P<0.05 compared with group M (n=20).

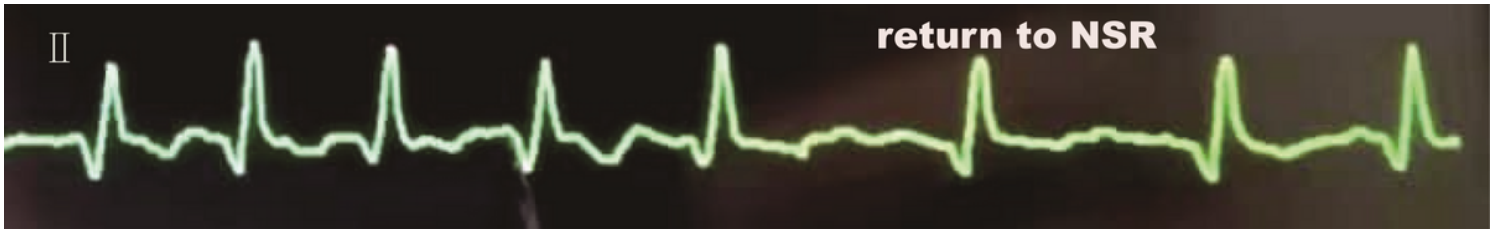
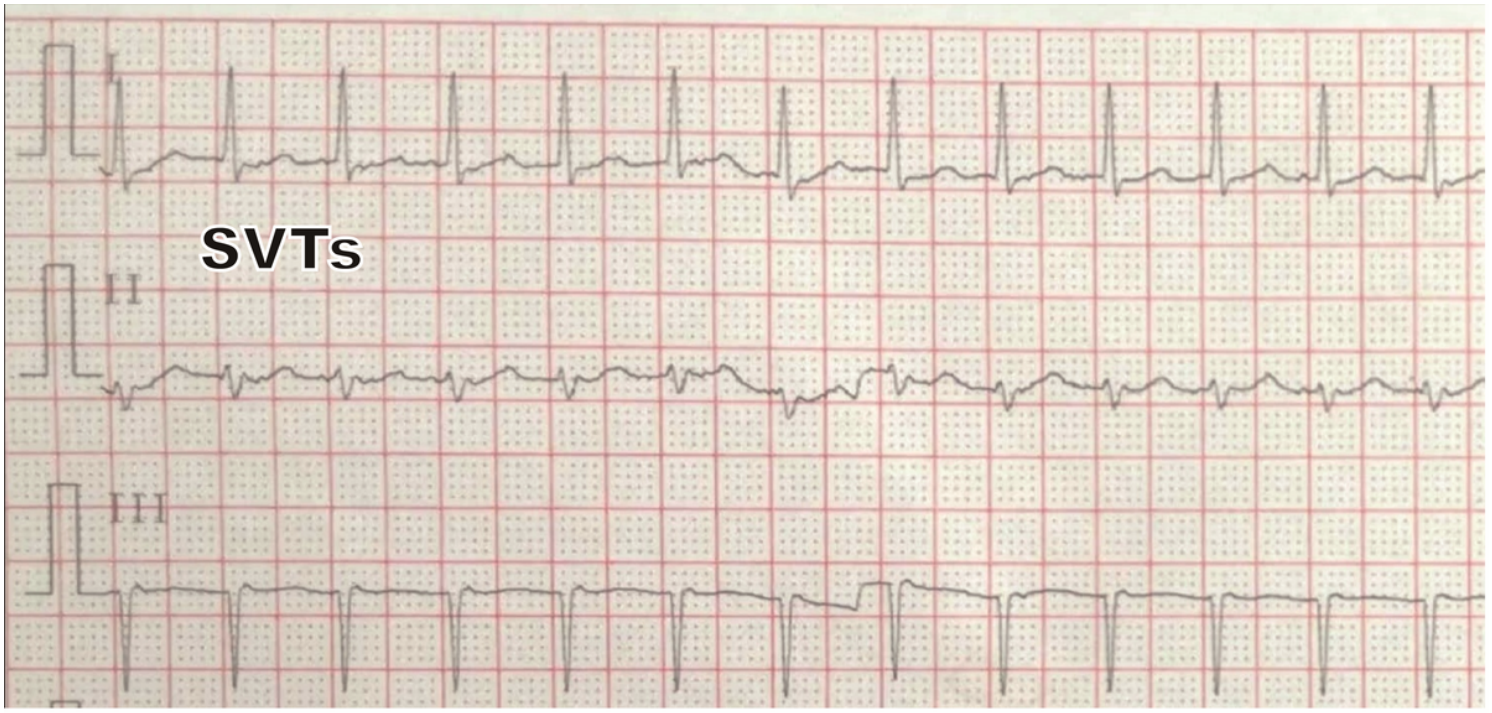


Figure 2

SVTs returned to NRS 4 minutes after infusion DEXm



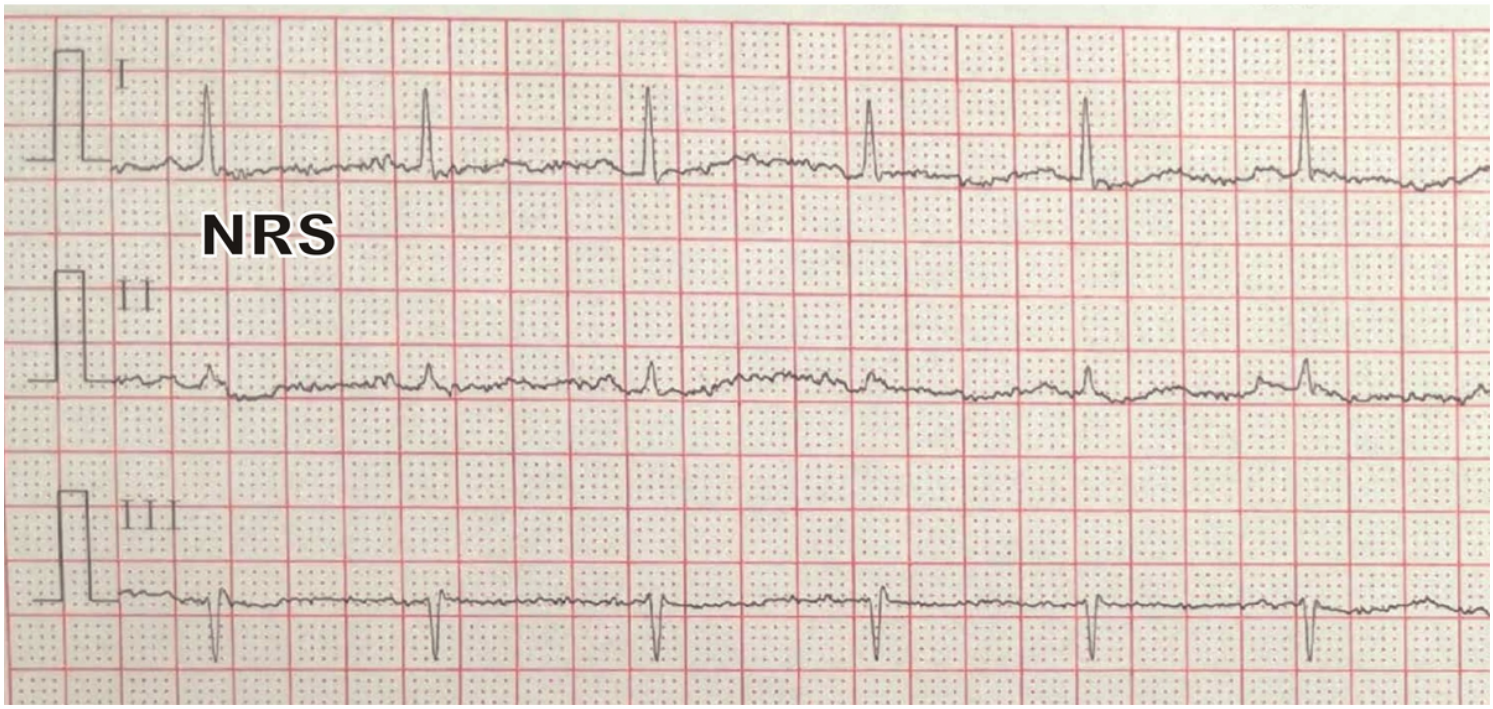


Figure 3

SVTs returned to NSR 9 minutes after infusion DEXm

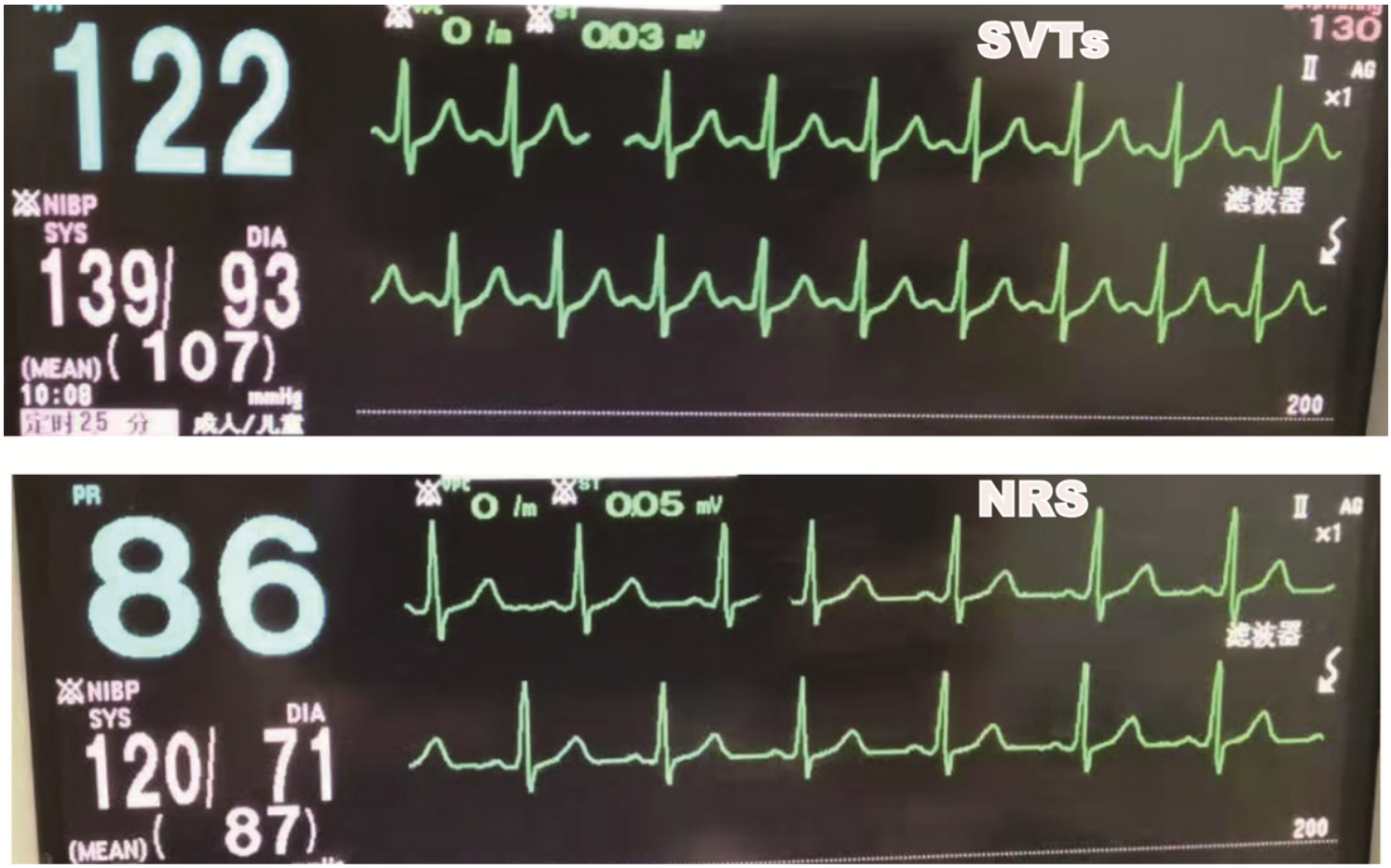


Figure 4

SVTs returned to NSR 13 minutes after infusion DEXm

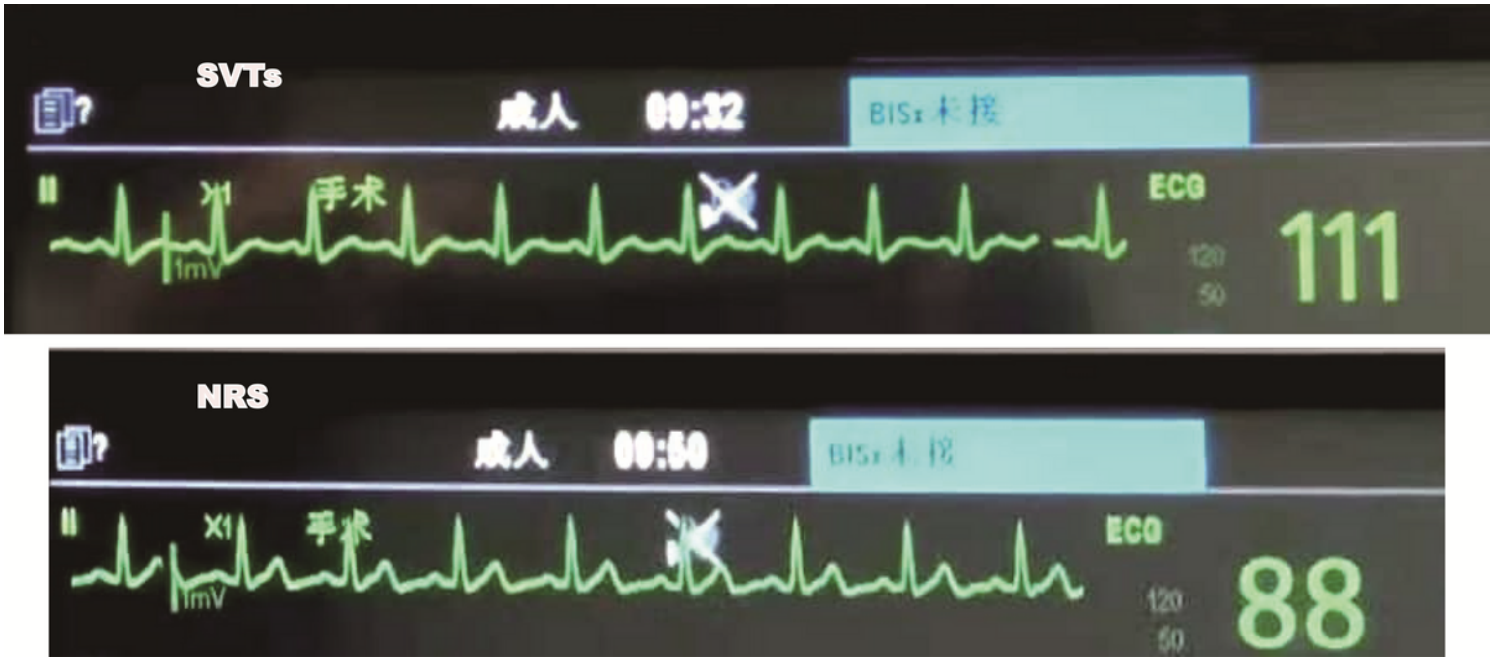


Figure 5

SVTs returned to NSR 18 minutes after infusion DEXm

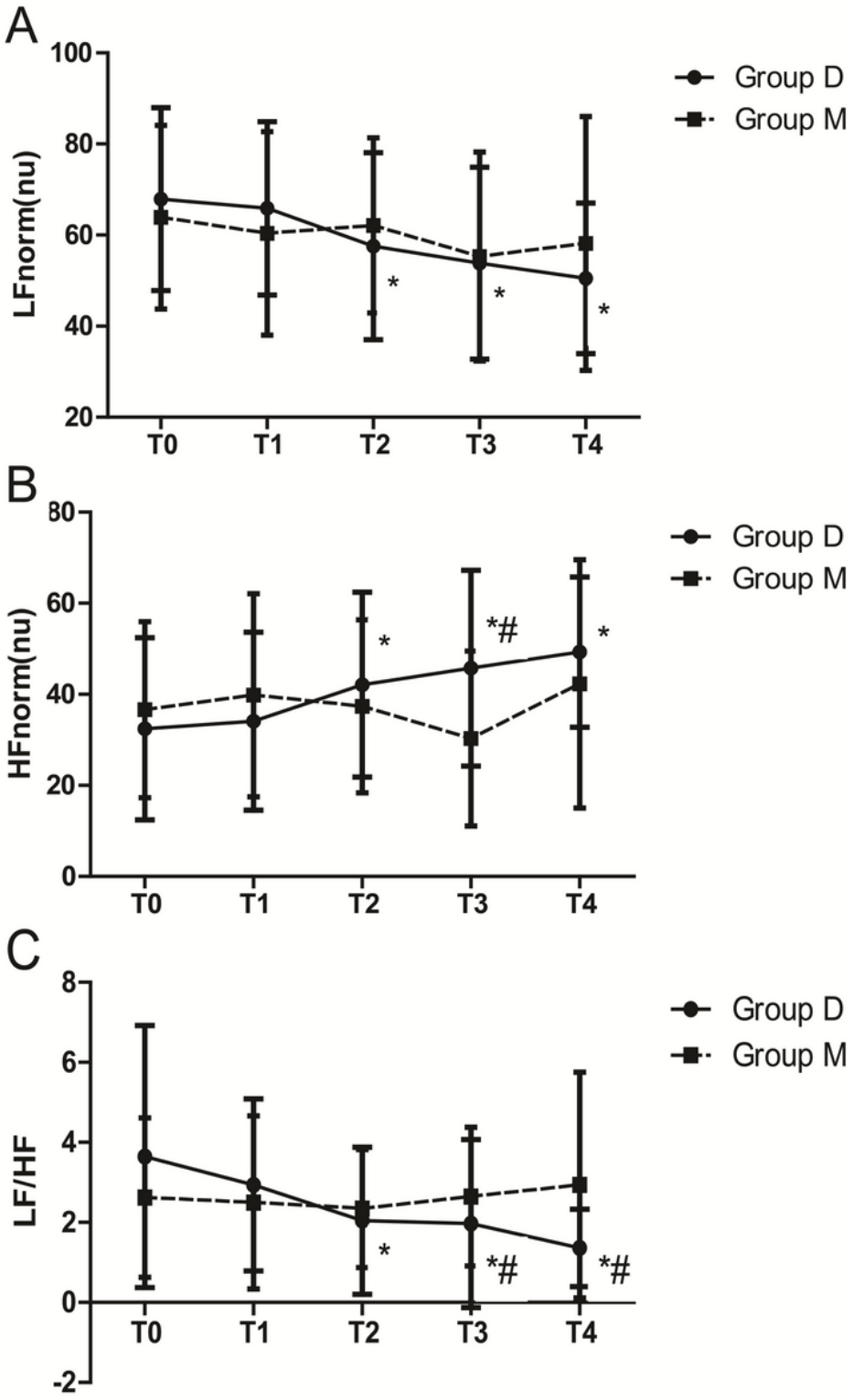


Figure 6

The changes in LFnorm, HFnorm, and LF/HF from T0 to T4 in both groups. *P<0.05 compared with T0, #P<0.05 compared with group M (n=20).