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ORIGINAL ARTICLE

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Suppressive mechanism of gastric motility by whole-body vibration

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Abstract ***Objective:*** To investigate the mechanism of gastric motility suppression by exposure to whole-body vibration (WBV). ***Methods:*** The gastric motility was evaluated by electrogastrography (EGG) under food intake and autonomic nerve blocking agents in ten healthy volunteers. Sinusoidal vertical vibration with a frequency of 4 Hz (1.0 ms^{-2} rms) was given to the subject for 10 min. ***Results:*** The amplitude of EGG wave and the power spectrum corresponding to the slow wave component was remarkably decreased by vibration exposure. Food intake enhanced the gastric motility about 2.5-fold in the power spectral density. During and after vibration exposure, the response mode was similar to those at fasting states. Under the influence of anticholinergic (scopolamine) and alpha-adrenergic blocking agents (prazosin), the power spectra were decreased. A further decrease was observed during vibration exposure. A beta-adrenergic blocking agent (propranolol) led to a marked increase in the amplitude of EGG and its power spectrum. With pretreatment by a beta-adrenergic blocking agent, however, vibration exposure reduced both of them. ***Conclusions:*** These results suggest that short-term exposure to WBV can suppress the gastric myoelectric activity, the responses on which may be mediating by neurohumoral effects as well as the mechanical effect of WBV.

Key words Electrogastrography (EGG) · Food intake · Gastric motility · Whole-body vibration

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Introduction

An increased prevalence of gastrointestinal symptoms in workers with whole-body vibration (WBV) exposure has been reported in some epidemiological studies (Seidel and Heide 1986; Miyashita et al. 1992). It is an interesting problem whether exposure to WBV is a specific risk factor for gastric disorders such as gastric neurosis and nonulcerative dyspepsia. These are functional disorders associated with abnormalities of gastric motility (Talley et al. 1994). Although there are a few experimental studies on acute exposure to WBV concerning the human gastric motility in healthy subjects, the findings have been inconsistent (Kjellberg and Wikström 1987; Ishitake et al. 1998). The responses of the gastric motility to WBV exposure remain unclear.

The gastric slow motion, namely peristalsis, originates from the corpus of the stomach and propagates through the longitudinal muscle fibers to the pylorus. Cutaneous electrogastrography (EGG) measures the electric activity of gastric smooth muscles and also is a potentially useful and noninvasive technique for evaluating the gastric motility. Physiologically, the gastric electric activity consists of the pacemaker potential (electric control activity) and the spike potential (electric response activity) (Guyton 1991). The spike potentials by the contraction of muscles of the antrum are superimposed on the pacemaker potential in a cutaneous EGG measurement. The indices of the frequency components and their power spectra provide reliable information about the gastric motility. The human gastric signals are divided into three components: bradygastria (0.5–2.0 cpm), slow wave (2.0–5.0 cpm), and tachygastria (5.0–9.0 cpm) (Chen and McCallum 1993). In particular, the change of the power density in the slow wave component may reflect the contractile activity of the stomach (Smout et al. 1980; Chen et al. 1994). The EGG has been widely used as a diagnostic method for functional disorders associated with abnormalities of the gastric motility (Geldof et al. 1987; Cucchiara et al.

1992; Jebbink et al. 1995; Parkman et al. 1997). In our recent experiments, brief exposure to WBV suppressed the slow wave component (Ishitake et al. 1998). The aim of this study was to clarify the possible mechanism of the gastric motility suppressed by short-term exposure to WBV by means of cutaneous EGG.

Materials and methods

Subjects and study protocol

Ten male healthy volunteers participated in this study. They had no history of relevant disorders of the gastrointestinal tract, including problems of digestion. The mean age was 22.4 ± 1.7 (SD) years (range 21–26 years) and the mean body mass index was 24.0 ± 2.6 (SD) (range 20.5–28.7). Prior to the study, the experimental procedures were carefully explained to all the subjects, and informed written consent was obtained from them. The subjects were asked not to eat and drink after a regular lunch. The mean fasting time was 4 h. The EGG was continuously recorded before, during and after vibration exposure for 10 min in each subject. Each subject was tested in two stomach conditions: in fasting state and filled with a regular solid meal (80 g, 135 cm^3).

These experimental procedures were carried out on different days for each subject. Three agents for autonomic nerve blocking (scopolamine butylbromide 10 mg; prazosin hydrochloride 1 mg; propranolol hydrochloride 20 mg) were administered orally to two subjects each on different experimental days. Sixty minutes after the administration, exposure to WBV was commenced.

During the experiments, the noise level induced by the electromagnetic shaker was 64–66 dB(A). The ambient temperature was maintained at 23 °C–25 °C.

Vibration exposure

The subjects were asked to sit on a hard flat seat without a backrest. Each individual subject chose a comfortable posture. The vibration stimulus was produced by using an electromagnetic shaker (ASE-385; Akashi, Japan). A sinusoidal vibration with a frequency of 4 Hz and an acceleration magnitude of 1.0 ms^{-2} (rms) was applied to the subjects for 10 min.

Measurement of EGG

After gentle abrasion of the skin to enhance the electrical conduction, two disposable Ag/AgCl electrodes (Vitrode, Nihon Kohden, Japan), 6 cm apart horizontally, were placed half way between the xiphoid and the umbilicus. A reference electrode was affixed to the right upper quadrant of the abdomen. The EGG signals were amplified with a pre-amplifier (AB621-G, Nihon Kohden, Japan). The time constant was 5 s. The high-frequency cutoff was set at 0.2 Hz to minimize interference from nongastric signals. The EGG signals were simultaneously digitized at 2 Hz by an analog-to-digital converter and filtered to remove noises of frequency more than 9 cycles/min (cpm) and less than 0.9 cpm.

Data analysis

The frequency analysis of EGG was done using fast Fourier transform (FFT). Power spectrum was calculated every 10 min at different recording periods. Two EGG parameters were used for evaluation: (1) the dominant frequency of the EGG, which may indicate a peak frequency; (2) the relative powers (%) of the slow wave component (2–5 cpm) and tachygastria component (5–9 cpm) were divided by the sum of the powers from 0.9 to 9 cpm. Components of slow wave and tachygastria were expressed as a percentage of total power.

Statistics

Data are expressed as medians (25%, 75%) because of the lack of normality in the distribution. The data of different recording periods for vibration exposure in the same subject were statistically compared using the Wilcoxon signed-rank test. Differences were considered to be significant at $P < 0.05$.

Results

Figure 1 shows a representative example of EGG waveform and its power spectrum at fasting state at the different recording periods before, during, and after vibration exposure. Before vibration, two peaks in the power spectral density were observed. The major component was around 3 cpm in frequency, the so-called slow wave component. The small and fast wave component was the frequency of 5–9 cpm, so-called tachygastria. All subjects showed a dominant slow wave component with the frequency of 2–5 cpm. During vibration exposure, the amplitude of EGG waveform of both components was associated with a remarkable decrease of power spectrum. When vibration exposure ceased, the amplitudes of EGG and its power spectrum around the slow wave markedly increased.

A modification of the gastric motility under the influence of food intake and vibration exposure was observed (Fig. 2). Food intake enhanced the gastric motility, showing a dominant increase of the amplitude in EGG waveform and the slow wave of its power spectral density. During and after vibration exposure, the response modes were similar to those at fasting state. However, the responses due to vibration exposure were larger than those at fasting state. Table 1 summarized the effect of WBV on EGG at the conditions of fasting and food intake. At both conditions, the relative powers (%) of the controls were almost same. Food intake produced a significant increase in the relative power of the slow wave and a significant decrease of the tachygastria component. The total power of EGG increased about 2.5 times (median). After food intake, the effects of vibration exposure were almost the same as in the fasting state. During vibration exposure, the relative power of tachygastria in the fasting state was significantly greater than in the food intake condition. In contrast, the relative power of the slow wave at fasting state was significantly smaller as compared to that at food intake condition.

Figure 3 shows typical responses to WBV exposure under the influence of various drugs. Pretreatment with an anticholinergic (scopolamine) and with an alpha-adrenergic blocking agent (prazosin) resulted in a decrease in the power spectra. In contrast, a remarkable increase in the power spectrum was observed after the administration of a beta-adrenergic blocking agent (propranolol). Under the influence of these drugs, the exposure to WBV caused a decrease in both components of the power spectrum following food intake. After exposure, the responses were almost the same in the three drug conditions.

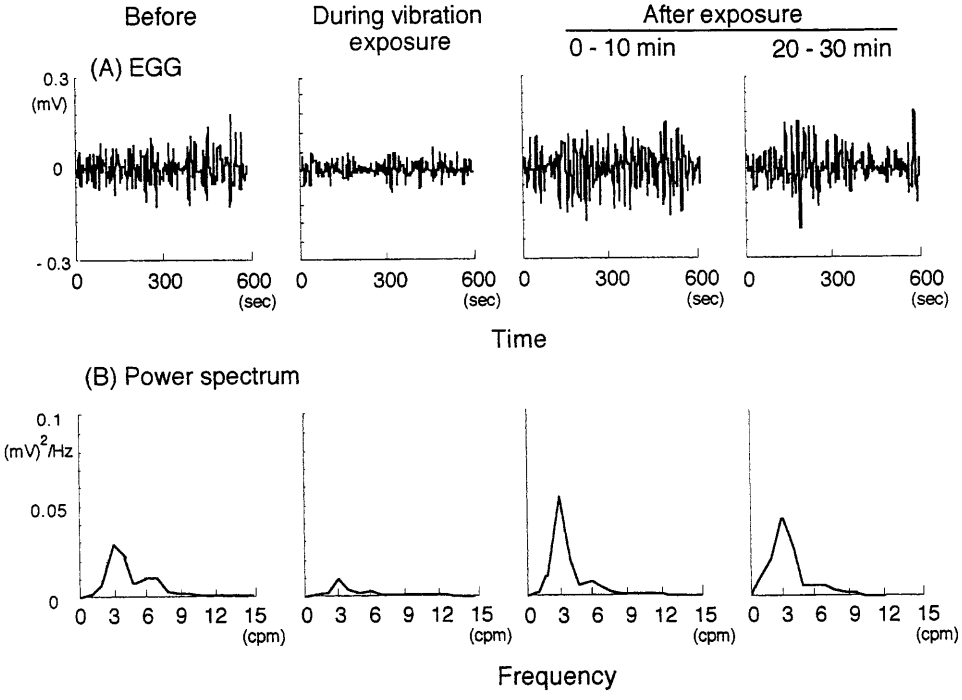


Fig. 1 A representative case of changes in electrogastrograms (EGG) (**A**) and their power spectra (**B**) on acute exposure to whole-body vibration in fasting state

Discussion

Brief exposure to WBV (4 Hz, 10 min) can reduce the power spectral density of the slow wave component of EGG, suggesting suppression of the gastric motility (Smout et al. 1980). Even a higher WBV frequency with short-term exposure (10 Hz, 5 min) reduced the power density of the slow wave component (Ishitake et al. 1998). Kjellberg and Wikström (1987), however, observed a biphasic phenomenon with an initial increase and thereafter a gradual decrease in the power density with the frequencies of 3, 5.4 and 7.8 cpm by WBV of 3 Hz. They suggested that the gastric motility was affected by WBV. The difference between our findings and

Fig. 2 The changes of EGG (**A**) and their power spectra (**B**) under the influence of food intake and acute exposure to whole-body vibration

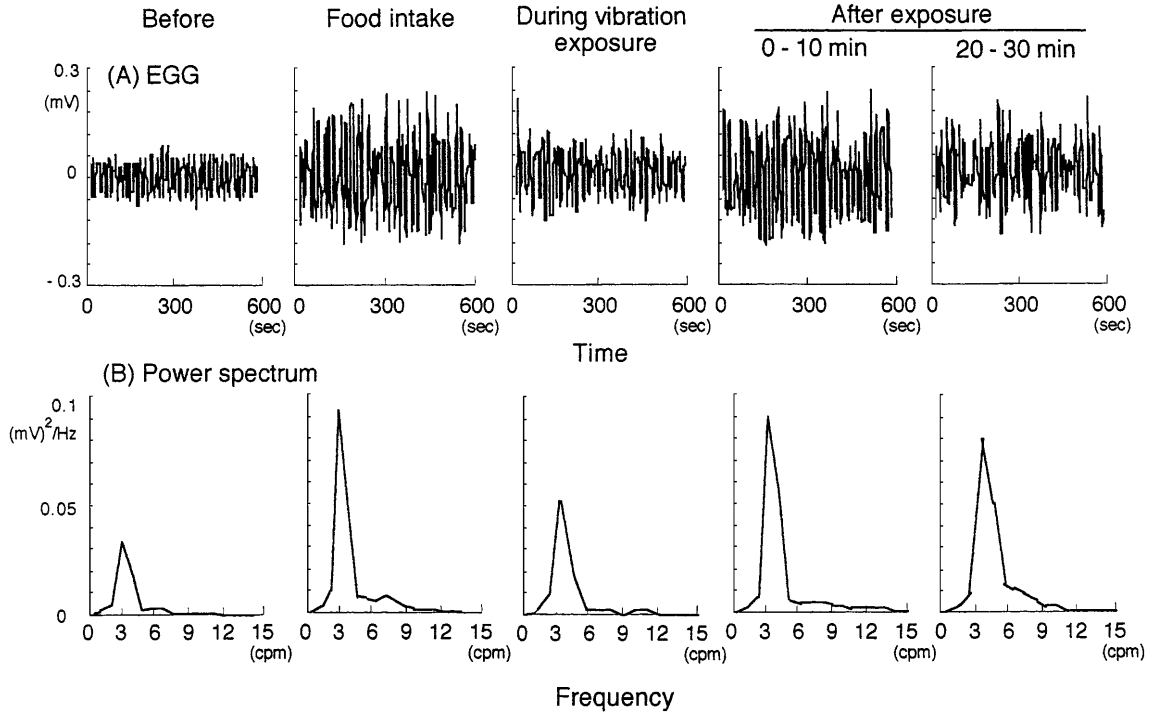


Table 1 Effect of whole-body vibration on EGG in fasting state and following food intake. Relative power indicates the proportion of total power comprised by each component. Total power is calculated as a percentage of the control. The values are medians (25%, 75%)

Condition	Control	Food intake	During vibration	After vibration
Fasting state				
Dominant frequency (cpm)	3.3 (2.8, 3.3)	–	2.8 (2.6, 3.0)	2.8 (2.8, 3.3)
Relative power (%)				
Slow wave	70.0 (60.2, 73.9)	–	64.8 (55.9, 73.5)	71.8 (62.5, 72.9)
Tachygastria	17.5 (12.6, 24.6)	–	25.0 (17.2, 32.7)	16.2 (13.1, 21.0)
Total power (%)	100.0	–	80.5 (65.0, 97.0)	104.1 (60.0, 140.5)
Food intake				
Dominant frequency (cpm)	2.8 (2.8, 3.3)	3.3 (2.8, 3.3)	2.8 (2.8, 3.3)	3.3 (3.3, 3.3)
Relative power (%)				
Slow wave	73.2 (64.0, 78.7)	85.1 (70.8, 86.9)	79.5 (70.5, 84.8)	88.3 (76.0, 90.3)
Tachygastria	19.0 (15.1, 27.1)	10.3 (5.8, 18.0)	15.0 (8.2, 20.0)	11.2 (7.5, 15.0)
Total power (%)	100.0	255.6 (130.5, 363.0)	192.0 (105.0, 332.0)	253.6 (137.0, 354.0)

*$P < 0.05$, $n = 10$

theirs may depend on the experimental conditions of the stomach contents, with or without food intake. An increase in the amplitude of EGG after eating has been observed by many investigators (Chen and McCallum 1991; Levanon et al. 1998). Although they carried out their experiments with no control of food intake or fasting time, these two factors were strictly controlled in our experiment. There is a possibility of misinterpretation because of confused data at different conditions of food intake. Possible artifacts such as body movements and startle reflex should also be considered because there were no raw EGG waveform data in Kjellberg's report. According to our preliminary study, some artifacts due to body movement can actually produce an increase in the power spectrum of the slow wave component.

Food intake is, of course, a very important influence on gastric motility. It is widely recognized that a good correlation is noted among increased amplitude, frequency of slow wave and gastric contraction, resulting from the increase of the contractile activity due to eating (Sun et al. 1995). Similar responses modes were observed during exposure to vibration under the conditions of fasting state and food intake. The changes in relative power of slow wave at the condition of food intake were greater than in the fasting state. It is suggested that a suppressive effect of vibration exposure on gastric motility may be enhanced under the condition of food intake.

With respect to a mechanism suppressing gastric motility, we should consider the neurohumoral and mechanical factors which mainly regulate gastric motility (Guyton 1991). The neural control is provided by the autonomic nervous system, which includes both cholinergic and adrenergic nerve fibers. Stimulation of the parasympathetic nerves leads to an increase in the activity of the gastric motility. On the other hand, stimulation of the sympathetic nerves inhibits gastric motility. The slow wave component may totally reflect the gastric electrical activity, including the muscle tone and contractile activities (Smout et al. 1980; Chen et al. 1994). From the pharmacological viewpoint, an anticholinergic agent can suppress the activity of muscle contraction. An alpha-adrenergic blocking agent may suppress the contraction of pylorus ring, and also a beta-adrenergic blocking agent would increase the muscle tone (Goodman Gilman et al. 1990). These pharmacological actions resulted in the suppression of the amplitude of EGG and its power spectra by the anticholinergic (Imai et al. 1998) and alpha-blocking agents, and the beta-blocking agent induced increases in the same parameters in the present study.

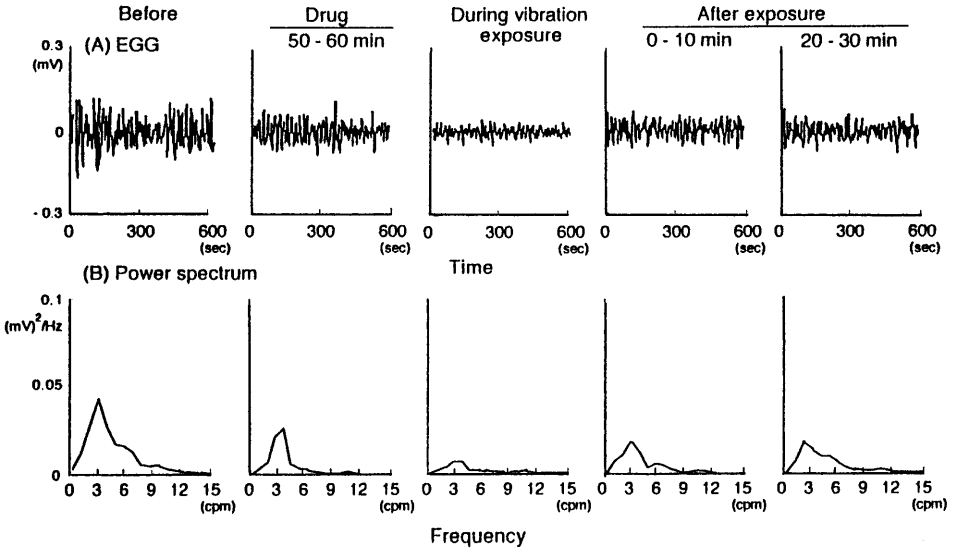
Exposure to WBV produced a further reduction in the activity of the slow wave component following pretreatment with these drugs. The reduction of slow wave component by vibration may be due to not only the autonomic nervous system but also humoral and mechanical factors. As for the humoral factors, the control of the secretion of gastric juice is stimulated by gastrin and the parasympathetic nervous system. Gastrin released from the pyloric glands can enhance gastric motility and stimulate hydrochloric acid secretion. Parasympathetic stimulation increases the secretion of acid, mucus and pepsinogen by the action of acetylcholine. Tactile stimulation of the surface of the stomach mucosa can also influence the gastric secretion. Exposure to WBV may have a mechanical effect on the stomach wall, leading to increased gastric secretion while gastric motility is not affected (Dupuis and Christ 1966). Suppression of the secretion of gastric acid by an H_2 receptor antagonist is associated with inhibition of gastric motility (Parkman et al. 1998). As there are few convincing data about a relationship between exposure to WBV and gastric secretion, further investigations should be carried out.

The movement of organs are increased by WBV with frequencies of 3–5 Hz and 7–10 Hz (Dupuis and Zerlett 1986). This suggests that passive gastric movement by

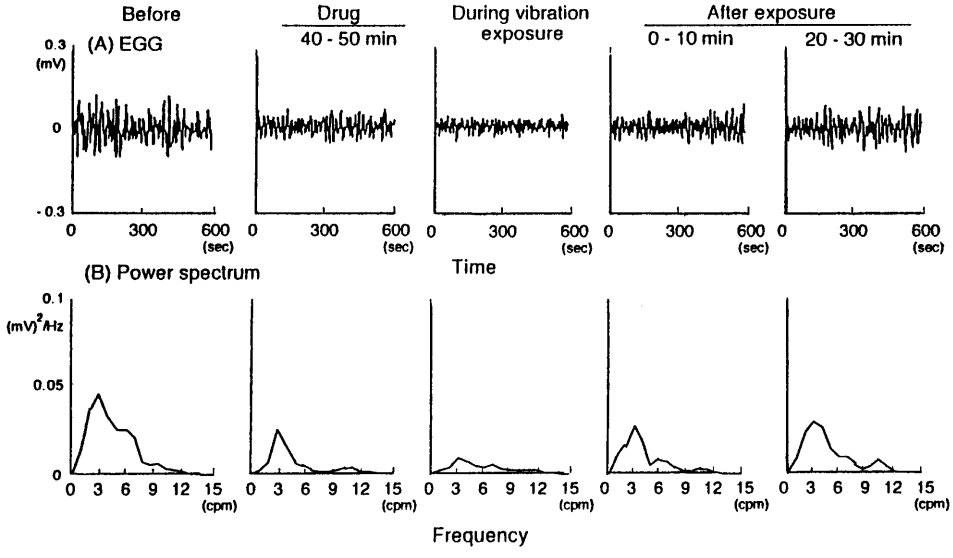
▶

Fig. 3 The changes of EGG (**A**) and their power spectra (**B**) under drug intervention (*1* anticholinergic agent, *2* alpha-blocking agent, *3* beta-blocking agent) and exposure to whole-body vibration

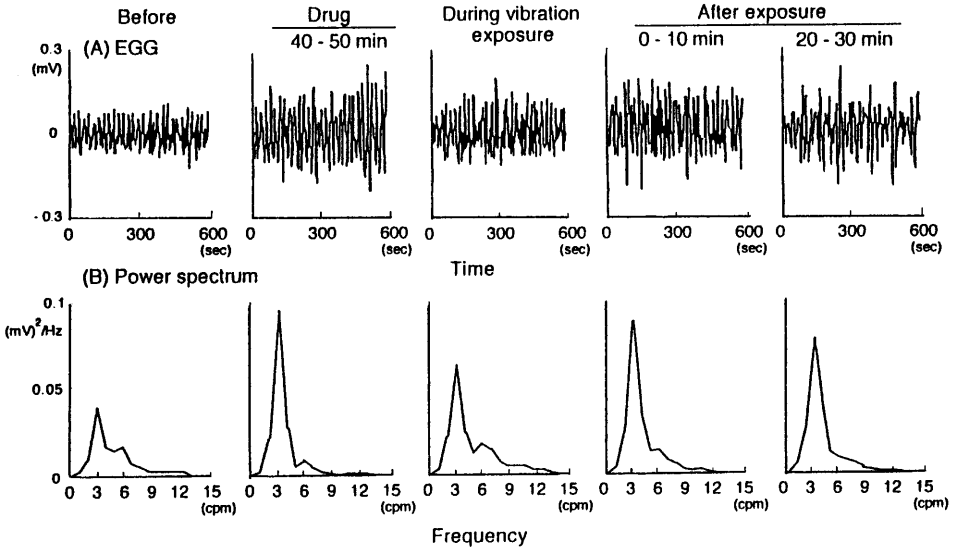
(1) Anticholinergic agent (scopolamine, 10mg)



(2) Alpha - blocking agent (prazosin, 1mg)



(3) Beta - blocking agent (propranolol, 20mg)



WBV reaches a maximum around resonance frequencies and produces some excitatory effects on gastric motility and secretion. Although no subject complained of discomfort during WBV exposure in the present study, we should consider the effect of the resonance frequency on gastric motility, which the biodynamic transmission of vibrations depends on. In conclusion, acute exposure to WBV may suppress the activity of contractile smooth muscle. The responses may be mediated by neurohumoral effects as well as the mechanical effect.

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