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ORIGINAL ARTICLE

Blood pressure and age associated with silent autonomic dysreflexia during urodynamic examinations in patients with spinal cord injury

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Study design: An observational study.
Objectives: To investigate the factors associated with silent autonomic dysreflexia (AD) during urodynamics (UD) in spinal cord injury (SCI) patients.
Setting: Taichung city, Taiwan, ROC.
Methods: Blood pressure (BP) and symptoms of AD were continuously monitored during UD, and systolic blood pressure (SBP) elevations >20 mmHg was considered an AD reaction. AD patients were divided into a symptomatic group and a silent group (without AD symptoms), and variables (basic demographic data, hemodynamic data and UD parameters) were compared.
Results: A total of 42 patients, 21 in symptomatic and 21 in silent AD groups, were analyzed. Symptomatic group had significantly greater diastolic blood pressure (DBP) increment and rates of SBP/DBP changes (change of BP divided by duration) as compared to the silent group (29.5 *vs* 21.7 mmHg; 4.8 *vs* 2.3 mmHg min^{-1} and 2.8 *vs* 1.4 mmHg min^{-1}) ($P<0.05$). The cutoff points of these three variables, determined by receiver operating characteristic analysis, are: DBP increment of 21 mmHg and rates of SBP/DBP change of 2.27 and 1.16 mmHg min^{-1}. Age had a weak negative correlation with DBP change (Pearson's $r=-0.377$, $P=0.048$) and the silent group was significantly older than the symptomatic group (49.4 *vs* 40.1 years, $P<0.05$).
Conclusion: Patients with more symptomatic AD tended to have significant DBP elevation and more rapid SBP/DBP increments, and this was negatively correlated with age. Aging decreases AD symptoms and the magnitude of DBP elevation, possibly through the mechanism of decreased baroreceptor sensitivity. BP monitoring during UD and other invasive procedures is strongly recommended, especially for elderly SCI patients.
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Keywords: autonomic dysreflexia; baroreceptors; blood pressure; spinal cord injuries; urodynamics

INTRODUCTION

Patients with supra-sacral spinal cord injury (SCI) are reported to be susceptible to autonomic dysreflexia (AD), especially those injured at the T6 level or higher, and have a prevalence of nearly half to three-quarters.[1,2] AD is not an all-or-none reaction. It varies in intensity from asymptomatic to a life-threatening emergency.[1,3] Serious morbidity, like consciousness change, visual disturbances, seizures, intracranial hemorrhage or even death, is primarily associated with sudden and marked rises in blood pressure (BP).[4–6] Symptoms (that is, headache, facial flushing, chills or sweating) are usually used as warning signs of BP elevation, although sometimes there is no presenting symptoms when BP is elevated, a condition referred to as silent AD.[6] In such cases, patients and physicians may miss the ideal time to manage the underlying strong afferent stimuli until serious sequelae ensue.

As stimuli from the lower urinary tract are the most common causes of AD, reportedly in 75–85% of cases,[2,3] the routine urodynamic (UD) study of these patients offers a controlled environment to observe the AD reactions. Several previous researches have investigated BP responses in UD examinations to further understand AD reactions and the associated factors.[3,6–9] Some of these have also found that the proportion of patients presenting with silent AD ranges from 35 to 43%.[6,8,9] However, only one, the study by Linsenmeyer *et al.*,[6] investigated the differential variables between symptomatic and silent AD. They found that no factor was significant, including BP values, levels of neurologic injury, length of injury, voiding pressures and bladder capacity. However, that study used a fixed level of BP (systolic BP 160 mmHg and diastolic BP 90 mmHg) as a definition of AD, which is different from more recent concepts that define increments of 20–40 mmHg from resting SBP as an AD episode.[10–12]

As silent AD may be an important clinical concern regarding the health of the SCI patients, the present study sought to discover the factors associated with AD symptoms and investigate the mechanisms

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underlying silent AD. In this study, BP and its symptoms were monitored throughout each UD examination, and an SBP increment >20 mmHg was defined as an AD reaction.

MATERIALS AND METHODS

From September 2010 to March 2012, 132 adult supra-sacral SCI patients who received UD examinations from Chung Shan Medical University Hospital were screened. The inclusion criteria were 18–65 years of age, neurologic injury level at T6 or above with stable condition, and not in the spinal shock stage (judged by increased muscle tone and presence of detrusor contraction or sphincter spasticity on UD). The exclusion criteria included neurologic diseases other than SCI, previous genito-urinary disease or surgery, current urinary tract infection, history of cardiovascular disease (including hypertension and peripheral vascular disease) and multiple injury levels. After screening, 101 patients were included for further investigation.

The classification of neurologic injury types (level and completeness) were according to the American Spinal Injury Association criteria.[13] An SBP elevation >20 mmHg was defined as AD reaction, and these patients were enrolled for the final analysis.[12]

The execution and reading of UD examinations followed the standards of the International Continence Society.[14] Cystourethrometry was performed by a triple-lumen catheter with continuous filling of isotonic saline at a rate of 30 ml min^{-1}. Electromyography of the external urethral sphincter was obtained via concentric needle electrodes. Filling was stopped if one of the following conditions presented: (1) patient reported a sensation of fullness, (2) spontaneous urine leakage, (3) infused volume reached 500 ml, (4) BP reached a dangerous level (SBP 180 mmHg or DBP 110 mmHg) and (5) intolerable AD symptoms.

BP, pulse rate and any AD symptoms (that is, headache, facial flushing, chills or sweating) were recorded before UD examination, every 2 minutes during bladder filling/voiding and after the examination. The BP and pulse rate measurements were obtained from an automatic sphygmomanometer with an inflatable cuff wrapped around one of the patient's upper arms. Each measurement took about 30–40 s. Maximal SBP and DBP changes were calculated as maximal values subtracted by the baseline value. Maximal changes of SBP/DBP divided by duration of time was defined as the rates of SBP/DBP change.

Other variables suspected of having an association with the presentation of AD symptoms were also compared between the symptomatic and silent AD groups. These include gender, age, injury duration, injury level, completeness of injury, maximal intra-vesical pressure, maximal urethral pressure, onset volume and duration of detrusor contraction (DC), bladder compliance, and types of detrusor sphincter dyssynergia (DSD). Bladder compliance was calculated from the period between the initiation of filling and before the start of any DC or the end of filling, if there was no DC. The DSD was classified into intermittent and continuous types according to the consistency of sphincter contraction.[15] Duration of DC was the interval between the onset of DC and urine leakage or termination of examination.

In the patients with elevated BP during UD examination, their BP usually increased in a continuous and progressive way until the bladder was evacuated. In order to better understand the stimuli most strongly associated with AD reaction during examination, SBP difference before and after each UD event (that is, onset of DC, onset of DSD, maximal urethral pressure, maximal intra-vesical pressure, urine leakage, and maximal capacity) were compared. It is calculated as the first SBP record after an UD event subtracted by the last record before this event.

Differences in distribution of categorical variables between symptomatic and silent AD were analyzed by χ^2-test, whereas differences in continuous variables between these two groups were analyzed by *t*-test. If any continuous variable showed significant difference between these two groups, we used receiver operating characteristic curve analysis to calculate the best cutoff point (that is, maximal value of sensitivity plus specificity) between them. Differences in BP changes of UD events were analyzed by repeat measure analysis of variance (ANOVA). The level of significance was set at $P<0.05$.

Statement of ethics

The hospital's Ethics Committee approved the study and all patients provided prior written informed consent.

RESULTS

A total of 42 patients were selected by the aforementioned criteria and those with SBP increments >20 mmHg entered the final statistical analysis. The prevalence of AD in our SCI patients with injury level at T6 or above was 41.7% (42/101). Among these 42 patients, 21 (50%) complained of one or more AD symptoms (symptomatic AD group) and the remaining 21 (50%) had no complaints when BP elevated (silent AD group). The presenting symptoms were sweating in 10, headache in 9, facial flushing in 2 and chills in 2. The basic data of these 42 patients and the comparisons of these variables between the symptomatic and silent AD groups were shown in Table 1. The symptomatic group was significantly younger than the silent group (40.1 ± 11.7 *vs* 49.1 ± 10.4 years). There was no difference in other basic variables. The other 59 patients without BP elevation >20 mmHg during UD had no complaints of AD symptoms.

Comparisons of hemodynamic and UD parameters between the symptomatic and silent AD group were shown in Tables 2 and 3. Symptomatic AD patients showed more significant DBP increments (29.5 ± 9.4 *vs* 21.7 ± 7.9 mmHg) and greater rate of SBP/DBP elevation than the silent AD patients (rates of SBP change 4.8 ± 2.8 *vs* 2.3 ± 1.3 mmHg min^{-1}; rates of DBP change 2.8 ± 1.1 *vs* 1.4 ± 0.9 mmHg min^{-1}) ($P<0.05$). Receiver operating characteristic-curve analysis showed that the best cutoff points to predict patients having AD symptoms are: DBP increment of 21 mmHg (sensitivity 86; specificity 69%), rate of SBP change of 2.27 mmHg min^{-1} (sensitivity 79; specificity 80%) and rate of DBP change of 1.16 mmHg min^{-1} (sensitivity 100; specificity 69%). (Figure 1) The symptomatic group had non-significant increases in maximal SBP/DBP value, SBP increment, maximal urethral pressure, bladder stiffness and chance of continuous DSD. A sage was a factor associated

Table 1 Comparison of basic demographic data between symptomatic and silent AD groups

	Total	*Symptomatic AD*	*Silent AD*
Number	42	21	21
Age	44.8 ± 12.9	40.1 ± 11.7	49.4 ± 11.7
Mean ± s.d. (range)	(22–65)	(22–63)*	(27-65)*
Gender			
Male	35	18	17
Female	7	3	4
Injury duration	18.3 ± 38.1	15.8 ± 18	20.6 ± 45.9
(months)	(1.5–180)	(2–60)	(1.5–180)
Mean ± s.d. (range)			
Injury level			
Cervical	35	18	17
Thoracic	7	3	4
Completeness of injury			
Complete	18	10	8
Incomplete	24	11	13

Abbreviations: AD, autonomic dysreflexia; s.d., standard deviation.
*$P<0.05$.

with symptomatic presentation, the correlations of age and other hemodynamic factors (that is, baseline and maximal BP, maximal changes of BP and rates of BP elevation) were further analyzed. The only correlation found between age and hemodynamic factors was a weak one with DBP change. (Pearson's $r = -0.377$, $P = 0.048$).

The results of comparing SBP changes during different UD events were shown in Table 4. We found SBP increase was most prominent at the onset of DC and DSD. The AD symptoms did not always present simultaneously during the periods of significant BP elevation and half appeared after BP elevation.

Table 2 Comparison of hemodynamic parameters between symptomatic and silent groups

	Total	*Symptomatic AD*	*Silent AD*	P *value*
Baseline SBP (mmHg)				
Mean ± s.d.	116.2 ± 14.0	115.5 ± 16.5	116.9 ± 10.3	0.798
Range	70 ~ 140	70 ~ 139	81 ~ 140	
Baseline DBP (mmHg)				
Mean ± s.d.	76.4 ± 9.9	76.5 ± 14.4	76.3 ± 7.5	0.755
Range	42 ~ 92	42 ~ 92	51 ~ 85	
Maximal SBP (mmHg)				
Mean ± s.d.	163.6 ± 24.7	168.4 ± 32.7	159.1 ± 13.9	0.266
Range	105 ~ 208	105 ~ 208	121 ~ 179	
Maximal DBP (mmHg)				
Mean ± s.d.	101.4 ± 14.1	104.7 ± 16.3	98.1 ± 10.1	0.235
Range	72 ~ 123	72 ~ 123	79 ~ 115	
Maximal SBP change (mmHg)				
Mean ± s.d.	45.4 ± 18.1	51.6 ± 24.3	40.6 ± 13.6	0.159
Range	22 ~ 94	22 ~ 94	23 ~ 66	
Maximal DBP change (mmHg)				
Mean ± s.d.	24.9 ± 9.7	29.5 ± 9.4*	21.7 ± 7.9*	0.042*
Range	11 ~ 46	11 ~ 46	12 ~ 35	
Maximal PR change (beats min^{-1})				
Mean ± s.d.	−5.7 ± 11.9	−6.8 ± 14.7	−4.8 ± 8.5	0.641
Range	−20 ~ 32	−20 ~ 32	−19 ~ 18	
Rate of SBP change (mmHg min^{-1})				
Mean ± s.d.	3.5 ± 2.6	4.8 ± 2.8*	2.3 ± 1.3*	0.004*
Range	1.25 ~ 11.5	1.55 ~ 11.5	1.25 ~ 6.60	
Rate of DBP change (mmHg min^{-1})				
Mean ± s.d.	2.1 ± 1.2	2.8 ± 1.1*	1.4 ± 0.9*	0.001*
Range	0.31 ~ 5.33	1.31 ~ 5.33	0.31 ~ 3.50	

Abbreviations: AD, autonomic dysreflexia; DBP, diastolic blood pressure; SBP, systolic blood pressure; PR, pulse rate; s.d., standard deviation.
*P<0.05.

DISCUSSION

In this study, the prevalence of AD is 41.7% in our SCI patients with injury level at T6 or above. This is similar to a previous report using the same definition of AD reaction (SBP increment >20 mmHg).[12] Among these patients with signs of AD, 50% were symptomatic and 50% were silent. This percentage of silent AD is higher than three previous studies that reported 35, 38 and 43% respectively.[6,8,9] This discrepancy may be due to the different criteria of AD in these studies. Two of them used definite levels of SBP/DBP values as 160/100 and 150/100 mmHg, respectively, whereas one used an SBP increment percentage of 30% of baseline value. As tetraplegic SCI

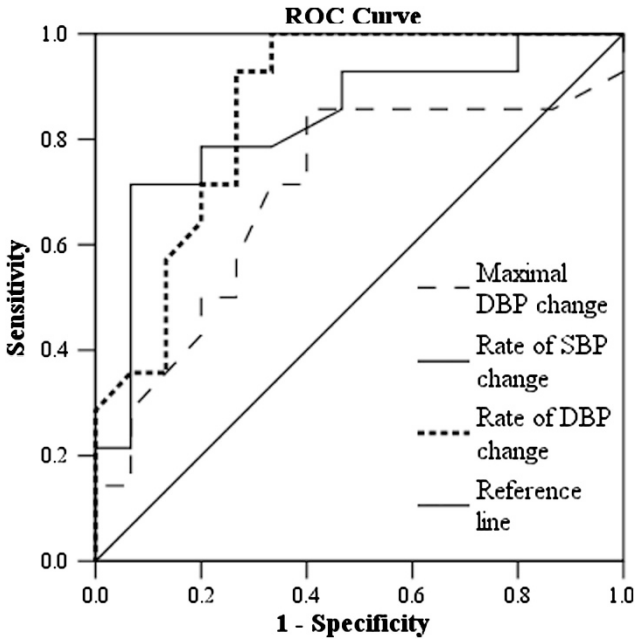

Figure 1 Receiver operating characteristic curve analysis of maximal DBP change, rate of SBP change, and rate of DBP change.

Table 3 Comparison of UD parameters between symptomatic and silent groups

	Total	*Symptomatic AD*	*Silent AD*	P *value*
Volume of DC onset (ml)	193.9 ± 111.5	204.1 ± 106.0	184.1 ± 103.8	0.768
Duration of DC (min)	7.0 ± 4.1	6.4 ± 3.4	7.4 ± 4.5	0.481
Maximal intravesical pressure (cmH2O)	76.3 ± 33.6	78.6 ± 31.0	74.1 ± 36.1	0.659
Maximal urethral pressure (cmH2O)	85.6 ± 37.0	86.9 ± 35.6	75.1 ± 33.7	0.081
Compliance (ml cm^{-1}H2O)	25.1 ± 21.7	22.9 ± 15.2	29.8 ± 24.2	0.145
Type of DSD (no.)				0.054
No DSD	1	0	1	
Intermittent DSD	10	2	8	
Continuous DSD	31	19	12	

Abbreviations: AD, autonomic dysreflexia; DC, detrusor contraction; DSD, detrusor sphincter dyssynergia; UD, urodynamic.

Table 4 Comparisons of SBP changes during UD events

	Onset of DC	*Onset of DSD*	*Max Pure*	*Max Pves*	*Urine leakage*	*Maximal capacity*
SBP changes (mmHg) Mean ± s.d.	13.7 ± 12.2*	13.1 ± 13.5*	4.6 ± 13.8	5.0 ± 12.8	−1.2 ± 14.5	−3.1 ± 12.0

Abbreviations: DC, detrusor contraction; DSD, detrusor sphincter dyssynergia; Max Pure, maximal urethral pressure; Max Pves, maximal intravesical pressure; SBP, systolic blood pressure; s.d., standard deviation.
*$P<0.05$.

patients usually have lower BP level than able-bodied persons, often at the range of 90/60 mmHg, the criteria of AD in these three studies are stricter than that of the current study and thus, might result in a higher rate of symptomatic AD and lower rate of silent AD. Moreover, two of these studies mentioned that some patients in the group that they defined as normotensive showed AD symptoms.[6,9] No one in the non-AD group in the present study had any AD symptoms.

Both this study and the previous report by Linsenmeyer *et al.*[6] reveal that the absolute levels of BP is not crucial to symptomatic presentation. Although the symptomatic AD group here has slightly higher values of maximal SBP and DBP than the silent AD group, the difference is not significant. That is to say, silent AD patients may have dangerously high levels of SBP, reaching 180 mmHg in this study (Table 2). On the other hand, we found the magnitude of BP increments, especially DBP, and the rates of BP changes are factors associated with the presentation of AD symptoms. After further receiver operating characteristiccurve analysis, patients with DBP increment reaching 21 mmHg, rate of SBP change reaching 2.27 mmHg min^{-1} and rate of DBP change reaching 1.16 mmHg min^{-1}, were found to have greater chance to be symptomatic. On the contrary, patients with BP changes lower than these values tended to be silent. We think this phenomenon could be partially explained by the physiology of baroreceptor reflex.

AD is a phenomenon initiated by a sudden rise in sympathetic tone with BP elevation induced by strong afferent stimuli below the injury level. While BP is elevated, some patients may experience uncomfortable symptoms, including headache, sweating and flushing, which may be associated with passively increased blood flow or vasodilatation above the lesion levels.[1,2,16] In this chain of reaction, baroreceptors mediated reflex arcs might have some important roles.[1,2,16] Baroreceptors are mechano-sensitive terminals triggered by both the magnitude and rate of change in arterial pressure level, which is usually referred to as the mean arterial pressure, a composition of 1/3 SBP and 2/3 DBP.[17] This may explain in part why magnitude of DBP increment, as well as the rate of BP changes, have a more important role than the magnitude of SBP changes in inducing symptoms.

In the present study, many factors, including injury patterns (level, duration and completeness) and UD parameters (onset/duration of DC, maximal intra-vesical/urethral pressure, compliance and type of DSD), are found to be not associated with AD symptoms. This finding is similar to that in the study of Linsenmeyer *et al.*[6], wherein the variables included were level and length of injury, bladder capacity and voiding pressure. However, the current study notes that patients without symptoms are significantly older than those with symptoms, and this may result partly from the weak negative correlation of age with DBP change (Pearson's $r = -0.377$). Thus, older patients have less DBP increase during UD and lower chances of being symptomatic. The effect of age on BP has been reported on in previous literature reports, noting that despite the continuous increase in SBP with age, DBP reaches its maximum at the age of 55 years and then begins to fall.[18] Stiffening of the aorta with diminished elastic recoil during aging may cause the runoff of blood during systole, resulting in reduced blood volume within the aorta and lower diastolic pressure.[19] The other possible cause of aging effect on symptomatic presentation is the reduction of baroreceptor sensitivity when patients get older.[20] These results serve as a reminder that older SCI patients have higher risk of silent AD and should be closely monitored as regards their BP during invasive procedures or even during voiding and defecation.

During UD examination, a previous research study reported that BP increase begins mostly at the peak of uninhibited DC or at maximal bladder capacity.[9] As elevation of BP during UD examination is a gradual but continuous pattern, it is difficult to determine the beginning or onset of BP increment. By comparing SBP difference before and after each UD event, this study reveals that the most prominent changes in SBP elevation occur at the onsets of DC and DSD. This phenomenon may be supported by the theory of Perkash *et al.*[7] in 1979. They proposed that AD is a spinal reflex mediated by afferents arising from the bladder wall near the bladder neck. These structures are noncompliant in patients with abnormal holding reflex-sphincter dyssynergia and convey excessive stimulus impulses when being stretched. On the other hand, as AD is a chain of reaction, symptoms present simultaneously with or later than BP elevation, as shown in the current study.

This study has some limitations that are worth noting. First, the total number of included subjects was only 42 and some variables seemed different but not significant. Second, a fixed infusion rate of 30 ml min^{-1} was used. As such, it could not be seen if the infusion rate was an influencing factor of symptomatic presentation. As AD is thought to be induced by stretching of the bladder wall or neck, the infusion rate may have some influence on AD symptoms. Lastly, AD symptoms were recorded according to subjective complaints. More objective measurements like sympathetic skin response or heart-rate variability could be used in the future.

CONCLUSIONS

Under the definition of SBP elevation >20 mmHg, SCI patients, injured at or above T6 level, with AD reactions have a 50% rate of silent AD. Elevation of BP is mostly significant at the onsets of DC and DSD during UD examination. Patients with symptomatic AD were associated with more significant DBP elevation and more rapid SBP/DBP increase. Aging patients had greater chance to have silent AD and this might be explained with decreased DBP elevation and possibly blunting of baroreceptors. BP monitoring during UD and other invasive procedures or even during voiding, is strongly recommended especially for aged SCI patients.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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