World Journal of Gastroenterology

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DOI: 10.3748/wjg.v2.iSuppl1.13

World J Gastroenterol 1996 September 25; 2(Suppl1): 13-14
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

Clinical applications of electrogastrography

Kenneth L Koch

Kenneth L Koch, MD, Gastroenterology Division, Department of Medicine, The Pennsylvania State University, Hershey, PA, United States

Author contributions: The author solely contributed to the work.

Original title: *China National Journal of New Gastroenterology* (1995-1997) renamed *World Journal of Gastroenterology* (1998-).

Received: November 1, 1995
Revised: January 27, 1996
Accepted: March 1, 1996
Published online: September 15, 1996

Koch KL. Clinical applications of electrogastrography. *World J Gastroenterol* 1996; 2(Suppl1): 13-14 Available from: URL: http://www.wjgnet.com/1007-9327/full/v2/iSuppl1/13.htm DOI: http://dx.doi.org/10.3748/wjg.v2.iSuppl1.13

BASIC MYOELECTRICAL ACTIVITIES OF THE STOMACH

Slow waves

The stomach is a neuromuscular organ which has intrinsic electrical activities that are modulated by the parasympathetic, sympathetic and enteric nervous systems and gastrointestinal hormones. Neuromuscular activities of the stomach generate basic electrical phenomena termed "electrical control activity"[1]. The outer layer of the circular muscle of the stomach wall is considered a source of these electrical oscillations; The interstitial cells of Cajal may also be the source of the gastric slow wave.

Gastric slow waves originate in a "pacemaker region" located on the greater curvature of the stomach near the junction of the fundus and proximal gastric corpus. The slow waves are propagated distally and circumferentially and ultimately migrate towards the pylorus at a rate of one propagated wave front every 20 s in man. Thus, the normal gastric slow wave frequency occurs at 3 cycles per minute[2]. As the gastric slow wave approaches the distal antrum and pylorus, a new slow wave develops in the pacemaker region.

The slow wave controls the timing and propagation of gastric peristalses, which are produced by the contraction of the circular muscle layer. Contractions of the circular muscle layer may not occlude the lumen, to strong lumen-obliterating contractions which are seen in the fasting state.

Spike potentials

Spike potentials or electrical response activity are the electrical events which occur during circular muscle contraction. Spike potentials occur only when slow wave depolarization has brought the circular muscle to its excitation threshold. When excitation threshold is reached, spike potentials and contraction of the circular muscle layer occur[1]. Thus, the linkage of the slow wave with spike potential activity underlies gastric peristaltic contractions which migrate from proximal stomach to pylorus at a rate of 3 per minute. Spike activity and gastric contractility are modulated by a host of factors including parasympathetic and sympathetic activity, ongoing hormonal changes in the fasting or postprandial state, a wide range of physical and nutrient factors which stimulate the stomach through intragastric or extragastric mechanisms, and a variety of emotional or stress-related stimuli.

ELECTROGASTROGRAPHY-A NON-INVASIVE METHOD TO RECORD GASTRIC MYOELECTRICAL ACTIVITY

Alvarez recorded sinusoidal 3 cycle per minute (cpm) waves with electrodes place d over the upper abdomen in 1922[3]. He called these recordings electrogastrograms (EGG), and predicted that abnormalities of the gastric myoelectrical rhythm may be related to upper gastrointestinal symptoms and gastric dysfunction. The normal gastric frequency range is between 2.4-3.6 cpm[4]. Other frequency ranges of interest are 3.6-9.9 cpm, an abnormally rapid frequency range termed tachygastria[4]. The EGG frequencies from 1-2.4 cpm are slower than normal, and may be abnormal if they dominate the recording[4]. These very slow EGG rhythms are termed bradygastrias. Finally, the 10-15 cpm range encompasses the known duodenal frequencies in man, which are from 10-13 cpm. In addition to frequency analysis and the percentage of power in the various frequency bands, the power in these bands can be compared before and after a stimulus such as a meal or drug.

ABNORMAL MYOELECTRICAL ACTIVITY OF THE STOMACH GASTRIC DYSRHYTHMIAS

Alvarez predicted that electrical abnormalities of the stomach may be related to abnormal digestive symptoms and abnormal gastric function. In 1980, You *et al*[5] described a series of patients with unexplained nausea and vomiting and dysrhythmias. The rhythms ranged from regular 6-7 cpm tachygastrias to very chaotic, fast rhythms termed tachyarrhythmias. Abell *et al*[6] showed that glucagon infusions evoked gastric dysrhythmias and nausea in healthy volunteers. The gastric dysrhythmias recorded with mucosal electrodes correlated with cutaneous signals which showed the same gastric dysrhythmias. Recent studies confirmed the excellent correlation between normal 3 cpm rhythm and arrhythmias recorded from surface EGG electrodes and serosal electrical recordings[7,8]. Stern and Koch showed that the shift from the normal 3 cpm electrical rhythm to tachyarrhythmias was associated with onset of nausea

during motion sickness[9].

CLINICAL APPLICATIONS OF ELECTROGASTROGRAPHY

Evaluation of patients with nausea and vomitig

Gastric tachyarrhythmias and bradygastrias have been recorded in diabetics with gastroparesis and in patients with idiopathic gastroparesis[10-12]. Abnormal EGGs reflect gastric dysrhythmias and are predictive of delayed gastric emptying. Over 90% of the patients with the gastric dysrhythmias had delayed gastric emptying[12].

Gastric dysrhythmias have also been recorded in patients with bulimia nervosa, anorexia nervosa, and in patients with functional dyspepsia and normal gastric emptying rates. Gastric dysrhythmias have been recorded in patients with nausea of pregnancy[13].

Normal 3 cpm EGG patterns were recorded in patients with documented gastroparesis and mechanical obstructions of the upper gastrointestinal tract[14]. Normal 3 cpm EGG rhythms and gastroparesis has also been recorded in patients with diabetes mellitus; These patients have very few upper gastrointestinal tract symptoms. Domperidone restored the normal 3 cpm EGG rhythm and reduced symptoms, but gastroparesis was unchanged, suggesting gastric electromechanical dissociation[10].

Finally, normal 3 cpm EGG rhythm and normal gastric emptying suggests that the patient's nausea and vomiting may not be related to gastric electrical or mechanical dysfunction. In these instances, the EGG pattern is helpful in that it suggests that non-gastric causes of nausea and vomiting should be sought. Patients with occult reflux esophagitis, chronic cholecystitis, and patients with psychogenic nausea have this normal pattern.

Evaluation of GI motility disorders in children

Electrography is an excellent technique for the study of gastric myoelectrical activity in children because it is non-invasive and safe. Studies of EGG activity in premature infants and newborns have been successfully accomplished[15]. EGG recordings in children with gastrointestinal pseudo-obstruction revealed patterns of myogenic and neurogenic dysfunction[16]. Children with n on-ulcer dyspepsia have a variety of gastric dysrhythmias which are eradicated with cisapride treatment[17].

Evaluation of motion sickness

Gastric dysrhythmias occur several minutes before subjects report the nausea of motion sickness[9,18]. Gastric dysrhythmias have been recorded during vection-induced nausea, rotating chair stimuli and during KC-135 "Vomit Comet" studies. EGG techniques also lend themselves for the study of gastric physiology in space. The effects of various drugs and foods on gastric dysrhythmias m ay be studied using EGG technology.

Evaluation of nauseogenic drugs and anti-nauseate drugs

gastric dysrhythmias and nausea occur after the infusion of glucagon, epinephrine, progesterone, and morphine. Gastroprokinetic drugs such as domperidone and cisapride correct gastric dysrhythmias and improve symptoms of nausea. Ondansetron, a-HT_3 antagonist, given during established morphine-induced nausea, eradicates gastric dysrhythmias and restores normal 3 cpm EGG activity.

SUMMARY

Gastric dysrhythmias are recorded in a variety of clinical syndromes and experimental conditions in which gastric motility disorders and symptoms of nausea are present. The eradication of the dysrhythmia and the restoration of the norm al 3 cpm gastric electrical activity are associated with improvement in symptoms, particularly nausea. Gastric dysrhythmias also interferes with the normal mixing and emptying of a meal. The clinical importance of gastric myoelectrical abnormalities continues to emerge. Much additional work is necessary to determine the mechanisms whereby gastric dysrhythmias occur and to investigate therapies designed to eradicate these abnormal gastric myoelectrical events.

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E- Editor: Liu WX

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