Gastrointestinal Implants Seminar

Nada Al Ahmadi - EPFL
Email: nada.alahmadi@epfl.ch

Michaela Rosinska - EPFL
Email: michaela.rosinska@epfl.ch

Giulia Cortelazzo - EPFL
Email: giulia.cortelazzo@epfl.ch

***Abstract*—In this paper we present alternative treatments to gastrointestinal disorders. Firstly we outline the complex physiology of the GI tract is presented, then some common illnesses related to the GI are discussed, focusing on motility disorders, gastroparesis, obesity, diabetes and constipation. Subsequently, electrical stimulation is proposed as an alternative novel treatment, with an overview of different stimulation strategies, implemented in the existing commercial devices. Lastly, a case study is presented, showing the potential of the novel device for the development of personalized GI treatment.**

I. Introduction

Gastrointestinal diseases are a serious concern to human health, affecting millions of individuals with symptoms ranging from impaired motility to severe digestive dysfunction. Traditional treatments like lifestyle modification, medication, and surgery have limitations and are often not effective, highlighting the need for alternative therapeutic techniques. Electrical stimulation of the gastrointestinal tract has been reported since the 1910s, while later clinical investigations demonstrated potential benefits for conditions such as gastroparesis and obesity. Follows a review of current commercial gastrointestinal stimulation technologies and the description of a wireless system-on-chip–based implant, that opens the doors to further exploration of gastrointestinal neurostimulation thanks to high programmability and long lifetime.

A. Gastrointestinal Physiology

The human digestive system is mainly composed of the gastrointestinal (GI) tract, which is the system of organs responsible for digestion, absorption of food, and evacuation of waste. It extends from the mouth to the anus and comprises several organs forming the tube through which liquid and solid food are processed (esophagus, stomach, intestines, etc.) [1]. Additional organs, producing digestive enzymes injected to the main digestive path, account for the second part of the digestive system (pancreas and liver for example).

Despite the variety of organs involved in the GI tract, the fundamental structure of their tissues, composed of concentric functional layers, remains the same. The inner layer is the mucosa through which nutrients are absorbed and transferred to the blood vessels in the submucosal layer. It is surrounded by the muscularis externa (or muscularis propria), that is made of two layers of smooth muscles. They are responsible for the peristalsis, which is the contraction and relaxation of the two muscular layers, allowing food to move through the digestive tube. The muscles are circular in the first layer, and longitudinal in the second layer. For the stomach, a third muscular layer, the inner oblique, is assisting the segmentation process, which is the mixing of the stomach content [1]. The outer membrane is the serosa. It contains cells secreting lubricating fluid that reduces the friction within the tract.

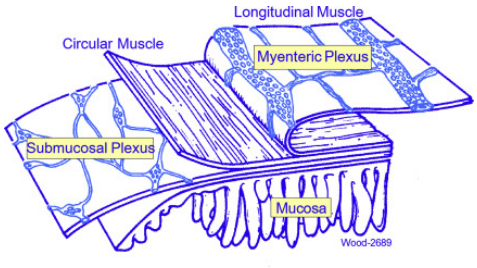

Fig. 1. Diagram of a section of the gastrointestinal tract [2]

The neural network associated with the GI tract is the Enteric Nervous System (ENS). It is a subdivision of the autonomous nervous system (ANS), which controls the involuntary and automatic body functions. The two major subnetworks innervating the GI tract are the myenteric (or Auerbach's) plexus and the submucosal (or Meissner's) plexus. The submucosal plexus, absent from the esophagus and with only a few innervations in the stomach, is located in the submucosa, [3]. It controls the absorption and lubrication of the digestive tube, activating the secretion of digestive fluid and enzymes [4]. The myenteric plexus is between the two muscular layers of the muscularis externa (see Fig.1) along the whole GI tract. It is a dense network of ganglia, where each ganglion contains tens of nerve cell bodies and hundreds of glial cells. Interstitial cells of Cajal (ICC) are also located in close vicinity of the myenteric plexus, their distribution specific to each organ of the GI tract [5]. ICC are pacemaker cells that generate slow waves, inducing depolarization in smooth muscles [6]. This depolarization doesn't instigate muscle contraction, but only synchronizes the firing of action potentials. The frequency varies between 3 waves per minute in the stomach to 16 waves per minute in the small intestine [7]. Together with the ganglia, they control the peristalsis through alternative stimulation and inhibition of smooth muscles.

The ENS controls GI functions mostly independently, but it is regulated extrinsicly by the Sympathetic and Parasympathetic Nervous Systems (respectively SNS and PNS). It has therefore a bidirectionnal connection to the Central Nervous System (CNS) through the vagal and pelvic nerves for the PNS and sympathetic pathways for the SNS [8]. Afferent neurons of the myenteric plexus communicate with interneurons, that are sending signals to efferent nerves and stimulating action

potentials (spikes) in smooth muscles. Contractions are then induced when these spikes are fired during the depolarization phase of the slow waves generated by ICCs [9]. One family of afferent neurons are Intrinsic Primary Afferent Neurons (IPANs), that are excited by mechanical and chemical signals. Myenteric neural activity is influenced by parasympathetic and sympathetic stimulations through the release of neurotransmitters that either excite (for parasympathetic stimulation) or inhibit (for sympathetic stimulation) the associated afferent neurons, thus controlling motility.

B. Illnesses

1) Gastrointestinal motility disorders: A variety of conditions can lead to GI motility disorders, by affecting muscles (myopathies) or neural circuits (neuropathies) [10]. They are divided into two groups depending on the section of the GI tract affected. Upper GI motility disorders affect the GI tract between the esophagus and the first part of the small intestines, and lower GI motility disorders affect small and large intestines and rectum.

2) Gastroparesis: It is an upper GI motility disorder where, in absence of any physical obstruction of the digestive tube, the stomach is emptying too slowly. Possible symptoms include: nausea, vomiting, early satiety and abdominal pain. For 50% of cases, the disease is idiopathic (spontaneous). The other 50% of cases appear after a surgery, an infection, due to diabetes or other diseases. Patients showed a reduced number of ganglia, ICCs and enteric neurons. An accumulation of immune cells (inflammatory infiltrates) was also observed in tissues [11]. For a healthy individual, slow gastric waves are stably propagating to the stomach after a meal. Gastroparesis is characterized by an anomaly of these slow waves (either higher, lower or uneven frequency) [12].

3) Obesity & Diabetes: Obesity is a chronic disease, qualified as epidemic with more than 1 billion people in the world afflicted[1] in 2022. The variety of causes include genetic, environmental, behavioral and metabolic factors [13]. The amount of body fat associated to obesity, increases the risk of type 2 diabetes linearly. Obesity influences the functions of insulin and β-cell. It is directly related to type 2 diabetes, caused by insulin resistance and a decrease of β-cells insulin secretions, leading to unregulated high glucose levels [14].

4) Constipation: It is a lower GI motility disorder affecting 20% of the adult population. Mechanical obstructions, metabolic conditions (diabetes), various myopathies and neuropathies are associated with constipation [15].

C. Solutions

1) Standard treatments: Treatment for gut-related diseases or symptoms typically falls into three main categories.

- **Lifestyle changes:** These often produce inconsistent and limited results. For example, managing obesity through lifestyle alone can be extremely challenging, and such changes may be impractical or only minimally effective over long periods of time.
- **Medication:** Our understanding of the underlying biological and chemical mechanisms for many gut-related symptoms remains incomplete. As a result, medications may not work for all patients (refractory cases) or may be unsafe for long-term use.
- **Surgery:** This is an invasive and permanent solution, carrying significant risks, including the potential for mortality.

There is a growing need for a solution that offers a less invasive alternative, that can be reversed and efficient without omitting the need for assessment of patient suitability for temporary implants.

2) Electrical stimulation: Historically, gastric stimulations were studied for treatment of gastroparesis when medications weren't successful. Two GI stimulation techniques are used for upper GI motility disorders. The first one is the gastric pacing method that uses physiologic lower frequencies and long pulses of higher energy. The second, the Enterra therapy, uses higher frequencies and short pulses of low energy [16].

- **Long pulse width and low frequencies** First studies on dogs showed the possibility of varying the frequency of the gastric slow waves between 2 and 8 cycles per minute [17]. Retrograde GI pacing was then studied on human volunteers with temporary electrodes placed on the lower stomach. A stimulation at 9 cycles minute was used which reduced water intake by 13% and food intake by 16% [18].
- **Short pulse width and high frequencies** Applied to obesity treatments, the Transcend gastric pacemaker was used in multiple studies with the following parameters : 10 mA amplitude, 208 ms pulse width, 40 Hz frequency (2s on, 3s off) [19]. The maximal percentage of excess body weight lost in this study was 40% over 2 years.
  Satiety is a key factor when addressing weight loss. It is influenced by hormonal activity as specific hormone levels are commonly changing after natural weight loss. It was observed that patients with pacemaker doesn't experience the expected rise of these hormones after meals [20], which could come from blockage of the efferent vagal nerve [21]. Animal studies on rabbits [22] and pigs [23] explored this hypothesis. The first study blocked efferent vagal impulses over 4 weeks, which resulted in decreased body weight by 12% and food intake by 40%. A stimulation of the afferent vagus was suggested by the change of heart rate. The second study stimulated both efferent and afferent vagal nerves, and confirmed the decrease of food intake and body weight. Stimulations were made at 170 mV amplitude, 1 Hz frequency and 170 ms impulse duration.

Sacral Nerve Stimulation (SNS) initially developed for urinary incontinence has been found applicable for lower GI motility disorders. Studies showed a reduction of symptoms for IBS [24], fecal incontinence [25] and constipation [26]. The detailed mechanism of operation of the SNS isn't known yet. The ENS is thought to be involved, as SNS may stimulate

[1]Body Mass Index (BMI) > 30

efferent nerves (pelvic nerve) and efferent somatic fibers.

D. Implants

Careful patient selection is crucial for the success of the implants, as not all individuals respond effectively to these devices. Certain patients may be insensitive to the stimulations, while others may exhibit behaviours that could undermine the intended therapeutic outcomes.

Insertion of the implants can be done endoscopically and percutaneously. Both are minimally invasive approaches that reduce recovery time, scarring, and risks compared to traditional open surgery. Endoscopic insertion is done through the mouth of the patient to the stomach and percutaneous insertion is done through the skin via a small puncture or incision.
Placement of the device can be done on the bowel, on the stomach (on the inside or the outside wall of the stomach) to stimulate the vagal nerve and as a cuff around the anterior and posterior vagal trunk.
The placement of the implant is absolutely critical in order to get satisfying results. The simulated areas have different effects on the patients and displacement may result in ineffective treatment. In 2010, a large study of 60 patients delivered poor results due to issues in placement of devices [27].

1) Existing technologies: Several implantable devices for gastrointestinal neurostimulation have been developed, but only a few have been approved for clinical use [16]. The the Enterra® system delivers low frequency, high energy stimulation, effectively reducing nausea and vomiting in patients with severe gastric motility disorders [28]. The Transcend™ pacemaker was used to block efferent vagal activity and slow gastric emptying by long pulse width, low frequency stimulation and short pulse width, high frequency stimulation. However, clinical studies showed no significant difference with sham therampy, leading to the withdrawal of the device [16]. Another therapy that targets the vagal trunks directly, through cuff electrodes, is the Maestro Rechargeable System, using VBLOCK® technology. This device has been approved by the FDA for weight loss in 2015 [29]. The Tantalus™ is implanted laparoscopically, by placing two pairs of electrodes in the gastric antrum and two others in the fundus, signaling the beginning of the meal. The stimulation enhances spontaneous gastric contractions, modulating gastric emptying.
These devices have several limitations. Mostly relying on battery, their size is considerable and their lifetime is limited, while they typically offer only a limited set of stimulation patterns. Although the present data on gastric neuromodulation is indecisive, it suggests a promising therapeutical potential. This is what motivated the study of Yi-Kai Lo and Po=Min Wang on a on wireless system-on-chip implant, that allows highly programmable stimulation, with full duplex data communication. It also combines stimulation with continuous impedance based motility recording. [9]

2) Case study - Wireless implant:
Here, we present the Wireless Implant for Gastrointestinal Motility Disorders by the University of California developed in 2017 [9]. We additionally use the paper presented by the same team for component and technical details [30].

a) Design considerations: The American team observed that the implants currently available are limited and presented issues they would like to address.

- No implant is capable of producing a pulse width of more than $2ms$, limiting research and development of new effective devices.
- No implant on the market is capable of sensing the GI tract making follow-up limited. The need for material and trained staff for ultrasound analysis can be costly and cumbersome. Moreover, the inability of tracking the patient at all times may result in incomplete data and studies may miss major events.
- Current implants are usually bulky, making them invasive to the patient.

To address these issues, the new implant can be parametrized in frequency, amplitude and pulse width. The System on Chip (SoC) is capable of sensing the GI movement and transmitting the data to the user. The user can choose a signal with an amplitude ranging from $0.003A$ to $20A$, a frequency from $0.001Hz$ to $200Hz$ and a stimulation width from $0.01s$ to a defined value. The volume of the Extraluminal Gastrointestinal Modulation Device (EGMD) is of $\sim 0.5m^3$ with a weight of $\sim 0.7g$ making it minimally invasive.

b) System: The system of the implant comprises a Graphical User Interface (GUI) for modulation control and data monitoring and a Relay Device (RD) for linking wirelessly the user to EGMD and the EGMD. The EGMD comprises the SoC, the electrode array and the coils for data and power integrated into a flexible substrate of $8\mu m$. The wireless transmission allows for a smaller device than traditional peacemaker which allows for a less invasive device and data transmission (for monitoring, controlling and simplified follow-up). The wireless EGMD can also work almost indefinitely without needing replacement.

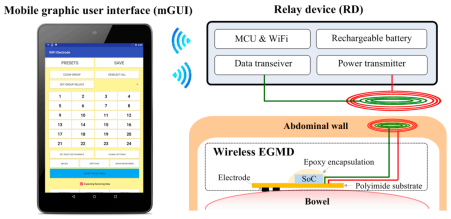

Fig. 2. General system with GUI, RD, power and data coils and GMD. [9]

c) Placement: The EGMD is inserted percutaneously on the surface of bowel and coils destined for power and data transfer are in a subcutaneous pocket in the abdominal wall as in Figure 2. The RD is carried by the patient for power and data transmission.

d) SoC: The SoC integrates several key functions, including a power converter, transreceiver, controller, motion recorder, and stimulation current drivers.
The power converter is used to get $\pm 1.8V$ and $\pm 12V$ voltages

in order to optimize the power consumption and allow the range of parameters we presented before. The stimulator needs $\pm 12V$ but the trans-receiver requires less power.

The receiver and transmitter use one coil in order to communicate and for better results, need separation during operation. To that aim, they have different signal frequencies, two different modulation schemes and different headers for starting each data sequence. A quasi full-duplex scheme was used so both receiver and transmitter can communicate simultaneously but not at the same frequency.

The controller used in this device is the NEural Command Signal Interface System (NECSIS). It is used to manage all signals coming from the motility recorder and user commands. It also manages signals going to the stimulator and data transmitted to the user.

The stimulation used for the device is a biphasic current controlled stimulation. A great choice as the current delivered to the tissue can be easily controlled. The control signal is inputted into the stimulation current control and with a level shifter and a HV output stage, they create the required stimulation. A charge cancellation switch is used as a current sink in order to discharge the electrodes.

The electrode records the bioimpedence to assess intestinal motility.The rhythmic impedance changes correspond to the slow-wave cycle of the muscle contraction. The signal is recorded into 12 channels selected by the demultiplexer, filtered into a high pass filter and finally converted into digital for transmission.

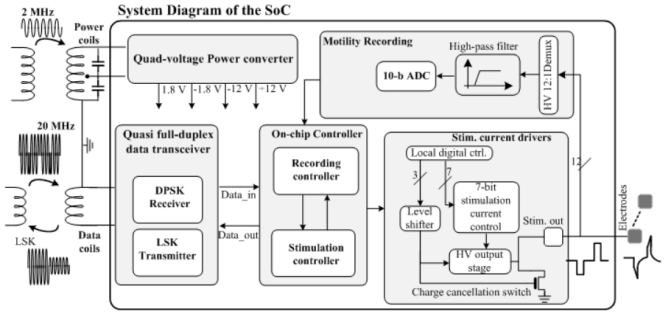

Fig. 3. Block diagram of the SoC with input and output [9]

e) Electrode array and package: The implant is made of 12 channels of 40 electrodes where each of them measures $46.7\mu m$ and each array measures $0.5mmx0.2mm$. The package used is a heterogenous System-in-Package (SiP) in order to size down the device. A biocompatible insulating epoxy covers the device to protect it. The entire EGMD has a volume of $\sim 0.5cm^3$ if the coils are aligned.

f) Experimental results: The device was evaluated through a bench-top characterization and in vivo experiments in rodent and porcine models. In bench-top testing, the SoC, driven by an external $16MHz$ oscillator to reduce noise [30], was controlled through a custom GUI in C#. Wireless power and bidirectional data communication were achieved with a Class E low power amplifier, a DPSK transmitter and a LSK receiver, through an inductive link. The stimulator successfully generated highly programmable current pulse trains with variable intensities, pulse widths, and leading polarities. In order to find the parameters to avoid undesired electrochemical reactions, cyclic voltammetry was performed in a $0.1M\ NaCl$ solution. The water window of $[-0,9V\ 1V]$ and the charge storage capacity of the electrode of $9.19\mu C$ were found. In vivo testing was performed in anaesthetized healthy Lewis rats by placing the electrode on the exposed caecum. The motility recording through multiple electrodes stimulating at $3\mu A$ and $0.1ms$ at $100Hz$ was confirmed by comparison with a pressure sensor. Stimuli of $1mA$ and $1ms$ at $100Hz$ evoked local contractions, but peristalsis was absent in benzalkonium chloride–induced aganglionic and slow-wave-blocked intestinal segments. Response time of smooth muscle contraction after stimulation in normal bowels decreased with increasing stimulation intensity, but decreased with higher frequencies. This suggests that different neural pathways were activated by different stimulation parameters. Pilot testing on intubate, anaesthetized juvenile porcine models, demonstrated the system's ability to induce smooth muscle contraction and motility measurement. The electrodes, placed on the serosal surface, allowed the recording of the impedance variation, but no peristalsis was observed. Studies on the porcine model are ongoing, to investigate the optimal stimulation parameters and assess the therapeutic potential of EGMD.

g) Competing technologies: Existing GI stimulation devices are predominantly powered by non-rechargeable batteries, increasing their volume, that ranges from 3.3 to $53.4cm^3$. These devices usually provide current stimulation, except for [31], but with limited parameter flexibility. Continuous stimulation is also rarely available, but it is demonstrated in [32]. Motility recording is sometimes integrated, but with other trade-offs, like in the piezoelectric system [31], where the sensing is given by bulky and energy demanding off the shelf electronics for the impedance analysis. The system presented in [33] provides motility recording through EGG and is wirelessly powered, but it is has larger dimensions and less freedom in terms of stimulation parameters. On the contrary, the previously presented device provides extremely configurable stimulation patterns, wireless programmability and motility recording, while maintaining a reduced size, allowing a one-time minimally invasive surgery.

II. Conclusion

Gastrointestinal motility disorders remain a major clinical challenge. Although electrical stimulation has emerged as a promising solution, the current implants remain bulky, with limited functionalities and without integrated sensing. The presented EGMD device promises to be a flexible stimulator with real time monitoring that would allow the needed further exploration in the field.

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