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ORIGINAL PAPER

# Discharge patterns of abdominal and pudendal nerves during induced defecation in anesthetized cats

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**Abstract** Defecation is thought to be achieved not only by contraction of the colon, but also by a rise in intra-abdominal pressure. In this study we recorded the discharges of nerves innervating the abdominal (Abd) muscles, diaphragm, external anal sphincter (EAS) muscle and pelvic floor (PF) muscles during induced defecation evoked by distention of an expellable balloon to reveal defecation-related muscle activities. The discharges of the Abd muscle and phrenic (Phr) nerves increased when rectal pressure increased. The discharges of the EAS and PF nerves usually increased in proportion to the pressure in the rectum and maintained a constant activity level, although some trials showed inhibition. The results suggest that activities of these muscles increase the intra-abdominal pressure.

**Keywords** Abdominal nerve · Defecation · Intra-abdominal pressure · Pudendal nerve

## Introduction

The abdominal (Abd) muscles consist of the external oblique, internal oblique, transverse abdominis and rectus abdominis and are innervated by motoneurons located in the ventral horn of the lower thoracic and lumbar spinal cord [1]. The Abd muscles play an important role in respiration, especially the control of expiration [2–4], vomiting [3], posture and walking [5–7]. When feces are expelled from the colon during defecation, not only contraction of the colon, but also a rise in intra-abdominal pressure due to contraction of the Abd muscles and diaphragm are thought to be important factors. The Abd muscles contribute to increases of intra-abdominal pressure [3] during various activities, such as speaking, coughing, laughing and weight lifting, which are also associated with increased activity of the external anal sphincter (EAS) muscle [8, 9].

Excretion movements, such as defecation and urination, are one of the functions indispensable to constant maintenance of a living body. These movements are produced mainly by cooperative activity of the hypogastric nerve (sympathetic nerve), pelvic nerve (parasympathetic nerve) and the pudendal nerve (somatic nerve). Defecation is achieved through an interplay of the parasympathetic neurons controlling the smooth muscle of the rectum and the internal anal sphincter (IAS) muscle, and the sacral somatic motoneurons controlling the EAS muscle. It is believed that increased pressure in the colon and relaxation of the IAS and EAS muscles induce defecation. Because inappropriate activation or relaxation of sphincteric muscles results in significant clinical dysfunction, such as fecal incontinence due to non-contraction of EAS muscles [10], abnormal responses of the EAS and IAS due to local anorectal disorders [11], absence of the relaxation of IAS [12] and spasticity and dyssynergia of EAS due to removal of supraspinal control [13], appropriate recruitment of the striated muscles in defecation is very important. The EAS and pelvic floor (PF) muscles are innervated by the

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pudendal nerve, which originates from Onuf's nucleus. Onuf's nucleus extends from the middle *S*1 to the upper *S*2 segments of the spinal cord in cats [14, 15]. EAS motoneurons are localized in the dorsomedial part of the nucleus, and perineal motoneurons are localized in the intermediate zone of the nucleus [14, 16–19].

Defecation is initiated when rectal contents distend the rectum until the stretch receptors in the rectal wall are stimulated [20]. It is thought that the EAS, a striated muscle encircling the anus, is normally in a state of contraction, but relaxes for defecation to permit the evacuation of the rectal contents. Electromyographic studies of the EAS muscle demonstrated the persistence of tonic activity during rest, suppression of activity with colon distention and a phasic response with stimulation of the anus [8, 21]. The fundamental function of some muscles in defecation was studied in humans using electromyogram (EMG) recordings, which also demonstrated the tonic contraction in the normal state and relaxation of the EAS muscles during defecation [8, 22]. However, what role the Abd muscles and diaphragm play during defecation is not known, although these muscles are thought to be important for increasing the intra-abdominal pressure. In this study, we recorded the discharges of nerves innervating the Abd muscles, diaphragm, EAS muscle and PF muscles during induced defecation evoked by distention of the colon with an expellable balloon to reveal defecation-related muscle activities. We recorded nerve discharges rather than EMG since movement artifacts interfere with EMG recordings, especially when feces pass near the EAS muscle.

## Methods

### General procedures

Experiments were performed on 8 adult cats weighing 2.3–3.3 kg. For the initial surgical procedures anesthesia was induced with 2–4 % halothane delivered in a mixture of oxygen and room air in an induction box. The trachea was intubated, and tracheal pressure was monitored through the intubated tube. Animals were maintained with 1–2 % halothane. The femoral artery and cephalic vein were cannulated to monitor blood pressure and to administer drugs, respectively. Arterial blood pressure was maintained at 100–130 mmHg. Subcutaneous temperature was maintained at 37–38 °C by using a heating pad. The level of anesthesia was monitored with pedal withdrawal reflex testing and systemic arterial blood pressure.

The nerves innervating the Abd muscles were dissected free, ligated, cut distally and placed on bipolar cuff electrodes to record nerve discharges. These were the lateral branch of the iliohypogastric nerve, innervating the external oblique muscle, and the medial branch of the iliohypogastric nerve, innervating the internal oblique, transverse abdominal and rectus abdominal muscles. The phrenic (Phr) nerve innervating the diaphragm was also placed on bipolar cuff electrodes. The pudendal nerve divides into three branches: the EAS nerve innervates the EAS muscle, the perineal nerve innervates the PF muscles, and the dorsal nerve of the penis innervates the external urethral sphincter muscle. The EAS and PF nerves were cut and placed on bipolar cuff electrodes (left side).

After the surgery, a mixture of urethane (100 mg/kg) and α-chloralose (50 mg/kg) was administered intravenously while gaseous anesthesia was gradually removed. One animal was immobilized by the intravenous administration of pancuronium bromide (Mioblock; Sankyo, Organon) and was maintained on artificial ventilation adjusted to maintain the end-expired $CO_2$ level at 4–6 %. This animal was used to check the interference of movement artifacts on nerve discharge recording during induced defecation.

All the experimental procedures were approved by the Animal Ethics Committee of Kyorin University and Ibaraki Prefectural University of Health Sciences and were in accordance with the guiding principles for the care and use of animals in the field of physiological sciences of the Physiological Society of Japan.

### Recording of nerve discharges during induced defecation

Defecation was evoked by infusing about 50 ml of warm water into a balloon inserted about 5 cm into the colon through the anus using paraffin liquid. The balloon was made by tying a normal-size condom over a silicone tube. The quantity of warm water was determined by observing suitable rectal pressure. Once water had been poured into the balloon, activities of the Abd, Phr, EAS and PF nerves increased, and the balloon began to be expelled. We considered this to be an induced defecation that was near a normal state. The pressure in the rectum and the activity of each nerve were recorded during induced defecation. In one animal, recordings were also made when the anus was closed with a clamp, preventing the balloon from being expelled. In this condition, the nerves discharge at a higher level, and changes in activity induced by increased intra-abdominal pressure can be more clearly differentiated. In other trials in this animal, the exit of the anus was closed slightly to mimic the condition when the feces are hard. Activity of each nerve was recorded, amplified and band-pass filtered (150 Hz–3 kHz, AB-651 J, Nihon Kohden, Japan), and the pressure within the balloon was also recorded and amplified (AP-601 G, Nihon Kohden, Japan). All signals were saved on a digital recorder (PC-208AX,

Sony, Japan). After the experiment, the data were digitized at 10 kHz (Micro1401mkII, ADC12, Cambridge Electronic Design, Cambridge, UK) for the analyses, and the nerve discharges were full-wave rectified using the rectify function and were integrated using the smooth function with a time constant of 0.01 s with computer software (Spike 2, Cambridge Electronic Design, Cambridge, UK). Baseline nerve activity was taken as the average rate for the 5 s prior to inflation of the balloon. The onset latencies of nerve responses to increasing rectal pressure were calculated as the time from the onset of the pressure increase to the onset of increasing nerve discharges, determined as the time when the integrated activity exceeded the baseline activity by 4 standard deviations. The peak response latencies were calculated as the time from the peak of the rectal pressure to the peak of the integrated nerve discharge. Additionally, the covariation of nerve activity and rectal pressure was documented by cross-correlations using integrated nerve discharges. Data are expressed as mean ± SD. Differences are considered significant at $p < 0.05$ with a Student's $t$ test.

## Result

### Nerve discharges during induced defecation

The activities of the Abd, Phr, EAS and PF nerves were not affected by movement artifacts during induced defecation. Activities recorded in one inactivated animal after injection of pancuronium bromide were not significantly different from those in animals anesthetized with only a mixture of urethane and α-chloralose.

Seventy-four trials of increasing rectal pressure were recorded in 8 cats. Branches of the first lumbar spinal nerve [lateral branch of the iliohypogastric (Abd lat) nerve, medial branch of the iliohypogastric (Abd med) nerve], EAS nerve and PF nerve were recorded in all 8 cats. Branches of the second lumbar spinal nerve were recorded in 3 (the Abd lat and Abd med nerves) or 5 (Phr nerve) cats. When a nerve discharged repetitively during induced defecation, the first onset was measured. Induced defecation weakened over time when evoked continuously. Consequently, successive trials were separated by 10–30 min and checked for discharge return. Trials in which the Abd lat, Abd med, EAS and PF nerves all did not discharge, which occurred occasionally, were excluded.

During induced defecation, activities of the Abd lat nerves were recorded in 94 trials and of Abd med nerves in 95 trials. Nerve activity was facilitated in 60 of 94 (64 %) and 62 of 95 (65 %) trials, respectively. The nerves exhibited no response in the remainder of the trials. Activities of Phr nerves were recorded in 23 trials, with increased discharge in 20 trials and no response in 3. Activities of the EAS and PF nerves were recorded in 74 trials; the nerves were facilitated in 42 of 74 (57 %) and 42 of 74 (57 %) trials, respectively (Table 1). The EAS and PF nerves were inhibited after the rise in rectal pressure in 22 (30 %) and 15 (20 %) trials, respectively (Table 1; Fig. 1b). No response was observed in the remaining 10 (13 %) and 17 (23 %) trials, respectively (Table 1). Representative activity of each nerve during induced defecation is shown in Fig. 1a. Abd nerve discharge increased as the rectal pressure increased, while Phr nerve discharge increased just before the peak of rectal pressure (↙Fig. 1a). Increased discharge of EAS and PF nerves could occur toward the end of the rise in rectal pressure (in 55 and 59 % of 74 trials, respectively), probably to expel feces. Increased discharge of EAS and PF nerves could occur toward the end of the rise in rectal pressure also in trials that showed suppressions (59 % of 22 trials and 93 % of 15 trials, respectively, Fig. 1b).

The mean latencies of nerve discharge to the onset of rectal pressure (Fig. 2) were 3.87 ± 4.41 s (0.18–22.65 s, $n = 56$) for the Abd lat nerves, 4.10 ± 3.78 s (0.38–19.74 s, $n = 62$) for the Abd med nerves, 5.82 ± 4.45 s (−0.14 to 16.12 s, $n = 52$) for the EAS nerves and 4.92 ± 3.80 s (−0.96 to 16.03 s, $n = 56$) for the PF nerves. The onsets for the EAS and PF nerves ranged throughout the period of increasing rectal pressure and often occurred near or later than peak pressure. On the other hand, the onset of Abd nerve activity always occurred within 5 s after rectal pressure started to increase. However, there was no significant difference in the onset latencies of pudendal and Abd nerve activities ($p > 0.05$). The latency of Phr nerve responses, measureable in only 13 trials because of overlapping respiratory activity, was 4.17 ± 2.49 s (1.14–9.07 s, $n = 13$). The activity of the Phr nerves bore a strong resemblance to activity of the Abd nerve.

Latencies of peak discharge relative to peak rectal pressure are shown in Fig. 3. When a nerve discharged repetitively during induced defecation, the peak of the largest burst was taken as the response peak. Mean peak latencies were −0.59 ± 1.49 s (−4.25 to 5.81 s, $n = 56$) for the Abd lat nerves, −0.67 ± 1.45 s (−4.67 to 3.98 s, $n = 62$) for the Abd med nerves, 0.93 ± 3.96 s (−20.41 to 9.65 s, $n = 50$) for the EAS nerves and 0.15 ± 4.87 s (−20.41 to 8.64 s, $n = 46$) for the PF nerves. Peak Abd nerve activities were predominantly before defecation onset, while the peaks of EAS and PF nerve activity tended to be after defecation onset. This difference was significant ($p < 0.05$). The mean peak latency for the Phr nerves was −1.86 ± 3.38 s (−9.73 to 2.82 s, $n = 20$). Peak Phr and Abd nerve activities were very similar.

**Table 1** Nerve activities during induced defecation

| | Facilitation | Suppression | No response | Total |
|---|---|---|---|---|
| EAS nerve | 42 (57 %) | 22 (30 %) | 10 (13 %) | 74 |
| PF nerve | 42 (57 %) | 15 (20 %) | 17 (23 %) | 74 |
| Abd lat nerve | 60 (64 %) | 0 | 34 (36 %) | 94 (L1;72, L2; 22) |
| Abd med nerve | 62 (65 %) | 0 | 33 (35 %) | 95 (L1;73, L2; 22) |
| Phrenic nerve | 20 (87 %) | 0 | 3 (13 %) | 23 |

Seventy-four trials recorded in 8 cats, the numbers of trials (% the rate of the 74 trials), *facilitation* increase of discharge, *suppression* decrease of discharge, *no response* no change

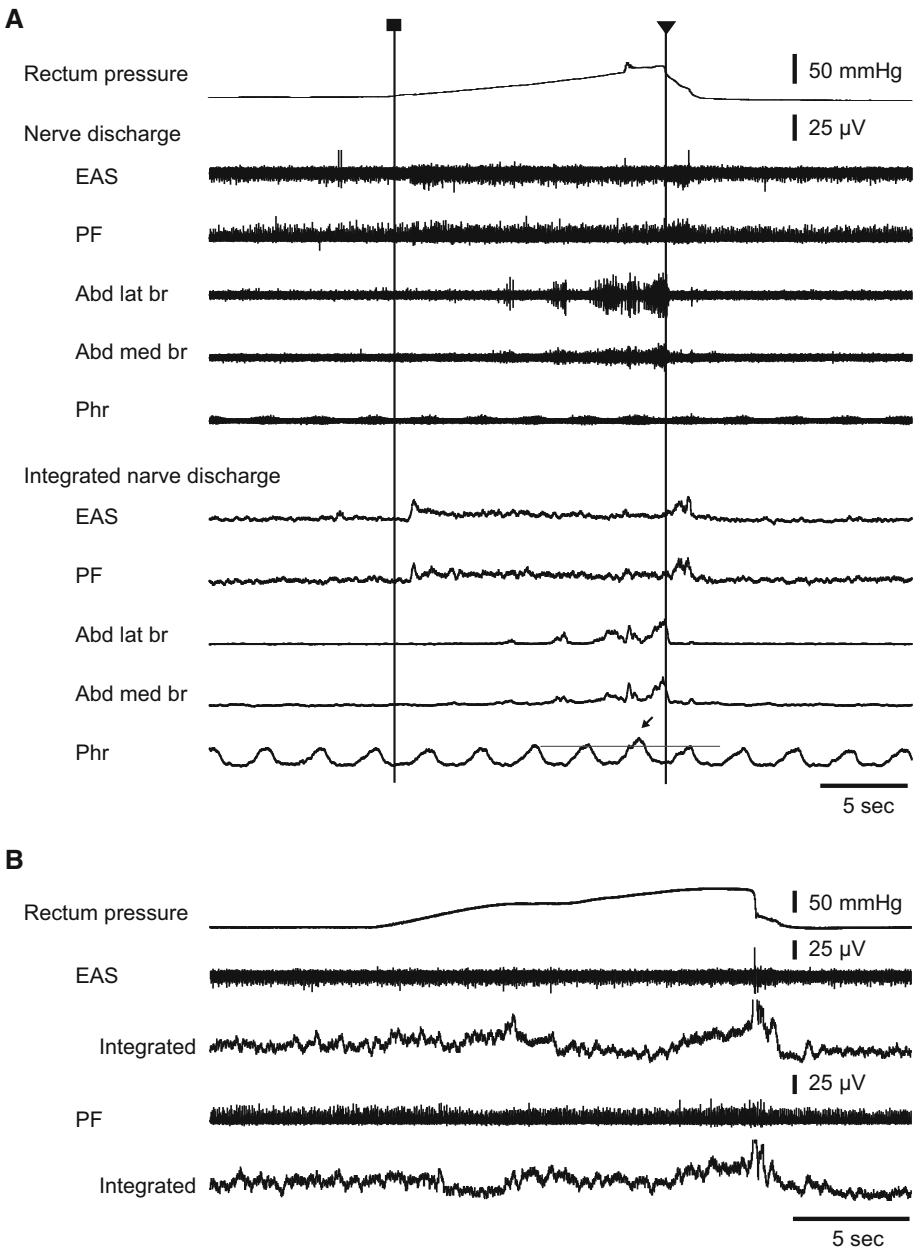


**Fig. 1** **a** Example of nerve discharges and integrated nerve activity during fictive defecation. Increase of intrarectal pressure induces action potentials in branches of the pudendal, abdominal and phrenic nerves. *Filled square* indicates the onset of the pressure rise in the rectum; *filled triangle* indicates the time of the peak rectal pressure;↙ indicates the change of Phr nerve discharge. **b** Example of EAS and PF nerve activity showing inhibitions during the increase of rectal pressure and showing increased discharges at the end of the increase of rectal pressure

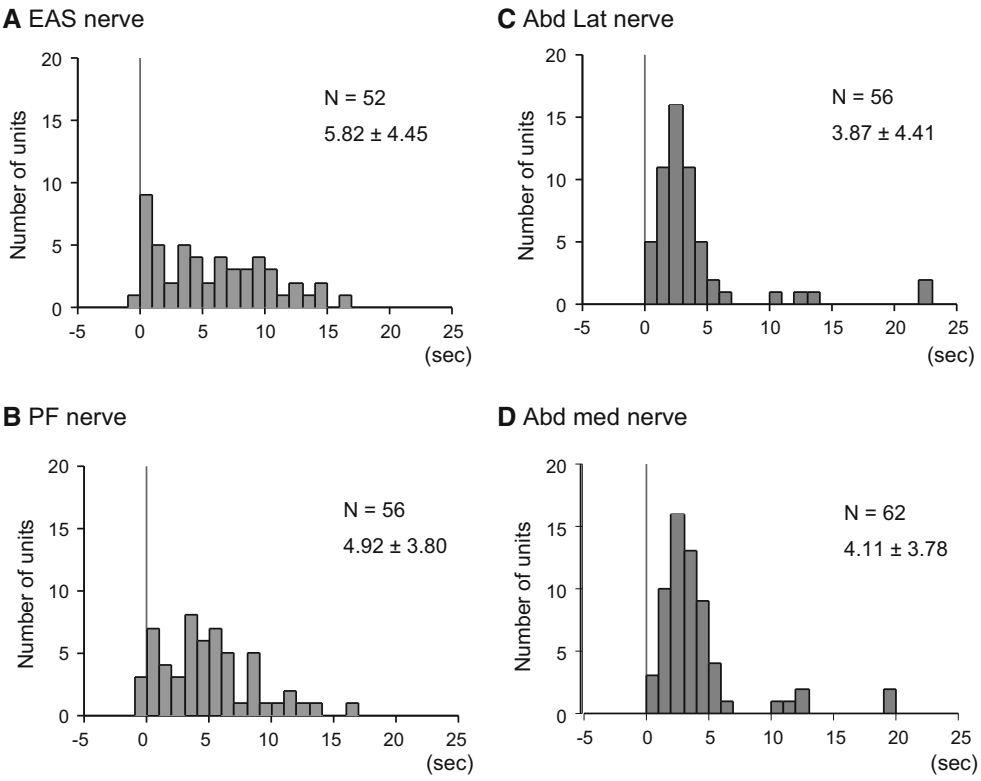


**Fig. 2** Time between the onsets of discharges in the 4 nerves and the onset of the pressure rise in the rectum (*filled square* in Fig. 1). **a** EAS nerve, **b** PF nerve, **c** Abd Lat nerve, **d** Abd med nerve. The size of the bin was 1 s. The *vertical lines* indicate the number of units per 1 s. The *horizontal lines* indicate the latencies of nerve discharge to the onset of rectal pressure. The *zero lines* indicate the onset of rectal pressure. *n* number of units; numbers are mean ± SD

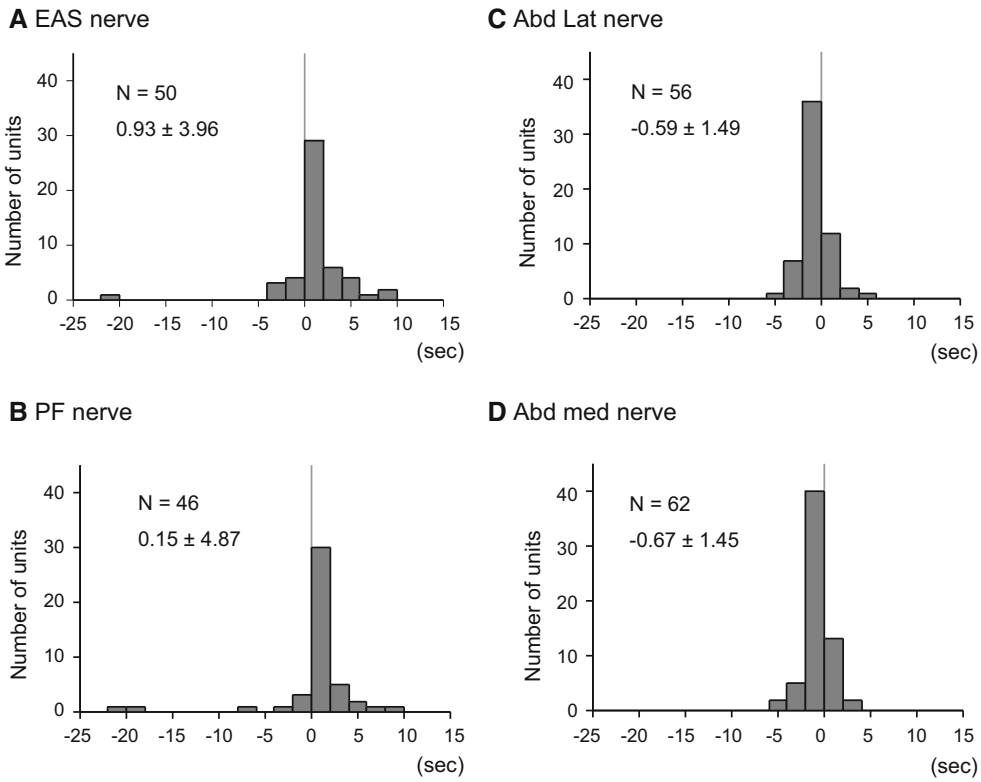


**Fig. 3** Time between the response peaks of the 4 nerves and the peak of the pressure rise in the rectum (*filled triangle* in Fig. 2). **a** EAS nerve, **b** PF nerve, **c** Abd Lat nerve, **d** Abd med nerve. The size of the bin was 2 s. The *vertical lines* indicate the number of units per 2 s. The *horizontal lines* indicate the latencies of nerve discharge to the onset of rectal pressure. The *zero lines* indicate the onset of rectal pressure. *n* number of units; numbers are mean ± SD

Nerve discharge during interruption of defecation

In order to examine the activities of Abd, Phr, EAS and PF nerves more precisely, the anus was closed so that the balloon could not be expelled. Five trials were performed on one cat after the completion of the induced defecation trials described above. Representative activity of each nerve during induced defecation with the anus closed is shown in Fig. 4a. The mean response latencies were 0.12 ± 0.28 s (−0.16 to 0.52 s, $n = 5$) for the Abd lat nerve, −0.12 ± 0.19 s (−0.39 to 0.11 s, $n = 10$) for the Abd med nerve, 0.46 ± 0.52 s (−0.10 to 1.09 s, $n = 5$) for the Phr nerve, 0.28 ± 0.24 s (0–0.64 s, $n = 5$) for the EAS nerve and 0.35 ± 0.24 s (0–0.64 s, $n = 5$) for the PF nerve. There was no significant difference between the onsets of the Abd nerves, Phr nerve and pudendal nerves ($p > 0.05$).

The mean peak latencies of nerve discharge were −0.21 ± 0.14 s (−0.46 to −0.06 s, $n = 8$) for the Abd lat

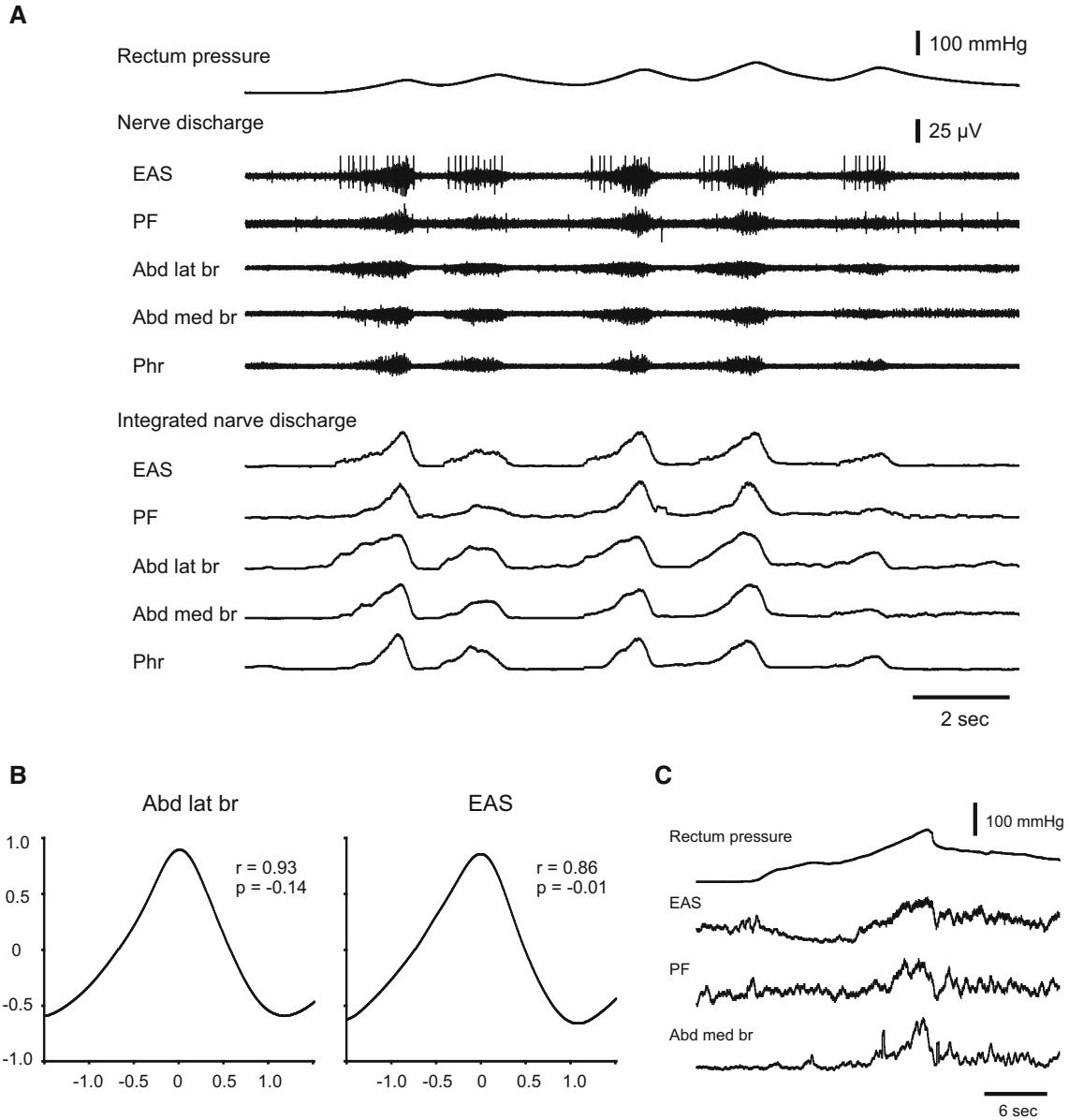


**Fig. 4** **a** Nerve discharges and integrated nerve activity during a trial when the anus was closed. The peaks of the pudendal and abdominal nerve activities gather just before the peak times of rectal pressures. **b** Cross-correlations between the integrated nerve activity and rectal pressure. *Left* Cross-correlation between the Abd lat nerve and rectal pressure; *right* cross-correlation between the EAS nerve and rectal pressure. *r* coefficient of correlation; *p* delays of the peak values between the nerve discharges and the pressure signal. **c** Integrated nerve activity when the exit of the anus was closed slightly. Since Abd lat br and Phr discharges resembled those of Abd med br, they were omitted

nerve, −0.28 ± 0.14 s (−0.51 to −0.04 s, $n = 10$) for the Abd med nerve, −0.28 ± 0.14 s (−0.48 to −0.13 s, $n = 5$) for the Phr nerve, −0.13 ± 0.10 s (−0.28 to −0.01 s, $n = 5$) for the EAS nerve and −0.18 ± 0.12 s (−0.29 to −0.04 s, $n = 5$) for the PF nerve. The peak times of the Abd, Phr and pudendal nerve were not significantly different ($p > 0.05$). The peak of the Abd, Phr and pudendal nerve activities tended to occur just before the peak rectal pressure.

In order to document the covariation of nerve activity and rectal pressure, cross-correlations between the integrated nerve discharges and pressure signal were calculated (Fig. 4b). The peak values were 0.70 ± 0.15 (0.51–0.93, $n = 6$) for the Abd lat nerve, 0.70 ± 0.17 (0.48–0.91, $n = 5$) for the Abd med nerve, 0.68 ± 0.09 (0.56–0.77, $n = 3$) for the Phr nerves, 0.70 ± 0.11 (0.60–0.86, $n = 3$) for the EAS nerve and 0.69 ± 0.11 (0.61 to −0.85, $n = 3$) for the PF nerve. All cross-correlations had high correlation coefficients. The delays of the peak values were −0.29 ± 0.15 s (−0.48 to −0.06, $n = 6$) for the Abd lat nerve, −0.27 ± 0.22 s (−0.64 to 0, $n = 5$) for the Abd med nerve, −0.27 ± 0.12 s (−0.39 to −0.10, $n = 3$) for the Phr nerve, −0.25 ± 0.20 s (−0.49 to −0.01, $n = 3$) for the EAS nerve and −0.23 ± 0.19 s (−0.48 to −0.01, $n = 3$) for the PF nerve. The delays of the peak values for the Abd, Phr and pudendal nerves were not significantly different ($p > 0.05$). Action potentials of the Abd nerve, Phr nerve and pudendal nerves seem to be initiated reflexively by increases of rectal pressure.

The time course of nerve activity during induced defecation was studied when the exit of the anus was partially closed. Three trials were performed. Representative activity of each nerve is shown in Fig. 4c. The EAS nerve was inhibited at the beginning of the rise in rectal pressure followed by a simultaneous increase in the activities of the Abd, PF and EAS nerves as rectal pressure approached the peak.

## Discussion

### Nerve discharges during induced defecation

Activities of the abdominal (Abd), phrenic (Phr), external anal sphincter (EAS) and pelvic floor (PF) nerves were recorded during induced defecation, and the relative timing of increased rectal pressure and increased nerve discharges was measured. Abd and Phr nerve discharges increased as the rectal pressure increased in 64 and 87 % of trials, respectively,. Co-contraction of the Abd muscles and diaphragm should increase the intra-abdominal pressure. Abd and Phr nerve discharges tended to be before the peak of rectal pressure, whereas the peak activities in the EAS and PF nerves were mostly after peak rectal pressure. Raised rectal pressure was always accompanied by increased activities of the EAS and Abd muscles. The EMG of the Abd muscles exhibited increases in the early phase of defecation and that of the EAS and PF showed increases in the later phase of defecation [8]. It is likely that when rectal pressure increases, the Abd muscles and diaphragm, EAS muscle and PF muscles temporally cooperate to raise the intra-abdominal pressure and assist in the expulsion of feces.

The EAS nerve discharged tonically before and after the rise in rectal pressure, with a phasic facilitation during the pressure increase. The EAS muscle, an orbicular striated muscle located in the distal colon and innervated by the pudendal nerve, acts to close the anal canal. The motor fibers of the right and left halves of the EAS muscle have an overlapping spatial distribution. The tonic discharge from the EAS muscle is due to a spinal reflex with afferent and efferent impulses conducted via the pudendal nerve. Unilateral transection of the pudendal nerve does not abolish the tonic discharge from any portion of the sphincter [21]. In this study, the pudendal nerve on one side of the animal was cut and placed on a bipolar electrode, and the nerve on the other side was intact. Therefore, the record of nerve activity was little influenced. Induced defecation weakened gradually when evoked continuously, and trials in which all the Abd lat, Abd med, EAS and PF nerves did not discharge occurred occasionally. This seems to be caused by an adaptation of the reflex since they happened after repeated trials, and the adaptation seems to be different for each muscle.

The EAS activities were inhibited during defecation in cats and humans [8, 12, 21, 23]; however, the EAS nerve activities were inhibited in 30 % of trials in our study. The slightest contact of the finger on the anal margin [8] and the insertion and removal of a glass probe from the anal canal [26] produced an immediate burst of activity. We suggest that this activity of the EAS nerve might contribute to the maintenance of the circular shape of the anal canal during passage of feces and resize the feces to allow an adequate volume to pass. Because the anal canal is a bit narrower than the rectum, it would hyperextend during defection, and soft tissues might be injured if the EAS muscle was always completely relaxed. It may be that when the abdominal muscles contract and abdominal pressure increases, a defecation reflex is induced, the IAS muscle loosens, and the EAS muscle is inhibited or adjusts the anal canal according to the state of its contents.

### Nerve discharge during interrupting defecation

When the anus was closed, the activities of the Abd, Phr and pudendal nerves increased as the rectal pressure went

up, and there was no significant difference between the time of the nerves' onsets or peak activities. The integrated discharges of all nerves were highly correlated with the rectal pressure. Activities of the Abd, Phr and pudendal nerves seem to be initiated reflexively as increased rectal pressure stimulates mechanoreceptors in the rectal wall. In order to observe the activity of the Abd, Phr, and pudendal nerves as the balloon was slowly expelled, the exit of the anus was closed slightly. In this case, the EAS nerve discharge was inhibited as the rectal pressure began to rise and then resumed as the pressure reached its peak. The activity of the Abd, Phr and PF nerves simultaneously increased toward the peak of pressure. These data strongly support our view that the co-activity of the Abd muscles, diaphragm and PF muscles raises the intra-abdominal pressure. When the exit of the anus was partially closed to mimic the condition when the feces are hard, the EAS nerve was inhibited. When the feces is relatively hard, the EAS muscle may be inhibited. When a defecation reflex is induced, the EAS muscle may be inhibited or activated according to the state of its contents. Bishop et al. indicated that the activity of the EAS muscle was inhibited by distension of the colon in decerebrate cats [2, 23]. Concerning having had little inhibition of the EAS during induced defecation in our study, the flexibility of the balloon might have been too soft to induce the inhibition. Urethane and α-chloralose anesthesia might also have influenced the activity of the EAS muscle. The inhibitory response of the EAS might be blocked under the influence of anesthesia.

## Summary

We recorded the discharges of nerves innervating the abdominal (Abd) muscles, diaphragm, external anal sphincter (EAS) muscle and pelvic floor (PF) muscle during induced defecation evoked by distention of an expellable balloon to reveal defecation-related muscle activities. The discharges of the Abd muscle and phrenic (Phr) nerves increased when rectal pressure increased. The discharges of the EAS and PF nerves increased in proportion to the pressure of the rectum, and some trials showed inhibition. When the anus was closed, the activities of the Abd, Phr, EAS and PF nerves increased as the rectal pressure rose. Abd and Phr nerve discharges tended to be before the peak of rectal pressure, whereas the peak activities in the EAS and PF nerves were mostly after peak rectal pressure. It is likely that when the rectal pressure increases, the Abd muscles and the diaphragm, EAS muscle and PF muscles temporally cooperate to raise the intra-abdominal pressure and assist with expulsion of feces. When the exit of the anus was closed slightly to mimic the condition when the feces are hard, EAS nerve discharge was inhibited, and the Abd, Phr and PF nerves discharges increased simultaneously. The results suggest that the co-activity of the Abd muscles, diaphragm and PF muscles raises the intra-abdominal pressure. When a defecation reflex is induced, the EAS muscle may be inhibited or activated according to the state of its contents.

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