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Clinical Study on Evaluation of Autonomic Nervous Dysfunction Based on Imaging Urodynamic Examination with Slow Filling and Synchronous Blood Pressure Monitoring in the Patients with Cervicothoracic Spinal Cord Injury

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Abstract

Objective: Explore the rule of autonomic nervous dysfunction in the patients with urination disorder after high level spinal cord injury, and seek a safe, objective and accurate method to evaluate autonomic nervous function. **Patients and Method:** 48 patients with dysuria after cervicothoracic SCI were selected. Before, during and after imaging urodynamic examination with slow filling in supine position, blood pressure and ECG were monitored simultaneously. The symptoms of sweating, shivering, headache, flushing and chills were observed and recorded. The study of the relationship among the changes of blood pressure, heart rate and urodynamic indexes and the above symptoms was analyzed. **Results:** They were divided into three groups: group A (no obvious abnormality), group B (hyperactivity) and group C (hypoactivity) according to their BP, HR and existing the symptoms or not. **Conclusion:** The incidence of autonomic dysfunction in the high level SCI patients with dysuria was very high (79.17%), most of them were hyperactivity, and a few were low function. The changes of SBP and DBP in the hypoactivity group all appeared an increasing and then declining trend, while the change of HR in the low function one was lower than normal and decreased continuously. The main inducements of AD are neurogenic detrusor overactivity, detrusor sphincter dyssynergia, elevated abdominal pressure and abnormal bladder sensitivity. The asymptomatic patients had a higher occurrence rate (43.75%). Only by imaging urodynamic examination with slow filling and synchronous blood pressure monitoring, can autonomic nervous function of

the patients be evaluated safely, objectively, early and accurately.

Keywords

High Level Spinal Cord Injury, Autonomic Nervous Function, Imaging Urodynamic Examination, Slow Filling, Synchronous Blood Pressure Monitoring

1. Introduction

The Patients with dysuria of high level spinal cord injury (SCI) above T6 often have autonomic nervous dysfunction, such as sudden significant increasement of blood pressure, tachycardia, or transient hypotension, bradycardia, and or accompanied by sweating, shivering, headache, blushing, chills, hyperspasmia of muscles below the injury level and other symptoms [1] [2]. Severe cases have the risk of cerebral hemorrhage [3], retinal hemorrhage [4], or seizures, and even cardiac arrest [5], which is one of the most serious complications after SCI. It may even aggravate the neurological symptoms of the patients [6] and affect the rehabilitation process. How can the blood pressure and heart rate change dynamically when autonomic nerve dysfunction occurs in such patients? What is the relationship between it and bladder filling (intestinal irritation)? What are the corresponding mechanisms and main incentives? How to evaluate autonomic nervous function safely and scientifically? The above issues urgently need to be studied and discussed. Therefore, it is very important to clarify the change rule of autonomic nerve dysfunction in the patients with dysuria after high level SCI, to explore its causes, to quantify autonomic nerve function of the patients scientifically, to find a safe, objective, early and accurate method to evaluate autonomic nerve function of them in clinic.

2. Patients and Method

From July 2017 to July 2020, 48 in patients with dysuria after cervicothoracic SCI in our hospital were selected.

The inclusion criteria: 1) 18 ≤ age ≤ 60 years old; 2) Dysuria caused by ASIA classification (A-D grade) of cervical and thoracic spinal cord injury; 3) Course of disease ≥ 1 month; 4) No urinary calculi; 5) No urinary tract tumor; 6) No obvious stress urinary incontinence; 7) No history of urinary tract, bladder and prostate surgery; 8) No serious urinary tract infection and there is a stable condition within a week; 9) Signed the informed consent.

The exclusion criteria: 1) Age < 18 or age > 60 years old; 2) Dysuria caused by injury of lumbar spinal cord and below level; 3) Urination disorder caused by cuaniocerebral diseases; 4) Course of disease < 1 month; 5) Have urinary calculi; 6) Have urinary tract tumor; 7) Have obvious stress urinary incontinence; 8) Have history of urinary tract, bladder and prostate surgery; 9) The symptomatic urinary tract infection occurred within the past week; 10) Those who have been

unstable condition within a week; 11) People who are allergic to iohexol; 12) People with allergies; 13) People who refuse to participate in the study.

The research method: Firstly, the patients were given adequate bowel preparation: They were given cleaning enema once at the night before the examination and once at four hours before the examination on the same day. The enema solution was a mixture of 100 ml normal saline and 40 ml glycerin. Then after their skin preparation in the perineum area, all patients underwent imaging urodynamic examination with slow filling in zero degree supine position. 10% iohexol sodium chloride solution was continuously pumped into the bladder manometer tube. The bladder filling rate was 8 - 10 ml/min. Before, during and after examination, blood pressure and ECG were monitored. Among them, the blood pressure and heart rate during examination are the corresponding values of the leakage point of the patients, or the immediate values when the patients had related discomfort symptoms, or the values at the time of stopping bladder filling. Observed and recorded whether the patients had sweating, shivering, headache, blushing, chills and other symptoms.

Inspection instrument: a set of German Siemens imaging urodynamic examination equipment, a wireless Bluetooth urodynamic instrument from Canada Labry, an ECG monitor of China Shenzhen Jinlu UT4000B.

Statistical methods: SPSS 21.0 software was used for data analysis. The measurement data were represented by mean ± SD, and the count data were indicated by two or three categories. T-test was used for the measurement data, $P < 0.05$ was statistically significant.

3. Results

The patients were divided into three groups according to their BP, HR and existing the symptoms of autonomic nervous dysfunction or not: group A (no obvious abnormality of autonomic nervous function): before, during and after examination, their BP and HR were normal, without accompanying symptoms; group B (hyperactivity of it): both their SBP and DBP rised more than 20% during examination than before, and at least they were accompanied with one of the five symptoms; and group C (hypoactivity of it): they had lower HR (BP) than normal, and or they were accompanied with one of the five symptoms. The values of BP and HR of the patients were shown in **Table 1**. For the sociodemographic and clinical characteristics, see **Table 2**. The data of age, disease course, bladder safety capacity and bladder compliance was listed in **Table 3**. T-test of age, disease course, bladder safe volume and compliance was used to compare between three groups in **Table 4**. T-test of BP, HR was used to contrast between three groups in **Table 5**. The incidence of three groups' patients was evinced in **Table 6**. The results of urodynamic examination were manifested in **Table 7**. The results of a typical case' blood pressure synchronous monitoring were demonstrated in **Table 8**. Group B' SBP trend of change was appeared in **Figure 1**; Group B' DBP trend of change was indicated in **Figure 2**. Group C' HR trend

Table 1. Values of BP and HR of the patients (mean ± SD).

	SBP (mmHg)			DBP (mmHg)			HR (Times/min)		
	SBP1	SBP2	SBP3	DBP1	DBP2	DBP3	HR1	HR2	HR3
A	112.25 ± 4.86	113.00 ± 11.81	116.38 ± 8.99	69.63 ± 5.34	71.25 ± 10.55	74.75 ± 6.67	66.00 ± 3.82	64.75 ± 4.37	66.38 ± 4.69
B	117.13 ± 16.78	144.57 ± 29.67	134.10 ± 18.68	76.40 ± 14.85	92.80 ± 22.50	86.27 ± 14.36	66.03 ± 14.90	66.30 ± 18.70	66.27 ± 12.51
C	103.38 ± 9.59	120.63 ± 5.66	110.13 ± 11.91	60.50 ± 7.87	74.63 ± 6.89	70.75 ± 10.86	59.63 ± 8.85	58.00 ± 8.78	56.50 ± 7.23

Table 2. Sociodemographic and clinical characteristics of the patients.

Group		A	B	C
Gender	M	5	23	7
	F	3	7	1
Hypertension history	No	8	26	8
	Yes	0	3	0
Hypotension history intermittently	No	8	29	7
	Yes	0	1	1
Plane of SCI (%)	Cervical segment	5 (62.50%)	28 (93.33%)	7 (87.50%)
	Upper thoracic segment (≤T6)	3 (37.50%)	1 (3.33%)	0 (0.00%)
	Lower thoracic segment (>T6)	0 (0.00%)	1 (3.33%)	1 (12.50%)
ASIA grades of SCI (%)	Complete	3 (37.50%)	13 (43.33%)	1 (12.50%)
	Incomplete	5 (62.50%)	17 (56.67%)	7 (87.50%)
Concomitant symptoms (%)	No	8 (100%)	15 (50.00%)	3 (37.50%)
	Yes	0 (0.00%)	15 (50.00%)	5 (62.50%)
Hydronephrosis (%)	No	7 (87.50%)	28 (93.33%)	7 (87.50%)
	Yes	1 (12.50%)	2 (6.67%)	1 (12.50%)

Table 3. Patients' age, disease course, bladder safety capacity and bladder compliance (mean ± SD).

Group	A	B	C
Age (years)	42.25 ± 7.92	37.07 ± 10.73	40.13 ± 14.84
Disease course (months)	12.50 ± 13.30	26.03 ± 13.91	31.75 ± 36.10
Bladder safety capacity (ml)	134.13 ± 178.90	211.17 ± 137.83	200.63 ± 116.89
Bladder compliance (ml/cmH_2O)	19.66 ± 48.65	18.52 ± 26.57	27.51 ± 45.22

Table 4. Inter-group comparison of the patients' age, disease course, bladder safe volume and bladder compliance (t-test).

p	Age	Disease course	Bladder safety capacity	Bladder compliance
A-B	0.212*	0.355*	0.195*	0.929*
A-C	0.726*	0.179*	0.394*	0.743*
B-C	0.513*	0.716*	0.844*	0.472*

*: $p > 0.05$.

Table 5. Inter-group comparison of the patients' BP and HR (t-test).

p	SBP (mmHg)		DBP (mmHg)		HR (Times/min)	
A-B	SBP1	0.425*	DBP1	0.217*	HR1	0.995*
	SBP2	0.006***	DBP2	0.013**	HR2	0.819*
	SBP3	0.014**	DBP3	0.035**	HR3	0.981*
A-C	SBP1	0.035**	DBP1	0.017**	HR1	0.082*
	SBP2	0.122*	DBP2	0.461*	HR2	0.072*
	SBP3	0.256*	DBP3	0.390*	HR3	0.006***
B-C	SBP1	0.034**	DBP1	0.006***	HR1	0.226*
	SBP2	0.031**	DBP2	0.032**	HR2	0.234*
	SBP3	0.002***	DBP3	0.007***	HR3	0.043**

*: $p > 0.05$; **: $p < 0.05$; ***: $p < 0.01$.

Table 6. Incidence of three groups' patients.

Group	A	B	C
Number	8	30	8
Percentage (%)	16.67	62.50	16.67
Total (B + C)		79.17	

Table 7. Results of the patients' urodynamic examination.

Group		A	B	C
Detrusor type (%)	Overactivity	5 (62.50%)	23 (76.67%)	7 (87.50%)
	Bladder atony	3 (37.50%)	7 (23.33%)	1 (12.50%)
Bladder compliance (%)	Decreasement	7 (87.50%)	23 (76.67%)	5 (62.50%)
	Increasement	1 (12.50%)	5 (16.67%)	1 (12.50%)
External urethral sphincter-detrusor (%)	Normal	0 (0.00%)	2 (6.67%)	2 (25.00%)
	Incoordination	6 (75.00%)	22 (73.33%)	5 (62.50%)
	Achalasia	2 (25.00%)	8 (26.67%)	3 (37.50%)
Bladder sensation (%)	Sensitive	4 (50.00%)	17 (56.67%)	5 (62.50%)
	lost	4 (50.00%)	9 (30.00%)	2 (25.00%)
	Declining	0 (0.00%)	4 (13.33%)	1 (12.50%)
Higher abdominal pressure (%)	No	6 (75.0%)	14 (46.67%)	1 (12.50%)
	Yes	2 (25.00%)	16 (53.33%)	7 (87.50%)

Table 8. Results of a typical case' blood pressure synchronous monitoring.

	SBP (mmHg)	DBP (mmHg)	HR (Times/min)
Start filling	113	70	77
Stop filling	137	87	65
End examination	130	82	66

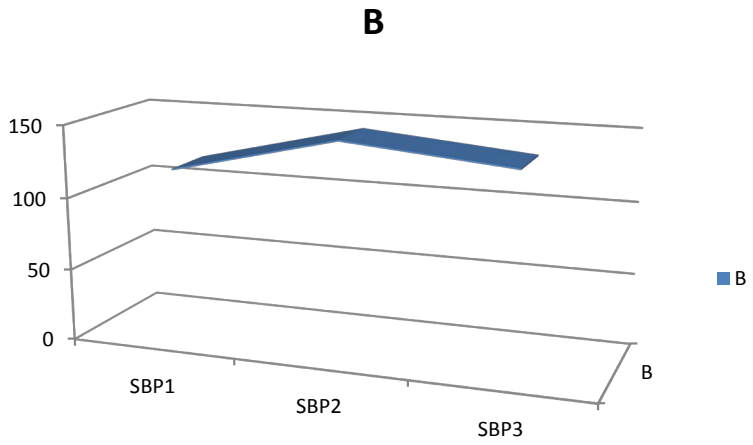

Figure 1. Trend chart of SBP in group B.

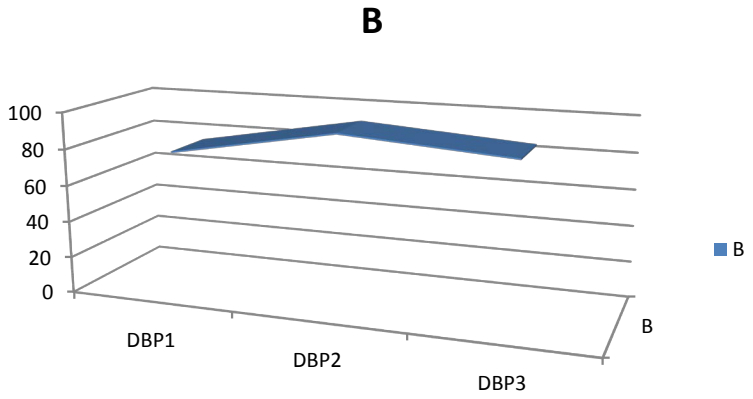

Figure 2. Trend chart of DBP in group B.

of change was revealed in **Figure 3**. The urodynamic graph of a typical case was disclosed in **Figure 4**.

Table 4 exposed that there was no significant difference in age, disease course, bladder safety capacity and compliance between three groups (T-test) ($P > 0.05$). However, from **Table 5**, compared of BP and HR between three groups (T-test), it can be seen that SBP2 of group A-B had extremely prominent discrepancy ($P < 0.01$); SBP3, DBP2 and DBP3 of it had marked distinction ($P < 0.05$). HR3 of group A-C was greatly remarkable different ($P < 0.01$), SBP1 and DBP1 of it had obvious dissimilarity ($P < 0.05$). The diversity between group B-C was more evident. **Figure 1**, **Figure 2** and **Table 1** revealed that the changes of SBP and DBP of group B were increased earlier and then decreased. **Figure 3** and **Table 1** exhibited that the changes of HR of group C was lower than normal and declined continuously. **Figure 4** suggested that the abdominal pressure was significantly higher than the bladder pressure, from the whole course of inspection. **Table 6** displayed the incidence of autonomic dysfunction of the patients with dysuria after high level SCI was very high (about 79.17%, adding B and C), most of them were hyperactivity (62.50%), and a few were hypoactivity (16.67%). **Table 7** manifested: The incidence of NDO [7], low compliant bladder (62.50% - 87.50%),

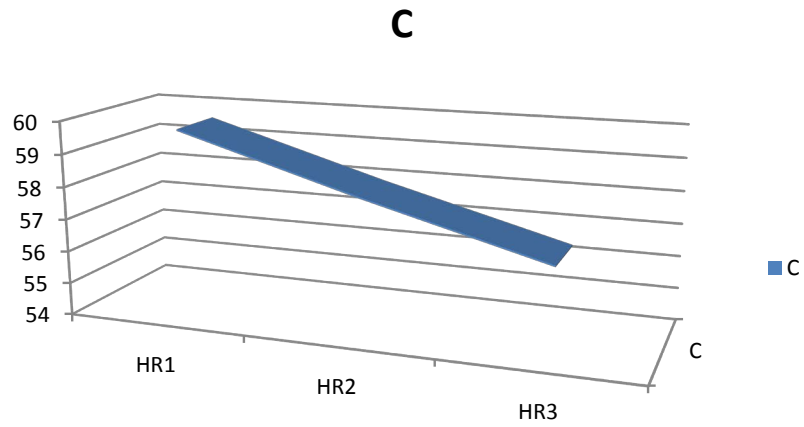

Figure 3. Trend chart of HR in group C.

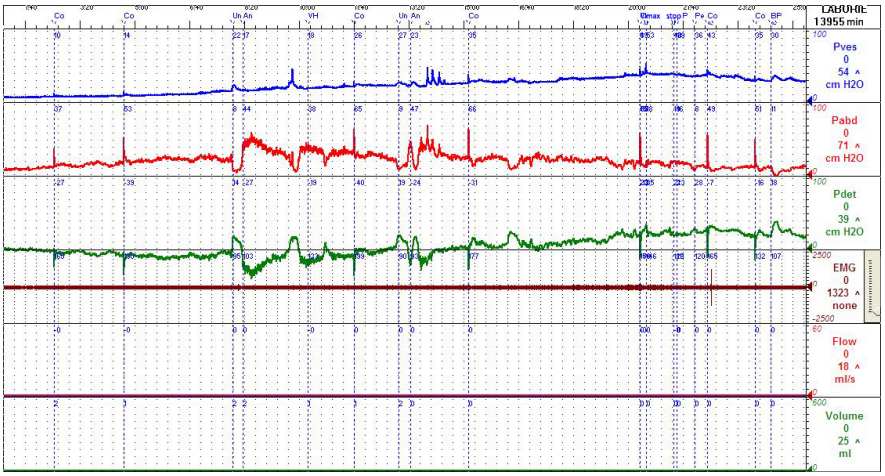

Figure 4. Urodynamic graph of a typical case.

NDSD [8] of group B and C was very high, that of high abdominal pressure of group B, C (53.33%, 87.50%) was evidently more quantity than that of group A (25.00%), the ratio of bladder disappearance of group B, C (30.00%, 25.00%) was fewer than that of group A (50.00%). From **Table 8**, compared with the baseline, it can be demonstrated that the values of SBP and DBP of a typical case at he time of stopping filling increased more than 20 mmHg. From **Table 2**, we found out that the incidence of symptoms without autonomic nervous dysfunction of group B and C was not low (50.00%, 37.50%), and the average incidence was 43.75%.

4. Discussion

Paraplegic patients with high level SCI may have abnormal autonomic reflex or autonomic dysreflexia (AD), especially in the patients with T6 or higher plane, the incidence is nearly 1/2 - 3/4, the latest research shows that: as high as 93% [9], the patients with cervical SCI are more common. Its severity is mainly related to the sudden and distinct increase of BP: such as conscious change, visual impairment, seizures, intracranial hemorrhage, and even death. Headache, facial

flushing, chest tightness, palpitation, sweating and other symptoms are often used as a warning sign of elevated BP. Nevertheless, sometimes patients have elevated BP without any symptoms, which is called "asymptomatic AD" [10]. In this case, patients and doctors may have missed the best time to manage the potential powerful afferent stimuli, which may lead to severe sequelae. In fact, this "silent killer" is more harmful to patients. The Patients with high level SCI can also present hypotension, orthostatic hypotension (OH) [11] [12], reflex bradycardia and cardiac arrest. Available data indicate that the incidence of bradycardia and cardiac arrest of SCI patients can be as high as 30%, and the average HR is 54 times/min, including some patients with severe bradycardia less than 50 times/min [13]. Therefore, we should pay enough attention to the occurrence of serious cardiovascular complications, such as bradycardia and cardiac arrest, especially in the patients with high level paraplegia. At present, circulatory disturbance has become the second leading cause of death in the patients with chronic SCI. Therefore, it is necessary and urgent to study the autonomic dysfunction in the patients with SCI.

The study demonstrated that the incidence of autonomic dysfunction in the high level SCI patients with dysuria was very high (almost 80.00%), most of them were hyperactivity (62.50%), and a few were low function (16.67%). Among them, the increasement of SBP and DBP in the hyperfunction group was greater than 20% of the baseline value when there was no attack, and they all showed a rise firstly and then a fall; the decline of HR (BP) in the hypoactivity one was lower than normal and decreased continuously, before, during and after examination, and or two groups of patients were accompanied with one of the five symptoms: sweating, shivering, headache, facial congestion, chills [14].

Indeed, In addition to bladder overinflation caused by NDO and NDSD is the main causes of AD, the results in **Table 7** suggest that the increase of abdominal pressure is also the main cause of AD. Previous studies have shown that the most common cause of AD is bladder dilation, accounting for 75% - 85%; the second common cause is intestinal irritation, such as fecal impaction, accounting for 20% [15] [16]. Most patients with dysuria of SCI are accompanied by neurogenic intestinal dysfunction. Therefore, with the increament of abdominal pressure caused by bladder filling and intestinal irritation, the conduction through visceral sensory nerve: pelvic nerve and pudendal nerve to the sacral spinal cord can also cause the above AD performance [17]. In this study, the patients of group B and C had more transient paroxysmal increasement of abdominal pressure than ones of group A, and or they were accompanied by transient enhancement of the myoelectric amplitude of the external urethral sphincter. Most of patients' abdominal pressure fluctuated within 10 - 30 cm H_2O for a short period of time, even as high as about 70 cm H_2O, as shown in the urodynamic map of a typical case of SCI (T3 AIS grade B). Although good bowel preparations (cleaning enema twice) had been done before the examination, the patient' bladder was filled slowly in the supine position, and detrusor was recorded uninhibitory contraction during the urine storage period, the maximum detrusor pressure is 35 cm

H_2O, without significant increase, the peak value of abdominal pressure was 71 cm H_2O. Simultaneous cystography showed that when the bladder was filled to 100 ml, it presented the X-ray appearance of neurogenic bladder protruded to the right. The results of synchronous blood pressure monitoring in **Table 8** indicated that the values of SBP and DBP at the time of stopping filling raised by more than 20 mmHg compared to the values at the beginning of filling, even though the patient had no symptoms of AD. Imaging urodynamic diagnosis: bladder sensation disappeared; detrusor was overactivity in the storage period, detrusor contraction was weakness in the voiding period; bladder compliance decreased; bladder safety capacity, urinary bladder capacity was about 356 ml; partial incordination was existed between external urethral sphincter and detrusor; rectal pressure increased significantly; there was no vesicoureteral reflux; autonomic hyperfunction. In **Table 7**, the incidence of high abdominal pressure of group B and C was noticeably higher than that of group A, illustrating that elevated abdominal pressure was actually very common in AD patients.

The results in **Table 7** also imply that the patients with abnormal bladder sensitivity are more likely to induce AD. Under normal circumstances, bladder filling or intestinal stimulation can cause sympathetic nerve excitation, increase blood pressure, stimulate aorta and carotid baroreceptor to regulate and normalize blood pressure; however, the patients with SCI can not reduce blood pressure through visceral vasodilation, the brainstem also can not transmit inhibitory impulses to the sympathetic nerves, If they were accompanied by hypersensitivity of the bladder below the injury level, AD is more likely to occur, and the sympathetic nerve mechanism is dominant, Elevated blood pressure is a clinical manifestation [18]; After the aortic and carotid baroreceptors are stimulated, the impulse is uploaded to the cardiovascular motor center of the brain stem, which can also cause parasympathetic dominance at the level above the injury, excitation of the vagus nerve, thus slowing down the heart rate, but the blood pressure is not necessarily lower, the situation of decline in heart rate is rare [19]. In **Table 1** and **Table 6** of this study, the results of group B and C are consistent with this.

It can be seen from **Table 2** that the average incidence of asymptomatic AD is higher (43.75%), which indicates that asymptomatic AD has great potential harm to the patients with chronic SCI [20]. Therefore, by accurately and objectively evaluating whether the autonomic nervous function of the patients is abnormal, early detection and identification of such asymptomatic patients, timely, scientific and standardized management of bladder and intestinal tract, and comprehensive treatment can effectively, maximally avoid acute attack of AD or deterioration of symptoms in the patients with SCI, reduce or ward off the occurrence of clinical malignant emergencies.

For the SCI patients with urination disorder, the autonomic nerve function is mostly hyperactive and blood pressure tends to rise. Therefore, during the whole process of image urodynamic examination, it is necessary to pay attention to the speed of bladder perfusion should be slow rather than fast, that is, slow filling,

also known as physiological filling refers to the speed of bladder filling is less than 10 ml/min [21], and synchronized blood pressure monitoring is performed to observe and record the patients' blood pressure and heart rate dynamic changes, before, during and after the examination, as well as whether there are five main symptoms of sweating, chills, headache, flushing and chills. Only in this way can the autonomic nervous function of patients be evaluated safely, objectively, early and accurately.

5. Conclusion

The incidence of autonomic dysfunction in the high level SCI patients with dysuria was very high (nearly 80.00%), most of them were hyperactivity, and a few were low function. The changes of SBP and DBP in the hypoactivity group all appeared an increasing and then declining trend, while the change of HR in the low function one was lower than normal and decreased continuously. The main inducements of AD are neurogenic detrusor overactivity, detrusor sphincter dyssynergia, elevated abdominal pressure and abnormal bladder sensitivity. The asymptomatic patients had a higher occurrence rate (43.75%). Only by imaging urodynamic examination with slow filling and synchronous blood pressure monitoring, can autonomic nervous function of the patients be evaluated safely, objectively, early and accurately.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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