Acta Medica Okayama

Volume 61, *Issue* 4 2007 *Article* 6

AUGUST 2007

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Abstract

The purpose of the present study was to investigate the relationship between bowel maneuvers and autonomic dysreflexia (AD) in patients with cervical spinal cord injuries (CSCI). Fifteen consecutive, clinically stable patients with CSCI participated. We evaluated changes in blood pressure (BP), pulse rate (PR) and classic symptoms of AD before, during and after a bowel program involving the manual removal of stool in lateral recumbency. The insertion of rectal medication induced a significant increase in systolic BP, which persisted during additional digital rectal stimulation. Furthermore, the manual removal of stool induced AD, with maximal increases of systolic BP (169.1(+-)19.5 mmHg, mean(+-)SD). However, the insertion of a finger into the anus after the end of stool flow did not cause a further increase in systolic BP. Systolic BP recovered to pre-program values within 5 min after defecation. Our study demonstrated that the combined effects of rectal and/or anal sphincter distension and uninhibited rectal contraction in response to the manual removal of stool might induce AD. We recommend avoiding, if at all possible, the manual removal of stool in order to prevent AD in patients with CSCI.

KEYWORDS: spinal cord injury, autonomic dysrefl exia, blood pressure, bowel program

Acta Med. Okayama, 2007
Vol. 61, No. 4, pp. 221–227

Acta Medica Okayama
http://www.lib.okayama-u.ac.jp/www/acta/

Original Article

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The purpose of the present study was to investigate the relationship between bowel maneuvers and autonomic dysreflexia (AD) in patients with cervical spinal cord injuries (CSCI). Fifteen consecutive, clinically stable patients with CSCI participated. We evaluated changes in blood pressure (BP), pulse rate (PR) and classic symptoms of AD before, during and after a bowel program involving the manual removal of stool in lateral recumbency. The insertion of rectal medication induced a significant increase in systolic BP, which persisted during additional digital rectal stimulation. Furthermore, the manual removal of stool induced AD, with maximal increases of systolic BP (169.1 ± 19.5 mmHg, mean ± SD). However, the insertion of a finger into the anus after the end of stool flow did not cause a further increase in systolic BP. Systolic BP recovered to pre-program values within 5 min after defecation. Our study demonstrated that the combined effects of rectal and/or anal sphincter distension and uninhibited rectal contraction in response to the manual removal of stool might induce AD. We recommend avoiding, if at all possible, the manual removal of stool in order to prevent AD in patients with CSCI.

Key words: spinal cord injury, autonomic dysreflexia, blood pressure, bowel program

Autonomic dysreflexia (AD) is commonly caused by bladder distension, bowel distension and defecation [1], and the fact that the associated hypertension may cause seizures, intracerebral hemorrhage or death is of particular concern [2]. Clinically, AD is characterized by, 1) a sudden increase in blood pressure (BP) with compensatory bradycardia, 2) headache, and 3) profuse sweating and vasodilatation with skin flushing above the level of the spinal cord injury (SCI) [3]. AD occurs in patients with an SCI above the greater splanchnic outflow, usually above T6 [2]. Proprioceptive and noxious stimuli below the level of injury may induce an episode of AD.

Bowel dysfunction in patients with SCI is a significant problem that often limits activities of daily living, social activity and quality of life. Fear of bowel accidents frequently restricts patients from activities outside their home [4]. Patients with SCI have to adopt many strategies to maintain adequate

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bowel management. The most widely used bowel programs include: 1) oral laxatives, 2) rectal medication, 3) gentle digital stimulation, and 4) manual removal of stool.

Recently, Kirshblum *et al.* [5] reported that silent AD occurred frequently in patients with SCI above T6 during a routine bowel program [5]. Therefore, it is particularly important for patients with cervical spinal cord injury (CSCI) to be able to recognize AD during their bowel program. However, to our knowledge, there are no published data on the relationship between bowel maneuvers and AD. The purpose of the present study was to investigate the clinical symptoms and signs of AD during and after a bowel program in patients with CSCI.

Materials and Methods

Subjects. The experimental protocol was approved by the Research Ethics Committee at our institution and all subjects were required to sign an informed consent form. Fifteen consecutive inpatients (14 men and 1 woman) at our rehabilitation unit with CSCI were prospectively studied. They underwent a physical examination and none were found to have hypertension, diabetes mellitus, pneumonia, ischemic heart disease, or renal dysfunction. We excluded patients with infection and inflammation, including urinary tract infection or a pressure ulcer, both of which might induce AD. The mean age in these subjects was 40.9 ± 15.4 years ($\pm$ SD, range, 19–64). The neurological findings were classified according to the American Spinal Cord Injury Association (ASIA), the standard for neurological and functional classification of SCI, and the anthropometric characteristics of the subjects are shown in Table 1. Thirteen patients were classified as having an ASIA A injury [6], and 2 an ASIA B injury [6]. One patient had a C2 level injury, 3 patients a C4 level, 5 patients a C5 level, 5 patients a C6 level and one patient a C7 level. None of the patients took medications known to interfere with BP or the autonomic nervous system. The mean duration of injury in these subjects was 45.8 ± 42.0 months ($\pm$ SD, range, 4.83–144.6). All patients were in a clinically stable state.

The participants were dependent on medication and the assistance of the health care staff to perform their bowel program. Nine patients had the bowel program twice a week, and 6 patients had it 3 times a week. All participants regularly took oral laxatives. Eight patients took a glycerin enema and 9 patients took suppositories containing sodium bicarbonate and potassium bitartrate in their bowel program. Urine voiding was achieved by either clean intermittent catheterization ($n = 8$) or indwelling urethral or suprapubic catheter ($n = 7$).

Bowel program and experimental protocol. Ten min prior to the bowel program, the subject's bladder was emptied to eliminate AD caused by distension as a potential confounder, and the subject was then placed in a lateral recumbent position. The subject wore an automated vital sign-recording device (Vital Signs Monitor, model 5200P; Welch Allyn Medical Products, 4341 State St Rd, PO Box 220, Skaneateles Falls, NY, USA) on the arm to monitor systolic and diastolic BP as well as pulse rate (PR). From 5 min before commencement of the bowel program (pre-program period), systolic and diastolic BP and PR were measured every 1 min. One well-trained nurse conducted the bowel program for all subjects. The bowel program was then begun, and the same vital signs were monitored every 1 min during the procedure, every 1 min until 5 min after the program, and again at 30 min and finally the next day at the same time (recovery period).

At the start of the bowel program, the medication

Table 1 Clinical features of the 15 study participants

Patient	Age (years)	Sex	Duration of injury (month)	Level of SCI	ASIA Impairment scale
A	50	M	11.2	C4	A
B	23	M	25.4	C5	A
C	19	M	4.83	C6	A
D	58	M	17.9	C4	A
E	27	M	105.7	C6	A
F	25	M	21.3	C6	B
G	54	F	75.8	C7	A
H	50	M	10.1	C6	A
I	29	M	32.8	C2	A
J	55	M	11.4	C5	A
K	24	M	52.1	C5	A
L	47	M	69.3	C5	A
M	55	M	98.7	C5	B
N	33	M	144.6	C4	A
O	64	M	6.54	C6	A

was inserted gently into the rectum, acting as an initialization stimulus (Table 2). After commencement of stool flow, digital stimulation was performed to induce augmentation of stool flow. Digital rectal stimulation with manual removal of stool was often necessary for participants and the distance of digital insertion into the anus was 6 cm. The end of defecation was signaled by the cessation of flatus and stool flow, and palpable internal sphincter closure [4]. The defecation period was defined as the period between the start and end of stool flow [4]. Symptoms of AD that were inquired about included headache, sweating or flushing above the level of injury, nasal congestion, blurred vision, anxiety, or any other unusual symptom that may have been experienced during previous episodes of AD unrelated to the bowel program [5]. The criteria for AD were determined to have been met when the systolic BP reached 20–40 mmHg above the baseline measurement for SBP [7]. It has been recommended that pharmacologic management to reduce systolic BP be considered if the elevated systolic BP equals or exceeds 150 mmHg despite removing the suspected cause of AD [8]. In our study, we stopped bowel maneuvers temporarily if the systolic BP reached 160 mmHg. If this BP continued for 5 min we did not resume the bowel program.

On another day when defecation and the bowel program were not performed, systolic and diastolic BP and PR were monitored at the same time points in an identical posture (lateral recumbency) as in the control study.

Statistical analysis. Data were expressed as mean ± SD. Analysis of variance (ANOVA) was used for comparison within and between groups. When ANOVA showed significant differences ($p < 0.05$), Fisher's test was used to determine differences between 2 time periods and between 2 groups. A p value < 0.05 denoted the presence of a significant difference between 2 groups.

Results

All subjects completed the bowel programs. Fig. 1 demonstrates typical changes in systolic and diastolic BP and PR throughout the bowel program in a subject with complete tetraplegia at the C7 level. Systolic BP, diastolic BP and PR during the pre-program period in subjects with CSCI were not significantly different compared with the control study. In contrast, all subjects in this study exhibited a significant rise in systolic and diastolic BP (as AD is defined) during the bowel program (Figs. 2, 3). In the control study, all

Table 2 Results of bowel program

Patient	Rectal medication	Frequency of bowel care (/Week)	Defecation period (minute)	Amount of stool (ml)	Main nature of stool	AD symptoms
A	S	3	14	300	S	None
B	E	2	27	300	S	None
C	None	2	23	200	H	Goose bumps
D	E	3	9	200	H	None
E	S	2	31	100	S	Headache
F	S	3	21	200	H	None
G	S	2	21	400	H	None
H	S	2	35	200	N	None
I	E	2	26	200	N	Chills, Sweating
J	S&E	2	13	200	N	None
K	S&E	2	26	200	N	Chills, Goose bumps
L	E	3	21	200	N	Headache
M	S&E	2	29	600	N	None
N	E	3	34	200	S	None
O	S	3	4	200	H	None

Rectal medication: S, Suppository; E, Enema.
Main nature of stool: S, Soft; H, Hard; N, Normal.

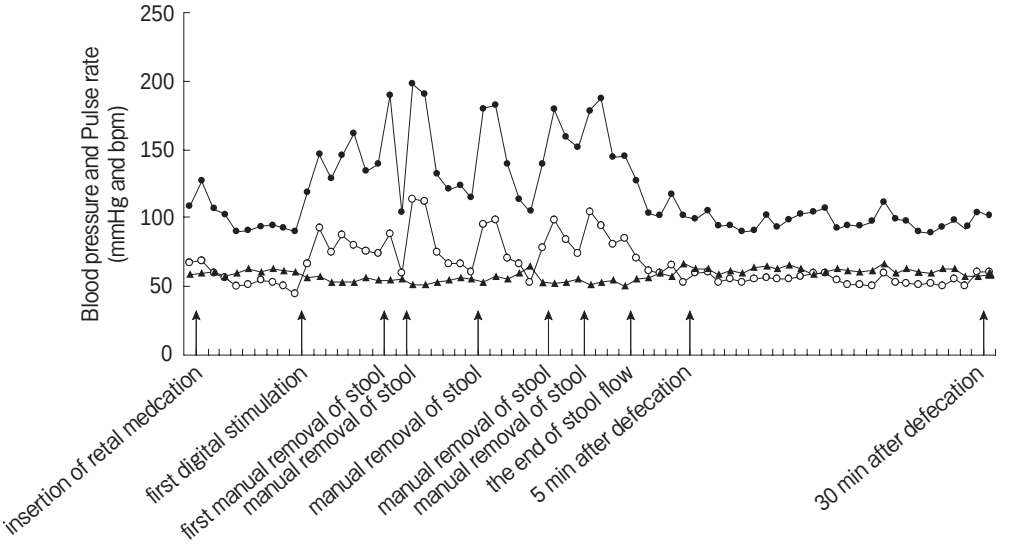

Fig. 1 Typical changes in BP and PR during the bowel program in a patient with CSCI. A 54-year-old woman with complete tetraplegia (ASIA impairment scale A) at the C7 level developed autonomic dysreflexia without classic symptoms during defecation. Closed circles, systolic blood pressure (mmHg); open circles, diastolic blood pressure (mmHg); closed triangles, pulse rate (beats per min [bpm]).

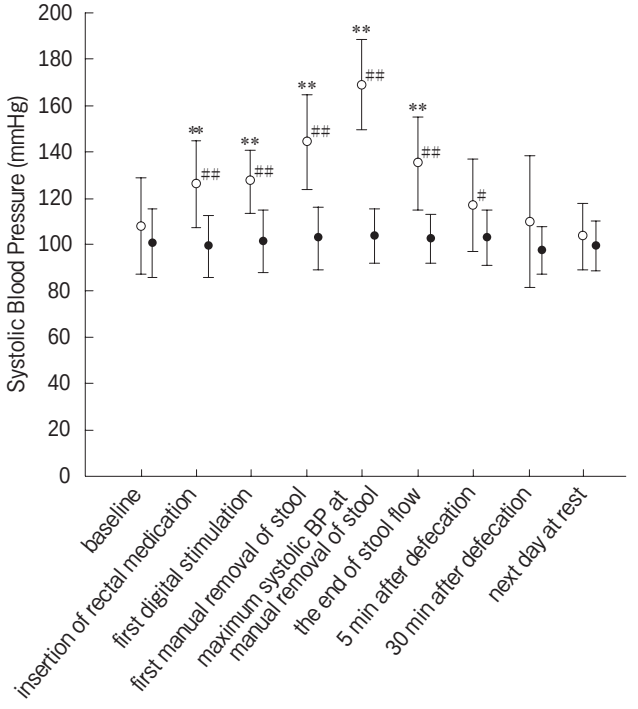

Fig. 2 Mean changes in systolic blood pressure during the bowel program. Open symbols: blood pressure during the bowel program, closed symbols: blood pressure at rest (control). Data are mean ± SD. $^{**}p < 0.01$, compared with pre-program (baseline) values, $^{\#\#}p < 0.01$, $^{\#}p < 0.05$, compared with control data.

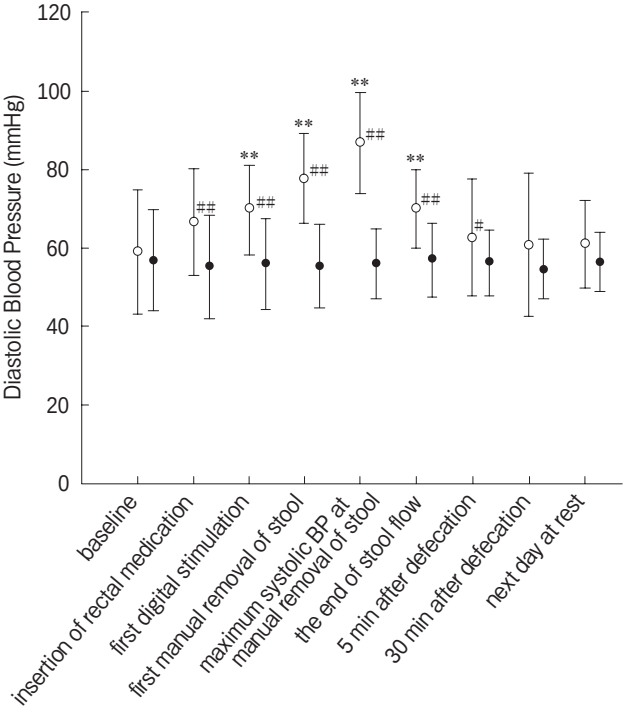

Fig. 3 Mean changes in diastolic blood pressure during the bowel program. Open symbols: blood pressure during the bowel program, closed symbols: blood pressure at rest (control). Data are mean ± SD. $^{**}p < 0.01$, compared with pre-program values, $^{\#\#}p < 0.01$, $^{\#}p < 0.05$, compared with control data.

measurements remained constant throughout the same period. The defecation period was 22.3 ± 8.72 min (Table 2). The length of time and percentage the patients were dysreflexic during the defecation period were 13.0 ± 9.02 min and $61.3 \pm 29.3\%$, respectively.

The insertion of rectal medication induced a significant increase in systolic BP ($p < 0.01$), which persisted during additional digital rectal stimulation ($p < 0.01$). The significant increase in diastolic BP was found at the first digital rectal stimulation ($p < 0.01$). Furthermore, with the onset of manual stool removal, both systolic and diastolic BP significantly increased from 108.1 ± 20.8 mmHg and 58.9 ± 15.7 mmHg to 169.1 ± 19.5 mmHg and 86.7 ± 12.9 mmHg, respectively, compared with the pre-program period ($p < 0.01$). In 14 subjects with CSCI, the maximum systolic and diastolic BP were recorded during the manual removal of stool, while the maximum systolic and diastolic BP in one subject was recorded during the insertion of rectal medication. At 5 min after defecation, both systolic and diastolic BP had returned to pre-program values, but remained significantly higher compared with the same period during the control study. The insertion of a finger to confirm the end of defecation did not induce an increase of BP consistent with AD. PR decreased significantly at the point of first digital stimulation compared with the pre-program period ($p < 0.05$) and returned to pre-program values at the end of stool flow (Fig. 4)

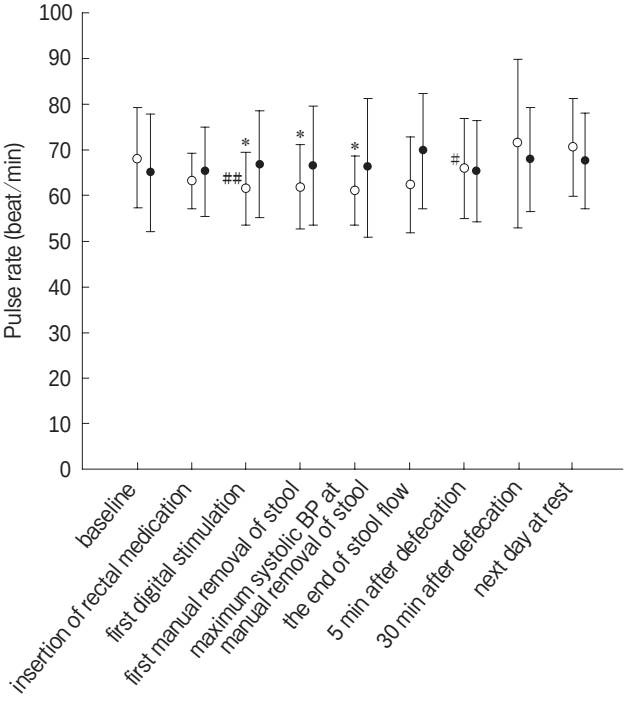

Fig. 4 Mean changes in pulse rate during the bowel program. Open symbols: pulse rate during the bowel program, closed symbols: pulse rate at rest (control). Data are mean ± SD. $^{*}p < 0.05$, compared with pre-program values, $^{\#\#}p < 0.01$, $^{\#}p < 0.05$, compared with control data.

Five patients complained of symptoms of AD during the time a significant rise in BP occurred, whereas 10 patients did not show any symptoms of AD during the same time period (Table 3). The symptoms of AD included chills (n = 2), goose bumps (n = 2), sweating (n = 1) and headache (n = 2). In 4 patients with symptomatic AD, the maximum systolic and diastolic BP coincided with the appearance of AD symptoms, but one patient complained of symptoms during the period of significant increase of BP but not at the peak of BP. In the 5 patients who had significantly increased BP with symptoms of AD, the maximum systolic BP and diastolic BP were 163 ± 15.7 mmHg and 83 ± 14.3 mmHg, respectively. In the 10 patients who had significantly increased BP without symptoms of AD, the maximum systolic BP and diastolic BP were 173 ± 18.3 mmHg and 86 ± 17.4 mmHg, respectively. There was no significant difference in BP between the 2 groups.

Table 3 Maximum systolic blood pressure and symptoms of autonomic dysreflexia during bowel program

Patient	Baseline	Maximum systolic BP during bowel program	AD symptoms
A	122	166	None
B	73	165	None
C	101	132	Goose bumps
D	106	142	None
E	133	176	Headache
F	139	176	None
G	109	198	None
H	67	197	None
I	109	172	Chills, Sweating
J	83	162	None
K	126	168	Chills, Goose bumps
L	130	165	Headache
M	105	177	None
N	97	154	None
O	121	197	None

Discussion

The present study demonstrated that, 1) the insertion of rectal medication induced a significant rise of systolic BP, 2) systolic and diastolic BP maximally increased during manual removal of stool and PR significantly decreased at that time, 3) systolic and diastolic BP recovered within 5 min of the end of stool flow, and 4) insertion of a finger into the anal canal after the end of stool flow was not associated with significant increases in systolic and diastolic BP. Therefore, mechanical and pharmacological stimulation of the anus during rectal distension with stool appears to induce symptomatic or silent AD.

In patients with chronic CSCI, the stimuli might arise from activation of thermoreceptors in the skin, and a reflex sympathetic discharge through the isolated spinal cord resulted in a more profound rise in mean BP during ice-water immersion [9]. A small amount of cold stimulation in CSCI subjects induced AD [9]. Although there is little information about the type of receptors that are activated in hollow organs necessary to induce AD, distension of hollow organs such as the bladder and the bowel is the most common cause of AD [10]. Cosman *et al.* [11] suggested that anoscopy, which involved anal sphincter stretching, was a stronger stimulus for AD than sigmoidoscopy, which involved rectosigmoid distension, although both anal sphincter stretch and rectosigmoid distension were associated with increased systolic BP. In the present study, the manual removal of stool could distend the anal sphincter and resulted in a maximal increase of BP in patients with CSCI. However, after the end of stool flow, insertion of a finger did not induce a significant increase of BP. Linsenmeyer *et al.* [2] emphasized that the increase in BP in patients with uninhibited bladder contractions was not caused by bladder distension *per se*, but by bladder distension triggering an uninhibited bladder contraction [2]. Therefore, the combined effects of rectal and/or anal sphincter distension and uninhibited rectal contraction in response to the manual removal of stool might activate mechanoreceptors and induce a greater increase of BP. Uninhibited rectal contraction might be induced more easily during stool impaction.

Of the 15 subjects who showed a positive pressor response during the bowel program, 10 (66.7%) had no symptoms of AD and were unaware of BP elevation. Linsenmeyer *et al.* [2] investigated whether symptoms of AD correlated with an elevation in BP during voiding in men with SCI and reported that of 35 hypertensive patients, 15 (43%) had no symptoms of AD. Kirshblum *et al.* [5] examined the existence and frequency of silent AD in 10 subjects with complete SCI above the neurological level of T6 and reported that none of the subjects experienced any of the classic symptoms of AD. In the present study, there was no significant difference in maximum BP between patients with and without AD symptoms.

It has been reported that acute elevation in BP is a sign of AD and might induce serious events including seizures, intracerebral and subarachnoid hemorrhage [10]. AD is accompanied by alterations in heart rate, which may in some cases be pathological [10]. Pulse rate may sometimes be irregular, and arrhythmias such as premature ventricular contractions, bigeminy and second degree A-V block have been reported [10]. We are afraid that heart failure and other cerebro-vascular attack might be induced as long-term complications of AD. Several methods have been suggested to clinically prevent AD in patients with SCI at or above T6. The initiation of a high fiber diet and use of stool softeners, gentle, small volume oil retention enemas, 2% lidocaine jelly into the rectum and very gentle insertion of glycerin suppositories may alleviate the constipation and avoid AD [12, 13]. Although the use of stool softeners, oil retention enemas, and the gentle insertion of suppositories were performed in the present study, AD was observed in all subjects. The manual removal of stool is dangerous since it may damage the anorectal mucosa and anal sphincter in this anesthetized area [14]. We recommend avoiding, as much as possible, the manual removal of stool in order to prevent AD in patients with SCI at or above T6.

Acknowledgments. The authors are grateful to Dr. F. G. Issa (www.word-medex.com.au) for the careful reading and editing of this manuscript.

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