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ORIGINAL ARTICLE

Topical anesthesia blunts the pressor response induced by bowel manipulation in subjects with cervical spinal cord injury

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Design: Prospective double-blind study.
Objective: Manual emptying of rectal contents is necessary for patients with spinal cord injury to prevent bowel obstruction; however, this maneuver induces a rise in blood pressure (BP) and autonomic dysreflexia (AD). The purpose of this prospective double-blind study was to investigate whether topical anorectal anesthesia attenuates the BP rise and AD during bowel manipulation in patients with cervical spinal cord injury (CSCI).
Setting: Kibikogen Rehabilitation Center for Employment Injuries.
Methods: The study subjects were 25 consecutive clinically stable patients with CSCI. Each subject received a complete bowel program involving manual removal of stool in lateral recumbency, after topical application of lidocaine or placebo jelly to the anorectal area. Systolic and diastolic BP, heart rate and symptoms of AD were recorded before, during and after bowel manipulation.
Results: Systolic BP was significantly lower during the lidocaine arm of the study compared with placebo at insertion of rectal medication, digital stimulation, beginning of stool flow, manual removal of stool, end of stool flow and at 5-min after emptying. The mean maximal increase in systolic BP during lidocaine treatment (33.2 ± 14.6 mm Hg) was less than during placebo (50.2 ± 19.5 mm Hg, $P<0.001$).
Conclusion: On the basis of our findings, we recommend induction of lidocaine jelly immediately before rectal manipulation in patients with CSCI to minimize the incidence and severity of AD.
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Keywords: spinal cord injury; lidocaine; rehabilitation; blood pressure; autonomic dysreflexia

Introduction

Spinal cord injury (SCI) above the fifth to sixth thoracic level is sometimes complicated by autonomic dysreflexia (AD).[1] The most common causes of AD are bladder distension, bowel distension and defecation.[2]

Patients with SCI have to adopt many strategies to maintain adequate bowel management. The most widely used bowel programs include: (1) oral laxatives, (2) rectal suppositories, (3) gentle digital stimulation and (4) manual removal of stool. Lynch *et al*.[3] reported that of 467 patients with SCI, manual evacuation was used regularly by 67% of patients with complete cord injuries and 25% of those with incomplete injuries. Manual removal of stool is necessary for patients with SCI to prevent bowel obstruction.

We previously investigated the changes in blood pressure (BP), heart rate (HR) and classic symptoms of AD before, during and after bowel programs involving manual removal of stool in lateral recumbency in patients with cervical SCI (CSCI).[4] Manual removal of rectal contents induced AD, with large increases in both systolic and diastolic BP, while insertion of a finger into the anal canal at the end of stool flow did not cause a further increase in systolic or diastolic BP.[4] Therefore, techniques that could reduce AD symptoms or dampen the rise in BP during bowel programs in patients with SCI are desirable.

AD in CSCI subjects is considered a reflex sympathetic discharge through the isolated spinal cord as the lack of supraspinal vasomotor control is responsible for the profound rise in BP.[5] Cosman *et al*.[6] found that instillation of lidocaine into the rectum did not prevent AD during stretching of the anal sphincter or during gaseous distention of the rectosigmoid induced by anorectal procedures such as flexible sigmoidoscopy and anoscopy. On the other hand, their follow-up study[7] found that lidocaine anal block prevented AD during anorectal procedures. We hypothesized that bowel program (digital stimulation) is not as strong a stimulus as anorectal procedures (with gaseous distention).

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Therefore, lidocaine may be more effective under the former procedures. In the present study, we evaluated the effects of topical anorectal anesthesia on the incidence and severity of AD in patients with CSCI during the actual conduct of bowel programs.

Methods

Participants

The experimental protocol was approved by the Research Ethics Committee of Kibikogen Rehabilitation Center for Employment Injuries and all subjects were required to sign an informed consent form. The subjects of this prospective study were 25 consecutive inpatients (22 men and 3 women) with CSCI. None of the subjects had hypertension or had allergy to local anesthetics, and none was on any antihypertensive medications that might influence AD–BP responses. Patients with infection or inflammation such as urinary tract infection, or pressure ulcers that might induce AD, were excluded from this study. Table 1 summarizes the anthropometric features of the participating subjects. Neurological classification was described according to the American Spinal Cord Injury Association (ASIA), the standard for neurological and functional classification of SCI.[8] Twenty patients were classified as having an ASIA A injuries and five ASIA B injuries. The neurological motor level was C4 in five patients, C5 in eight patients, C6 in seven patients and C7 in five patients. At study entry, the mean time since injury was 23.4 ± 36.4 months (± s.d., range: 3–172 months). All subjects were in a clinically stable state.

Table 1 Descriptive data for the 25 subjects

Subjects	*Age (years)*	*Gender*	*Duration of injury (month)*	*Level of SCI*	*ASIA impairment scale*
1	42	M	16	C5	A
2	27	M	8	C5	A
3	31	M	9	C5	A
4	26	M	13	C7	A
5	18	M	9	C5	A
6	30	M	7	C6	B
7	28	M	5	C6	A
8	36	M	13	C4	A
9	32	M	172	C5	A
10	23	M	12	C5	A
11	21	F	20	C4	B
12	19	F	9	C7	A
13	16	F	5	C4	A
14	55	M	9	C6	A
15	36	M	7	C6	A
16	50	M	9	C7	A
17	28	M	80	C5	A
18	32	M	3	C6	B
19	58	M	8	C4	A
20	18	M	14	C6	B
21	51	M	21	C4	A
22	38	M	6	C7	A
23	26	M	59	C6	A
24	29	M	57	C7	B
25	37	M	14	C5	A

Abbreviations: ASIA, American Spinal Cord Injury Association; F, female; M, male; SCI, spinal cord injury.

Twenty-three participants regularly took oral laxatives while two did not take any. Twenty-one patients took glycerin enemas; 4 patients took suppositories containing sodium bicarbonate and potassium bitartrate during the study.

Bowel program and experimental protocol

In total, 25 subjects received the bowel program described below under two conditions on two different testing days. The two treatment conditions, local anesthetic (lidocaine) lubricant and non-lidocaine jelly (placebo jelly), were double blinded. The interval between the control and lidocaine arms of the study was 7 days. At 15 min prior to each bowel program, the subject emptied the bladder to eliminate possible AD induced by bladder distension. Then the subject was placed in the lateral recumbent position. The cuff of an automated vital sign-recording device (sphygmomanometer BP-203i, Omron Colin Co., Tokyo) was placed around the right arm, and the recording of systolic and diastolic BP as well as HR started after a 5-min acclimatization period. From 10 min before the commencement of the bowel program, systolic and diastolic BP and HR were monitored once every minute. Ten ml of 2% Lidocaine or placebo jelly was instilled into the anal canal. At 5 min after jelly application, the following procedures were performed in the described sequence: insertion of medication into the rectum, digital stimulation, observation of beginning of stool flow and manual removal of the stool. The bowel program was terminated upon completion of stool flow and manual confirmation of rectal emptiness. Systolic and diastolic BP and HR were recorded until 30 min after the end of the bowel program and were simultaneously recorded at same time the next day.

One well-trained nurse who was blinded to the contents of the jelly applied the bowel program for all subjects. Digital stimulation was performed to initiate and augment stool flow. Digital-rectal stimulation with manual removal of stool was often necessary for rectal evacuation. The end of defecation was signaled by cessation of flatus and stool flow, and palpable closure of the internal anal sphincter.[9]

The clinical signs and symptoms of AD investigated in the present study included headache, sweating or flushing above the level of injury, nasal congestion, blurred vision, anxiety or any other unusual symptoms that may have been experienced during previous episodes of AD unrelated to the bowel program.[10] Karlsson[1] described criteria for AD were as follows: increase in systolic BP by at least 20%, combined with at least one of the following symptoms: sweating or chills or cutis anserine or headache or flushing.

In the present study, we stopped bowel maneuvers temporarily when the systolic BP reached 160 mm Hg.

Statistical analysis

The average values of systolic and diastolic BP and HR over a 5-min period before jelly application represented the control values and the average values of these parameters were calculated over each 1-min period of insertion of rectal medication, digital stimulation, observation of stool flow

beginning, and the first manual removal of stool, the end of stool flow, and at 5 min after the end of stool flow, 30 min after the end of stool flow and the same starting time on the next day.

Data were expressed as mean ± s.d. Analysis of variance (ANOVA) for repeated measurements was used for comparison within and between the two treatments. When ANOVA showed significant differences ($P<0.05$), Scheffe's test was used to determine differences between two time periods and between two treatment groups. Differences between lidocaine and placebo regarding stool volume and consistency were determined using the paired *t*-test and Wilcoxon signed-rank sum test, respectively. A *P*-value <0.05 denoted the presence of a significant difference between the two treatment groups. All statistical analyses were performed using SPSS (version 11.5, SPSS Inc., Chicago, IL, USA).

Results

There were no significant differences in the stool volume ($P=0.23$) and consistency ($P=0.71$) between the lidocaine and placebo treatments. Systolic BP increased significantly during manual removal of the stool ($P<0.01$) but returned to the baseline value at the end of stool flow in the lidocaine treatment. However, in the placebo treatment, systolic BP increased significantly during the insertion of rectal medication ($P<0.01$) and was still elevated at the end of stool flow ($P<0.05$); however, it returned to the baseline value at 5 min after defecation. Systolic BP values during the insertion of rectal medication, digital stimulation, beginning of stool flow, manual removal of stool, end of stool flow and 5 min after defecation were significantly lower in the lidocaine treatment group than the respective values of the placebo treatment group ($P<0.01$, $P<0.01$, $P<0.01$, $P<0.01$, $P<0.05$ and $P<0.05$, respectively; Figure 1). The maximal systolic BP recorded in the lidocaine treatment was lower than that of the placebo treatment for all subjects ($P<0.001$).

Diastolic BP (Figure 2) did not change significantly throughout the rectal program in the lidocaine treatment. On the other hand, diastolic BP in the placebo treatment increased significantly during digital stimulation ($P<0.01$) and remained elevated at beginning of stool flow ($P<0.01$) and manual removal of stool ($P<0.01$) but returned to the baseline value at the end of stool flow. The diastolic BP in the lidocaine treatment was significantly lower than in the placebo treatment during the insertion of rectal medication, digital stimulation, beginning of stool flow, manual removal of stool and 5 min after defecation ($P<0.01$, $P<0.01$, $P<0.05$, $P<0.01$ and $P<0.05$, respectively). Thus, the BP of patients with AD crisis during the bowel program (Figure 1) decreased spontaneously at the end of each bowel maneuver.

In both treatment groups, HR did not change throughout the rectal program, although it tended to decrease during digital stimulation, beginning of stool flow and manual removal of stool in both treatment groups (Figure 3).

A total of 10 patients in the placebo treatment group and 4 patients in the lidocaine treatment group reported symptoms of AD (Table 2). Subject 15 developed AD-related symptoms during bowel manipulation in the lidocaine

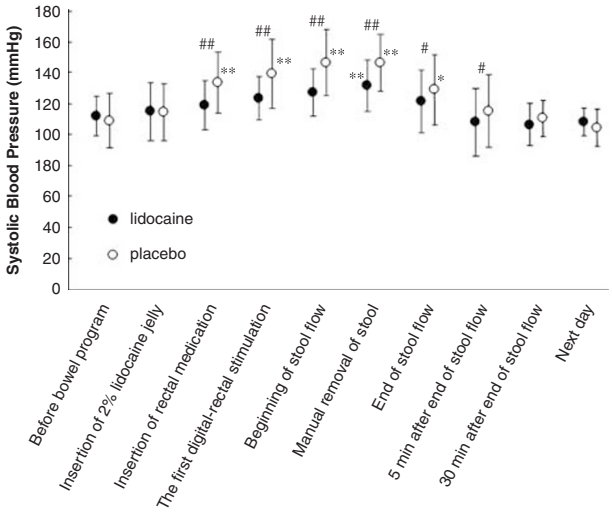

Figure 1 Systolic blood pressure during the bowel manipulation program. Data are mean ± s.d. *$P<0.05$, **$P<0.01$ compared with baseline values. #$P<0.05$, ##$P<0.01$ compared with placebo data.

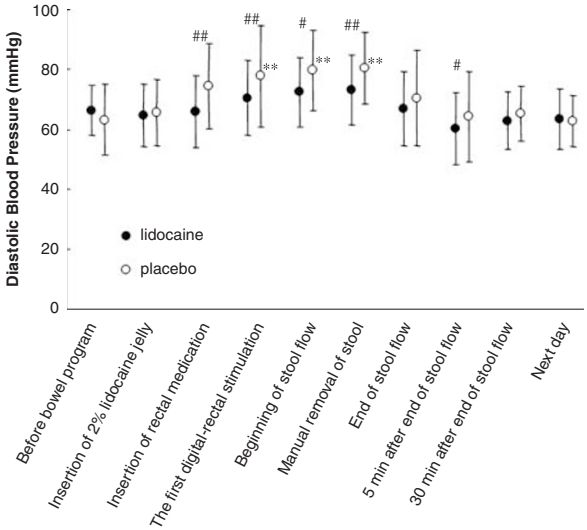

Figure 2 Diastolic blood pressure during the bowel manipulation program. Data are mean ± s.d. **$P<0.01$ compared with baseline values. #$P<0.05$, ##$P<0.01$ compared with placebo data.

treatment session, but no such symptoms were reported by the placebo treatment study.

The Δ changes in systolic BP during rectal manipulation in three subjects (subjects 1, 13 and 15) were higher during the lidocaine treatment session than those of placebo study. Sodium bicarbonate and potassium bitartrate suppositories exert their action by releasing carbon dioxide, which causes gaseous colonic distension, and in turn induces bowel motion. As subjects 4, 13, 20 and 21 used sodium bicarbonate and potassium bitartrate suppositories, the higher delta changes in systolic BP during lidocaine were not related to these medications. For the entire group, however, the maximum rise in systolic BP during the lidocaine study (33.2 ± 14.6 mm Hg) was significantly

($P<0.001$) lower than during the placebo study (50.2 ± 19.5 mm Hg).

Discussion

The present study is the first prospective double-blind study that evaluated the effects of topical anorectal anesthesia for AD during actual bowel manipulation in subjects with SCI. The major finding of this study is that anorectal anesthesia reduced BP during AD induced by bowel manipulation in CSCI. On the basis of our findings, we recommend that patients with CSCI should use lidocaine jelly immediately before bowel programs.

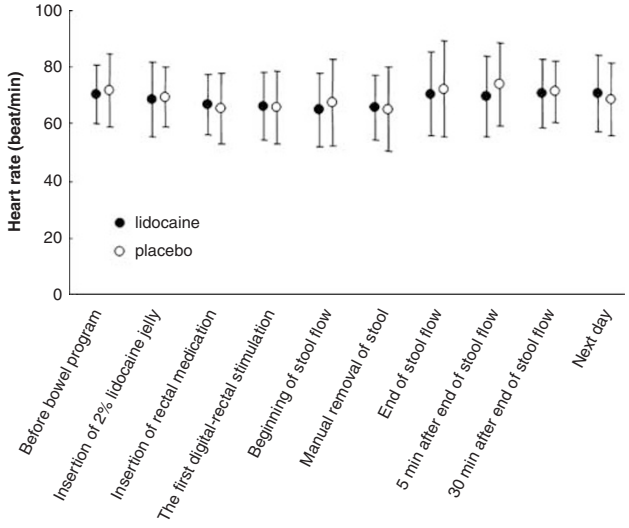

Figure 3 Heart rate during the bowel manipulation program. Data are mean ± s.d.

Cosman *et al*.[6] reported that in patients with SCI at or above T6, instillation of lidocaine jelly into the anal canal did not limit AD during flexible sigmoidoscopy and anoscopy. However, the present study demonstrated that lidocaine jelly attenuated AD responses during bowel program. In our study, a well-trained nurse gently performed the bowel maneuver; therefore, we suggest that the extent of anal and rectal stimulation was less than that achieved in the study of Cosman *et al*.[6] This may explain why lidocaine was effective in our study and not in the above study.

The AD reaction is provoked by peripheral afferent stimulation below the lesion level, which reaches the isolated spinal cord.[1] 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 large intestine is perhaps the most common cause.[1]

In another study, Cosman and Vu[7] suggested that somatic stimulation (anal sphincter stretch) might be more important than visceral stimulation (rectosigmoid distension), because injection of lidocaine blunted the AD response while topical lidocaine anesthesia did not. The results of our study indicate that lidocaine blocks anal and rectal afferents

Table 2 Changes in systolic BP and AD symptoms and signs

Subjects	*ΔChange in systolic BP (mm Hg)*		*% Change in systolic BP (%)*		*AD symptoms*	
	Lidocaine	*Placebo*	*Lidocaine*	*Placebo*	*Lidocaine*	*Placebo*
1	22	10	20	7	None	None
2	30	38	24	29	None	Headache
3	47	62	50	67	None	None
4	45	50	38	38	Headache	Headache
5	26	56	25	52	None	None
6	43	48	52	57	None	None
7	28	30	28	29	None	Flushing
8	49	68	51	60	None	Flushing
9	36	46	34	47	None	None
10	13	38	10	30	None	None
11	22	46	20	42	None	None
12	15	57	13	54	None	Headache, flushing
13	54	48	67	51	None	Flushing, goose bumps
14	13	22	12	22	None	None
15	43	39	49	40	Goose bumps	None
16	8	16	8	16	None	None
17	38	76	40	104	None	None
18	38	77	39	86	None	None
19	33	86	30	116	None	None
20	25	47	21	39	None	Headache
21	46	56	43	50	None	None
22	44	47	41	39	Headache	Headache
23	30	79	27	69	None	None
24	16	41	15	41	None	Headache
25	66	73	63	74	Flushing	Flushing

Abbreviations: AD, autonomic dysreflexia; BP, blood pressure.
ΔChange in Systolic BP = Systolic BP during rectal manipulation−systolic BP at baseline. % Change in Systolic BP = 100 × (Δchange in Systolic BP/systolic BP at baseline).

and blockage of these afferents indirectly contributed to the blunted pressor response.

The present study showed that topical anesthesia minimized the incidence and severity of AD symptoms such as headache, flushing and goose bumps as well as signs of AD (Table 2). Of these symptoms, headache is regarded the consequence of dilation of pain-sensitive intracranial arteries, which has not been shown to correlate with the severity of hypertension.[11] As lidocaine blocks rectal and anal afferents, the indirect consequence of this is reduced afferent contribution to the isolated spinal cord. Therefore, it follows that as the exaggerated sympathetic response is diminished, other autonomic symptoms such as headache and flushing are also reduced.

The present study identified a blunted pressor response (BP increase) but AD still occurred with lidocaine during manual removal of stool. Our results suggest that lidocaine does not completely prevent AD but rather reduces its severity in CSCI individuals. It is noteworthy, however, that subjects 1, 13 and 15 showed higher BP readings following lidocaine application compared with placebo. Therefore, it is incorrect to assume that lidocaine can prevent AD completely in all individuals.

On the basis of findings of the present study, we recommend the use of lidocaine and gentle bowel maneuver to minimize the severity of AD during bowel manipulation involving manual removal of stool in subjects with CSCI, with the exception of those known to be allergic to lidocaine.

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