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Perinatal taurine exposure programs patterns of autonomic nerve activity responses to tooth pulp stimulation in adult male rats

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Abstract

Perinatal taurine excess or deficit influences adult health and disease, especially relative to the autonomic nervous system. This study tests the hypothesis that perinatal taurine exposure influences adult autonomic nervous system control of arterial pressure in response to acute electrical tooth pulp stimulation. Female Sprague-Dawley rats were fed normal rat chow with 3% β -alanine (taurine depletion, TD), 3% taurine (taurine supplementation, TS) or water alone (control, C) from conception to weaning. Their male offspring were fed normal rat chow and tap water throughout the experiment. At 8–10 weeks of age, blood chemistry, arterial pressure, heart rate and renal sympathetic nerve activity were measured in anesthetized rats. Age, body weight, mean arterial pressure, heart rate, plasma electrolytes, blood urea nitrogen, plasma creatinine and plasma cortisol were not significantly different among the three groups. Before tooth pulp stimulation, low (0.3–0.5 Hz) and high frequency (0.5–4.0 Hz) power spectral densities of arterial pressure were not significantly different among groups, while the power spectral densities of renal sympathetic nerve activity were significantly decreased in TD compared to control rats. Tooth pulp stimulation did not change arterial pressure, heart rate, renal sympathetic nerve and arterial pressure power spectral densities in the 0.3–4.0 Hz spectrum or renal sympathetic nerve firing rate in any group. In contrast, perinatal taurine imbalance disturbed very low frequency power spectral densities of both arterial pressure and renal sympathetic nerve activity (below 0.1 Hz), both before and after the tooth pulp stimulation. The power densities of TS were most sensitive to ganglionic blockade and central adrenergic inhibition, while those of TD were sensitive to both central and peripheral adrenergic inhibition. The present data indicate that perinatal taurine imbalance can lead to aberrant autonomic nervous system responses in adult male rats.

Keywords

arterial pressure; autonomic nervous system; dental pain; perinatal taurine; renal sympathetic nerve; tooth pulp stimulation; rat

1.1 INTRODUCTION

The perinatal environment can greatly influence adult health and disease. Perinatal programming can occur at any time from conception to weaning (i.e., from pregnancy through lactation), and perinatal life encompasses most of the critical periods during which such programming occurs, e.g., embryogenesis, placental development, organogenesis, prepartum maturation of the fetus and sucking period or lactation (Zhang et al. 2011). Since different organs develop at different rates, timing is important in specifying the organs affected by this programming. Nutritional environment *in utero* and early life has been widely studied. Maternal under-nutrition produces low birth weight and has long-term effects on the offspring (Godfrey and Barker 2001). Adult abnormalities from under-nutrition include physiological changes and other diseases, especially coronary heart disease, hypertension, diabetes, impaired glucose tolerance and renal dysfunction. Some of these changes can be permanent and can transfer to next generation (primarily via epigenetic mechanisms). In addition, maternal over-nutrition causes developmental adaptation and can have long-term effects in adult life (Hanson and Gluckman 2008).

Previous studies suggest that neonatal taurine may program the adult nervous system, particularly the central regulators of the autonomic nervous system. For example, neonatal administration of taurine modifies the hippocampal function and can affect learning and memory in later life (Franconi et al. 2004; Suge et al. 2007). Perinatal taurine depletion impairs autonomic nervous system regulation of arterial pressure in adult rat offspring (Roisommuti et al., 2009). In such animals, the sympathetic nervous system can be markedly overactive in response to a high sugar diet (Roisommuti et al. 2009a; Thaeomor et al. 2010). Baroreceptor reflex function also decreases in these animals, and this perinatal programming by taurine can lead to other cardiovascular dysfunction in the adult life, e.g. hypertension and diabetes mellitus (Aerts and Van Assche 2002).

Peripheral autonomic nerve activity is closely regulated by central autonomic nervous system and can be modulated by external and internal factors via sensory input pathways, e.g. baroreceptors, chemoreceptors, proprioceptors and nociceptors. The sympathetic nerves differentially supply various organs that have unique functions. In the kidney, peripheral sensory activation may differentially affect sympathetic nerve fibers that supply a specific part of the kidneys (DiBona 2005a). This can occur despite a similar response of whole sympathetic nerve stimulation of the organ. Analysis by nerve recording and power spectral density analysis of integrated sympathetic nerve activity can assist in dissecting the underlying mechanisms of these effects.

Dental pulp pain or toothache is known to induce cardiovascular reflex responses (Sousa and Lindsey 2009). Increases in arterial pressure and heart rate caused by dental pain possibly lead to clinical complications particularly in subjects prone to develop cardiovascular

diseases (Brotman et al. 2007; Montebugnoli et al. 2004). Electrical tooth pulp stimulation can stimulate either sympathetic or parasympathetic nerve activity. Like most pain conveyed by trigeminal nerves, dental pain primarily activates parasympathetic rather than sympathetic vasodilator nerve activity to orofacial vessels (Drummond 1995). Thus, orofacial vasodilation is commonly observed during facial or dental surgery. In addition, orofacial pain may induce vasoconstriction in the nose and finger skin, probably via sympathetic nerve activation (Kempainen et al. 2001). Taurine can reduce pain (Renno et al. 2008; Terada et al. 2011), but it is still unclear whether it has any physiological function in dental pain responses and what the underlying mechanisms are. This study tests the hypothesis that perinatal taurine exposure influences adult autonomic nervous system control in response to acute electrical tooth pulp stimulation.

1.2 METHODS

1.2.1 Animal preparation and treatment

Sprague-Dawley (SD) rats were obtained from The Animal Unit of Faculty of Medicine, Khon Kaen University and maintained at constant humidity ($60 \pm 5\%$), temperature ($24 \pm 1^\circ\text{C}$), and light cycle (06.00–18.00 h). Female SD rats were fed with normal rat chow and water *ad libitum* from conception to weaning. They were divided into 3 groups by being fed with tap water alone for control (C), 3% β -alanine in tap water for taurine depletion (TD) or 3% taurine in tap water for taurine supplementation (TS). After weaning, all male offspring were fed with normal rat chow and tap water alone throughout the experiment. All rats were studied at 8–10 weeks of age.

The experimental protocol was approved by the Animal Ethics Committee of Khon Kaen University (Khon Kaen, Thailand), based on the Ethic of Animal Experimentation of National Research Council of Thailand (Record No. AEKKU 21/2553). The study was conducted in accordance with The USA National Institutes of Health Guidelines.

1.2.2 Experimental protocol

At 8–10 weeks of age, rats were anesthetized by thiopental (50 mg/kg body weight, i.p.), were tracheostomized, and their femoral artery and vein were inserted with polyethylene tube (PE-50 fused to PE-10). The arterial catheter was connected to a pressure transducer for continuous recording of arterial pressure pulse (BioPac Systems, Goleta, California, USA) and the venous one was connected to an infusion pump for fluid and drug injection. After 20–30 minutes recovery, a blood sample (1.0 ml) was drawn from the arterial catheter and the blood loss was replaced with an equal volume of saline. After laparotomy and inserting a stainless steel electrode (12 M Ω , 0.01 Taper, A-M System, Sequim, Washington, USA), renal sympathetic nerve activity was continuously recorded by connecting the electrode to DAM-80 amplifier (World Precision Instruments, Sarasota, Florida, USA) and BioPac Systems, respectively. Multi unit recording of renal nerve activity was conducted only on nerve units that responded to changes in arterial pressure following sodium nitroprusside infusion.

Body temperature was servo-control at $37\pm0.5^{\circ}\text{C}$ by a rectal probe connected to a temperature regulator controlling an overhead heating lamp. After 20–30 minutes recovery, the responses of arterial pressure, heart rate and renal nerve activity to tooth pulp stimulation at different voltages and frequencies were performed before and after adrenergic blockades. The blockades were ordered from hexamethonium (a ganglionic blocker, 10 mg/kg, i.v.), clonidine (a central α_2 -adrenergic receptor agonist, 5 $\mu\text{g/kg}$, i.v.) and prazosin (a peripheral α_1 -adrenergic receptor antagonist, 5 $\mu\text{g/kg}$, i.v.), respectively. Full recovery periods were allowed between blockades. These doses of drugs were reported to completely block the sympathetic nerve activity in rats (Mozaffi *et al.*, 1996). At the end of experiment, all animals were terminated by a high dose of thiopental anesthesia.

1.2.3 Electrical tooth pulp stimulation

Left lower incisor of the rat was separated using a rubber dam sheet. Then a custom-made silver electrode filmed with electrode gel was placed on the left lower incisor (as a surface electrode) and a ground lead was attached to the left cheek. Before stimulation, resting period was provided to get a steady state of arterial pressure and renal sympathetic nerve activity. The electrical stimulation was applied to the tooth pulp via the surface electrode connected to a current isolator controlled by a Grass stimulator (Grass Technologies, West Warwick, Road Island, USA) at 2 ms pulse duration and 5 s delay. The current strength (0.1–1.5 mA) was adjusted by step increasing voltage of 10, 20 and 30, respectively. In each voltage, the stimulating frequencies were set to 1, 5, 10, 20 and 40 Hz, respectively. Each test was set to 1 minute electrical stimulation and 2 minutes resting.

1.2.4 Data analyses

All recorded data were analyzed by Acknowledge software version 3.9.1 (BioPac Systems, Goleta, California, USA). The percent power spectral densities of low frequency (0.3–0.5 Hz, LF) or high frequency (0.5–4.0 Hz, HF) to the total power of LF plus HF indicate sympathetic and parasympathetic nerve activity, respectively (Roysommuti S 2009; Thaeomor A 2010). The patterns of the power spectral densities of renal sympathetic nerve activity and arterial pressure were analyzed individually by using Fast Fourier Transformation analysis. For each study, the graph of power spectrum was averaged from 3–10 rats by using Acknowledge software.

Plasma electrolytes (sodium, potassium, chloride and bicarbonate), blood urea nitrogen, plasma creatinine and plasma cortisol levels were analyzed by the Srinagarind Hospital Laboratory Unit (Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand). Hematocrit was determined by a standard technique and blood sugar by standard glucostrips and a glucometer (Accu-Check Advantage II, Roche, Indianapolis, Indiana, USA).

1.2.5 Statistical analyses

All data are expressed as mean $\pm$ SEM. Statistical comparisons among groups were tested by using one-way ANOVA and a post hoc Duncan's Multi-Range test. Student's paired t-test was used to compare values within a group. Significant criteria were p-value of less than 0.05.

1.3 RESULTS

1.3.1 General baseline data

Compared to control treatment, perinatal taurine depletion or supplementation did not alter body weight, blood urea nitrogen, plasma creatinine, blood electrolytes, hematocrit, blood sugar, plasma cortisol, mean arterial pressure, heart rate, low and high frequency power spectral densities of arterial pressure and renal sympathetic nerve firing rate in adult animals (Table 1). Electrical tooth pulp stimulation at different voltages and frequencies had minimal effects on arterial pressure and heart rate in the present study (data not shown). These responses were well preserved in all groups after any adrenergic blockade. In all groups, mean arterial pressures significantly decreased nearly equally after adrenergic drug administration, and the values remained low during tooth pulp stimulation.

1.3.2 Power spectral density of arterial pressure

Perinatal taurine depletion or supplementation did not affect baseline sympathetic and parasympathetic control of arterial pressure, as indicated by the similar low and high frequency power spectral densities of arterial pressure in experimental compared to control rats (Table 2). Ganglionic blockade by hexamethonium, central α_2 -adrenergic stimulation by clonidine and peripheral α_1 -adrenergic blockade by prazosin each significantly decreased the sympathetic component of low frequency power densities to a similar extent, i.e., no significant differences between groups. Parasympathetic nerve activity is not significantly altered by these treatments, and the high frequency power densities remained close to baseline values.

Tooth pulp stimulation at different voltages and frequencies increased low frequency power spectral densities of arterial pressure similarly in all the three groups, and high frequency power spectral densities were minimally affected (Fig. 1). The optimal strength of repetitive stimulation was 20 volts at 20 Hz. Surprisingly, these responses were still observed after adrenergic blockade (Fig. 2); however, the adrenergic dependent patterns of very low frequency spectral densities (below 0.1 Hz) differ from those observed in baseline spectral densities. Although acute hexamethonium infusion decreased these densities in the three groups, the effect on densities in TS vs. TD and control were decreased. Clonidine infusion decreased the very low frequency power spectral densities during tooth pulp stimulation much more in control than in TS and TD groups. In addition, prazosin markedly decreased these very low frequency densities in TD during tooth pulp stimulation.

1.3.3 Power spectral density of renal sympathetic nerve activity

Despite similar renal sympathetic nerve activity (Table 1), perinatal taurine depletion (but not supplementation) significantly decreased low and increased high frequency power spectral densities of integrated renal nerve amplitudes when compared to control treatment (Table 3). Unlike arterial pressure power spectrum, renal nerve activity spectral densities at frequencies of 0.3–4.0 Hz were not altered by central and peripheral adrenergic blockade; however, perinatal taurine supplementation (TS group) markedly depressed very low frequency power densities of renal nerve activity (below 0.1 Hz) when compared to control and TD rats.

In the present study, tooth pulp stimulation at different voltages and frequencies had minimal effect on renal sympathetic nerve firing rates (increased by ~5% in all groups; data not shown). In addition, these responses were not modified by adrenergic blockade, but such stimulation altered the patterns of renal sympathetic nerve activity (Fig. 3). Perinatal taurine depletion or supplementation markedly increased power spectral densities at the frequency spectra below 0.2 Hz when compared to control taurine treatment. The highest increase was observed in TD rats, and the response distributed in nearly all frequencies within these spectra. Electrical tooth pulp stimulation increased the TS (vs. TD) power density of integrated renal nerve activity more at lower frequencies (below 0.1 Hz).

During tooth pulp stimulation, the very low frequency power spectral densities of control animals were heightened by adrenergic inhibition (Fig. 4). In contrast, the power density of TS during tooth pulp stimulation was flattened by any adrenergic blockade, while that of TD was decreased only by clonidine and prazosin treatment.

1.4 Discussion

Perinatal taurine depletion predisposes adult rats to respond to a high sugar diet with sympathetic nerve overactivity and baroreceptor reflex dysregulation (Roysommuti et al. 2009a; Thaeomor et al. 2010). In contrast, perinatal taurine supplementation leads to minimal effects in adults (Roysommuti et al. 2009b). The present data further indicate that perinatal taurine imbalance alters the pattern of autonomic nervous system function at very low frequency components of arterial pressure and renal sympathetic nerve activity. This phenomenon is modified by tooth pulp stimulation, which models pain-induced autonomic nerve responses. In addition, the present study suggests that both central and peripheral adrenergic mechanisms contribute to the effects of perinatal taurine exposure on the autonomic responses to tooth pulp stimulation in adults.

Peripheral autonomic nerve outflow is primarily regulated by the central nervous system, particularly hypothalamus and medulla, and many lines of evidence indicate that the sympathetic nerve response to stimuli is not uniform (Ninomiya et al. 1973; Ninomiya and Irisawa 1975). To test the overall sympathetic and parasympathetic nervous system control of arterial pressure, many indirect non-invasive techniques have been used, including power spectral analysis of arterial pressure pulse and heart rate (Malliani et al. 1994; Parati et al. 1995). In rats, the power spectral density of arterial pressure is closely correlated with the autonomic nerve activity, directly measured in many organs, and in humans, power spectrum of heart rate variability is closely correlated. The frequency spectra that coincide to the autonomic control of cardiovascular system are usually within 0.2–8.0 Hz in rats (Persson et al. 1992) and 0.05–0.15 or 0.3–4.0 Hz in humans and dogs (Furlan et al. 1993; Malliani et al. 1994; Pagani et al. 1984).

Previous experiments in our laboratory suggested that frequency of arterial pressure power spectral density between 0.3–4.0 Hz is indicative of autonomic nervous system control of arterial pressure in rats (Roysommuti et al. 2009a; Thaeomor et al. 2010). The present study indicates that the arterial pressure power density at frequencies below 0.2 Hz may better model the effect of perinatal taurine exposure on the sympathetic nerve control of arterial

pressure. This hypothesis is strengthened by the present finding that either ganglionic blockade by hexamethonium or central sympathetic inhibition by α_2 -adrenergic agonist clonidine depresses the power density at this frequency spectrum. This phenomenon was not observed in the frequency spectrum of 0.3–4.0 Hz.

Several lines of evidence indicate that peripheral nociceptive stimulation increases sympathetic activity to heart, blood vessel and kidney. However, the cardiovascular responses to pain are rather complex. Stimulation of orofacial pain fibers can increase both sympathetic and parasympathetic nerve activity (Andrew and Matthews 2002; Kemppainen et al. 2001). Tooth pulp stimulation predominately activates the parasympathetic vasodilator nerve to orofacial skin and mucosal blood vessels (Kemppainen et al. 2001; Satoh-Kuriwada et al. 2003). Sympathetic nerve activity to the heart is also activated in some cases (Allen and Pronych 1997; Schaller 2004; Sousa and Lindsey 2009). The present study is the first to indicate that electrical tooth pulp stimulation at the strengths and frequencies that have minimal effect on arterial pressure and heart rate does not alter renal sympathetic nerve firing rates but does affect its pattern of response. Our data also suggests that perinatal taurine imbalance increases the synchronization of sympathetic outflow to the kidneys in response to the tooth pulp stimulation. This effect was also reported in rats following heat stimulation of their tails (DiBona and Sawin 1999). The increased response of renal sympathetic nerve to tooth pulp stimulation may confirm previous experiments that indicated high sugar intake induces sympathetic overactivity in perinatal taurine depleted, adult rats (Roysommuti et al. 2009a; Thaeomor et al. 2010).

In TD compared to TS rats, the power spectral densities of renal sympathetic nerve activity after hexamethonium or clonidine either at baseline or during tooth pulp stimulation were different. TD was more sensitive to clonidine, while TS responded well to both hexamethonium and clonidine. Clonidine acts mainly at the rostral ventrolateral medulla (RVLM) to inhibit peripheral sympathetic outflow (Guyenet et al. 2001; Madden and Sved 2003), while hexamethonium blocks both parasympathetic and sympathetic outflows. These data suggest that perinatal taurine depletion has significant affects in the RVLM or related areas.

Several lines of evidence have confirmed that renal sympathetic nerve supplies three main renal structures including renin releasing juxtaglomerular cells, renal tubules and renal vessels. Peripheral sensory inputs (i.e., hypotension, pain or heat) may alter both firing rates and patterns of renal sympathetic nerve activity (DiBona and Sawin 1999). Similar total firing frequency of the renal nerve can bring about different patterns of renal responses, depending on the selective action on different fibers in the nerve. Renin secretion responds actively at very low frequency of renal nerve activity, while renal tubular sodium reabsorption and renal blood flow respond at higher frequency ranges (DiBona 2005b). The present study indicates that perinatal taurine exposure with or without tooth pulp stimulation primarily affects the very low frequency component of renal nerve activity spectrum, suggesting it programs mainly the sympathetic-mediated renal renin secretion. This notation is supported by the previous data from our laboratory indicating that perinatal taurine depletion affects the renin-angiotensin system (Thaeomor et al. 2010).

1.5 CONCLUSION

In summary, perinatal taurine excess or deficiency can alter autonomic nervous system and renal function in adult animals. In the present study, tooth pulp stimulation at strengths and frequencies that do not affect arterial pressure, heart rate, renal nerve firing rate or arterial pressure power spectral densities of 0.3–4.0 Hz (the standard measures of autonomic nerve control of arterial pressure) were shown to greatly alter the pattern of very low frequency components (i.e., below 0.2 Hz) of both renal nerve activity and arterial pressure. These effects are altered differently by adrenergic blockade. Thus, this data indicates that perinatal taurine depletion can significantly affect aspects of autonomic nervous system activity in adult animals and this could significantly alter adult physiological function.

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Abbreviations

AP	arterial pressure
C	control
ETPS	electrical tooth pulp stimulation
HR	heart rate
HF	high frequency
LF	low frequency
MAP	mean arterial pressure
RA	renal sympathetic nerve activity
SD	Sprague-Dawley
TD	taurine depletion
TS	taurine supplementation

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Fig. 1. Patterns of arterial pressure variability before (left) and after (right) tooth pulp stimulation in control (C), taurine depleted (TD) and taurine supplemented (TS) groups. Each graph is averaged from 9–10 rats (AP, arterial pressure; ETPS, electrical tooth pulp stimulation).

Fig. 2. Patterns of arterial pressure variability during tooth pulp stimulation before adrenergic blockades (top-left), with hexamethonium (top-right), with clonidine (bottom-left) or with prazosin (bottom-right) in control (C), taurine depleted (TD) and taurine supplemented (TS) groups. Each graph is averaged from 3–10 rats (AP, arterial pressure; ETPS, electrical tooth pulp stimulation).

Fig. 3. Patterns of renal nerve activity variability before (left) and after (right) tooth pulp stimulation in control (C), taurine depleted (TD) and taurine supplemented (TS) groups. Each graph is averaged from 9–10 rats (RA, renal sympathetic nerve activity; ETPS, electrical tooth pulp stimulation).

Fig. 4.

Patterns of renal nerve activity variability during tooth pulp stimulation before adrenergic blockades (top-left), with hexamethonium (top-right), with clonidine (bottom-left) or with prazosin (bottom-right) in control (C), taurine depleted (TD) and taurine supplemented (TS) groups. Each graph is averaged from 3–10 rats (RA, renal sympathetic nerve activity; ETPS, electrical tooth pulp stimulation).

Table 1

Basic parameters of the rats in all experimental groups

Parameters	C	TD	TS
<i>n</i>	10	10	9
Age (weeks)	9.2 ± 0.3	9.2 ± 0.2	9.1 ± 0.2
Body weight (g)	284 ± 4	284.1 ± 11	277 ± 8
Blood urea nitrogen (mg/dl)	26.31 ± 0.94	22.99 ± 0.80	26.65 ± 2.49
Creatinine (mg/dl)	0.22 ± 0.01	0.20 ± 0.02	0.23 ± 0.02
Sodium (mEq/l)	142.80 ± 1.43	141.00 ± 0.50	142.50 ± 1.40
Potassium (mEq/l)	4.22 ± 0.23	3.87 ± 0.11	4.15 ± 0.23
Bicarbonate (mEq/l)	23.56 ± 0.38	22.84 ± 0.89	21.56 ± 0.37
Chloride (mEq/l)	103.90 ± 1.21	105.11 ± 0.77	105.88 ± 1.63
Hematocrit (%)	47.10 ± 0.46	46.90 ± 0.60	46.44 ± 0.93
Blood sugar (mg/dl)	107.80 ± 3.43	106.11 ± 3.37	95.22 ± 6.18
Plasma cortisol (µg/dl)	1.71 ± 0.19	2.04 ± 0.22	1.94 ± 0.18
MAP (mm Hg)	104 ± 8	103 ± 6	108 ± 6
HR (bpm)	369 ± 14	377 ± 9	377 ± 19
RA (spikes/second)	52.73 ± 8.11	55.87 ± 6.06	58.48 ± 10.38

Values are mean ± SEM. All parameters were not significantly different among groups. C, control group; TD, taurine depleted group; TS, taurine supplemented group; MAP, mean arterial pressure; HR, heart rate; RA, renal sympathetic nerve activity. There were no significant differences among groups.

Table 2

Power spectral density of arterial pressure before (baseline) and after drug administration

Parameters	C	TD	TS
Baseline			
LF/(LF + HF) (%)	2.6 ± 0.6	4.2 ± 1.3	2.7 ± 0.7
HF/(LF + HF) (%)	97.4 ± 0.6	95.8 ± 1.3	97.3 ± 0.7
Hexamethonium			
LF/(LF + HF) (%)	0.4 ± 0.1 [£]	0.2 ± 0.1 [£]	0.1 ± 0.0 [£]
HF/(LF + HF) (%)	99.6 ± 0.1 [¥]	99.8 ± 0.1 [¥]	99.9 ± 0.0 [¥]
Clonidine			
LF/(LF + HF) (%)	0.2 ± 0.0 [£]	0.4 ± 0.2 [£]	0.2 ± 0.0 [£]
HF/(LF + HF) (%)	99.8 ± 0.0 [¥]	99.6 ± 0.2 [¥]	99.8 ± 0.0 [¥]
Prazosin			
LF/(LF + HF) (%)	0.3 ± 0.1 [£]	0.5 ± 0.2 [£]	0.3 ± 0.1 [£]
HF/(LF + HF) (%)	99.7 ± 0.1 [¥]	99.5 ± 0.2 [¥]	99.7 ± 0.1 [¥]

Values are mean ± SEM. C, control group; TD, taurine depleted group; TS, taurine supplemented group; LF, low frequency (0.3–0.5 Hz); HF, high frequency (0.5–4.0 Hz);

[£], [¥] $P < 0.05$ compared to LF and HF baselines of same groups, respectively.

Table 3

Power spectral densities of renal sympathetic nerve activity before (baseline) and after drug administration

Parameters	C	TD	TS
Baseline			
LF/(LF + HF) (%)	6.6 ± 0.2	0.1 ± 0.2 *	3.4 ± 1.0
HF/(LF + HF) (%)	93.4 ± 0.2	99.0 ± 0.2 *	96.6 ± 1.0
Hexamethonium			
LF/(LF + HF) (%)	4.8 ± 1.7	1.5 ± 0.3	5.8 ± 2.1
HF/(LF + HF) (%)	95.2 ± 1.7	98.5 ± 0.3	94.2 ± 2.1
Clonidine			
LF/(LF + HF) (%)	4.6 ± 1.5	5.6 ± 4.1	2.9 ± 1.1
HF/(LF + HF) (%)	95.4 ± 1.5	94.4 ± 4.1	97.1 ± 1.1
Prazosin			
LF/(LF + HF) (%)	8.1 ± 2.1	4.7 ± 1.0	1.9 ± 1.2
HF/(LF + HF) (%)	91.9 ± 2.1	95.3 ± 1.0	98.1 ± 1.2

Values are mean ± SEM. C, control group; TD, taurine depleted group; TS, taurine supplemented group; LF, low frequency (0.3–0.5 Hz); HF, high-frequency (0.5–4.0 Hz);

* $P < 0.05$ compared to C.