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Review

Gastrointestinal motility disorders and acupuncture

Jieyun Yin, Jiande D.Z. Chen *

Division of Gastroenterology, Department of Internal Medicine, University of Texas Medical Branch, Galveston, TX, USA

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ABSTRACT

During the last decades, numerous studies have been performed to investigate the effects and mechanisms of acupuncture or electroacupuncture (EA) on gastrointestinal motility and patients with functional gastrointestinal diseases. A PubMed search was performed on this topic and all available studies published in English have been reviewed and evaluated. This review is organized based on the gastrointestinal organ (from the esophagus to the colon), components of gastrointestinal motility and the functional diseases related to specific motility disorders. It was found that the effects of acupuncture or EA on gastrointestinal motility were fairly consistent and the major acupuncture points used in these studies were ST36 and PC6. Gastric motility has been mostly studied, whereas much less information is available on the effect of EA on small and large intestinal motility or related disorders. A number of clinical studies have been published, investigating the therapeutic effects of EA on a number of functional gastrointestinal diseases, such as gastroesophageal reflux, functional dyspepsia and irritable bowel syndrome. However, the findings of these clinical studies were inconclusive. In summary, acupuncture or EA is able to alter gastrointestinal motility functions and improve gastrointestinal motility disorders. However, more studies are needed to establish the therapeutic roles of EA in treating functional gastrointestinal diseases.

Contents

* Corresponding author. Division of Gastroenterology, Route 0655, Galveston, TX 77555-0655, USA.
E-mail address: jianchen@utmb.edu (J.D.Z. Chen).

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1. Introduction

Acupuncture is a traditional Chinese medicine treatment and has been practiced empirically in China for several millennia. The existence of acupuncture is believed to have been at least 4000 years. Acupuncture is accomplished by inserting the tips of thin, stainless steel needles on specific points (called acupoints) through the skin. Conventional acupuncture or called "manual acupuncture" involves the manipulation of the inserted needles by hand, such as lifting, thrusting, twisting, twirling or other complex combinations. Electroacupuncture (EA) is a modification of this technique that stimulates acupoints with electrical current instead of manual manipulations, and appears to have more consistently reproducible results in both clinical and research settings (Li et al., 1992; Lux et al., 1994). Transcutaneous electroacupuncture (TEA) is a method of delivering electrical current via cutaneous electrodes placed at acupoints. This method is noninvasive and similar to transcutaneous electrical nerve stimulation (TENS) in which the cutaneous electrodes can be placed anywhere, not necessarily at acupoints. TEA and TENS are similar because acupoints are commonly located in the vicinity of nerve dermatomes.

Acupuncture is being increasingly accepted by practitioners and patients in the West as well, especially during the last three decades (Goldstein and Cox, 1977; Li and Chung, 1976; NIH, 1998). Both conventional acupuncture and EA have been used for a variety of ailments, particularly for the relief of pain (Cheng and Pomeranz, 1979; Pomeranz and Warma, 1988). It has been confirmed that acupuncture or EA has therapeutic effects for postoperative dental pain, postoperative and chemotherapy-induced nausea and vomiting (NIH, 1998). During the last decade, a considerable number of studies have investigated the efficacy of EA for the treatment of functional gastrointestinal disorders. Human and animal studies were conducted to explore the effects of EA on gastrointestinal secretion, sensation, motility and myoelectrical activity (Diehl, 1999; Li et al., 1992). In healthy volunteers, EA decreased basal acid output as well as sham feeding-induced (vagally mediated) acid output, but had no effects on the pentagastrin-stimulated acid output (Tougas et al., 1992). In rats with stress-induced gastric ulcer, EA was able to protect the stomach by thickening gastric mucosal barrier, stabilizing mast cells and decreasing the gastrin level in gastric mucosa (Shen et al., 1995).

Recently, a large number of studies have been performed to explore the efficacy of EA/TEA for the treatment of gastrointestinal motility disorders, and improvement in gastrointestinal symptoms has been reported in patients with various disorders associated with gastrointestinal motility (Chang et al., 2001; Li and Chung, 1976; Lin et al., 1997; Ouyang et al., 2004a; Takahashi, 2006). The aim of this review is to evaluate the efficacy and mechanisms of acupuncture or TEA on gastrointestinal motility disorders in both laboratory and clinical settings. PubMed search was performed using the combination of acupuncture with each of the following: esophageal motility, lower esophageal sphincter, gastroesophageal reflux, gastric motility, gastric accommodation, gastric myoelectrical activity, gastric slow waves, electrogastrography, antral contractions, gastric emptying, functional dyspepsia, gastroparesis, small intestinal contractions, small intestinal transit, colonic transit, visceral sensation, irritable syndrome, and constipation and diarrhea. Only articles published in English were reviewed and evaluated in this review.

2. Acupuncture and esophageal motility

2.1. Physiology of esophageal motility

The esophagus is a conduit that serves to transport swallowed contents from the oropharynx to the stomach. At the level of the gastroesophageal junction (GEJ), there is a ring-shaped thickening of the muscle layer known as the lower esophageal sphincter (LES). The LES creates and maintains a high-pressure zone at the GEJ by tonic contractions, augmented by contractions of the crural diaphragm. The LES functions as a barrier preventing the reflux of gastric content into the esophagus. The action of swallowing can initiate peristaltic contractions from striated esophageal muscles that sweep along the esophageal body. The tonically contracted LES relaxes with the onset of peristalsis due to the simultaneous activation of the inhibitory nerves in the myenteric plexus, and remains relaxed until the peristaltic contraction closes the sphincter (Yamata et al., 1995).

Abnormalities of esophageal motility are classified based on the LES function and contractile patterns of the esophageal body, including diffused esophageal spasm, ineffective esophageal motility disorder, non-specific esophageal motility disorder, hypotensive esophageal motility, and achalasia (Nebel et al., 1976). Diffuse esophageal spasm is characterized clinically by intermittent chest pain and dysphagia. Chest pain can vary from mild to crushing, extend to the back and jaw, and last from seconds to minutes. Dysphagia can be due to solids or liquids and often occur more commonly with ingestion of either very cold or very hot foods (Chen et al., 1989). Manometrically, an ineffective esophageal motility this disorder is characterized with a low amplitude of contractions in the esophageal body. It is often seen in patients with scleroderma or gastroesophageal reflux disease (GERD) (Bassotti et al., 1997). The diagnosis of non-specific esophageal motility is often used in the evaluation of patients with dysphagia and/or chest pain who has abnormal findings in esophageal motility tracing, but does not fulfill the fixed criteria for other discrete diagnosis (Kahrilas, 2000). Achalasia is a disorder of both the LES and smooth musculature of the esophageal body. In patients with achalasia, the primary problems are a failure of the LES to relax completely during swallowing and a failure of the esophageal smooth muscle to produce peristalsis adequately (Koshy and Nostrant, 1997). Diseases associated with esophageal motility disorders include functional dysphagia, noncardiac chest pain and GERD (Clouse et al., 1999; Kemp et al., 1986; Nebel et al., 1976).

2.2. EA and esophageal motility

Recently, a number of studies have reported the effects of EA on esophageal motility disorders. In one study, EA at ST36 was found to increase LES pressure (LESP) and the peak amplitude of esophageal peristalsis in cats with myotomy (Shuai et al., 2008). In another study, EA at PC 6 was found to significantly reduce the frequency of transient lower esophageal relaxations (TLESRs) induced by gastric distension in normal cats (Wang et al., 2007). In healthy volunteers, EA at PC 6 decreased the number of TLESRs induced by gastric distension by approximately 40%, but had no effects on basal LES pressure, the residual pressure during TLESRs and the duration of TLESRs (Zou et al., 2005). Chang et al. studied the effect of transcutaneous stimulation (TNS) on esophageal motility in healthy volunteers and found that TNS improved LES relaxation by 11.3% and increased percent of peristaltic contractions by 4.3% during swallow (Chang et al., 1996). In a study using dynamic scintigraphy, acceleration in esophageal transit was noted with auriculoacupuncture in patients suffering from cervical vertebopathy (Hep et al., 1999).

2.3. EA and GERD

GERD is characterized by excessive reflux of gastric content (acid, pepsin, etc.) into the esophagus causing symptoms of heartburn and acid regurgitation, and mucosal inflammation and injuries. The development of GERD is usually associated with a decreased LESP, increased TLESRs and decreased esophageal clearance capacity (Xing et al., 2004a). It has been reported that 44% of the adult population complain of GERD-related symptoms in the U.S. (Fass et al., 2001; Locke et al., 1997). Surprisingly, however, little has been reported in the literature on the efficacy of EA on GERD. Only one recent study by

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Dickman et al. investigated the effect of EA on GERD by comparing the effect of EA added to a conventional proton pump inhibitor (PPI) therapy with that of doubling the PPI dose in patients with refractory GERD. It was found that addition of acupuncture at ST36, PC6, SP9, CV12 and CV17 to the conventional PPI therapy was more effective than doubling PPI dose in reducing the symptoms of heartburn and acid regurgitation (Dickman et al., 2007). Apparently, more clinical studies are necessary to investigate the role of EA in the management of GERD.

3. Effects and mechanisms EA on gastric motility

3.1. Physiology of gastric motility

Gastric motility is one of the most critical physiological functions of the human gut. Without coordinated motility, digestion and absorption of dietary nutrients cannot take place. To accomplish its functions effectively, the gut needs to generate not just simple contractions but contractions that are coordinated to produce transit of luminal contents (peristalsis). Gastric motility functions include gastric accommodation, gastric myoelectrical activity (pacemaking activity), gastric contractions and gastric emptying described as follows:

a) Gastric accommodation. When food enters the stomach, the proximal part relaxes during eating to accommodate the ingested food without producing a large increase in gastric pressure, this reflex is called "gastric accommodation" and is believed to be involved in the regulation of food intake (Gilja et al., 1996; Kim et al., 2001b; Tack et al., 1998). The extent of gastric accommodation has been normally evaluated with Barostat and expressed as an increase in gastric volume in response to a meal (Kim et al., 2001a).
b) Gastric myoelectrical activity. Beginning from the proximal one third and distal two thirds of the stomach to the pylorus, there is gastric myoelectrical activity consisting of two components, slow waves and spike potentials (Chen et al., 1995). The slow wave is omnipresent and occurs at regular intervals whether or not the stomach contracts. It originates in the proximal stomach and propagates distally toward the pylorus. The gastric slow wave determines the maximum frequency, propagation velocity and propagation direction of gastric contractions. When spike potentials (equivalent to action potentials in single cells) are superimposed on the gastric slow waves, a strong lumen-occluded contraction occurs. The normal frequency of the gastric slow wave is about 3 cycles/min (cpm) in humans and 5 cpm in dogs. A noninvasive method similar to electrocardiography, called electrogastrography, has been developed and applied to detect gastric slow waves using abdominal surface electrodes (Chen et al., 1995).
c) Gastric contractions and gastric emptying. Coordinated and distally propagated gastric contractions are called gastric peristalsis. The gastric contraction is stronger in the antral area than the proximal stomach and is believed to play an important role in the regulation of solid gastric emptying. In healthy humans, the ingested food is usually emptied by 50% or more at 2 h after the meal and by 95% or more at 4 h after a solid meal (Tougas et al., 2000). In the postprandial period, there is electromechanical coupling: every slow wave is associated with one contraction. When the stomach is empty, the pattern of gastric contractions changes. The gastric contract pattern in the fasting state undergoes a cycle of periodic fluctuation divided into three phases: Phase I (no contractions, 40–60 min), Phase II (intermittent contractions, 20–40 min) and Phase III (regular rhythmic contractions, 2–10 min) (Yamata et al., 1995).

3.2. Functional dyspepsia (FD) and gastroparesis

Functional dyspepsia (FD) and gastroparesis are two common gastric motility disorders. Functional dyspepsia is characterized by symptoms of postprandial fullness, early satiation, epigastric pain and burning, in the absence of a readily identifiable organic cause (Tack et al., 2006). Gastroparesis is defined as severely delayed gastric emptying in the absence of mechanical obstruction, and classified as diabetic, postoperative and idiopathic according to its etiology (Abell et al., 2006). Main pathophysiologies of functional dyspepsia and gastroparesis include visceral hypersensitivity, impaired gastric accommodation and impaired gastric motility (antral hypomotility, impaired coordination, gastric dysrhythmia and delayed gastric emptying) (Chen et al., 1995; Malagelada et al., 1980; Tack, 2007; Tack et al., 1998, 2006).

3.3. Effect of EA on gastric accommodation

In the literature, few papers were found on the effect of EA on gastric accommodation (Ouyang et al., 2004b). It was reported that EA at ST36 restored vagotomy-induced impaired gastric accommodation in dogs but showed no effects on gastric accommodation in normal dogs. Impaired accommodation is often seen in patients with FD or gastroparesis. It is especially common in patients with diabetic gastroparesis due to autonomic neuropathy as the accommodation reflex is mediated via the vagal and nitrergic mechanisms. The ameliorating effect of EA on vagotomy-induced impairment in gastric accommodation suggests the therapeutic potential of EA for FD or gastroparetic patients with impaired gastric accommodation. In a rodent study with the use of strain gauge transducers, Tada et al. reported that EA induced gastric relaxation in anesthetized rats (Tada et al., 2003). Clinical studies are needed to investigate whether these findings in the animals can be applied to humans.

3.4. Effects of EA on gastric slow waves

Effects of EA on gastric slow waves have been extensively studied in both animals and humans, apparently attributed to the availability of the noninvasive method of electrogastrography. In dogs with duodenal or rectal distention, EA at ST36 increased the regularity of gastric slow waves (Chen et al., 2008; Ouyang et al., 2002), and the effect was found to be mediated via the opioid and vagal pathways (Chen et al., 2008; Ouyang et al., 2002). In healthy volunteers, EA was reported to enhance the percentage of normal 2–4 cpm slow waves (Chang et al., 2002; Chou et al., 2003; Lin et al., 1997), and alter the frequency of gastric slow waves (Shiotani et al., 2004). In addition, the effect of EA on gastric slow wave frequency was EA site-specific: EA at PC 6 alone and EA at ST36 alone showed opposite effects on gastric slow wave frequency, whereas EA at both PC6 and ST36 decreased slow wave frequency (Shiotani et al., 2004). The enhancement of gastric slow waves with EA was also noted in with TEA and acupressure (Chang et al., 2002; Stern et al., 2001). In patients with diabetes and gastric dysrhythmia, EA was found to increase the percentage of normal slow waves and decrease the percentage of tachygastria (Chang et al., 2001). The ameliorating effect of EA on gastric dysrhythmia reported in various clinical studies has been consistent and reproducible, indicating the robust role of EA for the treatment of gastric slow wave dysrhythmia. In an animal model, EA was reported to improve or normalize gastric dysrhythmia by increasing the vagal activity measured by heart rate variability, suggesting the involvement of the vagal pathway (Chen et al., 2008; Ouyang et al., 2002).

3.5. Effects of EA on gastric contractions

Gastric contractions play an important role in regulating gastric emptying. Gastric contractions can be measured by strain gauges (used in animals) and manometry (used clinically). The effects of EA on gastric contractions have been reported in rats (Iwa et al., 2007; Sato et al., 1993; Tatewaki et al., 2003), rabbits (Niu et al., 2007) and dogs (Chen et al., 2008; Ouyang et al., 2002). Sato et al. reported that

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in anesthetic rats, gastric contractions in the pyloric region were inhibited by acupuncture-like stimulation applied to the abdomen or lower chest region, and excited when the limbs were stimulated (Sato et al., 1993). Niu et al. reported that EA at ST36 significantly increased the number and amplitude of spikes assessed from gastric myoelectrical activity, indicative of increased gastric contractions, in rabbit, and that the effect was mediated via the cholinergic nerve (Niu et al., 2007). In dogs, EA was found to improve impaired antral contractions induced by rectal distension and the ameliorating effect involved the opioid pathway (Chen et al., 2008). These previous findings indicate that the stimulatory effect of EA on gastric contractions is consistent among different species. Inhibitory or dual effects of EA on gastric motility were also reported in a few studies (Qian and Lin, 1993; Tatewaki et al., 2003; Yuan et al., 1986; Zhou, 1986). In a rodent study with the measurement of gastric contractions using strain gauge transducers Tatewaki et al. reported that manual acupuncture at ST36 induced dual effects: stimulating gastric contractions in rats with hypomotility and inhibiting gastric contractions in rats with hypermotility. It was further reported that the stimulatory effect was medicated in part via the vagal and opioid pathway (Tatewaki et al., 2003). In general, the inhibitory or dual effects of EA were not as consistent as the excitatory effects of EA on gastric contractions; more data are needed to support the inhibitory or dual effects of EA on gastric contractions.

3.6. Effects of EA on gastric emptying

Acceleration of gastric emptying with acupuncture has been reported in both animals and humans (Iwa et al., 2006b; Ouyang et al., 2002; Tabosa et al., 2004; Wang et al., 2008; Xu et al., 2006). In rats with delayed gastric emptying induced by restraint stress, EA at ST36 was found to significantly improve gastric emptying of solids (Iwa et al., 2006b). Similar accelerative effect of EA on solid gastric emptying was also reported in normal rats (Tabosa et al., 2004). In dogs with delayed gastric emptying induced by duodenal distention, EA at PC6 and ST36 significantly accelerated gastric emptying and concurrently increased vagal activity assessed by the spectral analysis of the heart rate variability, suggesting a possible vagal mechanism (Ouyang et al., 2002). In patients with gastroparesis, EA at ST36 and PC6 accelerated solid gastric emptying measured by scintigraphy (Xu et al., 2006).

3.7. Application of EA in treating FD or gastroparesis

Compared to the animal studies on gastric motility with EA, little information is available on the application of EA in treating functional dyspepsia or gastroparesis. In a recent double-blind, cross-over study in 27 FD patients, TEA at ST36 and PC6 (twice weekly for a period of 2 weeks) reduced dyspepsia symptoms by 55% (Liu et al., 2008); an increase in vagal activity noninvasively assessed from the heart rate variability and in the plasma level of neuropeptide Y was also noted, suggesting the involvement of the vagal and hormonal pathways. In another controlled clinical study with 68 FD patients, manual acupuncture at acupoints resulted in a significant improvement in dyspeptic symptoms in comparison with acupuncture at non-acupoints (Park et al., 2009). In a non-controlled study involving 19 FD patients, chronic EA at ST36 and PC6 significantly reduced dyspeptic symptoms at both 2 weeks and 4 weeks after the treatment in the FD patients with normal gastric emptying, whereas acute EA at ST36 and PC6 improved gastric emptying in the patients with delayed gastric emptying in comparison with EA at non-acupoints (Xu et al., 2006). The acceleration of gastric emptying and improvement in dyspeptic symptoms with EA were also reported in a 2-week single-blinded controlled study involving 9 diabetic patients with symptoms suggestive of gastroparesis; in that study, EA was performed at ST36 and LI4 (Wang et al., 2008).

4. Effects and mechanisms of EA on intestinal motility and transit

4.1. Physiology of small intestinal motility

Small intestinal motility exhibits two distinct patterns: fasting and fed. The typical manifestation in the fasting state is the migrating motor complex (MMC). The MMC consists of three phases with considerably varying durations: Phases I, II and III. Phase I is a period of motor quiescence, representing 20% to 30% of the total cycle length. Phase II is characterized by intermittent and irregular contractions with a duration of 40% to 60% of the cycle length. Phase III is a 5–10 min period of intense, rhythmic contractions that propagate from the proximal to distal intestine. After a meal of sufficient nutrients, the fasting pattern of motility is switched to the fed pattern characterized by intermittent phasic contractions of irregular amplitude similar to those of Phase II of the MMC. Intestinal motility controls the transportation and absorption of the ingested nutrients. Intestinal dysmotility includes absence of the MMC, impairment of the MMC, such as impaired propagation of the MMC along the gut, postprandial hypomotility and hypermotility.

4.2. Effects of EA on intestinal motility in animals and humans

Little efforts have been made in the investigation of the effect of EA on small intestinal motility, probably attributed to the lack of non-invasive methods for the measurement of intestinal motility. In dogs with intestinal motility assessed by duplex Doppler sonography, EA at ST36 was reported to increase the frequency of intestinal movement by 20%, whereas EA at BL27 decreased the frequency of intestinal movement by 31% (Choi et al., 2001). In rats, EA at hindlimb acupoints (ST36 and SP6) significantly enhanced small intestinal transit assessed by counting plastic beads administered orally (Tabosa et al., 2004). In mice, intestinal contractions were enhanced with EA and the effect was blocked by atropine (Iwa and Sakita, 1994). In rabbits, EA at ST36 and SP6 reduced the inhibitory effect of morphine on duodenal peristalsis (Dai JL et al., 1993).

No convincing clinical studies are found in the literature showing the effect of EA on intestinal motility. In twenty healthy volunteers, EA at Siguan points (bilateral points LI4 and LR3) was shown to have little effects on small and large intestinal transit assessed radiographically (Yim et al., 2007). However, the sensitivity used for the assessment of the intestinal transit was questionable. In another study involving women with hysterectomy, acupressure was performed at PC6, ST36 and SP6 was found to improve gastrointestinal contractions in comparison with acupressure at sham points (Chen et al., 2003). However, the validity of this study is questionable as the acupressure was performed for only 3 min each time and the gastrointestinal contractions were assessed by a multifunctional stethoscope, a method that would not be approved by any expert working in the field of gastrointestinal motility. Apparently, clinical studies are needed to investigate the role of EA in treating patients with small intestinal motility disorders, such as postoperative ileus and chronic intestinal pseudo-obstruction. The invasive nature of the methods used in the assessment of intestinal contractions may explain the lack of clinical studies in this area.

5. Effects and mechanism of EA on colon motility and transit

5.1. Colonic motility

The colon functions mainly as a storage organ with moderate absorptive capacity for water, electrolytes, and nutrients. In the colon, there are individual phasic contractions and giant migrating contractions. The individual phasic contraction is the basic unit of contractile activity and occurs during the fasting and fed states. There are two types of individual contractions in the colon: short-duration and long-duration. Short-duration contractions last less than 15 s and the long-

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duration contractions last 40–60 s in the dog and human colon (Huizinga et al., 1985; Sarna et al., 1982; Sarna, 1991a; Sarna, 1984). Ingestion of a meal stimulates colonic motility (gastrocolonic reflex) and the colon (motility) goes to sleep when a person goes to sleep. The pattern of individual phasic contractions is complicated with a lack of specific dominant frequencies, probably associated with one of the main functions of the colon: storage. The bowel movement is achieved by the giant migrating contractions (Torsoli et al., 1971; Williams et al., 1987). The giant migrating contractions occur rarely, no more than once or twice a day in humans. Spontaneous mass movements and their associated giant migrating contractions occur mainly in the proximal colon, the mean migration distance in the canine colon is about 13 cm (Sarna, 1991a). Disrupted colonic motility has been associated with various functional diseases, such as irritable bowel syndrome (IBS), constipation and diarrhea.

5.2. EA on colonic motility

EA on colonic motility has been investigated in animal models. In conscious rats, EA at ST36 was reported to significantly increase contractility of the distal colon measured by manometry, and the stimulatory effect was mediated via the cholinergic pathway (Luo et al., 2008). Similar findings were also reported in an earlier rodent study: EA at ST36 increased colonic transit mediated via the sacral parasympathetic efferent pathway (pelvic nerve) (Iwa et al., 2006a). In contrast, in rats with restraint stress, EA was reported to inhibit stress-induced acceleration in colonic transit and the inhibitory effect was independent of the sympathetic pathway (Iwa et al., 2006b). In 17 children with chronic constipation, acupuncture at ST36, LI2 and LI4 gradually increased the frequency of bowel movement as well as the plasma opioid level during a 10-week treatment period (Broide et al., 2001).

5.3. EA and IBS

IBS is most common among various functional gastrointestinal disorders, affecting around 15% of the general population. IBS manifests by altered bowel habit with abdominal pain. No specific, bacterial, biochemical or morphological abnormality can be identified in these patients (Sarna, 1991b). A lowered sensory threshold to rectal distension is a hallmark of IBS patients (Bouin et al., 2002; Poitras et al., 2002). The therapeutic role of EA for IBS has not been established. A few studies have reported ameliorating effects of acupuncture on IBS symptoms whereas, others suggested purely placebo effects (Anastasi et al., 2009; Chan et al., 1997; Lembo et al., 2009; Rohrbock et al., 2004; Schneider et al., 2006). In an open-design pilot study, patients with IBS showed a significant improvement both in general well-being and in symptoms of bloating (Chan et al., 1997); in a randomized, sham/placebo-controlled trial in 29 IBS patients a significant improvement was observed in daily abdominal pain/discomfort, intestinal gas, bloating and stool consistency after 4-weeks of acupuncture of twice weekly at CV12, ST25 and CV6 et al. (Anastasi et al., 2009). In a controlled clinical trial of 43 IBS patients, Schneider et al. reported a significant improvement in global quality of life at the end of both acupuncture and sham-acupuncture treatment, and suggested a placebo effect of EA; the authors suggested that a study including 566 patients would be necessary to prove the efficacy of acupuncture over sham acupuncture (Schneider et al., 2006). The same group later reported a significant increase in parasympathetic tone with EA but not sham EA and suggested that different mechanisms may be involved in placebo and real-acupuncture driven symptom improvements in IBS patients (Schneider et al., 2007).

Unlike the improvement in IBS symptoms, the improvement in visceral sensation with EA is less controversial and has been consistently reported in both animals and humans (Cui et al., 2005; Xiao and Liu, 2004; Xing et al., 2004b; Xu et al., 2009). In a rodent model of IBS, EA at ST36 attenuated visceral hypersensitivity involving the opioid pathway and inhibited the enhanced excitability (attributed to neonatal injection of acidic acid) of colon specific dorsal root ganglion neurons (Xu et al., 2009). In patients with IBS, TEA at ST36 and PC6 increased the threshold of rectal sensation of gas but showed no effects on rectal tone or rectal compliance (Xing et al., 2004b). In another study with the treatment regimen of twice per week for 2 months, TEA at LI4 and ST36 improved IBS symptoms and abnormal rectal sensation in diarrhea-predominant IBS (Xiao and Liu, 2004).

Table 1
Effects of acupuncture on gastrointestinal motility.

Organ/functions	Finding	Methods	Subjects	Authors
Esophagus				
Contractions	Increased the peak of amplitude in esophageal peristalsis	EA at ST36	Cats	Shuai et al., 2008
LES	Increased LES pressure	EA at ST36	Cats	Shuai et al., 2008
TLESRs	Decreased the rate of LESRs	EA at PC6	Healthy human	Zou et al., 2005
GERD	Adding acupuncture is more effective in controlling symptoms than doubling PPI dose	Acupuncture at PC6, ST36, CV12, CV17, LR3 and SP9	Patients	Dickman et al., 2007
Stomach				
Accommodation	Restored impaired gastric accommodation by vagotomy	EA at ST36	Dogs with vagotomy	Ouyang, 2004b
Slow waves	Increased the percentage of regular slow waves	EA at ST36 and PC6	Healthy human	Lin et al., 1997; Chang et al., 2002
Contractions	Restored impaired antral contractions induced by rectal distension	EA at ST36	Dogs Rats	Chen et al., 2008; Tatewaki et al., 2003
Gastric emptying	Accelerated liquid gastric emptying delayed by duodenal balloon distention. Accelerated solid gastric emptying delayed by restraint stress	EA at ST36	Dogs Rats	Ouyang et al., 2002; Iwa et al., 2006b
FD	Chronic TEA improved dyspepsia symptoms and increased plasma neuropeptide Y level	TEA at ST36 and PC6	Patients	Liu et al., 2008
Gastroparesis	Accelerated solid gastric emptying in FD patients with delayed gastric emptying	EA at ST36 and PC6	Patients	Xu et al., 2006
Small intestine				
Contractions	Increased frequency of small intestinal motility	EA at ST36 and BL27	Dogs	Choi et al., 2001
Transit	Accelerated small intestinal transit	EA at ST36 and SP6	Rats	Tabosa et al., 2004
Colon				
Contractions	Increased contractility in distal colon	EA at ST36	Rats	Luo et al., 2008
Transit	Accelerated colonic transit. Inhibited accelerated colonic transit induced by restraint stress	EA at ST36 EA at ST36	Rats Rats	Iwa et al., 2006a,b
Sensation	Attenuated visceral hypersensitivity in rat model with IBS	EA at ST36	Rats	Xu et al., 2009
IBS	Both acupuncture and sham acupuncture improved symptoms, acupuncture in IBS is a placebo response	Acupuncture	Patients	Schneider et al., 2006

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6. Discussion and conclusion

Based on the evidence from the studies in both animals and humans, EA has the potential for treating gastrointestinal motility disorders. As shown in the summary table, EA increases LES pressure and reduces TLESRs, and therefore may be beneficial to patients with GERD. With regarding to gastric motility functions, it seems that EA enhances gastric accommodation, slow waves, contractions and emptying; suggesting a therapeutic potential for functional dyspepsia and gastroparesis. Little is reported on the effect of EA on small intestinal motility and therefore its role for treating patients with intestinal motility disorders has not been established. Similarly, not much is known on the effect of EA on colon motility and the therapeutic effects of EA for common functional bowel disorders, such as IBS, constipation and diarrhea, are not conclusive (Table 1).

Acupuncture or EA has advantages of being noninvasive and practiced for many years. Accordingly, ample clinical data are available on the application of EA for treating various disorders. With regard to its applications for the treatment of gastrointestinal motility diseases, however, there are a number of problems: 1) most of the available methods used for the assessment of gastrointestinal motility are noninvasive, making it less feasible for basic and clinical research; 2) patients with functional gastrointestinal disorders are heterogeneous with unclear pathophysiologies or pathogenesis, and therefore the outcome of the treatment of these patients is typically controversial not only with EA but also with other therapies; 3) based on our review of the literature, there are also issues including the methodology of EA, study design and outcome measurements. Different methods have been used for the implementation of acupuncture, including, manual acupuncture, EA, TEA and acupressure, and these different methodologies make it difficult for the comparison of the efficacy of EA. In some studies, the parameters of EA that are important for the success of the therapy (Han, 2003) were not mentioned or appropriately determined; whereas in other studies, the experimental designs were not adequate and the measurement methods were inadequate.

We believe that EA has a great therapeutic potential for treating gastrointestinal motility disorders and functional gastrointestinal diseases. Future clinical studies with EA should follow rigid scientific designs, optimize methodologies and apply cut-edge outcome measures. In most of clinical studies, symptoms (subjective) are primary endpoints and therefore the experiment must be controlled and blinded if feasible. For the implementation of EA, efforts should be made in the selection of acupoints and stimulation parameters, the duration and frequency of EA. In addition, physiological measurements should also be made in clinical studies to understand possible mechanisms and pathways involved with EA.

In conclusion, acupuncture or EA is able to alter gastrointestinal motility functions and improve gastrointestinal motility disorders. However, more studies are needed to establish the therapeutic roles of EA in treating functional gastrointestinal diseases, such as GERD, functional dyspepsia, IBS, constipation and diarrhea.

7. Uncited references

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Ouyang and Chen, 2004
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