Wireless Intra-body Communication for Implantable and Wearable Body Devices using Galvanic Coupling

A Dissertation Presented
by

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To my beloved son Kishan Prajeesh!
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List of Acronyms

BAN  Body Area Network
BLE  Bluetooth Low Energy
CSMA  Carrier Sense Multiple Access
CSMA/CA  Carrier Sense Multiple Access with Collision Avoidance
DSSS  Direct Sequence Spread Spectrum
EBN  Extra-body Network
ECG  Echocardiogram
EEG  Electroencephalogram
EMG  Electromyography
FCC  Federal Communications Commission
FES  Functional Electrical Stimulation
FM  Frequency Modulation
GC-IBN  Galvanic Coupled Intra-Body Network
HBC  Human Body Communication
HF  High Frequency
IBN  Intra-Body Networks
ISM  Industrial Scientific Medical
kbps  kilo bits per second
LF  Low Frequency
Mbps  mega bits per second
MF  Medium Frequency
MICs  Medical Implant Communication Service

NB  Narrow Band

PWM  Pulse Width Modulation

RF  high frequency radio

SAR  Specific Absorption Rate

TDMA  Time Division Multiple Access

UHF  Ultra High Frequency

UWB  Ultra Wide Band

VHF  Very High Frequency
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Abstract of the Dissertation

Wireless Intra-body Communication for Implantable and Wearable Body Devices using Galvanic Coupling

by
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Implantable body sensors and actuators (eg., drug delivery pumps and neural stimulators) are becoming commonplace promising unprecedented improvements in personalized medicine. These miniaturized devices need to communicate with each other (i) to share the real time health data for actuation purposes, (ii) to upload the physiological data to a remote monitoring entity, and (iii) to dynamically configure implant sensing tasks with new instructions. Over-the-air radio waves are largely absorbed in human tissues and also leak into the environment up to two meters from the body. In this thesis we design a low power galvanic coupling based intra-body communication network (GC-IBN) that involves transmitting electrical currents and leveraging the natural conduction of the body tissues.

We first derive and experimentally validate expressions for the gain of the body tissue channel by building (1) a three-dimensional multi-layered tissue equivalent circuit model, and (2) a two-port network model that adapts for different body parts by enabling rearrangement of the signal source and pickup locations flexibly. Our models are sensitive to nearly ten different parameters, including electrode separation and tissue thicknesses.

Towards safe deployment of GC-IBN, it is critical to verify that the power injected into the tissue and the amount of heat generated during the signal propagation are both within the permissible limits. We next estimate the bounds on induced current that can be safely coupled to tissue. This induced current density and the frequency dependent power dissipation are measured and quantified to verify the safe limits. We then analyze the best and worst case for the thermal energy distribution within tissues for varying transmission power levels, number of co-located transmitters and blood perfusion conditions through finite element simulation and experiments on a human tissue phantom.
We make the first contribution towards building an energy-efficient topology through optimal placement of data collection points/relays using measurement-driven tissue channel models for GC-IBN and the Weiszfeld algorithm. Further, we show that the energy consumption over the entire implant network is balanced in our approach, while also meeting the application needs.

Finally, we propose a new cross-layer protocol for GC-IBN to address the limited energy constraint and co-channel interference among implants. Initially, we devise a method that allows multiple implants to communicate individual sensed data to each other through CDMA code assignments, but delegate the computational burden of decoding only to the surface relays. Then, we devise a distributed beamforming approach that allows coordinated transmissions from the implants to the relays by considering the specific tissue path chosen and tissue heating-related safety constraints. This collision-free protocol prevents undue interference at neighboring implants, and is the first application of near-field distributed beamforming in human tissue.

In summary, this thesis addresses the unique challenges pertaining to signal propagation through human tissue and takes experimentally proven steps towards practical intra-body networking. The tissue channel models, algorithms, protocols and safety guidelines devised can be integrated to build continuous, safe and energy efficient intra-body wireless communication that connects multiple implants and wearables to external world, paving way for the emergence of revolutionary medical applications.
Chapter 1

Introduction

Humans are embracing assistive technologies to unprecedented levels, allowing augmentation of natural abilities and restoration of lost-bodily functions. For e.g., in October 2016, the cyborg Olympics planned in Zurich will combine revolutionary advances of technology and medicine, thereby enabling physically disabled but cyber-physically empowered athletes to compete. The key enabling engineering feature that makes all this possible is the use of wireless communication technology for on and intra-body implantable devices. This facilitates transfer of instructions from an external controller to an array of implanted electrode cuffs that directly wrap the paralysis causing degraded neural endings in the patient’s limb [1]. Through this process, the limb movements can be stimulated artificially through external stimulus, mimicking the natural brain-controlled motion. This situation is further complicated as the post stimulation response from the limb, along with the other related physiological parameters such as body stability and muscle exertion levels must be monitored by the embedded sensors. This data is then transferred back to the external controller, thereby closing the loop of stimulus-actuation-feedback.

While this is only one representative example, such interconnected devices that compose an Intra-Body Networks (IBN) give rise to the ability to communicate with the external world. IBNs are poised to usher in dramatic improvements in personalized medicine, implant-based in-situ monitoring, controlled drug delivery, and activity based neuro-muscular stimulation, among others. However, the state of the art for wireless technologies for intra-body communication rely on high frequency radio (RF) signals that are highly absorbed by the human tissues, making RF-based communication unsafe and energy inefficient. In this thesis, we propose an alternative wireless architecture for IBN using galvanic coupling (GC), in which low-frequency weak electrical currents are modulated with the information signal and directly coupled to the tissue. We call this paradigm as Galvanic Coupled
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Intra-Body Network (GC-IBN) as the signals propagate within the body using its natural conduction properties.

1.1 Implants empowered personalized health care

Traditionally, vital signs are measured using wired electrodes worn on the body surface. Such electrodes and the connected sensors hinder the body movements, and hence, cannot be worn for longer periods. The wired electrodes provide ephemeral observations, capturing a snapshot of the prevailing health condition that may not reflect the exact in-situ complexities. As opposed to this, implanted sensors, allow continuous, convenient and accurate local observations deep in the tissues without restricting user activities. A separate class of implanted devices called actuators that are drug delivery devices (for e.g., a pain relief diffusing agent implanted at the spine) or the neural stimulator at the nerve endings described earlier, offer unmatched and powerful therapeutic efficacy without any influence on other body parts. Today, the number of devices implanted directly inside the human organs, augmented by the ultra-miniaturization of body devices using flexible nano or MEMS technology are growing [2]. The general population is slowly opening up to the benefits of such in-situ monitoring, as continuous improvements are being demonstrated in the accuracy of sensing, security of communication and ease of deployment thanks to advancements in minimally invasive implant technology.

Working with implant heterogeneity:

Given the very large number of ailments that can affect a person, it is difficult to imagine one single type of implant being able to gather all the necessary data for correct diagnosis. These different types of implants further require different data rates, duty cycles, energy consumption levels, among others. We believe that future IBNs will necessary involve high levels of heterogeneity, as illustrated below using a simple example of a network that provides assistive services for elderly patient care.

Many applications for autonomous health monitoring in elderly populations have been developed using a multitude of off-the-shelf body devices, such as a body movement and activity detector using 3-axes accelerometer, magnetometer and gyroscope sensors, fall detection sensor, pain relieving implant (e.g., pstim [3]), bladder volume sensor and bladder control actuator [4]. Table [1.1] summarizes these different body devices along with the specific location of these devices within the
CHAPTER 1. INTRODUCTION

body, the data rate required to communicate and the direction of major information flow (uplink - from the sensors to IBN, or downlink - from IBN to the actuators). Although, it is not required to embed all the listed devices in a person, often a large subset of these devices may be relied upon for remote monitoring and health management.

The devices embedded in body handle data that are highly inter-correlated. For instance, when an elderly person experiences a fall, the deviations of the movement from previously established reference signatures will be reported by the activity sensor as well as by the fall detection sensor. The incident will also impact heart rate, blood pressure, perspiration rate and body temperature, possibly resulting in an unexpected spike. If the same area is undergoing a pain control treatment, then the drug delivery actuator may also initiate a proactive delivery of the required dosage. Thus, a new event will be recorded in multiple sensors leading to a surge in the body network traffic. This correlation in the physiological observations cannot be fully leveraged if these sensors are independently monitored. Instead, when the devices are connected by a single IBN holistically, the root cause for sudden surge in traffic can be easily identified and the available bandwidth can be efficiently utilized for monitoring or actuating the critical component (the body movements and heart rate in this case). In certain scenarios, the fall can also be prevented if the incongruity in movements or the body imbalance can be sensed pro-actively and the stability be restored with the help of a gait control stimulator or neuroprosthetic control stimulator (e.g., NEUWALK [5]). In this case, the entire control loop is built inside the body providing negligible communication latency and data handling errors ensuring timely response and care.

For the example showing the caregiving scenario for elderly subjects, more than 30% of the body devices are implantable, as illustrated in Fig. 1.1. However, there are several challenges associated with implant communication occurring through tissues. The transmission signal used to enable communication between implants and IBN, must propagate through tissues and hence, should use frequency spectrum where the signals are absorbed by the tissues largely composed of water. Additionally, there should be sufficient bandwidth for reliable communication, without elevating the tissue temperature even after continuous transmission durations. Also, the IBN built over the through tissue links must coordinate several embedded devices, each with distinct capabilities and requirements in terms of bandwidth and reliability. For instance, a temperature sensor has very low data rate requirement (≈ 10 bps), while an endoscopic image capturing capsule moving through the digestive track requires very high data rate (≈ 1 Mbps). Adding to the complexity, both the bandwidth availability and demand can vary dynamically based on the tissue channel condition and health condition respectively. As an example of changes in channel condition, a well hydrated tissue
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Table 1.1: Body devices (wearables and implants) used for elderly care (* - implanted devices)

<table>
<thead>
<tr>
<th>Device</th>
<th>In-body location</th>
<th>Data rate</th>
<th>Data flow direction</th>
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<tr>
<td>Temperature</td>
<td>Forehead</td>
<td>48 bps</td>
<td>Uplink</td>
</tr>
<tr>
<td>Heart Beat Rate</td>
<td>Finger tip</td>
<td>48 bps</td>
<td>Uplink</td>
</tr>
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<td>Pulse Oximetry</td>
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<tr>
<td>pH Sensor*</td>
<td>Implanted in oral cavity</td>
<td>48 bps</td>
<td>Uplink</td>
</tr>
<tr>
<td>CardioMEMS</td>
<td>Pulmonary artery</td>
<td>1.44 kbps</td>
<td>Uplink</td>
</tr>
<tr>
<td>Fall detection</td>
<td>Waist band</td>
<td>1 kbps</td>
<td>Uplink</td>
</tr>
<tr>
<td>Barostim Sensor*</td>
<td>Carotid artery of neck</td>
<td>48 bps</td>
<td>Uplink/downlink</td>
</tr>
<tr>
<td>Barostim stimulator*</td>
<td>Carotid artery at collar bone</td>
<td>48 bps</td>
<td>Uplink/downlink</td>
</tr>
<tr>
<td>Bladder volume sensor*</td>
<td>Bladder</td>
<td>48 bps</td>
<td>Uplink</td>
</tr>
<tr>
<td>Bladder control*</td>
<td>Under abdomen skin</td>
<td>48 bps</td>
<td>Uplink</td>
</tr>
<tr>
<td>pstim* - pain reliever</td>
<td>Ears (battery behind ears)</td>
<td>48 bps</td>
<td>Uplink/downlink</td>
</tr>
<tr>
<td>Artificial Retina*</td>
<td>Behind ear</td>
<td>36 kbps</td>
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<tr>
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<td>Uplink/downlink</td>
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<tr>
<td>Hearing Aid</td>
<td>middle-ear or cochlear</td>
<td>10 kbps</td>
<td>Uplink/downlink</td>
</tr>
<tr>
<td>1 point ECG</td>
<td>On chest</td>
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<td>Uplink</td>
</tr>
<tr>
<td>Glucose monitor</td>
<td>Beneath skin at abdomen</td>
<td>32 bps</td>
<td>Uplink/downlink</td>
</tr>
<tr>
<td>Insulin Pump*</td>
<td>Inside abdomen</td>
<td>32 bps</td>
<td>Downlink</td>
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<tr>
<td>Plethysmogram*</td>
<td>Vein implant</td>
<td>48 bps</td>
<td>Uplink</td>
</tr>
<tr>
<td>e-AR gait sensor</td>
<td>Ear canal</td>
<td>200 bps</td>
<td>Uplink</td>
</tr>
</tbody>
</table>

will have higher conductivity and hence improved channel gain compared to the dehydrated tissue. Routine activities such as fluid intake, exercising and showering could influence the network behavior. Hence a comprehensive tissue channel analysis accounting the influencing factors is required for the IBN design. There are several such unique challenges to be addressed and critical requirements to be managed by the IBN involving implants, which are taken as the subject of this dissertation.

1.2 Building blocks for implant communication

A large body of work concerning intra-body communication has focused on using high frequency radio (RF) signals originally intended for over the air communication. RF based IBN would incur heavy energy costs owing to high absorption within the human tissue. The communication also becomes environment-dependent with limited security as the signals extend to at least six feet around the body. Among the other candidate technologies for IBN, capacitive coupling needs a ground connection for the link set-up and hence, is suitable only for wearable devices. Inductive coupling technique have found applications in integrated wearable devices within clothing. However, they require coils on transmitter and receiver side, which requires the devices to have larger surface area
for sufficient coupling of the energy. Therefore they are not suitable for implants with small form factors. Fundamentally, all the above techniques use high frequency RF signals that are not suitable for propagation through tissues. A novel approach of using ultrasonic waves has been demonstrated recently which has many benefits of safety, proven technology, reduced energy consumption, and high directionality. However, links formed by ultrasound are susceptible to propagation delays and multipath effects, which in turn requires complex transmitter-receiver circuitry.

In contrast to these various signal propagation techniques, galvanic coupling (GC) of weak, low or medium frequency electrical currents to tissues is a different technique that we propose to use in this thesis for IBN. The GC-IBN paradigm safely transmits data through tissue using two orders of magnitude less energy than RF signals (see Chapter 2), and also without requiring grounded terminals, larger coils or complex circuitry. Galvanic coupled links can be established using simple transmitters and receivers as the signal propagation delay is negligible. Hence we believe galvanic coupling is the appropriate technology for building an IBN and we extensively develop both the theory and systems for realizing this technology.

1.3 Galvanic Coupled Intra-body Network

In GC-IBN the communication link is established using low/medium frequency (100 kHz—1 MHz) and low power (≤ 1 mW) electrical signals. The basic principle of establishing a galvanic coupled link through the tissue is given below.

**GC-IBN working principle**

In GC based communication, a pair of electrodes within a given IBN node couple the weak electric signal to the body tissues, which is initially modulated by the sensor data [6]. The induced
field in the tissue is well below the permissible limit of $25 \text{ mA/m}^2$ \cite{7,8}. Majority of the induced current that is coupled to the body passes through the return path of transmitter (represented by black arrow in Fig.\ref{fig:1.2} and a minor part (illustrated by gray arrows, which is $\approx 8 \text{ dB}$ for surface node and $\approx 5 \text{ dB}$ for implants) propagates through the body. The difference in induced potential is detected by the electrode pair of a receiver node. The receiver demodulates the signal to receive the sensor data. Note that there is no common ground required here, as in the case of capacitive coupling \cite{18}.

A characteristic feature of GC-IBN is that the signal has a dominant component propagating through the inner tissue layers, even when the transmitter is placed on the surface \cite{32,8}. Thus, apart from being more energy efficient compared to the peer technologies using RF links, capacitive coupling or induction coupling, the galvanic coupled communication is less impacted by external environment. In addition, the cost-effective GC transceivers provide continuous real time connectivity. A carefully designed coupling device operating with optimized signal amplitude and frequency gives rise to a dominant signal component that can be guided to traverse through specific part of the body. Thus, multiple concurrent transmissions along the same body becomes possible, leading to new challenges in interference-free operation. This behaviour differs from RF propagation in free space, wherein other transceivers must be silenced owing to the broadcast nature of the medium.
CHAPTER 1. INTRODUCTION

Advantages of GC-IBN

The various studies and experiments that we conducted on GC-IBN reveal several advantages of GC links over the existing peer technologies as listed below:

- The low frequency signal is not absorbed by the tissue, and hence, the elevation in the local tissue temperature is negligible. This ensures safe communication inside the body in contrast to the RF signals.
- The communication is energy efficient offering long battery life for implants.
- GC-IBN can coexist with other wireless applications as the operating frequency is entirely different from that of existing over-the-air RF technologies.
- GC links are short range links extending to nearly 30 cm through body and not more than a few centimetres external to the body. This body confined communication offers secured transmission and minimal environmental influence.
- GC links guarantee acceptable bandwidth for high data rate applications.
- GC-IBN requires simple protocols and transceiver circuitry allowing cost-effective devices and network deployments.
- Continuous unobtrusive real time connectivity is possible while permitting the patients to lead a normal life.

1.4 Thesis Contributions

In the following, we briefly describe the various challenges in building GC-IBN and highlight how they are addressed in the rest of the thesis.

Tissue equivalent channel models

Ideally for an authoritative study of the tissue channel, the signal propagation characterization must be done through real experiments. However, in-vivo tissue experiments involving human require several levels of permissions, which is challenging unless the GC-IBN is proven to be a matured technology. Hence, a unified analytical model for the channel gain for weak electrical signal propagation through various tissue layers is required. We build a three dimensional multi-layered...
tissue equivalent circuit model with the ability to reproduce results and to accurately predict the channel gain across the skin as well as across and through the inner body tissues as shown in chapter. The model constructed accommodates a variety of design parameters. We also build a two-port network based model that adapts to different body parts with different tissue arrangement. These models are then verified through extensive finite element simulations and published measurements in existing literature. The empirical measurements obtained from a test bed with porcine tissue are used to validate the models built. The chapter also presents a model sensitivity analysis performed with respect to multiple design parameters.

**Tissue safety assurance in GC-IBN**

Before deploying the GC-IBN technology for medical use, it is critical to ensure the tissue safety aspect in terms of the permissible limits on the magnetic and electric field strength coupled to the body tissue and the amount of heat generated in tissue during signal propagation. We conduct a safety analysis of GC-IBN in chapter first, in terms of the induced electrical ($E$) and magnetic ($H$) fields using the Maxwell’s boundary conditions derived at the electrode-tissue interface. Second, we obtain the tissue thermal distribution analysis for varying power levels, number of collocated transmitters and blood perfusion conditions using the finite element simulation and skin phantom based empirical set-up. Our analyses confirm the tissue temperature elevation being well below the suggested safe limit. We also derive the tissue thermo-regulation strategies under abnormal body conditions using suitable transmission duty cycle, transmission sequence, transmitters separation distance and number of concurrent transmissions that bounds the maximum tissue heating to a level that do not contribute towards core body temperature (within 1°C). The proposed duty cycling and sequencing strategies improve the bandwidth efficiency upto 85% providing sufficient bandwidth for the implant communication.

**In-body node positions & topology optimization**

The unique intra-body channel raises several questions on the topology of the implants and the external (i.e., on skin) data collection nodes. We make the first contributions in chapter towards (i) building an energy-efficient topology through optimal placement of data collection points/relays using measurement-driven tissue channel models, and (ii) balancing the energy consumption over the entire implant network so that the application needs are met. We achieve this via a two-phase
CHAPTER 1. INTRODUCTION

iterative clustering algorithm for the implants and formulate an optimization problem that decides the position of external data-gathering points.

Beam-forming in the Body: Energy-efficient and Collision-free Communication for Implants

Implants operate under challenging constraints of limited battery energy, heterogeneous tissue-dependent channel conditions and human-safety regulations. To ensure reliable and energy efficient communication for implants and to address the above mentioned challenges, we propose a new cross-layer protocol for GC-IBN. As the first step, we devise a method that allows multiple implants to communicate individual sensed data to each other through CDMA code assignments, but delegates the computational burden of decoding only to the on-body surface relays. Then, we devise a distributed beamforming approach that allows coordinated transmissions from the implants to the relays by considering the specific tissue path chosen and tissue heating-related safety constraints. This collision-free protocol that prevents undue interference at neighboring implants, especially for multiple deployments. This is the first application of near-field distributed beamforming in human tissue. Results reveal significant improvement in the network lifetime for implants of up to 79% compared to the galvanic coupled links without beamforming.

1.5 Outline of the Dissertation

The thesis is organized as follows.

In Chapter 2, we review the existing candidate technologies for IBN and summarize the research contributions related to GC-IBN.

Chapter 3 describes the construction of the three dimensional tissue equivalent models: circuit model for human forearm and two-port network model for human arm that are verified using the Finite Element Methods (FEM) based simulations and validated by the testbed using porcine tissue. Further, the sensitivity of the model is analyzed for ten different parameters influencing GC-IBN design.

Chapter 4 analyses the galvanic coupled tissue safety in terms of the induced $\vec{E}$ and $\vec{H}$ fields and the rise in tissue temperature. Suitable transmission strategies are proposed to regulate tissue temperature.
CHAPTER 1. INTRODUCTION

In chapter 5, an energy efficient and energy balanced GC-IBN topology is proposed along with a two-phase iterative algorithm for finding optimal positions of the signal pick-up points.

Chapter 6 outlines our proposed cross-layer protocol for GC-IBN using beam-forming that alleviates interference for neighboring nodes as well as offer dramatic life extension for implants.

Finally, Chapter 7 concludes the dissertation and presents the potential directions of future exploration in GC-IBN.
Chapter 2

Related Work On Intra-body Networks

Intra-body network interconnects multiple devices in and on the human body for collecting the physiological information from body sensors and dispensing instructions to the embedded actuators. We anticipate a well designed IBN to possess the following properties:

- The ability to handle heterogeneous real time data that is generated continuously in the body
- The ability to form safe, secure and reliable communication links through the heterogeneous tissue medium
- The ability to provide energy-efficient links that guarantee long life for the implants

Several existing technologies allow wireless communication among the body devices. In this chapter, we classify the related networking technologies based on the nature of signals used for communication into (i) electromagnetic IBN and (ii) ultrasonic IBN. We start from this high level classification as the tissue medium can carry both the electromagnetic (EM) waves and ultrasound waves. While the ultrasound waves are used in a specific narrow spectrum within body, the EM waves are deployed in various ranges of frequency. The communication in each frequency range is formally specified by a set of standards (e.g., ZigBee) and by specific mechanisms by which the signal is fed to the body (e.g., antenna radiation, galvanic coupling, induction coupling and capacitive coupling). We use the difference in frequency, standards and signal coupling mechanisms to classify the various forms of electromagnetic IBN as given in Fig[21]. We discuss the related contributions in each of these forms, highlight their pros and cons, and motivate the need for the galvanic coupling based IBN in this chapter.
2.1 Electromagnetic Intra-body Networks

The frequency of communication plays a key role in determining the quality and quantity of signal propagating through the tissues and also impacts the other primary communication parameters such as tissue absorption, scattering, and dissipation. We proceed with our frequency based classification as microwave IBN, HF-UHF IBN (gamma dispersion range) and LF/MF IBN (beta dispersion range), as given below. We do not consider the alpha dispersion range (10 Hz to a few kHz) for IBN, as some of the naturally occurring physiological signaling operates in this range (e.g., Electroencephalogram (EEG), Electromyography (EMG)).

2.1.1 Microwave signal based Intra-body Networks

While microwave EM signals are well suited for over the air communication reaching long distances, it is not the right choice for IBN communication for the following reasons: The higher range of frequency starting from High Frequency (HF) in radio spectrum to microwave spectrum has high Specific Absorption Rate (SAR) in tissues, resulting in tissue heating effects and high energy consumption. Also, at frequencies higher than 10 MHz, the signal wavelength becomes comparable to the human body dimensions making the body act as an antenna radiating EM energy to several meters around the body (refer Fig. 4.1). The energy radiated out cannot be used for in-body communication and hence is considered wasted that dramatically reduces the energy efficiency. The energy spreading out also incurs a high level of interference from plethora of surrounding wireless devices. This, in addition to the body’s shadowing effects, renders the communication link less secured and unreliable. The majority of existing Wi-Fi based IBN technologies operate in this frequency range.

Wi-Fi standards

The Wi-Fi or WirelessLAN technologies such as IEEE 802.11.a/b/g/n provide high speed Internet access for computing gadgets extending coverage to about 50 m indoors and 100 m outdoors at 2.4 GHz and/or 5 GHz Industrial Scientific Medical (ISM) bands. For IBNs, the retrieval of sensors for battery replacement becomes impractical, requiring efforts on reducing energy consumption for data aggregation and communication. We shall demonstrate subsequently in chapter 4 that IBN using RF signals raises the local tissue temperature, making it infeasible for extended durations of communication. Also, when there are multiple nodes in/on the same region of the body, each...
### CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

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*Figure 2.1: Communication technologies used for body area communication*

A sensor that requires continues and uninterrupted access needs a RF channel to communicate. Such a dedicated assignment of channels can potentially clog the already saturated hospital Wi-Fi.

There are many personal area networking standards in this ISM frequency range that provide the framework of protocols and physical layer specifications to enable low power body area communication. Few of them are highlighted below:

**Bluetooth Technology**

Classical bluetooth technology (BT) is a widely used technology for networked body devices, which is standardized by the task group 1 of IEEE 802.15. BT defines a packet-based framework with master-slave architecture for connecting upto 7 devices (slaves) in the personal operating space. It uses Ultra High Frequency (UHF) radio waves in the ISM band from 2.4 to 2.485 GHz supporting upto 1 Mbps data rate and covering upto a distance of 100 m. In the active state, a slave should be on for almost the whole duty cycle to keep it synchronized with the master. This consumes more energy with a peak current requirement of 30 mA with 1 W as reference.
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

Bluetooth Low Energy Technology

Bluetooth Low Energy (BLE) or Bluetooth Smart or Bluetooth 4.0 technology, developed for low energy devices such as the body sensors consumes only half of the peak current consumed in classical Bluetooth (15 mA) with a reference power reaching a maximum of 0.5 W. Through a simplified modulation approach, the maximum data rate offered is about 0.27 Mbps while the signal can covers around 100 m. [33] uses polling based connection set-up from the three axis accelerometer, gyroscope and magnetometer, where the node sends a request when a connection to the central coordinator is to be made.

Zigbee standard

Zigbee is another popular Wireless Personal Area Communication standard (WPAN) extensively used for body area networks ([34, 35, 36, 37]), internet of things and home automation. It is extended from the physical and data link layer specification in IEEE 802.15.4 with new network and application layers. The lower layers rely on Direct Sequence Spread Spectrum (DSSS) for interference mitigation and contention based Carrier Sense Multiple Access with Collision Avoidance (CSMA/CA) for channel access. Zigbee uses low power compared to Wi-Fi standards with low duty cycles and offers lower data rate of about 20 kbps at 868 MHz, 40 kbps at 915 MHz and 250 kbps at 2.4 GHz. The technology can cover a distance of upto 100 m and can connect upto 65,000 devices. [35] defines the hardware and software architecture for wireless health care network with provision for node naming, discovery, any-to-any ad-hoc routing, authentication and encryption.

IEEE 802.15.6 standard for BAN

The state-of-the-art body area communication is regulated by an exclusive standard IEEE 802.15.6 that accommodates both high frequency and microwave spectrum based physical layers. The Narrow Band (NB) PHY and the Ultra Wide Band (UWB) PHY operate in the microwave spectrum that use Wi-Fi, bluetooth or Zigbee standards [39] discussed above. The NB PHY protocol operates on 402 – 405 MHz, 863 – 956 MHz and 2.36 – 2.4 GHz spectrum for implants, wearable and medical links respectively, offering 0.1 – 1.0 Mbps data rates. The UWB PHY operates in the 3 – 5 GHz and 6 – 10 GHz bands occupying 499.2 MHz bandwidth and offers 0.4 – 12.5 Mbps data rate.

The HF based physical layer called Human Body Communication (HBC) PHY in IEEE 802.15.6 standard offers to connect utmost 256 devices per Body Area Network (BAN), using
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

5.25 MHz bandwidth in the 21 MHz band. HBC offers high data rate up to 1.3 Mbps, but consumes a high power of about 40 mW power in active mode. The HBC PHY offers direct access to extra-body wireless network up to a distance of 10 m (normally 2 – 5 m) as it does not confine inside the body. This spreading into the environment makes the signal prone to environmental influence and interference. Also the longer links consume more transmission energy.

2.1.2 HF/VHF Radio based Intra-body Networks

At the High Frequency (HF) or Very High Frequency (VHF) radio spectrum, the signal wavelength becomes comparable to body dimensions and hence the body acts as an antenna radiating the signal to a few meters (around 2 – 4 m) away from the body. The signal comprises of a strong displacement current and a weak conduction current and hence can be transferred to the body using loose coupling of electric field or magnetic field (or induction coupling). A simple form of electric field coupling is called as capacitive coupling, which is described as follows.

**Capacitive coupled IBN**

Capacitive coupling transfers signals to the body using differential pair of electrodes at both the transmitter (Tx) and receiver (Rx) side \[ 18 \]. The electrodes are not directly attached to the body but loosely coupled or placed in proximity with the reference terminal of both Tx and Rx that are connected to the ground. When a signal is applied across Tx electrodes, an electric field is induced proportional to the capacitance formed by electrodes and the tissue in between. The induced field spreads through the body surface as varying electrical potential, which is detected with respect to the reference electrode by another pair of receiving electrodes at another part of the body. The frequency of operation ranges in between 10 MHz and 50 MHz, offering data rate of around 48 kbps.

This set-up cannot be used to connect implants as it requires a guided return path of the signal in the form of a ground connection. The dominant signal path of capacitive coupled links is not inside the body but on the air as a surface wave extending to several feet around the body making it susceptible to environmental influences from other RF and electronic systems rising concerns on security. In addition, the capacitive plates themselves are likely to emit stray fields.

**Induction coupled IBN**

IBN established using magnetic or induction coupling uses a coil in the implant (acting as either transmitter or receiver) that is inductively coupled to another coil in the external device. When
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

A transmitting coil is excited, the varying magnetic field between the two parallel coils induces a change in voltage along the receiving coil. The quality of induced induction is proportional to the intensity of the current and voltage in the coils and hence the coils are to be placed closer to the body surface so as to reduce the signal fading with distance of separation. The induction link can be used for both energy transfer and data transfer. Possible data rate extend upto 204 kbps at the 13.5 MHz band [20].

Higher power transfer efficiency in induction coupled IBN requires larger coils in the devices that in turn would require devices with larger surface area. This is a challenge for the implants with small form factor. Also, the short range of communication requires perfect node alignment that is not always possible with implants. Moreover, the communication can only be initiated from the external to internal node. Incidental communication initiation from the internal node on account of an emergency condition cannot be supported by the inductive coupling based body communication.

2.2 Ultrasonic Intra-body Networks

Ultrasound signals propagate better in the water based medium and hence are considered as an effective alternative to microwave signals and HF/VHF radio signals for establishing IBN through human tissues [29]. The ultrasonic wave propagation is considered safe at frequencies around 100 kHz that offers reliable communication through a few centimeters in tissues. However, the transmitter-receiver circuitry requires suitable transducers that are difficult to miniaturize. Also, the presence of intermittent gaseous substance alters the channel characteristics that is undesirable for intra-body communication, particularly for the implanted scenario in the thoracic cavity.

The factors like heterogeneous tissues with irregular structure, higher propagation latency, and the multi-path propagation of ultrasound signals through tissues result in scattering of ultrasound waves. [29] introduces a Ultrasonic WideBand (UsWB) technology, comprising the physical and medium access control protocol layers. UsWB uses short information-bearing carrierless ultrasonic pulses that follows a time hopping pattern to combat the multipath and scattering effects. Thus, sophisticated signal processing techniques and transceiver hardware are required that increase the system complexity.
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2.3 Galvanic coupling based intra-body networks and background

Motivated by the features and limitations of the above methods, we adopt a new IBN paradigm where implants may communicate wirelessly using galvanic coupling (GC) of weak low frequency electrical signals to the tissues to enable IBN, called GC-IBN. Our choice of using galvanic coupling as method for transmission through tissues is motivated by the high water content within the human body, which facilitates the propagation of EM waves in Low Frequency (LF) or Medium Frequency (MF) range.

The signal can propagate through longer tissue paths leveraging the conduction properties of body tissues and lower signal absorption defined by the minimal SAR in this spectrum. The dominating conduction current keeps the signal confined within the body, with negligible quantity of signal leakage to environment. Thus the LF/MF based GC-IBN surmounts the problem of massive signal absorption in contrast to the RF links, making it energy-efficient as well as secured from the environmental influence (refer chapter 3.2 & Appendix B). Furthermore, in the LF/MF spectrum, the signal wavelength is larger than the human body dimensions, minimizing the undesirable effects from propagation latency, nulls, scattering and multipath fading effects. Thus, we believe using GC based links for IBN has an advantage over other existing technologies.

2.3.1 Related work on Galvanic Coupled IBN

The practice of injecting current through body has been used in health care applications such as Body Impedance Analysis (BIA) and diathermy for many decades. However, the usage of the human body as the medium of communication and the possibility of establishing communication links using LF/MF signals through tissues were not explored until late 90s. In 1997, Takash Handa et al. proposed the first low power AC micro current based body sensor network for wearable Echocardiogram (ECG) sensor. A contemporary work was proposed by Derek P. Lindsey et al. for implant to surface node communication leveraging the ionic conduction properties of human tissues. Through their communication set-up, the authors identified 37 kHz to be the optimal frequency of operation with 3 mA of input current. Subsequently, Michael Oberle, in his Ph.D. thesis, presented the galvanic coupled approach consuming 500 $\mu$W with improved spectral efficiency for implanted blood pressure sensor. Keisuke Hachisuka et al., used Frequency Modulation (FM) based transmitter-receiver designs working at 5 MHz and 10.7 MHz as the frequency of operation and transmitted ECG signals from chest to arm. Wegmuller et al., determined the maximum distance through tissue offering reliable channel gain in the frequency...
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

range in 10 to 27 MHz with an input current of 1 mA at 3.3 V. Using a tissue simulating phantom they demonstrated a data rate of 4.8 kbps. The authors presented a transmitter-receiver architecture for GC-IBN that is used for clinical trials with various electrode materials and adhesives.

Several contributions were made towards modeling the galvanic coupled tissue channel [41, 42, 43]. Wegmuller [30] proposed a tissue equivalent planar channel model comprising two directions of current flow through tissue. The model was then evaluated using an in-vivo measurement setup. However, efforts on building a comprehensive heterogeneous tissue channel with realistic current propagation directions, creating realistic network architectures of multiple embedded implants, formulating safety guaranteeing protocols and algorithms were not developed as part of prior work, thus framing the motivation of this thesis.

2.3.2 Related work on channel models

Well understood tissue channel characteristics would enable efficient network design and energy budget in GC-IBN. However, preliminary in-vivo tissue experiments are not practical prior proper GC-IBN design. While OTA-RF communication is well understood despite its high absorption level within the body, a unified analytical model for the channel gain for weak electrical signal propagation through various tissue layers remains in a nascent stage. Among the different techniques available for modeling the tissue electrical behavior, quasi-static approximations, full wave numerical techniques such as Finite Difference Time Domain Method (FDTD), Finite Element Analysis (FEA), and Equivalent Circuit Analysis (ECA) based modeling are the main approaches. The quasi-static field distribution analyses are computationally efficient. However, they only represent low frequency approximations to Maxwell’s equations and cannot be relied on for high frequency applications. Field analysis using FDTD and FEA are flexible and accurate but require a great deal of time for computing, and find limited application in a rapid deployment of an IBN. The ECA model offers a simple transfer function valid for a wide range of frequency, with the advantage of accurate and instantaneous gain computation making is feasible for IBN deployment for time-sensitive healthcare applications. Wegmuller proposed a two dimensional (planar) tissue equivalent channel model [46] and proposed a measurement setup for in vivo verification of the simulation results.

However, most of the existing approaches [47, 48, 49, 46] consider single tissue layer with limited flexibility, which we aim to overcome in our proposed work. Additionally, works that consider the multi-layer effect [49] include only bidirectional signal propagation paths (longitudinal and cross
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

paths) between transmitter and receiver. The direct path between the transmitter terminals that depends on the underlying tissue impedance is assumed to be measurable at the electrode attachment site [46, 50], which limits its practicality. Also, the transverse path from one tissue to other that depends on the tissue thickness is neglected. The tissue equivalent model needs to be asymmetric as opposed to the existing models to account for dissimilar dimensions, tissue heterogeneity, and non-identical electrode set-up at transmitter and receiver, which significantly complicates the analysis. These models have limited flexibility and are restricted from considering multiple possible paths of current in the three dimensional heterogeneous tissue medium and thus requires further investigation. We derive an extensive three dimensional model to analyze the electrical behavior of human tissues in Chapter.3.

2.3.3 Related work on analyzing human tissue safety

The primary concern regarding signal propagation through the human tissues is the safety for tissues. High frequency and high power signal transmission through tissues might lead to elevated body temperatures. Tissue heating caused by medical procedures such as diathermy, RF ablation, MRI and ultrasound procedures [64] have been extensively studied in the past. Similarly, heating effects of implants have been investigated in the high frequency range [65] and in the MHz and GHz frequency range [67] for understanding the Specific Absorption Rate (SAR) from the radiation effects. In the range 100 kHz to 1 MHz, tissues have higher conductivity and offer longer signal propagating paths. Owing to this reduced attenuation, and hence absorption, the rise in temperature is also on the lower side. While the tissue heating effect of the higher kHz range has been analyzed for high power applications such as Diathermy, the impact of low power galvanic coupled links with possible multiple concurrent transmitters has not yet been studied. We study the thermal distribution in human tissues with single and multiple concurrent transmitters in tissues as well as devise strategy to control medium access that avoids tissue heating when the blood flow rate is affected.

2.3.4 Related work on identifying optimal topology for GC-IBN

The short links in GC-IBN (≈ 30 cm [70]) and the varying body channels require dynamic cluster formation. However, analyzing all possible solutions for relay placement is an NP-hard problem, and the short time-scales suggest the use of heuristic approaches. We further discuss additional design considerations and topology optimizing techniques with node clustering in Chapter.refchap:topology.
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

**Tissue safety:** WSN has the option of using high power long range links to communicate directly with the sink or access point avoiding multi-hops [74]. This is not possible in GC-IBN owing to the tissue safety requirement of restricting to the pre-specified transmission power level. The maximum power transferred though the tissues is required to be lower than the safe level suggested by ICNIRP [8].

**Clustering constraints:** Equitable distribution of energy within classical WSNs is achieved by rotating the role of the cluster head (analogous to relay node, in our case) [66]. The GC-IBN is constrained to have the relay on the skin-surface, and hence the implanted nodes are no longer candidates for role switching. In the general case, WSN protocols assume the bulk of traffic flows in a single direction (i.e., transmit-only sensors and receive-only sink), while a typical GC-IBN with sensors and actuators involve bidirectional traffic. Moreover, as the GC-IBN comprises of non-redundant implants, the network is considered operational until the first implant runs out of energy. This is in contrast to the WSN scenario, where the cluster remains useful as long as a reduced subset of sensors is available.

**3-D propagation:** Traditional 3D clustering approaches like [75] handle all three dimensions equally. However, with the GC transmitter on surface, a receiver at tissue depth receives a stronger signal than a receiver on the skin surface at the same distance owing to the superior conducting properties of the inner tissues. Straightforward application of techniques, such as K-Means clustering that have been applied to terrestrial WSNs, do not account for the different propagation paths and are unlikely to perform the required optimal partition as the number of clusters and initial seed value are unknown. In order to use the instance based classification clustering such as k-Nearest Neighbors algorithm (kNN), the raw data is to be pre-processed for identifying the outliers (such as no node or isolated node condition).

**Relay positioning constraints:** Classical WSNs have uniform distribution of nodes, which also results in spread of cluster heads throughout the area under study [68]. However, implant locations are influenced by medical applications, and these may result in small pockets of deployment. Thus, the distribution of the relay points in this case is non-uniform. Moreover, relays must forward information among themselves, serving as a conduit for messages among the sensors, instead of direct communication between multiple implant pairs (e.g., $R_1$ and $R_2$ forward information, instead of $N_1$ and $N_2$ directly). This ensures lower energy consumption for the implants, but imposes constraints on the number of nodes connected to relays. Finally, earlier works on relay positioning for on-surface nodes are not suitable for implants [76], which makes the current problem scenario novel.
CHAPTER 2. RELATED WORK ON INTRA-BODY NETWORKS

2.3.5 Related work on beamforming inside the body

We propose a beamfoming based MAC access strategy an energy efficient approach to enable communication from implants to surface relays. Existing standards for Wireless Body Area Communication (WBAN), including IEEE 802.15.4 based LR-WPAN (Zigbee), IEEE 802.15.6 Human Body Communication (HBC) standard and Bluetooth low energy (BLE), assume that implants are similar to classical over-the-air wireless sensors. This is because in both cases, the nodes are battery powered, have small form factors, with low on-board resources. Classical CSMA/CA [78, 79] and channel hopping used in these standards impacts definite time of delivery, energy efficiency, and is unable to handle sudden spikes in traffic. The frame-length and inter-frame spacing are designed for high frequency signal propagation in the air medium over long distances (>2 m), rather than the low frequency short range communication (<50 cm) inside the body. Other overheads such as handshakes, channel sensing, scheduling, transitions from frequent sleep and wake-up states, among others, increase the processing complexity. An alternative form of intra-body links established using ultrasonic signals suffer from high multi-path delay and complex circuitry.

We note that the low rate and sparse traffic generated by implants under normal physiological conditions may become bursty when an abnormal event is observed, limiting utility of both contention-based and reservation-based access techniques. Hence, for contention and reservation-free access, we advocate the use of [80] that enables concurrent transmissions. However, CDMA multiplies the energy costs by using a high rate code, which in turn contributes to the net energy consumed per unit of useful data. Hence, in this paper we design smart energy-focusing strategies. Seminal contributions for conventional beamforming in far-field, high frequency signals exist [81]. However, the problem of beamforming for near-field and narrow band signals in a heterogeneous tissue-like medium has not been demonstrated so far, particularly for the low frequency signals (<1 MHz) used in GC-IBN. Coordinated beamforming using multiple separate antenna elements may be possible in many applications where implants are placed in close proximity of each other, such as for neuro-muscular stimulation or orthopedic sensors that merits further investigation on this topic [1, 82]. We explore the possibility of beamforming inside the body using galvanic coupled intra-body links in chapter 6.

In the rest of the thesis, we present our technical contribution to develop the galvanic coupled intra-body communication as a matured technology to be considered for implant communication.
Chapter 3

Tissue Equivalent Channel Models

For establishing communication links among the IBN nodes, the tissue channel needs to be analyzed for selecting optimal propagation characteristics in order to safely and reliably transfer information. Our work on an analytic model for building a reliable human tissue communication channel is motivated by the fact that in-vivo tissue experiments are not always possible, commercially available phantoms do not accurately reflect the tissue heterogeneity, and electrical propagation characteristics over a wide frequency range. Human body is composed of multi-layered tissues each with its own signal propagation characteristics. Tissue impedance calculations should include this multi-layer phenomenon for accurate channel estimations. The state of the art has been mainly restricted to a single tissue communication such as on-surface (i.e., with the transmitter and receiver placed on the skin), with a limited investigation in muscle [40], that analyzes only three directions of current flow. Our model completely changes this analysis using practical assumptions of the tissue electrical properties, where four directions of current flow (the additional direction involving current passing into lower/upper tissue layers) is possible. To the best of our knowledge, this comprehensive treatment of galvanic coupling-based channel model has not been derived before, and for successful communication between implanted sensors, it is essential for characterizing the transverse path from one tissue to another.

Moreover, for a detailed analysis on the implant data link through tissues, the communication channels along tissues needs to be characterized individually as skin to skin (S-S), skin to muscle (S-M), muscle to skin (M-S) and muscle to muscle (M-M) paths, among others. The field distribution arising out of the galvanic coupled multi-layered inner tissue that includes the above mentioned intra-body scenarios needs further investigation, as no reproducible analytic model exists that has been verified through experiments.
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We summarize our main contributions towards modeling tissue as communication channel as follows:

- We derive a three dimensional multi-layered human forearm Tissue Equivalent Circuit model (TEC) for analyzing the communication channel through the surface and inner tissue-layers. Our reproducible expression involves a large number of configurable parameters (over 10), which can comprehensively capture the various design intricacies of GC-IBN-based communication.

- Our theoretical approach is validated with previously conducted experiments for on-skin communication. Interestingly, our model indicates a tighter match with previously obtained measurements, than what was possible using existing models. We also include additional validations through measurement studies conducted on porcine tissue.

- For verifying the accuracy of the multi-tissue analysis, we construct a 3D model of the human forearm using finite element simulation. The simulator captures minute aspects of the signal propagation through the inner tissues. This allows the simulation to be used for quick analysis of future network designs for situations where intra-body testing is not immediately feasible.

- We analyze the model for various parameters like tissue thickness and electrode dimensions/separations and provide insights on suitable implant positions inside the tissues.

The rest of the chapter is organized as follows: We present the background work in Section 3.1 and formulate our analytical model based on a circuit equivalent construction for the human forearm in Section 3.2.3 with the corresponding simulation model and safe signal generation conditions described in Section 3.4. The model verification and analysis of the model parameters are given in Sections 3.5 and 3.6 respectively. Measurements based on porcine tissues are presented in Section 3.7.

3.1 Background & motivation

The existing approaches consider single tissue layer with limited flexibility, which we aim to overcome in our proposed work. Additionally, works that consider the multi-layer effect [49] include only bidirectional signal propagation paths (longitudinal and cross paths) between transmitter and receiver. The direct path between the transmitter terminals that depends on the underlying tissue impedance is assumed to be measurable at the electrode attachment site which limits its practicality.
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Also, the transverse path from one tissue to other that depends on the tissue thickness is neglected. The tissue equivalent model needs to be asymmetric as opposed to the existing models to account for dissimilar dimensions, tissue heterogeneity, and non-identical electrode set-up at transmitter and receiver, which significantly complicates the analysis. These models have limited flexibility and are restricted from considering multiple possible paths of current in the three dimensional heterogeneous tissue medium and thus requires further investigation. We derive an extensive three dimensional model to analyze the electrical behavior of human tissues.

We aim to build a Tissue Equivalent Circuit (TEC) model that should quickly provide an estimate of the channel gain based on the choices of input frequency, transmitter-receiver locations, distance and separation between their electrodes. Our model uses some easily obtained physiological factors, such as dimensions and hydration levels.

3.2 Three Dimensional Tissue Equivalent Circuit Model of Human Forearm

We specifically design the model for the human forearm, with the individual tissue impedance obtained from their electrical properties. The corresponding dimensions are average values for an adult male. We derive this model next using the frequency dependent electrical properties of live tissues.

3.2.1 Tissue Impedance:

Living tissue is composed of both movable charges and movement restricted dipoles. Hence, it can be characterized as an imperfect dielectric medium. When an array of electricity conducting cells are excited by an external electrical signal, each cell activates its neighbor, enabling signal propagation through different paths dictated by the cell structure and the frequency of operation. Low frequency signals cannot penetrate the high impedance cell membrane, and so it takes the circuitous path through extra-cellular fluid. As opposed to this, high frequency signals pass through intra-cellular fluid by penetrating the cell membrane. Thus, the cell membrane gives a capacitance effect, allowing the passage of only high frequency components.

Under 100 MHz, the dimensions of human body and implants are small compared to the signal wavelength, and hence, we undertake the analysis using lumped elements. Using the frequency dependent electrical properties of live tissues (conductivity \( \sigma \) and permittivity \( \epsilon \)), a
simple biological cell can be modeled with Resistance $R_{ext}$, $R_{int}$ (representing dissipation loss), and a capacitor $C_m$ (representing the charge holding ability), connected as shown in Fig. 3.1(b). We use the approach in [51] to derive the electrical properties of human tissues as given below.

$$\epsilon = \epsilon_0 \epsilon_r = \epsilon_0 (\epsilon' - j(\epsilon'' + \frac{\sigma}{\omega \epsilon_0}))$$  \hspace{1cm} (3.1)

where $\epsilon'$ is the dielectric constant and $\epsilon''$ is the out of phase loss factor, expressed in terms of complex permittivity ($\epsilon^*$) as,

$$\epsilon^* = \epsilon' - j\epsilon'' \hspace{1cm} (3.2)$$

$$\epsilon' = \epsilon_{\infty} + \frac{\epsilon_s - \epsilon_{\infty}}{1 + \omega^2 \tau^2} \hspace{1cm} (3.3)$$

$$\epsilon'' = \frac{(\epsilon_s - \epsilon_{\infty}) \omega \tau}{1 + \omega^2 \tau^2} \hspace{1cm} (3.4)$$

In the above set of equations, $\epsilon_{\infty}$ and $\epsilon_s$ are dielectric constants at very high and very low frequencies, $\omega$ is the angular frequency measured as $2\pi \times$ frequency and $\tau$ is the dielectric relaxation time given by $X/R$, where $X$ is the reactive component from capacitance effect.

Using (3.3) and (Appendix B.12), the tissue admittance using RC elements can be calculated as,

$$Y = G_{ext} + \frac{1}{R_{int} + jX_C} = F_W \left( \frac{\sigma M_1 + \frac{1}{\sigma \kappa M_1 + j\omega \epsilon M_2}}{\sigma M_1 + \frac{1}{\sigma \kappa M_1 + j\omega \epsilon M_2}} \right)$$  \hspace{1cm} (3.5)

where $Z$ is the impedance, $G$ is the conductance, $M_1$ is the ratio of cross sectional area (A) and length of the channel (L) decided by the direction of impedance measurement and while $M_2$ is the ratio of A and thickness of channel as explained in section 3.2.3. $F_W \in [1, 10]$ is the correction factor accounting for variation in dielectric properties with respect to tissue water content water distributions [52] that can be determined using non-invasive hydration testing and $\kappa$ is the ratio of external to internal cell resistance. We assume that the other tissue properties can be estimated without actual measurement such as tissue thickness approximation using body mass index (BMI), bio-electrical impedance analysis or triceps skin fold thickness.

### 3.2.2 Single Tissue Equivalent Model

Prior to the complete modeling of the forearm, the equivalent circuit of a single galvanic coupled tissue is calculated using four impedance. These impedance values are derived as follows, based on the four paths taken by an injected current. These are marked as P1, P2 and P3 in Fig 3.1(a)
Figure 3.1: (a) Equivalent circuit of a single tissue layer, (b) Equivalent circuit of single biological cell, (c) Equivalent circuit of electrode and coupling impedance, and (d) 3D Circuit Model for the forearm as a layered dielectric

for a single tissue layer, and the fourth path from one tissue layer to a neighbor is shown as P4 in Fig. 3.1(d).

- Path P1 is the primary return path offering the direct impedance $Z_D$ that channels the majority of current from the terminal to ground electrodes in the transmitter. In this case the factor $M_1$ given in (3.5) takes the form $(E_L \times T)/E_S$, where $E_L$ is a side of the square electrode, T is tissue thickness and $E_S$ is the terminal-reference electrodes separation distance in transmitter and in receiver that are assumed to be the same if not specified. To distinguish them if they are different, we use the representation $E_{ST}$ for the transmitter electrode separation and $E_{SR}$ for the receiver electrode separation.

- Path P2 serves as a pathway for a small portion of current directed towards the receiver electrodes through longitudinal impedance $Z_L$, between the transmitter and receiver electrodes. $M_1$ of $Z_L$ is calculated as $(E_L \times T)/D$, where, $D$ is the transmitter-receiver separation distance.

- Path P3 is the electric current propagation path from source terminal in transmitter to the reference terminal in receiver through cross impedance $Z_C$. $M_1$ in this case becomes $(\sqrt{2}E_L \times T)/(\sqrt{D^2 + E_{ST}^2})$. In all the above cases, $M_2$ is chosen to be the tissue thickness.

- Path P4 is the electric current propagation path to adjacent tissue layer through transverse
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 impedance $Z_T$. To compute this impedance, $M_1$ is substituted with $T/A_e$, where, $A_e$ is the electrode area. In this case, $E_S$ becomes the channel thickness.

We also include the effect of the coupling impedance offered by the contact between the electrode and the tissue interface in the derivation of channel characteristics, as it determines the amount of signal entering into the tissue. This impedance denoted as $Z_{Co}$ (refer Fig.3.1(a)), is calculated next.

**Electrode-Tissue Coupling Impedance**  The coupling impedance is a function of frequency, area of contact, tissue hydration, electrode material and surface treatment. To calculate the equivalent impedance at the electrode-tissue interface, we follow the approach in [53], where the interface is modeled as shown in Fig.4.2(b). Here,

$$\text{Re} = K_1 f^m / A_e \quad \text{and} \quad X_e = 1/wC_e = K_2 f^{m'} / A_e$$  \hspace{1cm} (3.6)

where, $f$ is the frequency of operation, $K_1$ depends on the electrode material. $K_2$ lies within the range $(0,1)$ based on the tissue hydration and surface treatment, $m$ and $m'$ are constants for diffusion control and for activation control. The dots in Fig.3.1(a) represents the possibility of attaching $Z_{Co}$ to any tissue based in the channel under study. For instance, along the S-M path, the coupling impedance, $Z_{Co}$, at the transmitter and receiver positions are included in the direct impedance $Z_D$ at each position. $Z_D$ at the transmitter side is represented as $Z_{DT}$, and that corresponding to the receiver side of the muscle is represented as $Z_{DR}$.

For developing a tractable model, we assume uniform transverse tissue thickness along the paths indicated by $\oplus$ in Fig.3.1(a). However, it is possible to introduce asymmetry in the model by varying the electrodes separation $E_S$, $E_D$ and/or $T$ at transmitter and receiver as analyzed in Section 3.6. Anisotropism can also be introduced into the model by assuming that the transverse impedance is larger than the longitudinal impedance [54].

3.2.3 Modeling for Forearm

We approximate a longitudinal section of galvanic coupled human forearm (refer Fig.3.2) as multi-layered dielectric block with four tissue layers - outer dry skin, fat, muscle and cortical bone (hard outer covering of bone) layers of thickness 1 mm, 7 mm, 15 mm and 20 mm respectively. The parameters such as $T$, $D$, $E_S$, and $E_L$ are added as variables in the impedance calculation. The benefit of this equivalent circuit analysis modeling approach is that it uses a simple first-approximation for
the voltages and currents that are likely to be observed at different points within the given tissue layer during signal propagation. The rectangular model (Fig.3.2(b)) enables direct and easier computation of impedances in individual directions. Moreover, it can be extended to any part of the body, such as thorax.

In the following multi-layer discussion, the superscript $i$ and $j$ denote a specific tissue layer, i.e., $i, j \in \{ S, F, M, B \}$, with the substitutions of $S$ for skin, $F$ for fat, $M$ for muscle, and $B$ for bone. The single tissue impedance $Z_D$ and $Z_L$ in Fig.3.1(a) become $Z_D^i$, $Z_L^i$, $Z_C^i$, and $Z_T$ takes the form $Z_{T}^{i-j}$, denoting path from layer $i$ to $j$. The circuit in Fig.3.1(d) is used to model the flow of current through skin, fat, muscle and bone in the forearm. The S-S path characteristics are studied with the transmitter electrodes (across nodes A and B) and receiver electrodes (across nodes C and D) both coupled on the skin surface (depicted dashed lines in Fig.3.1(d)). The transmitter and receiver are moved to the muscle tissue for analyzing the M-M path (shown as dot-dot-dash lines). The transmitter is coupled to the skin and receiver is moved to the muscle for the S-M path and vice-versa for the M-S path.

The circuit shown in Fig.3.1(d) has four tissue layers with 20 tensions (including the terminal branches) and 16 equations and is solved Kirchhoff’s Current Law (KCL). The four complex admittance values of each tissue are calculated using (3.5). The node with the source terminal attachment becomes the starting node and reference terminal of the transmitter is chosen as the reference node. The current equation for the first node A based on the difference in node voltage is
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given below.
\[
\frac{V_A - V_B}{Z_D} + \frac{V_A - V_C}{Z_L} + \frac{V_A - V_D}{Z_C} + \frac{V_A - V_E}{Z_T^{S-F}} = I
\]  
(3.7)

where \( V_X \) is the voltage estimated in node \( X \), \( \forall X \in \{ A, B, C, \ldots \} \), \( I \) is the input current given by \( V_{IN}/Z_{in} \) and \( Z_{in} \) is the input impedance across transmitter terminals. Similarly, using the following equations, the voltage difference detected on skin for S-S path can be solved across the nodes C and D.

\[
\frac{V_C - V_A}{Z_L} = \frac{V_C - V_B}{Z_C} + \frac{V_C - V_D}{Z_{DR}} + \frac{V_C - V_G}{Z_{S-F}} 
\]  
(3.8)

\[
\frac{V_A - V_D}{Z_C} = \frac{V_D - V_C}{Z_{DR}} + \frac{V_D - V_B}{Z_L} + \frac{V_D - V_H}{Z_{S-F}} 
\]  
(3.9)

For simpler calculations, the admittance of each loop is calculated and formulated as the admittance matrix \( M_G \) as shown below.

\[
M_G = \begin{bmatrix}
\sum_{i=1}^{n} \frac{1}{Z_{1i}} & -\frac{1}{Z_{12}} & \ldots & -\frac{1}{Z_{1n}} \\
-\frac{1}{Z_{21}} & \sum_{i=1}^{n} \frac{1}{Z_{2i}} & \ldots & -\frac{1}{Z_{2n}} \\
\vdots & \vdots & \ddots & \vdots \\
-\frac{1}{Z_{n1}} & -\frac{1}{Z_{n2}} & \ldots & \sum_{i=1}^{n} \frac{1}{Z_{ni}}
\end{bmatrix}
\]  
(3.10)

where \( Z_{nm} \) is the impedance between node \( n \) and node \( m \). The current at each point is calculated based on the following relation.

\[
M_G \hat{V} = \hat{I}
\]  
(3.11)

where \( \hat{V} \) is the vector with tensions that needs to be found, and \( \hat{I} \) is the vector with the sum of currents through each node. From the KCL node equations and the voltage vector \( \hat{V} \) and current vector \( \hat{I} \) representing the sum of currents entering or leaving node can be represented as

\[
\hat{V} = \begin{pmatrix}
V_1 \\
V_2 \\
\vdots \\
V_n
\end{pmatrix}
\quad \& \quad
\hat{I} = \begin{pmatrix}
I \\
0 \\
\vdots \\
0
\end{pmatrix}
\]

where \( V_n \) is the voltage at node \( n \). The position of I depends on the placement of the source. The voltage received across any of the branch between C-D, G-H, and so on in Fig 3.1(d) can be calculated based on the location of the receiver electrodes. The transfer function from the circuit in Fig 3.1(d) is
calculated using

\[ G(w, E_L, D, E_S, [T]) = 20 \log_{10} \left| \frac{V_o}{V_I} \right| \]  

(3.12)

where \([T]\) is the vector of tissue thicknesses for skin, fat, muscle and bone, \(V_o\) is the potential difference observed across the receiver electrodes and \(V_I\) is the source voltage. We tracked the phase shift information using the following equation.

\[ \text{Phase} = \arctan \left( \frac{\text{Im}(V_o)}{\text{Re}(V_o)} \right) \]  

(3.13)

The channel characteristics computed using the model thus derived are presented and verified in Section 3.5. It can be seen from the derivations that the model is more expressive and one can demonstrate the ability to analyze the impact of various network parameters such as electrode size, transmitter receiver separation, and tissue thickness among others on sensor placement and tissue channel performance.

### 3.3 2-Port Circuit Model

Individual tissues are represented as black-boxes in a 2-port model, constructed from the electrical properties of tissues that is configurable with tissue dimensions corresponding to a specific human subject. The principle benefit of this 2-port model is a simple first-approximation for the voltages and currents that are likely to be observed within the given tissue layer during communication. The black-box approximation makes it easier to add, remove or modify tissue dimensions within the model which otherwise is complicated and time consuming to alter the model appropriately for different parts of the body. The model works for signal propagation within the same tissue layer, adapts easily for different combinations of tissues, and describes the signal propagation through tissues from the input to output ports for a given choice of input frequency, transmitter-receiver distance (D), terminal separation (\(E_S\)) (refer Fig. 5.1(a)) and tissue thickness (T). As an added advantage, the port parameters enable direct observation of the scattering parameters at the electrode-tissue and tissue-tissue boundaries to assess the reflection and propagation at interfaces.

The 2-port equivalent of a single GC-coupled tissue is modeled using tissue impedance along the four propagation paths P1, P2, P3 (Fig. 5.1(a)) and P4 (Fig. 5.1(c)) taken by the injected current that are obtained as follows.

- P1 is the primary return path offering the direct impedance \(Z_D\) that channels the majority of
current from the terminal to reference electrodes in the transmitter. In this case \( M_1 \) given in (3.5) takes the form \( (E_L \times T)/E_S \), where \( E_L \) is a side of the square electrode, \( T \) is tissue thickness and \( E_S \) is the terminal-reference electrodes separation in transmitter/receiver.

- \( P_2 \) serves as a pathway for a portion of current directed towards the receiver electrodes through longitudinal impedance \( Z_L \), between the transmitter and receiver electrodes. \( M_1 \) of \( Z_L \) is calculated as \( (E_L \times T)/D \), where, \( D \) is the transmitter-receiver separation distance.

- \( P_3 \) is the current propagation path from source terminal in transmitter to the reference terminal in receiver through cross impedance \( Z_C \). Here, \( M_1 \) becomes \( (\sqrt{2}E_L \times T)/(\sqrt{D^2 + E_{ST}^2}) \).

In all the above cases, \( M_2 \) is chosen to be the tissue thickness.

- \( P_4 \) is the current propagation path to adjacent tissue layer through transverse impedance \( Z_T \).

To compute this impedance, \( M_1 \) is substituted with \( T/A_e \), where, \( A_e \) is the electrode area. In this case, \( M_2 \) is assigned the value of \( E_S \).

The coupling impedance offered by the electrode-tissue interface determines the amount of signal entering into the tissue. This impedance, denoted as \( Z_{Co} \) (refer Fig. 3.1(a)), is a function of frequency, area of contact, tissue hydration, electrode material and surface treatment and is modeled similar to the approach in [53] as

\[
R_{Co} = \rho_{Co}f^m/A_e \quad \text{and} \\
X_{Co} = 1/wC_{Co} = K_1\epsilon_{Co}f^m/A_e
\]

where, \( f \) is the frequency of operation, \( \rho_{Co} \) is the electrode and hydrogel resistivity, \( A_e \) is electrode area, \( K_1 \in (0, 1) \) is chosen based on the tissue hydration, \( \epsilon_{Co} \) depends on electrode permittivity, and \( m \) is the constant for activation control. The dots in Fig. 3.1(a) represents the possibility of attaching \( Z_{Co} \) to any tissue based on the tissue path under study. For instance, in the skin to muscle path, the coupling impedance is included in input impedance of skin and output impedance of muscle.

Using the impedance defined above, the 2-port equivalent of a tissue can be defined in terms of Z-parameters represented as a \( 2 \times 2 \) matrix of complex numbers that obeys the relation, \( [V] = [Z][I] \) where \([V]' = [V_1 \ V_2]\) is the voltage vector, and \([I]' = [I_1 \ I_2]\) is the current vector at the input and
output ports. The Z parameter of each tissue can be calculated as,

\[
[Z] = \begin{bmatrix}
  Z_{11} & Z_{12} \\
  Z_{21} & Z_{22}
\end{bmatrix}
= \begin{bmatrix}
  \frac{2Z_1Z_2}{Z_1 + 2Z_2} & \frac{Z_1Z_2}{Z_1 + 2Z_2} \\
  \frac{Z_1Z_2}{Z_1 + 2Z_2} & \frac{Z_1Z_2 + Z_2^2}{Z_1 + 2Z_2}
\end{bmatrix}
\]

with \( Z_1 = \left( \frac{2Z_LZ_T}{Z_T - Z_L} \right) \) and \( Z_2 = \left( \frac{Z_TZ_D}{Z_T + Z_D} \right) \). For the tissue layer receiving signal from another layer, \( Z_1 \) becomes \( \left( \frac{2Z_LZ_T}{Z_T - Z_L} (1 + Z_T) \right) \), where \( Z_T \) is the transverse impedance offered by the interface between tissue layers. Similar to impedance parameters, we also use admittance parameters \([Y]\) for handling parallel impedance and ABCD parameters \([A]\) for handling cascaded impedance.

### 3.3.1 Human Arm Equivalent 2-port Network

We approximate the galvanic coupled human arm as layered dielectric block of tissues as given in Fig. 3.1(c). The model of 700 mm length has four tissue layers - outer dry skin, fat, muscle and cortical bone (hard outer covering of bone) of thickness 1 mm, 9 mm, 25 mm and 20 mm respectively. For developing a tractable model, we assume uniform tissue dimensions along the paths indicated by \( \oplus \) and \( \otimes \) in Fig. 3.1(a). However, it is possible to introduce asymmetry in the model by varying the electrodes separation \( E_S \), and/or \( T \) at transmitter and receiver.

In the following multi-layer discussion the superscript \( i \) and \( j \) denote a specific tissue layer, i.e., \( i, j \in \{ S, F, M, B \} \), with the substitutions of \( S \) for skin, \( F \) for fat, \( M \) for muscle, and \( B \) for bone. The single tissue impedance \( Z_D \) and \( Z_L \) in Fig. 3.1(a) become \( Z_D^{i-j} \) and \( Z_L^{i-j} \) and \( Z_T \) takes the form \( Z_T^{i-j} \), denoting path from layer \( i \) to \( j \). The transmitter electrodes, attached on the tissue, form the input port, and the receiver electrodes form the output port. This concept is clarified further with a sample case of the transmitter and receiver coupling locations on skin to muscle path (S-M path) is shown in Fig. 3.1(c). Figure 3.2 illustrates the location of coupling electrodes when on-surface nodes are moved into muscle tissue. We explain the development of the analytical models for skin to skin (S-S), muscle to muscle (M-M), skin to muscle (S-M) and muscle to skin (M-S) tissue paths of signal propagation next. We ignore the paths through fat and bone tissues as the implants are not commonly placed in these tissues.

- **S-S path model** Here, the transmitter and receiver sensor electrodes are positioned on the skin surface with coupling impedance \( Z_{Co} \). The current flow is through skin, and through the parallel paths in fat, muscle and bone layers. The input impedance of the fat layer includes the transverse
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Figure 3.3: Coupling impedance location for transmitter and receiver moving from on-skin to muscle

impedance from skin as the signal originates in skin. Similarly, the input impedance of muscle and
bone layers include the transverse impedance from the fat and muscle tissues, respectively. The
overall admittance is:

\[
[Y^{S-S}] = [Y^S] + [Y^F] + [Y^M] + [Y^B]
\]  

(3.14)

• **M-M path model** To study the channel response at the muscle layer, the transmitter and receiver
sensor electrodes are moved inside muscle tissue (M-M path), in which the dominant path of the
current also lies. Along the M-M path, the muscle tissue is in parallel to that of skin and fat tissues
and with the bone. In this case, the input impedance of fat and bone layers include the transverse
impedance from the muscle layer as the signal originates from the muscle. The input impedance of
skin layer includes the transverse impedance of muscle and fat tissues. The total channel admittance
at M-M path can be calculated as the parallel combination of individual tissues as given in S-S path
model.

• **S-M path model** When the transmitter is on skin and the receiver is in muscle, the impedance
offered by the S-M path is measured as the cascaded skin-fat-muscle ports that is in parallel to
that of bone. The transverse impedance is calculated as in the S-S case as the signal source is at
skin. Assuming \( A^S, A^F \) and \( A^M \) as the \( [A] \) parameters of skin, fat and muscle, the combined \( [A] \)
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Parameter for the cascaded skin-fat-muscle tissues is given by

\[
[A^{S,F,M}] = [A^S][A^F][A^M]
\]  \hspace{1cm} (3.15)

With the added parallel effect of bone tissue, the overall admittance of the S-M path is computed as follows.

\[
[Y^{S-M}] = \begin{bmatrix}
\frac{A_{22}^{S,F,M}}{A_{12}^{S,F,M}} & -\frac{\triangle[A^{S,F,M}]}{A_{12}^{S,F,M}} \\
-\frac{1}{A_{12}^{S,F,M}} & \frac{A_{11}^{S,F,M}}{A_{12}^{S,F,M}} \\
\end{bmatrix}
+ [Y^B]
\]  \hspace{1cm} (3.16)

**M-S path model** For the M-S path, with the source at muscle, the transverse impedance is calculated as in M-M path. Here, the ABCD parameter of cascaded muscle, fat and skin tissues is given by

\[
[A^{M,F,S}] = [A^M][A^F][A^S]
\]  \hspace{1cm} (3.17)

The admittance parameter of M-S path is obtained by computing the cascaded muscle, fat and skin tissue in parallel to the bone. The resulting expression is similar to the computation in (3.16). The voltage gain \(G(w, E_L, D, E_S, [T])\) can be calculated from the total equivalent Z parameters (inverse of Y parameter matrix) \([Z_{i-j}]\) as,

\[
\frac{V_{out}}{V_{in}} = \frac{Z_{21}^{i-j}Z_{Load}}{(Z_{11}^{i-j} + Z_{Co})(Z_{22}^{i-j} + Z_{Load}) - Z_{12}^{i-j}Z_{21}^{i-j}}
\]  \hspace{1cm} (3.18)

where \(i, j \in \{S, M\}\) for denoting S-S, S-M, M-S and M-M paths individually. The gains along the four paths above is plotted in Fig. 3.5 and analyzed in section 4.5.

The channel gain obtained for the four paths obtained using the 2-port model derived in Sec 3.3 at \(D = 15\) cm, \(E_S = 5\) cm, and \(E_L = 1\) cm are given in Fig. 3.5. The gain along the S-S path has a peak at 500 KHz and decreases beyond 1MHz.

**3.3.2 Performance of 2-port model**

**Results verification using literature:** The clinical trial results in [48] match exactly with our analytical results as the measurement set-up is similar to our model assumptions presented above. However, though our results matches closely with the other literature measurement at 100 KHz as shown in Fig 3.4, there is a difference of approximately 7 dB and 3 dB with [55] and [49] respectively.
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Figure 3.4: S-S path gain comparison from literature.

at 1 MHz.

The difference in gain in the latter two cases are likely due to the variations in the electrode dimensions, electrode material, physical tissue dimensions in the measurement set-ups. For instance, in [49], the skin thickness was assumed to be 1.5 mm with $E_S = 80$ mm while we assigned them 1 mm and 100 mm respectively. Similarly, in [55], the radius of the subject’s arm is 47.5 mm, which we modeled as 55 mm. There are other inherent measurement uncertainties including surface treatment at electrode attachment, tissue temperature and hydration levels that we model using parameters $F_W$ and $Z_{Ca}$ and are not specified in the corresponding literature. Also, the conductivity and permittivity of tissues also vary among individuals by +/- 0.1 S/m and +/- 0.05 S/m in the range of frequency used [56] that contribute to the difference between our results and the results in [55] and [49]. In order to overcome other measurement uncertainties in GC-IBN, an adaptive communication system is required that alters the transmission parameters based on the time dependent estimation of channel characteristics. For instance, estimating bounds on interference and allowing sufficient tolerance levels in transmission power control schemes would help alleviating the above mentioned uncertainty.

- **Tissue paths comparison:** Among the four paths, M-M path has the largest gain of $-24.5$ to $-22.5$ dB be due to the higher conductivity and larger volume of muscle. The gain is increasing with
frequency up to 800 KHz and falls low at higher frequencies. The higher gain in M-M path is also caused by the signal trapping in inner tissues with little dissipation to air. In the S-S path, however, owing to the immediate coupling with surrounding air, the maximum gain is only around $-41$ dB at 500 KHz. The lower values of dry skin conductivity and thickness also lead to higher attenuation in S-S path. The channel gain can be improved by using suitable surface treatments such as application of conductive gels at the electrode attachment location.

The signal originating on-skin primarily propagates through low resistant muscle. Therefore, with a source on skin, a receiver in muscle (S-M path) experiences better channel gain compared with an on-skin receiver (S-S path). The loss of S-M path being $\approx 10$ dB more than the M-M path is due to the initial loss at the skin tissue and the intermediate fat tissue with $Z_{T}^{S-F}$ and $Z_{T}^{F-M}$. Along the M-S path, the gain is high ($-38.5$ dB) at 200 kHz and decreases with frequency (to $-46$ dB) indicating higher gain than S-S path at frequencies lower than 200 kHz. At higher frequencies, M-S path offers the worst gain among all the paths. Hence, surface to implant communication through S-M path offers significant benefit at higher frequency while implant to on-surface communication performs better at lower frequency. At frequencies higher than 1 MHz, majority of the signal leaks from the tissue to the surrounding space that cannot be received by body nodes. For this reason, we
avoid the frequencies above 1 MHz and also the frequencies below 100 kHz that may comprise the body’s natural frequencies.

### 3.4 Simulation Framework for Model Verification

In this section, we describe the tissue modeling using the Ansys HFSS, which allows us to perform full-wave electromagnetic simulations for arbitrary 3-D models. It allows detailed computational analysis of field distribution at various locations inside the tissues using finite element analysis (FEA), and is especially useful when experimental results are not easily obtained for intra-body channels.

We model the forearm with dimensions as described in Section 3.2.3. A pair of copper cuboids of dimension $10 \times 10 \times 1 \text{mm}$ that is similar to TEC model is used as the terminal and reference electrodes. The electrodes are connected by a complex impedance defined lumped port. The source current of 1 mA is set at the lumped port (input). To 1 foot distance around the forearm model, we emulate a boundary as an open electrical circuit. The frequency dependent electrical properties of dielectric tissue blocks are configured using (B.4)-(B.12) derived in appendix for the frequency range 100 kHz to 1 MHz.

HFSS transforms the 3-D tissue model into a mesh of tetrahedron structures, with a high density of mesh points at critical positions like the electrode-tissue interface (Fig.3.6 (left)). We performed the analysis in terms of the equivalent electric and magnetic ($E$ and $H$) fields in simulation in contrast to current and voltage ($I$ and $V$) vectors in TEC model to estimate the channel gain. To determine the field strength across the above said tetrahedrons, complex EM field values at each vertex of tetrahedron is computed using Maxwell’s partial differential equations. The normal $E$ component on skin surface is measured as surface integral over an area equivalent to the surface area of a receiving electrode. The $H$ field is measured as surface integral of its tangential component.

The current through surface $S$ at distance $l$ from the source can be obtained from Ampere’s law as $I_{\perp S}(l) = \oint H . dl$.

From Fig.3.7(a), we see that the signal propagates disparately in each layer. For instance, along the lateral direction, the signal propagates only through a part of bone. However, in the muscle, the signal propagates through the entire tissue (refer Fig.3.7(a)). The signal strength at any point $P$ (refer Fig.3.7(b)) in a tissue depends on its electrical properties and on the distance between source $S$ and $P$ along the tissue and is independent of the distance from center of the cylinder ($r$), or the azimuth angle ($\theta$) between the line connecting center to $P$ and a reference plane. For this reason, we
approximate the curvature SP of the cylindrical arm as the Euclidean distance of rectangular tissues in TEC model in Section 3.2.3 (Fig. 3.2(b)). In order to achieve model conformance in the FEA cylindrical arm model, we estimate the angle of electrode separation, \( \theta \) as \( E_S/r \), where \( E_S \) is the Euclidean distance of electrode separation in TEC model. For emulating the signal received at the implanted sensor, we move the transmitter electrodes and port into muscle tissue (Fig. 3.6(right)). The \( E \) field strength measured across the receiver electrodes is used to calculate the output voltage. The gain through the tissues can be calculated as follows.

\[
G_E(dB) = 20 \log_{10} \left( \frac{E_{Detector}}{E_{Coupler}} \right)
\]  

(3.19)

The simulation is repeated for different \( E_S \) (distance between the terminal and reference electrodes), and D (different distances between the transmitter and receiver) for varying [T] (thickness of tissues) at frequencies ranging from 100kHz to 1MHz. The results are used to verify our TEC model as discussed in Section 3.5. In addition, using the FEM model, we derive the boundary conditions next that are necessary to ensure tissue safety.
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Figure 3.7: (a) Tissue signal distribution illustrating lower signal strength in bone and higher signal strength in muscle (b) Influence of $r$ and $\theta$.

3.5 Model Verification & Discussion

This section verifies the analytical model derived in (Section 3.2.3) using the simulator design from Section 3.4 as well as with prior experimental measurements for S-S path in literature. We use the clinical trial findings described in the existing work [50] and measurements in [47] for verifying the channel gain obtained through the S-S path and [43] for verifying the effect of varying the transmitter-receiver separation distance (D) on gain in M-S path. We conduct the evaluations on the following basis at different paths: (i) variation of gain with frequency, (ii) phase shift of the signal with frequency, and (iii) impact of frequency on energy dissipation.

The channel gain obtained from 100 kHz to 1 MHz with D being 100 mm and the electrode separations in transmitter $E_{ST}$ and receiver $E_{RT}$ being 50 mm using TEC model (3.12) and simulation model (3.19) are presented for the S-S and M-S in Fig 3.8 and for the S-M and M-M paths in Fig 3.9. The tissue dimensions are specified in section 3.2.3. The values we choose for $F_W$, $m$ and $m'$ are 0.7, −1.15 and −0.81 [53]. The channel gain obtained using TEC model (Fig 3.8) at 100 kHz is around $-50$ dB and drops by 10 dB at 1 MHz on the S-S path. We see good agreement among the TEC and simulation model plots and with prior experimental results from literature for
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the S-S path. The variation between the TEC model results and simulation results is less than 2 dB, verifying the accuracy of the model. The channel gain obtained for TEC model S-S path matches well with the clinical trials in [50], where the electrodes and tissue dimensions used are similar to the ones assumed in our analysis.

There is a difference of about 3 dB with the measurements from [47], which we attribute to the variation in the electrode dimension (circular electrode with radius 0.5 cm) and the usage of electrode conductive gel. There are other inherent measurement uncertainties associated with GC-IBN including tissue temperature, hydration levels and surface treatment that we capture using parameters $F_W$ and $Z_C$ for a typical adult, which are not specified in [47]. Moreover, the literature reports a variation of 2 dB among measurements on different days. The above mentioned reasons along with variation in $\sigma$ and $\epsilon$ values of tissues among individuals by $\pm 0.1 \text{ S/m}$ and $\pm 0.05$ respectively, in the range of frequency used [56] contribute to the difference between our results and those reported in [47].

We observe that the gain obtained in the muscle tissue is significantly higher than the S-S path by $\approx 24$ dB advantage in gain with $-26$ dB at 100 kHz, that drops by $\approx 4$ dB at 1 MHz, indicating better SNR and less frequency sensitivity in M-M path. Note that the S-S path gives a gain variation of $\approx 10$ dB in the range of frequency considered. The S-M and M-S paths have channel gain higher than the S-S path but lower than the M-M path. The S-M path with the receiver placed in muscle has atleast 12 dB more gain than the M-S path with the receiver on skin. As there are no published experimental data on the signal gain over the M-M, S-M & M-S paths to our best knowledge, our studies are limited to comparison between the analytical and theoretical models we have derived in this work.

- **Phase shift of the signal with frequency**: We next study the impact of tissue channel on the transmitted signal phase using (3.13), at S-S, S-M, M-S and M-M paths. Fig 3.10(a) shows the shift in phase when the signal frequency varies in the range of 100 kHz – 1 MHz. We observe that the phase shift on the S-S and M-S path varies from 16 to 20 degrees, whereas for the M-M and S-M paths, there is less than 7 degrees of shift in phase reinforcing that the muscle tissue serves as a better channel.

3.6 Model Sensitivity Analysis

The model proposed in this paper uses different variables as network design parameters such as tissue thicknesses, transmitter-receiver separation, electrode dimensions, and terminal
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Figure 3.8: S-S and M-S gain Vs frequency using tissue equivalent circuit model (TEC), simulation (FEA) and literature measurements; D=10 cm, $E_{ST}=E_{SR}=5$ cm

separations. A better understanding of the relationships between these parameters and the channel gain would help determining the placements of IBN nodes. For this purpose, we undertook a one-factor-at-a-time approach to study the influence of the key network parameters on channel gain in this section.

3.6.1 Effect of Tissue Thickness on Channel Gain

One of the important parameters that determine channel gain is the thickness of each tissue layer. In this section, we investigate the impact of fat and muscle tissue thickness on the signal gain. As sensors are often placed either on the skin (with non-invasive access) or in the muscle (best propagation characteristics), the intermediate fat tissue behavior and its thickness play a crucial role in determining the quantity of signal that transcends the tissue boundaries. For instance, the influence of tissue thickness as a parameter in transverse impedance $Z_T$ of the model is given by

$$Z_T = \frac{(T + \gamma)(\rho + i\omega\epsilon)}{\rho E_T^2(\rho + 2i\omega\epsilon)}$$  \hspace{1cm} (3.20)

where $\gamma$ denotes the change in tissue thickness from the average value considered in this paper. In general, fat acts as a barrier between skin and muscle tissues, allowing either tissue to retain the energy (for $\gamma > 0$) or allowing more current to pass through (for $\gamma < 0$). For the channel gain results
Figure 3.9: S-M and M-M gain Vs frequency using tissue equivalent circuit model (TEC), simulation (FEA) and literature measurements; D=10 cm, $E_{ST}=E_{SR}=5$ cm

given in Fig. 3.8 and Fig. 3.9 we considered an average value of forearm fat thickness as 7 mm. From the results of varying fat thickness in Fig. 3.10(b), it can be seen that for varying fat thickness from 0.5 mm to 60 mm with $D = 100$ mm, $E_S = 50$ mm at 100 kHz the M-M path shows no significant change in gain and performs better for all fat thicknesses assumed. The S-M path has a slight drop in gain by about 1 dB illustrating that for any fat thickness, the dominant part of signal propagates through the muscle. The M-S path gain also drops with fat thickness when there is no signal leakage from muscle to skin for thick fat. The S-S path gain drops for fat thickness between 1 and 3 cm and then improves towards the thin fat values when there is minimal leakage to the layers beneath the skin.

We can conclude that for a thick fat layer, the receiver should be positioned in the same tissue layer as the transmitter for better channel gain. As signal leakage is non-negligible for any fat thickness, simultaneous communication on the skin and within the muscle cannot coexist at the same frequency. Thus for multiple pair of co-located sensors and actuators placed on the skin as well as implanted within the muscle to be active, a multi-access scheme is required. For covering longer distances, and if the BMI values indicate thick fat layer, the M-M path is preferable. We undertake a similar study for varying muscle thickness and the results are given in Fig. 3.10(c). The gains along all the four considered paths increases with muscle thickness. In M-M, M-S and S-M paths, for
Figure 3.10: Sensitivity Analysis. Top row: (a)Phase Shift (left) Vs Frequency (b) Gain Vs Fat thickness (c) Gain Vs muscle thickness. Bottom row: (d) Gain Vs transmitter - receiver separation (D) (e) Gain Vs electrode separation in transmitter & receiver (E_s) (f) Gain Vs electrode misalignment (Δℓ)

40 mm increase in muscle thickness, the increase in gain is about 15 dB while in S-S path, the gain increases by 8 dB. Thus, networks formed in thicker muscle tissue offer better channel gains and cover longer distances.

### 3.6.2 Impact of Transmitter-Receiver Separation Distance

The maximum possible transmitter-receiver separation distance (D) that determines the quality of signal for communication is one of the primary factors in IBN design. Transmitted signals suffer a natural attenuation with distance owing to the increasing longitudinal impedance, Z_L. Using analytical model, the impact of variation in D in the longitudinal and cross impedance (Fig.3.11(a)) can be derived in terms of the network parameters considered in this section as,

\[
Z_L = \frac{D(T \rho + i \omega \epsilon D)}{A \rho (T \rho + 2i \omega \epsilon D)}
\]

and

\[
z_C = \frac{\sqrt{2(D^2 + E_S^2)(T^2 \rho + i \omega \epsilon (D^2 + E_S^2))}}{2 \rho E_L T (T^2 \rho + 2i \omega \epsilon (D^2 + E_S^2))}
\]
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The rate of change of $Z_L$ with respect to the change in $D$ is inversely proportional to $D$ that reflects similar trend in the channel gain calculation as illustrated in Fig. 3.10(d). For an increase in $D$ from 20 to 100 mm, the signal gain drops by around 18 dB in S-S path, about 10 dB in M-M path, and about 12 dB in S-M/M-S paths. This analysis would help determine the single-hop distance in body network design.

3.6.3 Impact of Electrodes Separation Distance

Fig. 3.11(b) illustrates variation in the electrode separation distance, $E_S$ of the transmitter and the receiver together. The effect of $E_S$ is prominent on the direct impedance $Z_D$ as given by the following relation.

$$z_D = \frac{E_S(D^2\rho + i\omega\epsilon E_S^2)}{\rho E_L D(D^2\rho + 2i\omega\epsilon E_S^2)} \tag{3.23}$$

The gain in all paths increases with $E_S$ as shown in Fig. 3.10(e). Moving the electrodes far apart, such as for the separation achieved by positioning one electrode on the top surface and the other one on the bottom surface of the forearm, the gain dramatically increases to a maximum of 25 dB. We observe similar trends when the separation distance is varied within muscle (i.e., the M-M case). For
instance, by parting the electrodes from 20 mm to 100 mm, the increase in gain is about 20 dB in S-S path, 5 dB in M-M path, and 8 dB in S-M and M-S paths for average fat width.

3.6.4 Effect of Transmitter and Receiver Alignment

In the above discussion, we considered equal distances between the electrodes of transmitter \( E_{ST} \) and receiver \( E_{SR} \) with the transmitter electrode pair perfectly aligned with that of receiver along the longitudinal direction as shown as dotted line in Fig 3.11(c). In this section, we assume the possibility of electrodes’ mis-alignment shown as dashed lines in Fig 3.11(c) deviated by \( \Delta \ell \) from aligned position and study its impact on the channel gain. \( \Delta \ell \) shown in Fig 3.11(c) illustrates only the position in-between the dotted lines that would reduce \( E_{SR} \) while it can also be a deviation outside the dotted lines that would increase \( E_{SR} \) further. The following equation shows the modified expression for \( Z_L \) that includes the influence of mis-alignment \( \Delta \ell \).

\[
Z_L = \frac{\sqrt{(D^2 + \Delta \ell^2)}(T \rho + i\omega \varepsilon \sqrt{(D^2 + \Delta \ell^2)})}{A \rho (T \rho + 2i\omega \varepsilon \sqrt{(D^2 + \Delta \ell^2)}))}
\] (3.24)

It is found that the gain decreases with \( \Delta \ell \) caused by the increase in \( Z_L \) as shown in (3.24) and in other impedance irrespective of the direction of deviation (inside or outside). Maximum gain is obtained for the perfect alignment (\( \Delta \ell = 0 \)) as observed in Fig 3.10(f).

3.6.5 Electrode dimensions:

The electrode size specified by \( E_L \) also has same effect as that of electrode separation, \( E_S \). It can be seen from the impedance relationships given in (3.22) and (3.23) that larger electrode dimensions could lead to higher gain. For instance, an increase of 10 mm in \( E_L \) of electrode brings in 8 dB of improvement in gain. However, larger on-skin or implanted nods may cause discomfort.

Table 3.1: Similarity in electrical properties of Porcine (P) & human(H) tissue

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Conductivity (S/m)</th>
<th>Permittivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin (dry)</td>
<td>0.00016 (P) 0.00045 (H)</td>
<td>965 (P) 1119.2 (H)</td>
</tr>
<tr>
<td>Fat</td>
<td>0.03 (P) 0.024 (H)</td>
<td>98 (P) 92.8 (H)</td>
</tr>
<tr>
<td>Muscle</td>
<td>0.25 (P) 0.36 (H)</td>
<td>9900 (P) 8089.2 (H)</td>
</tr>
</tbody>
</table>
Thus, a compromise between electrode size and gain can help decide the transmitter - receiver distance, the need for next hop relay nodes and their best possible location.

3.7 Model validation using Porcine Experiments

In addition to the verification of the proposed TEC model using simulation and literature measurements, we also performed empirical validation of our model using galvanic coupled channel gain measurements with porcine tissue as the transmission medium. The porcine tissue is considered for validating our analytical model because of the similarities between human and porcine tissues with respect to cutaneous blood supply, body surface areas, cellular turnover rate (28 – 30 days), lipid composition and also in their electrical properties. The porcine electrical properties match accurately with the Cole-Cole model [57]. Table 3.1 illustrates the similarity in electrical properties between human and porcine tissues. The analytical model was adapted to the electrical properties of the porcine tissue used in [57, 56, 58, 59].
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3.7.1 Measurement Set-up and Calculation

The porcine tissue sample obtained from a local slaughter house was extracted with skin, fat and muscle on from a pig weighing 260 pounds. Samples of dimension $34 \times 25 \times 5$ cm$^3$ were cut from the loin surrounding the hip bone and immediately used for our experiments. To ensure fixed and tight holding on the irregular tissue surface, we used the alligator clips (40 mm) as the electrodes at the two transmitter terminals and two receiver terminals. We modified the electrode material and dimension accordingly and removed the bone layer in TEC model to enable results comparison.

The skin was cleaned, slightly abraded and moistened on the location where the electrodes are to be attached. A portable bi-channel signal generator and oscilloscope were used for carrying out the experiment on-site. The block diagram for the basic connection and the actual experimental set-up are shown in Fig. 3.12 and Fig. 3.14 respectively. For isolating the transmitter and receiver, we used the OEP PT4 1:1 pulse transformers, one in between the signal generator and transmitter electrodes, and the other in between the receiver electrodes and oscilloscope.

Initially, we connect the input directly to the receiver and measure the signal across the receiver terminals without the tissue channel in-between to read the attenuation through transmitter and receiver electronics and the noise. We then introduce the tissue channel, and note the loss incurred through the tissue at 100 kHz and at 1 MHz. We extract the path loss through tissues using the transmitted signal strength, the received signal strength and the obtained channel attenuation.

We approximate the observed instantaneous ambient and electronic noise as Gaussian distribution with zero mean. The effect of noise is mitigated using time average of the periodic signal over $10^6$ signal oscillations as

$$V_o^2(t) = \frac{1}{T} \int_{t-T/2}^{t+T/2} V_o^2(t) dt$$  \hspace{1cm} (3.25)

where ‘‘ notation denotes the time averaged signal and $T$ is the signal duration for operating frequency between 100 kHz and 1 MHz.

3.7.2 Discussion on Experimental Results

The channel gain obtained using our analytical model, constructed without the bone layer, is compared against the real measurement made on the porcine tissue using the above given set-up and the average results obtained within 30 min and within 3 hrs of sacrifice are given in Fig. 3.13. The model gain along the M-M path out performs the S-S path by about 18 dB in 5 cm and 14 dB in 10 cm in both the test frequencies. The S-M and M-S path gains are close to each other as the muscle
tissue is also exposed to the air, like the skin tissue, and there is no reflection from the bone tissue. Hence we consider them together in this study as S-M/M-S path. The empirical results are close to the TEC model results, which validates our approach.

Albeit there are similarities between the human and porcine tissue, there are few differences that affect the accuracy of the TEC model. The porcine skin is relatively hairless and tightly attached to subcutaneous tissues. It is less vascular and also thicker. For instance, the stratum corneum of human skin is on average around 10 \( \mu m \) in thickness while that of porcine is 20 \( \mu m \). Similarly, the pH of porcine skin is 6 – 7 and that of human skin is 5. To add to this, the conductivity of muscle and fat varies from animal to animal by \( \pm 0.1 \) S/m and \( \pm 0.05 \) S/m in the range of frequency used \[56\]. Also, the change in tissue properties over time caused by the variation in tissue hydration level and temperature \[58\] as illustrated in Table 3.2 contributes to measurement uncertainties as discussed below.

When the tissue sample is freshly obtained (within 30 min), the S-S path offered 1 dB more than the TEC gain with dry skin (refer Table 3.2). This is likely due to the abrasion on skin, caused by the shaving process that helped reducing the skin impedance. The impedance was

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**Figure 3.13:** Porcine tissue experimental measurements Vs TEC model results


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Table 3.2: Change in $G$ with tissue state and duration after excision

<table>
<thead>
<tr>
<th>Duration after excision</th>
<th>State of tissue</th>
<th>Deviation(dB) from TEC model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>S-S</td>
</tr>
<tr>
<td>&lt;30 min</td>
<td>Dry</td>
<td>-1</td>
</tr>
<tr>
<td></td>
<td>Moistened</td>
<td>-4</td>
</tr>
<tr>
<td>2 to 3 hrs</td>
<td>Dry</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Moistened</td>
<td>-3</td>
</tr>
</tbody>
</table>

Further reduced when the locations of electrode attachment were moistened. However, the same measurements when observed after a couple of hours indicated a fall of 3 dB from the initial gain. Moistening the skin helped recovering 5 dB of gain compared with the dry tissue state. We obtained the average value of these measurements in each path and plotted them in Fig 3.13. There is a difference of 3 dB between the analytical model and empirical results, which is likely contributed by the above mentioned uncertainties, the reasons highlighted in Section 3.5 and due to the structural damage caused by excision.

3.8 Noise and Capacity Estimation

To fully determine the ability of the receiver to decode the signal and determine the achievable rate, the estimation of noise is of critical importance. Once this noise level is known, the signal to noise ratio (SNR) can be computed, and the impact of various modulation schemes can be studied. To quantify the noise level, we focus on a single pair of transmitter-receiver nodes. We model the noise by approximating the power spectral densities (psd) of thermal noise, electrode coupling noise and RF radiation interference as described below.

**Thermal Noise $N_T(f)$:** The thermal noise depends mainly on frequency and temperature can be calculated as:

$$N_T(f) = \sqrt{4KTR} \frac{W}{\sqrt{Hz}}$$

(3.26)

where $T$ is the absolute temperature in Kelvin, $K$ is Boltzmann constant, and $R$ is electrode and tissue resistance.

**Electrode coupling noise $N_E(f)$:** This noise occurs at the interface where the electrode is attached to the tissue. The skin-electrode interface noise can be related to the real part of the skin-electrode impedance, and is equivalent to the thermal noise at high frequencies. We use the
CHAPTER 3. TISSUE EQUIVALENT CHANNEL MODELS

Figure 3.14: Experimental set-up for galvanic coupling with porcine tissue

Table 3.3: Gain and Capacity for BPSK Modulation at $\varphi = 1e8$

<table>
<thead>
<tr>
<th>Distance (cm)</th>
<th>Frequency (KHz)</th>
<th>Gain (dB)</th>
<th>Channel Capacity (bps)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>S-S</td>
<td>M-S</td>
</tr>
<tr>
<td>10</td>
<td>100</td>
<td>-37</td>
<td>-30.5</td>
</tr>
<tr>
<td>10</td>
<td>1000</td>
<td>-38</td>
<td>-38.5</td>
</tr>
<tr>
<td>20</td>
<td>100</td>
<td>-52</td>
<td>-47</td>
</tr>
<tr>
<td>20</td>
<td>1000</td>
<td>-53</td>
<td>-55</td>
</tr>
</tbody>
</table>

Noise p.s.d of surface electrodes as approximated in [60] as:

$$N_E(f) = \frac{1}{f^\alpha}, \quad 1.5 < \alpha < 2.0$$

(3.27)

where $\alpha$ is a correction factor that depends on the gel type and skin properties. RF Radiation interference $I_o$: RF radiation from sources such as TV and radio broadcast signals, transmissions in the so called lost band (160 - 190 KHz), non-directional radio beacons (NDBs) (190 - 435 kHz), amateur radio (135.7 - 137.8 KHz) and top band radio (1.8 MHz - 2 MHz) that are in 100 KHz to 1 MHz range might be a potential source of interference. We approximate these interference sources
### Table 3.4: Gain and BER for BPSK Modulation at $\varphi = 1e8$

<table>
<thead>
<tr>
<th>Distance (cm)</th>
<th>Frequency (KHz)</th>
<th>Gain (dB)</th>
<th>BER (BPSK)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>S-S</td>
<td>M-S</td>
</tr>
<tr>
<td>10</td>
<td>100</td>
<td>-37</td>
<td>-30.5</td>
</tr>
<tr>
<td>10</td>
<td>1000</td>
<td>-38</td>
<td>-38.5</td>
</tr>
<tr>
<td>20</td>
<td>100</td>
<td>-52</td>
<td>-47</td>
</tr>
<tr>
<td>20</td>
<td>1000</td>
<td>-53</td>
<td>-55</td>
</tr>
<tr>
<td>10</td>
<td>1000</td>
<td>-38.5</td>
<td>-14.5</td>
</tr>
<tr>
<td>20</td>
<td>1000</td>
<td>-47</td>
<td>-58</td>
</tr>
</tbody>
</table>

As Additive White Gaussian Noise, by assuming that the contributions from each of these sources are independently and identically distributed (i.i.d) Gaussian random variable $N(0, \varphi^2)$, with zero-mean and standard deviation $\varphi$.

The channel’s Signal to Noise Ratio (SNR) and maximum capacity using Shannon-Hartley theorem can be calculated as

$$SNR_{i-j} = \frac{P_t \cdot G^{i-j}(w,E_L,D,E_S,T)}{(N_T + N_E + I_0) \cdot \Delta f},$$

where $P_t$ is the transmitter power and $\Delta f$ is the receiver bandwidth. The SNR estimated can be used to calculate bit error rate (BER) for a given modulation type, frequency and $D$.

For a node to communicate with sink at a distance, more than the reliable communication range (say from a node implanted at wrist to sink on shoulder), multi-hop paths using relays are required. Assuming a spanning tree that connects all the nodes in each part of body to a sink using relays, we call the link that connects an upper limb sub-tree (T) to sink A through a relay as the last hop link (LH). According to Max-flow Min-cut theorem, a minimum cut on LH, with capacity $C(LH)$, limits the maximum number of nodes in the sub-network and the maximum possible traffic at each node [61]. $C(LH)$, for various lengths and paths of LH using Shannon-Hartley theorem, can be determined as

$$C(LH) = W \log_2(1 + SNR^{i-j}) \text{[bits/s]},$$

where $W$ is transmission bandwidth. For the $N$ nodes in T to communicate reliably, the condition, $\sum_u R(u) < C(LH)$, where $R(u)$ is the total data rate to and from node $u$, $\forall u \in \{1, \ldots, N\}$ has to be satisfied. Assuming equal data rate $\hat{R}$ in all the $N$ nodes, the maximum possible $N$, $N_{Max}$ in T is limited by

$$N_{Max} < \frac{C(LH)}{\hat{R}}$$

**BER & link capacity:** Assuming a bandwidth of 100 kHz and 1 MHz, with input power of 1 mW, $\varphi$ of $1e8$ and SNR estimated using (3.28), the BER calculated for BPSK modulation technique is
given in Table 3.3 & 3.4 for 10 cm and 20 cm between the transmitter and receiver. The M-M path offers minimum BER for both 10 cm and 20 cm. On the other hand, a maximum BER of greater than unity is offered by the M-S path with $D = 20 \text{ cm}$ operating at 1 MHz. However, the same M-S path gives reliable communication with BER $< 10^{-4}$ at both frequencies with $D = 10 \text{ cm}$, which is similar to the S-S path. The S-M path performs better than S-S and M-S paths in terms of BER for the distances and frequencies considered in this analysis. At $D = 10 \text{ cm}$, the maximum capacity of 1.03 Mbps is achieved in M-M path with $f = 1 \text{ MHz}$. At $D = 20 \text{ cm}$, M-S path offers a minimum capacity of 26 kbps.

Threshold distance & $N_{\text{Max}}$: If communication is assumed to be reliable when BER $< 10^{-4}$, at 100 kHz S-S path gives better gain upto 13 cm. The $D_{\text{Th}}$ attainable along M-S, S-M and M-M paths are 19 cm, 24 cm and 32 cm respectively. Using (3.29), (3.30), $D_{\text{Th}}$ and capacity of GC-IBN links, we estimated $N_{\text{Max}}$ for BPSK modulation, at 100 kHz, $\varphi = 1e7$ and $1e8$, with $R=100$ & 10,000 bps and the results are given in Table 3.5. According to the results, at $\varphi = 1e7$, and $D = 20 \text{ cm}$, S-S path could connect only one node in the subnetwork with $R = 10 \text{ Kbps}$. However, there could be upto nearly 10,000 nodes in subnetwork on M-M path with $\varphi = 1e8$, $D = 10 \text{ cm}$, each with $R=100 \text{ bps}$. 

---

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<th>$\varphi$</th>
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<th>$D_{\text{LH}}=20 \text{ cm}$</th>
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Chapter 4

Ensuring Safe Signaling Through Tissues

For safe deployment of galvanic coupled intra-body networks, it is critical to verify that (i) the amount of energy coupled to the tissue and (ii) the heat generated within tissues during signal propagation stay within permissible bound. In this chapter, we first estimate the bounds on the induced current density at the point of coupling and then analyze the thermal distribution within tissues, for varying transmission power levels, number of collocated transmitters, and blood perfusion conditions using finite element based numerical simulation and skin-phantom based experiments. Based on the results, we derive the suitable transmission duty cycles, separation distances and number of concurrent sources that may co-exist without raising the tissue temperature. The proposed strategies provide up to four fold increase in bandwidth efficiency through concurrent transmissions, ensuring sufficient bandwidth for implant communications. Our major contributions in this chapter are as follows:

a) We determine the bounds of electrical energy that can be safely coupled to the live tissue.
b) We analyze the thermal distribution in tissues in the presence of single or multiple sources and demonstrate that the net temperature rise remains below 1°C, which can then be efficiently regulated by the body’s natural mechanism.
c) We investigate the sensitivity of the spatio-temporal temperature profile in association with the normal and extreme rates of blood perfusion.
d) We devise optimized duty cycles for the implanted sensors and a transmission sequencing policy to control the temperature increase, as well as to improve the per-node bandwidth efficiency.
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This chapter is organized as follows. We present the related background and motivation in Sec. 4.1. Sec. 4.2 defines the limits for the frequency of operation and electrical energy coupled to the live tissue. Sec. 4.3 presents the GC tissue heating model. Sec. 4.4 introduces the effects of varying perfusion and suggests appropriate duty cycles and transmission sequences. Sec. 4.5 summarizes the results of the simulation and empirical test beds.

4.1 Background & motivation

Heating effects of implants have been investigated in the high frequency range [65] and in the MHz and GHz frequency range [67] for understanding the Specific Absorption Rate (SAR) from the radiation effects. In the range 100 kHz to 1 MHz, tissues have higher conductivity and offer longer signal propagating paths. Owing to this reduced attenuation, and hence absorption, the rise in temperature is also on the lower side. While the tissue heating effect of the higher kHz range has been analyzed for high power applications such as Diathermy, the impact of low power galvanic coupled links with possible multiple concurrent transmitters has not yet been studied. We study the thermal distribution in human tissues with single and multiple concurrent transmitters in tissues as well as devise strategy to control medium access that avoids tissue heating when the blood flow rate is affected.

4.2 Bounds on operating frequency and energy coupled to the tissue

4.2.1 Choosing the operating frequency

To identify the ideal range of the transmission frequency, we consider two factors: (i) frequency of the signals naturally generated by the human body, and (ii) signal loss caused by dissipation for a given frequency within the tissue. The electrical signals within the human body including neural impulses, ECG, and EEG signals operate at a frequency lower than 50 kHz, and therefore, we avoid the frequencies ≤ 50 kHz for intra-body communication. As the channel characteristics are frequency dependent, we need to identify the ideal operating frequency that reduces signal loss.

The signals transmitted into the tissue results into two current components, i.e., the conduction current and displacement current as given in (4.9). At lower frequencies, the conduction current that is caused by the movement of charges is high. This enables energy detention inside
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the tissue, resulting in higher intensity at the receiver end. At higher frequencies above 1 MHz, the conductivity remains constant and therefore the conduction current also remains fixed. However, due to increase in capacitance effect the displacement current grows larger with frequency. This ultimately results in signal dissipating from the body into the surrounding region, possibly causing interference externally, as well as limiting the energy incident on the receiver electrode.

For instance, at 100 kHz, the H field in the surrounding the body is in the order of a few $\mu A/m$, extending to around 50 mm at the exterior. On the other hand, at 10 MHz, the H field surrounding the body is higher by two orders of magnitude, extending to about 3 feet away from the body (refer Fig 4.1). The signal spreading out of the body is considered wasted, as it cannot reliably be detected at the embedded receiver. Thus, the signal loss is minimized as long as the operating frequency is restricted in such a way that the conduction current dominates the displacement current. This is true when the relationship $\frac{\sigma \omega}{\varepsilon} > 1$ holds in all tissues, i.e., when we limit the frequency lower than 2 MHz. Thus, to ensure that the dissipation loss is at minimum, the maximum frequency of operation is set at 1 MHz.

Figure 4.1: H Field spreading out of body at (a) 100 kHz (b) 1 MHz (c) 10 MHz

4.2.2 Bounds on field strengths induced in tissue

We use the finite element based model to estimate the safe IC fields coupled to the tissue. Complex Magnitude of $E$ and $H$ fields on the surface of skin is measured using the surface integrated normal components on the coupling electrodes modeled as non-model 2-D squares on the tissue surface by

$$E_{\perp S} = \int_S (E \cdot \hat{n}) \hat{n}$$

(4.1)
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\[ H \perp_S = \oint_S (H \cdot \hat{n}) \cdot \hat{n} \]  \hspace{1cm} (4.2)

where \( S \) is surface of square, \( \hat{n} \) is unit vector perpendicular to the surface \( S \), and \( \cdot \) denotes the dot product. Squares of same size as electrodes are chosen to calculate the field gain as ratio of input and output fields. The current through the object at distance \( l \) from the source can be obtained from Ampere’s law as

\[ I_{\perp S} \text{ at } l = \oint H.dl \]  \hspace{1cm} (4.3)

To measure the field strengths inside the tissue, line models are used as non-model object as they could exactly capture the field that an implant lead could receive being in the same position. Tangential \( E \) and \( H \) components to these lines are measured inside the tissue as

\[ E_{\parallel S} = \oint_S -\hat{n} \cdot (\hat{n} \times E) \]  \hspace{1cm} (4.4)

\[ H_{\parallel S} = \oint_S -\hat{n} \cdot (\hat{n} \times H) \]  \hspace{1cm} (4.5)

where \( \times \) denotes the cross product.

In order to ensure that the induced current density in the model is safe for the human tissue, we perform the following analysis.

On exciting the electrodes with voltage \( V \), the potential difference inside the high conduc-
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tive electrode $\oint E.\,dl$ becomes zero. The total current flowing through the surface of electrode of uniform cross section is given by

$$I = \iint_{s} J.\,ds,$$

where $J$ is the current density. The electric field $E$ at the electrode surface can be decomposed into normal and tangential components as $E_{t,\text{Electrode}}$ and $E_{n,\text{Electrode}}$, where $E_{\text{Electrode}} = E_{t,\text{Electrode}} + E_{n,\text{Electrode}}$. At the equipotential electrode-tissue (conductor-dielectric) contact area, the tangential component of electric field $E_{t,\text{Electrode}}$ approaches zero \cite{62, 63} and the non-zero normal component becomes the field of excitation in the tissue given as:

$$E_{n,\text{Electrode}} = E_{n,\text{Tissue}}$$

The $E_{n,\text{Tissue}}$ is shown as $E_{n,\text{Skin}}$ in Fig.4.2. At any instance, the current density at the contact area will be the largest among all other parts of tissue as the signal flows radially away from the region of source and attenuates with distance. Therefore, the source region is the area, where the safety levels of current injection is to be confirmed to avoid tissue damage. To ensure the safe limit of exposure in the contact area of dimension $10\, \text{mm} \times 10\, \text{mm} \times 1\, \text{mm}$, we limit the current flowing through the tissue at the electrode contact area in such a way that,

$$I_{\text{contact-area}} = \iint_{s} J.\,ds = \iint_{s} \sigma . E.\,ds, \leq 1\, \text{mA}$$

where $s$ is the surface area of electrode and $J$ is the current density is given by,

$$J = (\sigma + j\omega\epsilon'') E$$

that includes both conduction and displacement currents.

We confirmed the safe current density level using simulation by measuring the magnitude of current density at the rectangular region in contact with source electrode (region of maximum exposure). For an input current of $1\, \text{mA}$ at $0.5\, \text{V}$, the observed value of $J$ is $0.6\, \text{mA}/\text{m}^2$ which is well below the safe limit. In case of multiple transmitters in IBN, the transmitters should be spatio-temporally separated in order to ensure that the cumulatively aggregated values of current density (due to multiple sources) does not exceed beyond the safe level. We illustrate this further in section 4.4.
4.3 Tissue Heating Model for Galvanic Coupling

In this section, we develop a model that will enable analyzing the spatio-temporal thermal distribution in the presence of single or multiple galvanic coupled transmitters.

4.3.1 Need for analysis on tissue thermal response

The principal side effect arising from signal propagation in tissues in this kHz frequency range is tissue heating. Rise in local tissue temperature upto 1 °C is safe, as it can be easily controlled by the body’s natural thermo-regulation system [8]. The main objective of this analysis is to show that the weak signal power used in a typical two-node GC link of length 15 – 20 cm, i.e., a 5 mA source signal results in a temperature rise that is much lower than 1 °C. We envision that multiple sensors and actuators, possibly forming an array of electrodes, may be embedded in the tissue for long term holistic monitoring, and many of them may exist in close proximity. This situation raises the following concerns:

(i) When there are multiple long-term concurrent sources, the aggregated signal level may inject more thermal energy than what can be safely handled, (ii) When the variable blood perfusion rate, i.e., the primary body temperature regulating factor, drops below to a very low value, the increase in the local tissue temperature may impact the core body temperature.

To address these concerns, accurate knowledge of tissue thermal distribution is required that will also enable planning of transmission duty cycles and sequences. In this paper, we use finite element based numerical analysis to study the thermal distribution when signal propagates through the tissues. This study, accompanied by experimental measurements and duty cycle selection approach, also helps to capture the otherwise rare real-life situations, such as reduced or high rate blood flow, and the resulting impact on tissue temperature in the presence of concurrent transmissions from several closely located sources.

4.3.2 Preliminaries

The tissue heating effect of the higher frequency signals used in RF applications have been extensively studied. However, the impact of low power and low frequency galvanic coupled links with possible multiple concurrent transmitters has not yet been studied.

We use the modified form of the classical Pennes Bioheat equation [69] for analyzing the
heat generated in resting forearm, in which the rate of spatial tissue heat accumulation is given by

$$\rho_i C_i \frac{\partial T}{\partial t} = \nabla . k_i \nabla T + Q_{src} - Q_b$$

(4.10)

where $\rho$, $C$ and $k$ are the thermal properties namely density, specific heat and thermal conductivity, $\frac{\partial T}{\partial t}$ is the rate of thermal variation, $\nabla . k_i \nabla T$ is the heat flux, $Q_{src}$ denotes the thermal sources and $Q_b$ indicates the thermal regulation by blood perfusion. Subscript $i$ indicates properties of a specific tissue $i$.

### 4.3.3 Metabolic & GC Transmitter Heat Source Model ($Q_{src}$):

Tissues that have an embedded GC transmitter also have various ambient sources of heat. The normal body temperature is regulated using the metabolic heat generated by tissues with a rate of generation $Q_m$ that varies based on the tissue type, part of the body, ambient temperature and activity level. When a GC transmitter embedded in tissue injects a signal in the frequency range of 100 kHz to 1 MHz, a flow of current is induced in the form of conductive power dissipation. The work done in overcoming the tissue impedance during the current flow is converted to heat at a rate $Q_{cndc}$. Thus, the specific value of $Q_{cndc}$ at a point in the tissue depends on the injected signal strength and the tissue channel gain. When an electrode positioned at a point $A$ injects the signal, we estimate the signal strength observed at point $B$ using the channel gain $g_{AB}$ obtained from our earlier work in developing tissue channel models in [70].

The dielectric nature of tissue gives rise to dielectric power dissipation with a heat generation rate $Q_{dier}$, which is caused by the molecular dipole rotation. While this phenomenon is significant in the GHz frequency range, the contribution of $Q_{dier}$ in the low kHz, as is used by galvanic coupled tissues, is negligible, especially when compared to the dominant component of $Q_{cndc}$. The aggregated rate of conduction and dissipation of heat generated at a point $B$ by a GC transmitter at point $A$ can be expressed as

$$Q_{cndc} + Q_{dier} = k_s (\sigma_i (w) + j w \epsilon_i (w)) P_{in} . g_{AB},$$

(4.11)

where $P_{in}$ is the power admitted at the electrodes, $\sigma_i$ is the conductivity of tissue $i$, $w = 2\pi f$ is the angular frequency, $f$ is the frequency and $\epsilon_i$ is the tissue permittivity. When multiple transmitters coexist in a given area of a tissue with non-negligible co-interference, the total heat generated can be
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computed from the aggregated power level, given by,

\[ Q_{cn} + Q_{diel} = k_s (\sigma_i(w) + j w \epsilon_i(w)) \sum N (P_{in} g_{AB}) \]  \hspace{1cm} (4.12)

With the metabolic, conductive and dielectric sources of thermal generation, \( Q_{src} \) in (4.10) can be expressed as

\[ Q_{src} = Q_m + Q_{cn} + Q_{diel} \]  \hspace{1cm} (4.13)

4.3.4 Impact of Blood Perfusion on GC Tissue Heating (\( Q_b \))

Perfusion is the rate of heat transfer between the tissue and the arterial blood stream that plays a critical role in determining the steady state tissue temperature. The blood perfusion \( Q_b \) is expressed in terms of temperature difference between the tissue and blood given by

\[ Q_b = w_b \rho_b C_b (T - T_a) \]  \hspace{1cm} (4.14)

where \( w_b \) is the blood flow rate, \( \rho_b \) and \( C_b \) are the density and specific heat capacity of blood and \( T_a \) is the arterial blood temperature.

4.3.5 Impact of Boundary conditions on GC Tissue Heating:

The thermal sources and sinks that are specifically deployed at certain surfaces (e.g., convection at the air-tissue interface) and parts of tissue (e.g., heat flux at electrode-tissue interface) can be modeled as individual boundary conditions as follows:

- Tissue-tissue interface: We assume zero heat flux at the boundaries that connect the tissue under study with other tissues, which when stated as zero Neumann boundary, takes the following form:

\[ -\vec{n} \cdot k_i \nabla T + q T = g \]  \hspace{1cm} (4.15)

where the parameters \( q \) & \( g \) are set to 0 and \( \vec{n} \) is the unit normal vector.

- Convection at air-tissue interface: The temperature at the skin surface exposed to air is influenced by the ambient temperature. We model the boundary condition at the air-tissue interface as the convectional heat exchange, which according to the Newton’s law of cooling is given by

\[ k_s \frac{\partial T}{\partial x} = h_{cnvc} (T_{amb} - T) \]  \hspace{1cm} (4.16)
where \( h_{cnvc} \) is the convection coefficient. The corresponding Neumann boundary in (4.15) has \( q = h_{cnvc} \) and \( g = h_{cnvc} T_{amb} \).

- Electrode-tissue interface: The heat generated inside the sensor nodes, the transceiver circuits and the electrodes are also dissipated to the surrounding tissue according to the classical Fourier’s law. We model this source of heat as a Dirichlet boundary heat source \( q_{circ} \) at all the five electrode faces in contact with the tissue given by,

\[
q_{circ} = k_e (\sigma_e P_{in} + T_n)
\]

(4.17)

where \( k_e, \rho_e \) and \( C_e \) are the thermal properties of electrode, \( \sigma_e \) is the electrode conductivity and \( T_n \) is the temperature generated inside the node circuitry.

We use the model presented here for studying the tissue thermal response when coupled to an embedded signal source.

### 4.4 Duty Cycle and Sequencing based Thermoregulation

Under the normal conditions, the thermal energy induced in tissue from GC transmission is close to the negligible amount (see Fig.4.7(a)) as the induced power is below the safe limit suggested by [8] and the induced thermal energy is at least two orders of magnitude lower than the suggested safe level of 1 °C. However, in rare physiological conditions, the perfusion \((w_b)\) may reach extreme values beyond the average maximum \(w^{max}_b\) and minimum \(w^{min}_b\) values. Moreover, multiple transmitters can be embedded close to each other and engage in concurrent transmissions. We consider three of such conditions in this section and devise appropriate duty cycles and transmission

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Figure 4.3: Transmission sequence for multiple transmitters

sequence.

**Poor Perfusion Effect:** Under critical situations such as tissue damage, arterial damage or development of pressure points from prolonged resting, $w_b$ drops too low to quickly regulate the local tissue temperatures. This results in accumulation of thermal energy at points close to or in contact with the transmitting electrodes. When $w_b$ drops to a low value, less than $w_b^{min}$, the medium access should be controlled to keep the local temperature elevation within bounds, particularly when there is no perfusion as discussed below.

- **Zero Perfusion:** When $w_b=0$, the temperature elevates steeply with a high rate, reaching steady state at $t=\infty$. In such state of perfusion, the local temperature elevation can be controlled by transmitting $(t_{ON})$ for very short period (e.g., 0.1 secs) with long Inter Transmission Pause (ITP) period, $t_{OFF}$ ($\approx 1$ min). The short $t_{ON}$ and long ITP phase offers a low bandwidth efficiency ($\eta$) computed as $\frac{t_{ON}}{t_{ON}+t_{OFF}}$. When there are concurrent transmissions, each transmission elevates the temperature of the entire tissue area under study (refer Fig.4.6(b)). Therefore, concurrent transmissions are not recommendable when $w_b=0$. Possibly, each node can take turns in utilizing the brief $t_{ON}$ for signaling emergency link down state caused by poor perfusion. Sharing of the narrow bandwidth among multiple transmitters further plummets the bandwidth efficiency per node ($\eta$), down as

$$\eta = \frac{1}{N} \frac{t_{ON}}{t_{ON} + t_{OFF}}, \quad \eta_{all} = \frac{t_{ON}}{t_{ON} + t_{OFF}}$$  \hspace{1cm} (4.18)

where $N$ is the number of collocated nodes and $\eta_{all}$ is the bandwidth efficiency offered in total for all $N$ nodes.

- **Non-zero Poor Perfusion:** For $0<w_b<w_b^{min}$, the time required for bringing the elevated temperature to base body temperature ($T_{base}$) is much less than that required for the zero perfusion state
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Figure 4.4: 3D models (a) one source with face numbers displayed (b) two sources (c) multiple (20 sources) and (d) the relaxed mesh for (a)

(≈ 5 to 10 secs from Fig 4.7(e)). Moreover, the temperature elevation is higher in the electrode neighborhood and gradually drops with the channel gain $g$ i.e., the temperature elevation for larger distances from electrode is negligible. We denote the maximum distance from electrode experiencing noticeable rise in temperature as $D_T$. These features can be leveraged towards improving $\eta$ as follows:

1. Duty Cycle Based Control: Long term exposure to elevated temperature would impact the global body temperature. Hence we set our primary objective in the poorly perfused tissue to control the period of exposure, $t_{ON}$ to a target temperature $T_{target}$. We use the net spatio-temporal local tissue temperature elevation ($T_{net}$) as a parameter to control the level of thermal exposure, computed as:

$$T_{net} = \int_{t_1}^{t_2} \int_V TdV dt < T_{target} \tag{4.19}$$

for $t_{ON} \in [t_1, t_2]$ secs, where, $V$ is the spatial domain. $T_{target}$ generally depends on $w_b$, $h_{envc}$ and tissue area (e.g., ankle or abdomen). When $T_{net} \geq T_{target}$, we pause the transmission with ITP phase. This approach would nullify any noticeable influence of the local temperature over the global temperature and would also enable longer $t_{ON}$ phases for single transmission case.

2. Duty cycle & Concurrency Control: The above strategy can be extended to concurrent multiple transmissions ($m \leq N$) if $T_{net} \leq T_{target}$ with the $t_{ON}$ periods of all the $m$ transmitters coinciding. When $T_{net}$ exceeds $T_{target}$, the ITP phase ensues to bring $T_{net} = 0$ and then the cycle repeats. The
per node and the total bandwidth efficiency in this case is improved from (4.18) by a factor $m$ as

$$\eta = \frac{m}{N} \frac{t_{ON}}{t_{ON} + t_{OFF}}, \quad \eta_{alt} = \frac{m^2}{N} \frac{t_{ON}}{t_{ON} + t_{OFF}}$$  \hspace{1cm} (4.20)$$

The above defined concurrent strategy can be realized by leveraging the electrode spatial diversity to distribute the local temperature rise evenly in the tissue. For instance when two concurrent transmissions are not sufficiently separated they would influence each other, i.e., if the first transmitter is in the ITP phase and the second transmission is ongoing, the temperature in the area between the first and second transmitters cannot reach $T_{base}$ (refer Fig. 4.7(d) in Sec. 4.5). To avoid this neighborhood effect, the concurrent transmitters should be spatially well separated, with distance greater than $D_T$. We formulate this condition as $x.d_{es} + y.d_e \geq D_T$, where $d_{es}$ is the electrode separation distance, $d_e$ is the electrode dimension, $(a, b)$ & $(c, d)$ are the row and column location of the electrodes 1 & 2 respectively, $x = \sqrt{(a-c)^2 + (b-d)^2}$ and $y = \sqrt{(a-c-1)^2 + (b-d-1)^2}$. For e.g., the distance between the electrodes in (1,1) and (5,1) in Fig. 4.4(c) is given by $4d_{es} + 3d_e$. A possible sequencing strategy is depicted in Fig. 4.3(e), where if the first cycle transmits from region 1 and then the second cycle should choose a node from the region 2 that is diagonally farther from the first transmitter. The third cycle should choose from the region 3 and so on.

**Very High Perfusion Effect:** High perfusion with $w_b > w_b^{max}$ indicates a state of tissue abnormality, possibly caused by any one of a multitude of factors such as infection, vasodilation and high blood pressure. During such conditions, a local tissue temperature rise would push up the perfusion rate further, aggravating the situation. To control this, we restrict $t_{ON}$ to a duration that bounds $T < 50\%$ of its steady state value, followed by the ITP phase. Very high values of $w_b$ offers short duration of both $t_{ON}$ and $t_{OFF}$ (refer Fig. 4.7(e)). Hence, better control of the temperature elevation can be obtained with quickly alternating $t_{OFF}$ and $t_{ON}$ phases enabling better bandwidth efficiency compared to the poor perfusion state (refer Table 4.2). Multiple concurrent transmitters in tissues with $w_b > w_b^{max}$ should share $t_{ON}$ among themselves to control the overall rise in local temperature, bringing down the per node bandwidth availability as in (4.18).

**Temperature Control with Normal Perfusion:** When $w_b$ is within the average limits, the temperature rise in the area surrounding the electrode reaches a steady state in a few seconds (refer Fig. 4.7(a) & (b)) that is in the safety limits. Also, the $t_{OFF}$ duration is shorter than the poor perfusion state. Hence, the medium can be accessed with extended $t_{ON}$ phases and shortened ITP phases. On this basis, we modify the duty cycle & concurrency control strategy proposed for non-zero poor perfused tissue as follows to further improve the per node bandwidth efficiency.
CHAPTER 4. ENSURING SAFE SIGNALING THROUGH TISSUES

Figure 4.5: Thermal distribution for $P_{in} = 10\,\text{mW}$ at face $F7$ for (a) No transmission (b) 1 transmission and (c) 2 concurrent transmissions

• **Reduced ITP phase:** Rather than waiting for the elevated temperature to drop to $T_{base}$, a transmitter can start its $t_{ON}$ phase when the temperature of the neighbor drops to 10% of the elevated value. This would significantly increase $\eta$ as the last 10% fall in temperature takes more than 25% of the total $t_{OFF}$ duration (refer Fig.4.7(e)). This residual temperature would eventually fall to $T_{base}$ in the next ITP phase that follows the $t_{ON}$ phase of its neighbor. The per node bandwidth efficiency and all node bandwidth efficiency in this case are given by

$$
\eta = \frac{t_{ON}}{n(t_{ON} + 0.75t_{OFF})}, \quad \eta_{all} = \frac{m}{n} \frac{t_{ON}}{t_{ON} + 0.75t_{OFF}}
$$

(4.21)

where $n \leq N$ is the number of transmitters in a neighborhood with distance of separation less than $D_T$. We analyze this strategy in Sec.4.5 using Fig.4.7(c).

• **Skipped ITP phase:** The minimum temperature elevation with normal perfusion can be exploited with successive $t_{ON}$ phases as illustrated in Fig.4.7(d) for the $n$ closely located transmitters. This successive transmission can be used in conjunction with the distant concurrent $m$ transmitters discussed in the non-zero poor perfusion state. The ITP phase follows the $n\, t_{ON}$ phases. During ITP phase, all the points reach $T_{base}$, ensuring good thermoregulation as well as improved $\eta$.

For instance, for a 24 transmitter grid in Fig.4.3 during the first $t_{ON}$ phase, the unshaded
6 transmitters in the first (left most) pattern transmit. Next we choose the 6 unshaded transmitters in the next pattern in Fig 4.3(b) that are located diagonally to the initial set of transmitters. This would enable the local temperature in the initial transmitters to lower ensuring $T_{\text{net}}$ to be below $T_{\text{target}}$.\eta in this case can be very high compared to any of the above mentioned strategies because of $m$ parallel $t_{ON}$ sessions and all $N$ transmitters sharing a single ITP phase:

$$\eta = \frac{t_{ON}}{nt_{ON} + t_{OFF}}, \quad \eta_{all} = \frac{mt_{ON}}{nt_{ON} + t_{OFF}}$$ \text{(4.22)}

**Duty Cycle Policy:** Based on the above discussion, our fitted duty cycle control policy on $t_{ON} \& t_{OFF}$ is summarized below for a specific scenario for the parameters in Table 4.2.

$$t_{ON} = \begin{cases} 
    t_{\text{min}}, & \forall w_b \leq w_b^{\text{min}} \\
    7.206 - \frac{1.164}{w_b^{\text{min}}}, & \forall w_b^{\text{min}} < w_b \leq w_b^{\text{max}} \\
    0.72 - 0.2w_b, & \forall w_b > w_b^{\text{max}} 
\end{cases} \text{ (4.23)}$$

$$t_{OFF} = \begin{cases} 
    1.7w_b^2 - 6.3w_b + 6.74, & \forall w_b \leq w_b^{\text{min}} \\
    0.4w_b^2 - 2.8w_b + 6.4, & \forall w_b^{\text{min}} < w_b \leq w_b^{\text{max}} \\
    t_{\text{max}}, & \forall w_b > w_b^{\text{max}} 
\end{cases} \text{ (4.24)}$$

We analyze these policies along with the thermal distribution results, duty cycle and sequencing strategies as well as the bandwidth efficiency achieved in the following section.

### 4.5 Simulation Results and Experimental Studies on GC Thermal Distribution

In this section, we explain our finite element based numerical simulation and skin phantom-based empirical approach for investigating the transmission duty cycle effects on tissue thermal distribution.

**Finite element based simulation set-up**

We model the three dimensional rectangular tissue slab of dimension $15 \times 20 \times 0.3 \text{ cm}^3$ using Autodesk 123D Design software. A cubical volume of dimension $1 \times 1 \times 0.05 \text{ cm}^3$ is carved out of the tissue block to mark the site of the electrode. We build the one electrode, two electrode
CHAPTER 4. ENSURING SAFE SIGNALING THROUGH TISSUES

and multielectrode models as illustrated in Fig.4.4(a),(b) and (c) respectively. In the multielectrode model, the electrodes are separated by 2.3 cm. Each electrode has five faces if on surface and six faces in case implanted. We import the model geometry to MATLAB, generate and redefine the tetrahedral mesh for finite element analysis as shown in Fig.4.4(d). The spatio-temporal thermal energy distribution in the model is obtained by solving (4.10) using the parabolic PDE solver over a given period of time. We set the parameter as given in Table.4.1 with the boundary conditions set individually as required for different conditions.

1. Model verification: Initially, we verify the steady state tissue temperature to be maintained at $T_{base} = 35^\circ C$, without the external signals. The electrode faces $F_1$ to $F_5$ in Fig.4.4(a) are set as insulating boundaries. We apply (4.16) to the tissue face $F_6$ to make it act as a convective boundary and assume that $Q_{src} = Q_m$. The resulting steady state temperature throughout the tissue is within $35 \pm 10^{-12}^\circ C$ as shown in Fig.4.5(a), thus ensuring accurate thermo-regulation of the tissue. It takes around 2 secs for the simulator to reach the steady state temperature. We therefore, allow 2 secs as the initial phase in each simulation procedure.

2. Thermal Distribution vs Transmit Power: To observe the tissue temperature variation during a transmission, we alter the boundary condition of the electrode faces (face $F_1$ to $F_5$ in Fig.4.4(a)) as
constant heat flux (4.17). For this analysis, \( Q_{src} \) takes the form in (4.13) to include the conductive and dissipative effect (4.11) of the current flowing through the tissue. Fig.4.5(b) shows the spatial temperature profile for \( P_{in}=10 \text{ mW} \) after 10 secs at the non-convective side (face \( F_7 \), behind \( F_6 \)), which experiences higher temperature elevation compared to the convective face. The temporal thermal elevation is depicted in Fig.4.7(a) for \( P_m=1 \text{ mW}, 10 \text{ mW} \) and \( 100 \text{ mW} \) and \( w_b=1.2 \) at points that are 0.5 cm and 2 cm away from the electrode. The maximum temperature is recorded at the area in contact with the electrode followed by the points in \( F_7 \) that are directly behind the contact area. The elevation in temperature is present only at the proximity of the electrode within an average radius, \( D_T \) of 3 cm at the convective face and 3.5 cm at the non-convective face, that reaches steady state in few seconds.

3. **Empirical validation using Skin Phantom:** We validate the tissue thermo-regulation model explained above using a skin phantom (7 × 7 × 0.3 × cm³) coupled with 2 sources. The experimental set-up is shown in Fig.4.6(a). We use the alligator clips (40 mm) as the exciting and ground electrodes and a high precision RTD thermometer to measure the rise in temperature. We modify the simulation

Figure 4.7: (a) Rise in Temperature (°C) with varying \( P_{in} \) for 1 transmitter and (b) for 2 transmitters (c) Reduced ITP for 2 transmitters; (d) Skipped ITP for 2 transmitters (e) \( t_{ON} \) & \( t_{OFF} \) vs \( w_b \); (f) \( T_{net} \) vs number of concurrent transmissions; (g) \( T \) for \( w_b = 0 \) and model validation using phantom experiment.
CHAPTER 4. ENSURING SAFE SIGNALING THROUGH TISSUES

Table 4.2: Duty cycle & bandwidth efficiency (t_{min}=0.1\,\text{secs}, \; t_{max}=2.2\,\text{secs}, \; T_{target}=20^{\circ}C, \; w_b^{min}=0.5 \; & \; w_b^{max}=1.5, \; m=4, \; n=6)

<table>
<thead>
<tr>
<th>State</th>
<th>w_b</th>
<th>t_{ON}</th>
<th>t_{OFF}</th>
<th>\eta (N=1)</th>
<th>\eta (N=24)</th>
<th>\eta_{all}</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.1</td>
<td>0.1s</td>
<td>50s</td>
<td>0.2%</td>
<td>0.01%</td>
<td>0.2%</td>
</tr>
<tr>
<td>Poor</td>
<td>0.1</td>
<td>0.1s</td>
<td>7s</td>
<td>1.43%</td>
<td>0.24%</td>
<td>0.96%</td>
</tr>
<tr>
<td>Perfusion</td>
<td>0.3</td>
<td>0.1s</td>
<td>6.2s</td>
<td>1.6%</td>
<td>0.27%</td>
<td>1.08%</td>
</tr>
<tr>
<td>Average</td>
<td>0.6</td>
<td>5.7s</td>
<td>5.6s</td>
<td>50.4%</td>
<td>14.3%</td>
<td>57.3%</td>
</tr>
<tr>
<td>Perfusion</td>
<td>1.0</td>
<td>6.05s</td>
<td>3.36s</td>
<td>64.3%</td>
<td>15.3%</td>
<td>61%</td>
</tr>
<tr>
<td>1.4</td>
<td>6.4s</td>
<td>3.3s</td>
<td></td>
<td>66%</td>
<td>15.4%</td>
<td>61.4%</td>
</tr>
<tr>
<td>Very high</td>
<td>1.6</td>
<td>0.4s</td>
<td>2.2</td>
<td>15.38%</td>
<td>0.64%</td>
<td>0.64%</td>
</tr>
<tr>
<td>Perfusion</td>
<td>2.0</td>
<td>0.32s</td>
<td>2.2</td>
<td>12.7%</td>
<td>0.53%</td>
<td>0.53%</td>
</tr>
</tbody>
</table>

model parameters (such as the tissue, electrode dimensions) with \( w_b = 0 \) to match with the phantom set-up.

We compare the temperature generated in the phantom for a 1 mW signal with that of the corresponding simulation model in Fig.4.7(g) upto 50 min. we observe that the temperature rise is linear in both simulation and experiment, with a slightly faster rate of rise in phantom. This is caused by deeper signal injection to the tissue through the sharp pins in the alligator clips while the simulation uses a flat surface electrode geometry. The close match in thermal distribution between the simulated and empirical set-up validates the proposed finite element model used for the analyses in this paper.

4. Two Concurrent Transmissions: Next, we extend this model for two concurrent transmissions by adding the constant heat flux \((4.17)\) for two electrodes in Fig.4.4(b). The \( Q_{src} \) term in \((4.13)\) uses the conduction and dissipation effect in \((4.12)\) for incorporating both the current sources in the computation. The spatial distribution of \( T \) after 10 secs at the non-convective side is shown in Fig.4.5(c), which has the maximum temperature rise same as that of the single transmission case. However, when the two electrodes are separated less than 3 cm (2.3 cm in Fig.4.5(c)), the heat density in the area between the two electrodes is higher than the rest of the surrounding tissue because of the heat aggregation. Fig.4.7(b) shows the temporal thermal elevation for \( P_{in}=1\,\text{mW}, 10\,\text{mW} \; \text{and} \; 100\,\text{mW} \; \text{and} \; w_b=1.2 \) at 0.5 cm (position A), 1 cm from electrode 1 towards the edge (position C) and at the mid point between the electrodes (position B). Note that at B, which is 0.15 cm farther from electrode 1 than C, the generated heat is atleast 0.01 °C higher, proving that separation between concurrent transmissions is a key factor in determining the local temperature rise.

5. Thermal Distribution vs Perfusion: In Fig.4.7(e) the steady state temperature for \( w_b=1.6 \) is
reached in <1 secs while that for \( w_b=0.4 \), takes \( \approx 9 \) secs. The worst case \( (w_b=0) \) is depicted in Fig. 4.7(g), where the temperature with 2 sources reach the 1 \(^\circ\)C in 6.6 hours, exceeding the safe limit. Fig. 4.7(f) compares \( T_{net} \) computed using (4.19) versus the number of concurrent transmitters in a tissue area over 20 secs when \( w_b \) is within the average limits for \( P_{in}=1 \) mW. For \( T_{target} = 20 \) \(^\circ\)C, the safe number of concurrent transmissions can go upto 9.

- **Reduced ITP phase:** Fig. 4.7(c) illustrates the duty cycle based thermoregulation for 2 transmissions. The temperature rises more at 0.5 cm from electrode 1 than at farther points during \( t_{ON}=[0, 2] \) secs and then drops to 90% of \( t_{base} \) during the next \( t_{OFF}=[2, 4] \) secs. During \( t_{ON}=[4, 6] \) secs, electrode 2 transmits rising the temperature near electrode 2 and in between the 2 electrodes. However, the temperature at point closer to electrode 1 continue to drop and reaches \( t_{base} \) in the next \( t_{OFF} \) slot. This technique improves the per node efficiency by atleast 10%.

- **Skipped ITP phase:** Fig. 4.7(d) displays the temperature control when the first ITP phase after the \( t_{ON} \) of electrode 1 is occupied by the \( t_{ON} \) of electrode 2. In the following ITP phase, all the points reach \( T_{base} \), ensuring good temperature control and improves \( \eta \) by atleast 3.5% and \( \eta_{alt} \) by 13% more than the Reduced ITP strategy. Table 4.2 summarizes bandwidth efficiencies obtained using skipped ITP phase for average perfusion state.
Chapter 5

Energy Efficient & Energy Balanced In-Body Topology

The last chapter illustrated that the galvanic coupled intra-body communication overcomes the problem of massive absorption of RF waves in the body. However, the unique intra-body channel raises several questions on the topology of the implants and the external (i.e., on skin) data collection nodes. This chapter makes the first contributions towards (i) building an energy-efficient topology through optimal placement of data collection points/relays using measurement-driven tissue channel models, and (ii) balancing the energy consumption over the entire implant network so that the application needs are met. We achieve this via a two-phase iterative clustering algorithm for the implants and formulate an optimization problem that decides the position of external data-gathering points. Our theoretical results are validated via simulations and experimental studies on real tissues, with demonstrated increase in the network lifetime.

- **Problem Definition.** Given the weak signal strength, the network performance of the GC-IBN depends on the length of the links, which is in turn determined by the position of the signal aggregation points or relays. While the GC channel has been modeled earlier in [70], there is no prior work on designing a practical network based on the channel observations. To address this gap, this paper develops a theoretical framework for designing a clustered network, where multiple implanted nodes at various depths inside body tissues ($N_2, \ldots, N_4$ in Fig. 5.1) are served by an external on-skin relay ($R_2$). The relays may also forward data to reach a specific relay ($R_1$) that has RF capability to connect with the outside world.

The clustering problem presented here is different from the extensive work for classical
wireless sensors that exists today in the following ways: (i) In an IBN, considering the sensor separation in terms of distance alone is not sufficient, but also the specific tissue conduction properties and their relative dimensions must be taken into account. (ii) For RF signals, we only need to avoid concurrent signal reception from two or more sources at the receiver that results in detection errors. Instead in the GC-IBN we need to prevent constructive signal combination at any intermediate point that goes beyond permissible limits for tissue safety. (iii) The relative depths at which the implants are located must be considered in clustering to ensure that the harder-to-reach implants have proportionally longer lifetime. (iv) There is no redundancy among nodes within the IBN, and hence, every node is important, and vital to the application. (v) The links between the implants and the external relays exhibit highly asymmetric behavior between uplink and downlink directions. (vi) Finally, there are varying traffic needs between different implants served by the same relay, which may necessitate proximity considerations for certain nodes.

• **Contributions.** The main contributions of this work are:

1. We propose the first theoretical clustering framework that provides clear guidelines for placing on-skin relay nodes for embedded implants, while considering the heterogeneous composition and 3-D characteristics of human tissue channels.

2. We derive an acceptable GC-IBN link length distribution that minimizes transmission power for all the nodes, as well as the number of relays, while meeting application demands. This ensures balanced energy consumption among all the nodes.
3. We evaluate the effectiveness of the proposed clustering framework using detailed simulation models and experimental studies involving porcine tissue.

The rest of this chapter is organized as follows: Sec. 5.1 summarizes the related background contributions and problem motivation. Sec. 5.2 introduces our GC-IBN system model. Sec. 5.3 explains our 2-phase clustering framework, which is then analyzed and evaluated in Sec. 5.4.

5.1 Background & Motivation

The comparatively short links in GC-IBN ($\approx 30$ cm $[70]$) and the varying body channels require dynamic cluster formation. However, analyzing all possible solutions for relay placement is an NP-hard problem, and the short time-scales suggest the use of heuristic approaches.

**Clustering constraints:** The GC-IBN is constrained to have relay on the skin-surface, and hence the implanted nodes are no longer candidates for role switching. A typical GC-IBN with sensors and actuators involve bidirectional traffic and comprises non-redundant implants. The network is considered operational until the first implant runs out of energy.

**3-D propagation:** In GC-IBN, when there is a transmitter on surface, a receiver at tissue depth (e.g., $R_2$ & $N_3$ in Fig. 5.1) receives a stronger signal than a receiver on the skin surface at the same distance (e.g., $R_2$ & $N_5$ in Fig. 5.1) owing to the superior conducting properties of the inner tissues. Straightforward application of techniques, such as K-Means clustering that have been applied to terrestrial WSNs, do not account for the different propagation paths and are unlikely to perform the required optimal partition as the number of clusters and initial seed value are unknown. In order to use the instance based classification clustering such as k-Nearest Neighbors algorithm (kNN), the raw data is to be pre-processed for identifying the outliers (such as no node or isolated node condition).

**Relay positioning constraints:** In GC-IBN, the implant locations are influenced by medical applications, and may result in small pockets of deployment. Thus, the distribution of the relay points in this case is non-uniform. Moreover, relays must forward information among themselves, serving as a conduit for messages among the sensors, instead of direct communication between multiple implant pairs (e.g., $R_1$ and $R_2$ forward information, instead of $N_1$ and $N_2$ directly). This ensures lower energy consumption for the implants, but imposes constraints on the number of nodes connected to relays.
5.2 GC-IBN System Model & Bounds

One of the relays function as data sink and other relays, apart from connecting the implants, forward the data towards the sink, forming a two-tier hierarchical architecture. We limit this work to optimize the intra-cluster topology that includes choosing the nodes participating in each cluster and estimating the relay position. We provide an overview of the clustering goals in this section, with the variables listed in Table I.

We assume a set \( \{N_1, ..., N_n\} \) of iid nodes embedded as implants in the body, or placed on the body surface, as shown in Fig.5.1. The position of a node \( N_m \) is represented by \( L_m = \{(x_m, y_m, z_m), T_m\} \), where \( T_m \in \{\text{skin}(S), \text{muscle}(M)\} \) is the tissue where \( N_m \) is present, and \( \{x_m, y_m, z_m\} \) represents the three dimensional coordinates. Specifically, \( z_m \in \{0, ..., D_i\} \) denotes the depth at which the node \( N_m \) is present in the tissue \( T_m \). \( D_i \forall i \in T \) is the thickness of chosen tissue layer.

Note that we limit this work to skin and muscle tissues, for the purpose of ease of explanation and given that implants are not generally embedded in fat or bone, but the steps can easily be extended to include fat or bone tissues.

The primary goal of clustering is to place the relay closer to implants (for short links) as well as to connect more nodes to the relay, even from neighboring clusters (cluster merging) so as to reduce the number of relays required (eg., as illustrated in clusters \( C_1, C_3 \& C_5 \) in Fig.5.2(b)). If the number of nodes exceeds the relay capacity (defined below), an additional relay is assigned to handle the overload situation (refer \( C_4 \) in Fig.5.2(b)). Intuitively, no relays are assigned to regions with no nodes (refer \( C_2 \) in Fig.5.2(b)). An isolated node that cannot be reached from existing relays should be allocated to a dedicated relay (similar to \( C_6 \) in Fig.5.2(b)).

The number of nodes in a cluster \( C_k \), \( \forall k \in \{1, ..., K\} \) is denoted as \( |C_k| \leq n \), where \(|.|\) denotes cardinality. \( K \) is number of clusters that contains \( I_k \leq |C_k| \) implants and \( |C_k|-I_k \) surface nodes. The relay \( R_k \) assigned for cluster \( C_k \) is on the skin surface at \( L_{R_k} \) depth (\( z_k=0 \)) and is reachable from all the \( |C_k| \) nodes through single-hop transmission. Fig.5.2 shows the scenario where the GC-IBN clusters extend over multiple tissues. We avoid representing the length of the link (\( \Lambda_{mR_k} \)) between the implant \( N_m \) and relay \( R_k \) purely in terms of Euclidean distance, considering the presence of heterogeneous tissues between the surface relay and the muscle implant (for surface nodes, the Euclidean assumption still holds). Instead, we approximate the length of the link to be homogeneously co-planar in muscle with the relay assumed to be vertically below the surface.
Figure 5.2: (a) Clustered GC-IBN (b) Clustering objectives (gray lines represent uniform grids and shaded blue area denotes optimized clusters)

containing $L_{R_k}$, and on the plane of the implant in the muscle at $L'_{R_k}$.

$$
\bar{\Lambda}_k = \left\{ \Lambda_{iR_k} = X^2 + Y^2 + Z^2, \quad T_i = \{S\}, i = \{1, \ldots, |C_k| - I_k\} \right\}
$$

\begin{equation}
\Lambda_{jR'^k} = X'^2 + Y'^2 + Z^2, \quad T_j = \{M\}, j = \{1, \ldots, I_k\}
\end{equation}

where $\bar{\Lambda}_k = \{\Lambda_{1R_k}, \ldots, \Lambda_{nR_k}\}$, $X = |x_i - x_{R_k}|$, $Y = |y_i - y_{R_k}|$, $X' = |x_j - x_{R'_{k}}|$, $Y' = |y_j - y_{R'_{k}}|$, $Z = z_{i,j}$ and $\{x_{R'_{k}}, y_{R'_{k}}\} \in L'_{R_k}$.

The channel gain between the node and the relay it is connected to can be estimated in terms of the link length as:

$$
\bar{g} = \left\{ \begin{array}{ll}
g_{iR_k} = f_{S-S}(\Lambda_{iR_k}), & i = \{1, \ldots, |C_k| - I_k\} \\
g_{jR_k} = f_{M-S}(\Lambda_{jR'^k}, z_j), & j = \{1, \ldots, I_k\},
\end{array} \right.
$$

where $\bar{g}$ is the channel gain vector corresponding to $\bar{\Lambda}_k$. For a surface node to the relay, that we term as skin to skin (S-S) scenario, let $f_{S-S}$ be the function mapping the Euclidean link length to the channel gain between them using the circuit based channel model built with the tissue electrical properties. These two nodes can be the on-skin sensor and the relay (eg., $N_5$ & $R_2$ in Fig 5.1), or
between two relays (eg., $R_1$ & $R_2$ in Fig.5.1). Similarly, $f_{M-S}$ maps the approximated implant-relay link length to the multi-tissue channel gain, given the path traverses muscle to skin (M-S). We achieve this by adding the skin and fat impedance to that of the muscle at $L'_{R_k}$ to obtain the heterogeneous M-S path gain. Functions $f_{S-S}$ & $f_{M-S}$ have been earlier developed in [70], which we use in this work. In Fig.5.1 $N_5$ is connected to $R_2$ through the S-S path, while the implants $N_2$, $N_3$ and $N_4$ communicate with $R_2$ via the M-S path.

As the first step towards our proposed heuristic clustering scheme, we establish the upper and lower bounds on the transmit power that are feasible, as this directly impacts the separation distance.

- **Lower bound on $P_t$:** The bit error rate must remain below the application demands. This can be achieved by ensuring the channel SNR remains above the desired SNR ($\delta_{mR_k}$) by controlling the minimum required transmit power $P_{tm}^{min}$ as:

$$P_{tm}^{min} = \begin{cases} \frac{\delta_{mR_k} N_o^{mR_k} f_m}{g_{mR_k}} & \forall m \in \{1, \ldots, |C_k|\}, \Lambda_{mR_k} > 0 \\ 0 & \forall m \in \{1, \ldots, |C_k|\}, \Lambda_{mR_k} = 0, \end{cases}$$

(5.3)
where $N_{mR_k}$ is the Gaussian noise P.S.D in $N_{m-R_k}$ path with zero mean, and $f_m$ is the receiver bandwidth. The condition $\Lambda_{mR_k}=0$ is possible only with surface nodes that also acts as a relay. We ignore this condition for further analysis.

- **Upper bound on $P_t$:** $P_{t_m}$ is bounded above by two factors. First, to ensure tissue safety, the maximum transmit power, $P_{t_m}^{max}$ must satisfy the following condition assuming a single transmission occurs at a time.

$$P_{t_m} \leq P_{t_s} \forall m \in \{1, \ldots, |C_k|\},$$  \hspace{1cm} (5.4)

where $P_{t_s}$ is the maximum safe power that can be transferred though the tissues [8]. Second, the lifetime of the implant must be sufficiently long. Assuming M-PSK modulation, the energy consumed by $N_{m}$ over a period $H_m$ can be estimated using the link budget calculation as,

$$E_{H_m}^{m} = \frac{E_{b}^{m} \eta_m H_m}{f_m \log_2 M_{m}^{'}} \forall m \in \{1, \ldots, |C_k|\},$$  \hspace{1cm} (5.5)

where $E_{b}^{m}$ is the bit transmission energy, $M'$ is the modulation level and $\eta_m$ is the data rate required in $N_{m}$ (Eg., $\eta$ of ECG nodes is 4 kbps). For lower values of $E_{b}^{m}$, the total energy consumption over $H_m$ will also be lower, indicating the possibility of extended $H_m$. With an initial energy store of $E_0$ for the battery life to extend beyond $H_m$, the following condition should be satisfied:

$$H_m \cdot P_{t_m} \leq E_0, \forall m \in \{1, \ldots, |C_k|\},$$  \hspace{1cm} (5.6)

Using (5.4), (5.5) and (5.6), the upper bound on $P_{t_m}$ for the given $H_m$ and $P_{t_s}$ can now be obtained as:

$$P_{t_m}^{max} = \min \{P_{t_s}, \frac{E_0}{H_m}\} \forall m \in \{1, \ldots, |C_k|\}$$  \hspace{1cm} (5.7)

**Bounds on $\Lambda$:** The maximum link distance that offers the desired node lifetime, SNR and BER, without exceeding $P_{t_s}$ is the threshold length, $\Lambda_{m}^{th}$ of node $N_{m}$, and estimated as:

$$\Lambda_{m}^{th} = \begin{cases} f_{S-S}^{'}(P_{t_m}^{max}, g_{mR_k}) & \forall m \in \{1, \ldots, |C_k| \} \\ f_{M-S}^{'}(P_{t_m}^{max}, g_{mR_k}) & \forall m \in \{1, \ldots, I_k\} \end{cases}$$  \hspace{1cm} (5.8)

where, $f_{S-S}^{'}$ & $f_{M-S}^{'}$ are the inverse functions of $f_{S-S}$ & $f_{M-S}$ along the S-S and M-S paths respectively, providing the length of the link that offers the gain $g_{mR_k}$ for $P_{t_m}^{max}$. We assume the threshold link length along S-S path, $\Lambda_{S-S}^{th}$ within the cluster as constant as the surface nodes need not
be that constrained in terms of energy replenishment. However, the threshold link length of the M-S path, \( \Lambda_{M-S}^{th} \triangleq \min \{ \Lambda_{1}^{th}, ..., \Lambda_{I_k}^{th} \} \) concerning the implants of a cluster vary in \((0, f'_{M-S}(Pt_s, g))\]
as implants with long life require shorter links and vice-verse. Hence, both single hop and reliable communication from \( N_m \) to \( R_k \) is feasible if

\[
0 < \Lambda_{mR_k} \leq \Lambda_{m}^{th}, \forall m \in \{1, ..., |C_k|\}, \forall k \in \{1, ..., K\}. \tag{5.9}
\]

- **Cluster capacity limit:** The overall bandwidth requirement of a cluster cannot exceed the capacity \((Q_o)\) of the outgoing link that connects \( R_k \) to the next hop surface relay or sink. Hence, \( C_k \) is restricted to the set of nodes with the sum of required bandwidth less than \( Q_o \). This bounds \( C_k \) as follows:

\[
1 \leq |C_k| \leq \max_{|C_k|} \left( \sum_{m=1}^{\left| C_k \right|} \eta_m \leq Q_o \right) \tag{5.10}
\]

### 5.2.1 Heterogeneity factors in GC-IBN

Given the choice of tissues where the implants are present, the path-loss and energy cost differ. In addition, the bandwidth requirements of the nodes are also not uniform (e.g., sensors may require higher uplink bandwidth while actuator may need higher downlink bandwidth). To address this scenario, we capture the various heterogeneity factors in a single-valued weight metric.

- **Heterogeneity from embedded tissue \((T)\):** Surface nodes \((T_m=\{S\})\) incur low energy-conservation costs \((C(S))\) as they are on the skin, hence easily accessible. Implants \((T_m=\{M\})\) incur a higher cost per unit of energy spent \((C(I))\) as they require invasive procedures for energy replenishment. For extended cluster lifetimes, the overall energy consumption of implants is to be minimized as \( C(I) < C(S) \) or

\[
\frac{1}{I_k} \sum_{i=1}^{I_k} Pt_i \ll \frac{1}{(|C_k| - I_k)} \sum_{s=1}^{\left| C_k \right| - I_k} Pt_s, \tag{5.11}
\]

for \( I_k \) implants and \(|C_k| - I_k \) surface nodes. Hence \( \Lambda_{mR_k} \) is to be weighed based on \((T_m, z_m)\) to ensure optimal clustering.

- **Heterogeneity from data rate \((\eta)\):** The difference in \( \eta \) among the cluster nodes suggests that nodes with higher data rates require longer duty cycles and consume more energy. For instance, assuming \( \{N_A & N_B \in C_k\} \) with \( \Lambda_{AR_k} = \Lambda_{BR_k} \) and \( \eta_A & \eta_B \) as respective required data rates,

\[
\text{if } \eta_A \propto \gamma \eta_B, \text{ then } Pt_A \propto \gamma Pt_B \tag{5.12}
\]
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for some constant $\gamma > 1$. This results in an undesirable variation in $P_t_m, \forall m \in \{1, \ldots, I_k\}$. To ensure equitable energy costs throughout the network with varying $\eta$, the on-skin relay should be ideally moved closer to the node with higher $\eta$ (i.e., closer to node $A$ in (5.12)) to compensate the additional $P_t (P_t_A)$ required. This can be achieved by weighing the links proportionally with respect to $\eta$.

5.2.2 Node weights Estimation

The critical heterogeneity factors $T$, $z$ and $\eta$ are integrated into an effective weighted link metric $w_m$ for each node estimated as,

$$w_m = \alpha^{(T_m + z_m)} - 1 \frac{\eta_m}{\sum_{i=1}^{|C_k|} \eta_i}, \forall m \in \{1, \ldots, |C_k|\}$$

(5.13)

$\forall k \in \{1, \ldots, K\}$, where the first term from the right weighs the nodes according to the tissues and depths, while second term modifies the weights based on the normalized data rate. $\alpha \in [1, 10]$ is the energy prioritizing factor chosen based on the difference desired between $C(I)$ and $C(S)$. Enumerating $T_m = \{S, M\}$ as $T_m = \{1, 2\}$, $\alpha = 1$ denotes $C(I) = C(S)$ that is suitable for setting up a short term GC-IBN, with equal life span for surface nodes and implants. On the other hand, $\alpha = 10$ sets much higher $C(I)$ suitable for long term deployments.

5.2.3 The Uniformity factor

As one of the design goals, we target proportional power consumption among all implants. To quantify this concept, we devise a quality metric $\hat{U}$ that determines an approximate percentage of residual energy in the remaining nodes when the first implant is lost.

$$\frac{\min(\Lambda_{mR_k})}{\max(\Lambda_{mR_k})} > \hat{U}, \forall m \in \{1, \ldots, I_k\}$$

(5.14)

For example, $\hat{U} = 0.8$ ensures that when $N_x \triangleq \max_x (P_t_x)$ gets depleted with $P_t_x.H_x = E_0$, the residual energy in other live implants will be $\leq 20\%$ of $E_0$. Accordingly, in our clustering framework, we restrict $C_k$ to include only those implants that satisfy (5.14) for an estimated relay position. The number of nodes satisfying the uniformity constraint might be increased if $\hat{U}$ is relaxed to a lower value (e.g. from 0.8 to 0.7). The influence of $\hat{U}$ on $K$ is analyzed in Sec 5.4 S5.
5.3 GC-IBN Cluster Topology Design

In this section, we propose our heuristic two-phase iterative clustering framework for GC-IBN. In the first phase or the Initial Cluster Approximation Phase (ICAP), we generate the initial approximation of $K$ and $C_k$ using the node positions. With no prior knowledge on $K$ or $L_{R_k}$, we use neighborhood learning that uses lower energy than conventional message passing techniques. In the second phase, we perform the Nearest Neighbor based Iterative Cluster Optimization (NICO) until the achieved clustering is deemed optimal. Thus, through this approach, the quantity, size and position of the clusters are iteratively optimized for energy efficiency under performance constraints.

5.3.1 Phase I. Initial Cluster Approximation Phase (ICAP):

In this phase, we use definitive cuboid grid based clustering as (i) it identifies and connects outliers while no nodes are left under-classified, a common possibility among density based approaches [75], and (ii) partitioning the node space into finite number of cuboids requires fixed number of executions that depends only on the number of cuboids irrespective of $n$. The cuboids are preferred over the spheres as the grid size is different in the third spatial dimension (i.e., height varies). In addition, the cuboid packing avoids area overlaps or gaps so that every node is considered only once for clustering.

Grid size estimation: Larger clusters (i.e., with higher ($|C_k|$)) require longer average link lengths, consuming more $Pt$, while smaller clusters increase the number of clusters $K$. In addition, the clusters should also satisfy the link length condition in (5.9). We use the unit-cuboid grids to identify the clusters of right size while separating the nodes by a maximum threshold distance of $\Lambda_{th} = \min(\Lambda_{th}^S - S, \Lambda_{th}^M - S)$.

**Theorem 5.3.1**: In a unit cuboid of length and width being $\lambda = \Lambda_{th}^k / \sqrt{2}$, the maximum S-S or M-S link length between the nodes and random relay positions satisfies $\Lambda_{max}^m_{R_k} \leq \Lambda_{th}^k$.

**Proof**: The link lengths of nodes in the grid are independent and have the same distribution. Let the maximum link length in $C_k$ be $\Lambda_{max}^m_{R_k}$. Using the cumulative distribution function of $\Lambda_{mR_k}$ (formulated in Appendix.1),

\[
P(\Lambda_{max}^m_{R_k} > \lambda \sqrt{2}) = P(\Lambda_{max}^m_{R_k} \leq \lambda \sqrt{2} + 1) - P(\Lambda_{max}^m_{R_k} \leq \lambda \sqrt{2})
\]

\[= F_{\Lambda}(\lambda \sqrt{2} + 1)^n - F_{\Lambda}(\lambda \sqrt{2})^n = 0\]

The grid size defined with $\lambda = \min(\Lambda_{th}^S - S, \Lambda_{th}^M - S)$ satisfies (5.9). Hence, we use $\lambda$ for partitioning.
with splitting index as
\[
\{ x, y, z \} = \{ X_1+a\lambda, Y_1+b\lambda, D \},
\] (5.15)
where \(a=\{0, 1, ..., \lceil (X_2-X_1)/\lambda \rceil \}, \) \(b=\{0, 1, ..., \lceil (Y_2-Y_1)/\lambda \rceil \}, \) \(\lceil . \rceil \) is the ceil function, \(D\) is the tissue thickness comprising of skin, fat and muscle, forming the third dimension and \(\{ X_1, Y_1 \} \) & \(\{ X_2, Y_2 \} \) are the surface dimensions of the body area of interest. Prior clustering, the \(n\) nodes in given body area are included in the ‘not-clustered list’, NL. The three dimensional grid virtually partitions the given tissue into \(K=(|a|-1)(|b|-1)\) cuboids or clusters (refer Fig 5.2(b)). The set of \(C_k = \{ N_i, \forall i \in \{1, ..., |C_k| \} : |C_k| \leq n \}\) nodes enclosed by a cuboid \(k\) participates in the cluster \(k\forall k \in \{1, .., K\}\). The NL is then updated to remove the clustered nodes as \(NL = NL \setminus C_k, \forall k \in \{1, .., K\}\). The so formed grids give an initial approximation on the number of clusters \(K\) in the given area.

**Lemma 5.3.1:** If \(C_1=\frac{\Lambda_{b-S}^h}{\sqrt{2}}, C_2=X_2-X_1, C_3=Y_2-Y_1, p \in [\lceil \frac{C_2}{C_1} \rceil, C_2]\) and \(q \in [\lceil \frac{C_3}{C_1} \rceil, C_3]\), the CDF of \(K\) is given by \(\frac{\mu(C_1-1)-(C_2-p)}{\mu(C_1-1)}\) \(\frac{q(C_1-1)-(C_3-q)}{q(C_1-1)}\).

**Proof:** Using (5.9) and the observation that \(\Lambda_{b-S}^h < \Lambda_{M-S}^h\) for the same \(Pt\) [70], we see that \(\lambda\) is uniformly distributed in \((0, \Lambda_{b-S}^h/\sqrt{2})\). Assuming the minimum value of \(\lambda\) as 1, the CDF of \(\lambda\) is given by
\[
F_\lambda = \begin{cases} 
0, & \lambda < 1 \\
\frac{\lambda-1}{C_1-1}, & 1 \leq \lambda \leq C_1 \\
1, & \lambda > C_1 
\end{cases}
\] (5.16)

Let \(P = |a| - 1\). The CDF of \(P\) can be derived using (5.15) as
\[
F_P(p) = F_\lambda\left(\frac{C_2}{p}\right) = \begin{cases} 
0, & p < \lceil \frac{C_2}{C_1} \rceil \\
1 - \frac{\frac{C_2}{C_1} - 1}{C_1 - 1}, & \lceil \frac{C_2}{C_1} \rceil \leq p \leq C_2 \\
1, & p > C_2 
\end{cases}
\] (5.17)
using variable transformation technique. If \(Q = |b| - 1\), then \(P\) and \(Q\) share similar distribution and are also independent. The joint CDF of \(F_K(P, Q)\) can be obtained from (5.17) as a joint distribution of \(P\) and \(Q\) as \(F_K(p, q) =
\[
\begin{cases} 
0, & p < \lceil \frac{C_2}{C_1} \rceil, q < \lceil \frac{C_3}{C_1} \rceil \\
[1 - \frac{\frac{C_2}{C_1} - 1}{C_1 - 1}] [1 - \frac{\frac{C_3}{C_1} - 1}{C_1 - 1}], & \lceil \frac{C_2}{C_1} \rceil \leq p \leq C_2, \lceil \frac{C_3}{C_1} \rceil \leq q \leq C_3 \\
1, & p > C_2, q > C_3 
\end{cases}
\]
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At the end of this phase, \( NL = \emptyset \) as all the nodes are included in some cluster.

5.3.2 Phase II. Nearest Neighbor based Iterative Cluster Optimization (NICO)

In this phase, we semi-locally optimize the clusters from the approximations obtained in ICAP by iteratively adjusting \( K \) and \( C_k, \forall k \in \{1, ..., K\} \). The NICO phase comprises of the following five steps that are iterated in sequence. The description below follows the flowchart given in Fig. 5.10.

**Step 1. Relay position optimization (\( \hat{L}_{R_k} \)):** The NICO phase starts with identifying the optimal relay position \( \hat{L}_{R_k} \) for each \( C_k, k \in \{1, ..., K\} \) towards minimizing \( Pt_m, \forall m \in \{1, ..., |C_k|\} \) and balancing \( Pt_m, \forall m \in \{1, ..., I_k\} \) while achieving the link shortening and cluster shrinking objectives given in Fig. 5.2(b). For instance in Fig. 5.1, the total link length, and hence, the path loss experienced by the implant \( N_2 \) can be reduced by bringing \( R_2 \) closer to \( N_2 \). A dramatic decline in \( Pt \) obtained by reducing the link length by just a few centimeters is described in Sec. 5.4 (S1). The optimal relay position \( \hat{L}_{R_k} \) that reduces the link length of multiple (or even all) nodes in a cluster using the weights in (5.13) can be estimated as,

\[
\hat{L}_{R_k} = \arg\min_{L_{R_k}} \sum_{m=1}^{|C_k|} w_m \Lambda_{mR_k}, \quad (5.18)
\]

s.t (5.18.i) \( \Lambda_{mR_k} \leq \Lambda_{M-S}^{th}, m \in [1, ..., I_k] \)

(5.18.ii) \( \Lambda_{nR_k} \leq \Lambda_{S-S}^{th}, n \in [1, ..., |C_k| - I_k] \)

for \( I_k \) implants and \( |C_k| - I_k \) surface nodes in \( C_k \). Constraints (5.18.i) & (5.18.ii) limits the link length to the threshold link lengths estimated for M-S and S-S paths. Although the estimated \( \hat{L}_{R_k} \) significantly improves node life than the conventional relay positions, it tends to overfit by penalizing a few nodes making their \( \Lambda_{mR_k} \) longer in the interest of minimizing the sum of link lengths. The issue becomes critical when implants are penalized. (5.13) weighs the implants heavier to avoid this. However, when there are multiple implants in the cluster \( (I_k > 1) \), few of them might be penalized. Hence, the optimization problem in (5.18) needs modification, so that it extends the battery life of all the nodes, as well as balances the implant energy.

The deviation in energy consumption among the implants is due to the variation in \( w \Lambda_{mR_k}, m \in \{1, ..., I_k\} \). Aiming only for the balanced residual energy would lead to near-zero deviation in \( w \Lambda_{mR_k} \) with much longer links. Hence, for each cluster, we reformulate (5.18) by (i) using a weighted \( L_1 \) norm to prevent overfitting, and (ii) by adding a log barrier function to restrict
the non-negative constraints as follows:

\[
\min \sum_{i=1}^{A} (w_i \Lambda_i R_k + \gamma w \| L_i - L_k R_k \|_1^1 - \mu \log(p_1+p_2)) \\
\text{s.t.} \begin{align*}
(5.19) & \Lambda^h_m S - \Lambda_m R_k + p_1 = 0, \quad m \in [1, \ldots, I_k] \\
(5.19) & \Lambda^h_{S-k} - \Lambda_{n R_k} + p_2 = 0, \quad n \in [1, \ldots, |C_k| - I_k]
\end{align*}
\]

where \(\gamma = (u-1)v\) is the \(L_1\) penalty parameter and \(\mu \log(p_1+p_2)\) is the barrier function with \(p_1, p_2\) as the slack variables. The binary variable \(u\) becomes 1 for \(I_k=0\) and becomes 0 for \(I_k>0\), i.e., when \(I_k=0\), \(L_k R_k\) relies only on the surface node positions. The binary variable \(v\) tailors the problem for \(I_k>1\) and \(I_k \leq 1\) conditions. The variable \(A=u|C_k|+(1-u)I_k\) tunes the objective function towards energy efficiency for cluster without implants and towards energy balance in cluster with implants.

The optimization problem is convex with \(L_1, L_2\) norms and affine constraints. We solve it using the interior point method that finds the feasible \(\hat{L}_k R\) in the descent direction, estimated from the Newton step on the equivalent Karush-Kuhn-Tucker equations (obtained via linear approximations) \[72\]. The Interior point method converges fast with fewer iterations towards precise solution \[71, 72\] but requires a suitable starting point. We estimate the initial relay position at the cluster centroid on surface obtained as \(\{ \frac{1}{|C_k|} \sum_{m=1}^{|C_k|} L_m(x_m), \frac{1}{|C_k|} \sum_{m=1}^{|C_k|} L_m(y_m), 0 \} \) with \(\mu = 0.1\). The affine constraints and the penalty function ensure feasible relay positions inside the simplex. Note that the heterogeneity conditions in \(5.11\) & \(5.12\) are implicitly handled by \(w\) and hence, are not repeated in the optimization problem.

**Step 2. Cluster Reformation:** In this step we verify if the clusters obtained above satisfy the limits on \(P_t, \Lambda_{m R_k}, |C_k|\) and \(\hat{U}\) derived in \(5.7\)-\(5.11\) and \(5.14\). The non-conforming nodes are removed from \(C_k\) and added back to NL for reclustering as follows. In a cluster that does not satisfy \(5.7\), \(5.9\), \(5.11\) and \(5.14\), the implant with the longest link length is removed from the cluster and added back to NL as follows.

\[
NL=\{NL \cup N_m : m=Max_m (\Lambda_{m R_k}) \}; \quad C_k=\{C_k \setminus N_m\} \\
\forall m \in \{1, \ldots, I_k\}. \quad \text{If} \ (5.10) \text{is not satisfied, then the node with } max(\eta_m), \forall m \in \{1, \ldots, |C_k|\} \text{ is removed from cluster and added back to NL.}
\]

\[
NL=\{NL \cup N_m : m=Max_m (\eta_m) \}; \quad C_k=\{C_k \setminus N_m\} \\
\forall m \in \{1, \ldots, I_k\}
\]
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Step 3. Nearest Relay Assignment: Next, we assign the nodes in NL to the closest cluster. We use a combination of Delaunay Triangulation and Nearest Neighbor algorithms for the purpose. Using the triangulations, the Voronoi region $V_k$ of a relay $R_k$, $\forall k \in \{1, \ldots, K\}$ is determined as the locus of the skin and muscle regions that has $R_k$ as the nearest neighbor.

$$V_k = \{L_m|\Lambda_{mR_k} \leq \Lambda_{mR_j}\}, \forall k, j \in \{1, \ldots, K\}, k \neq j$$ (5.22)

Using $V_k$ (refer Fig 5.3(c)), and the node position as query parameters, our Nearest Neighbor algorithm finds the relay offering shortest $\Lambda_{mR_k}$ and satisfying (5.7), (5.9)-(5.11) & (5.14). This step has a low complexity of $O(K \log(n))$ as it avoids the distance computation of every possible node-relay combination. When a node is equidistant from multiple relays, (such as the one enclosed in circle in Fig 5.3(c), we choose the relay with less load to assign the node.

Note that in this step, the $\Lambda_{mR_k}$ does not rely on the definitive grid dimensions but only on $\Lambda_{M-S}^{th}$ & $\Lambda_{S-S}^{th}$. Hence $\Lambda_{mR_k}$ can exceed the cuboid dimension $\lambda$ specified in ICAP. This enables cluster shrinking or expanding just to cover the conforming node positions. Also, $\Lambda_{mR_k}$ distributions for surface nodes differ from that of implants based on $\Lambda_{S-S}^{th}$ and $\Lambda_{M-S}^{th}$, as illustrated later in Fig 5.4.

Step 4. Cluster Reassignment & Merging: If the new $\hat{L}_{R_k}$ offers shorter $\Lambda_{mR_k}$ to a node in the neighborhood cluster $C_j$ as given below,

$$\Lambda_{xR_k}<\Lambda_{xR_j} : N_x \in C_j, k \neq j, \forall k, j \in \{1, \ldots, K\},$$ (5.23)

then $N_x$ is reassigned to $C_k$, if its inclusion satisfies (5.10). The link lengths can be compared using the generated Voronoi regions and Nearest Neighbor algorithm in Step 3. As a consequence of this reassignment in a semi-distributed fashion, if $C_j=\emptyset$, then it is deleted with no relay assignment satisfying the null cluster objective in Fig 5.2(b).

Step 5. Dedicated Relays: Finally, the nodes in NL that cannot be included in the existing clusters are assigned a dedicated relay, forming clusters with $|C_k|=1$ and increasing $K$ by the size of NL. This forms the basis of the lower bound in the cluster size defined in (5.10) that can be merged among themselves or with other clusters in Step 2 of next iteration, if the required conditions are satisfied (see Fig 5.2(b)). Any change in the cluster participation from the previous iteration (marked by $flag$ in Fig 5.10) requires a new run of the $L_{R_k}$ optimization, followed by the iterative execution of all the steps in NICO phase until the termination criteria (given below) are met.
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Termination & Correctness: Fixing the number of clustering iterations can result in additional iterations than that actually required. To overcome this and to ensure algorithm correctness, we define our NICO termination criteria as follows: (a) $NL = \{\emptyset\}$, indicating that every node participates in a cluster, (b) $K \leq n$, which is ensured in Step.2, where relays with no node assignments are removed, and (c) $flag = false$, indicating no cluster change in the current iteration. The resulting relay position is bound inside the simplex formed by node positions with links shorter than the threshold lengths (see Fig.5.9).

The time complexity of ICAP is $O(Kn)$ while that of NICO is $O(Kn \log(n/\epsilon)) + O(nK) + O(K \log n) + O(Kn) + O(n)$ and $O(n \log Kn)$ for $NL$ and $flag$ update with an iteration complexity of $O(\sqrt{nK})$[71]. Thus the overall worse case complexity of the framework is $O(n^{3/2}K^{2}\log n)$. The resulting $\Lambda_{mR_k}$ and $K$ are substantially reduced from that of the ICAP phase as illustrated in Fig.5.4 & Fig.5.9. The algorithm is executed offline prior network installation to estimate the relay positions. The NICO phase is repeated periodically after the implants are inserted to accommodate channel variations.

5.4 Performance Evaluation

In this section, we: (i) analyze the impact of shortening $\Lambda_{mR_k}$ using empirical measurements on porcine tissue (chosen for similarity in properties with human tissues) and verify the simulation parameters with actual measurements in scenarios S1 and S2; (ii) evaluate the proposed
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Figure 5.4: $\Lambda_{mR_k}$ distribution in ICAP and NICO phases

GC-IBN clustering and relay positioning framework on optimizing $\Lambda_{mR_k}$, $K$, $C_k$ and $P_t$ using a galvanic coupled human forearm simulation model that computes the channel gain using the tissue equivalent electrical parameters in a circuit model from [70]. Using the simulation, we compare the optimal link length obtained in the NICO phase with the sub-optimal link length obtained in ICAP phase in scenario S3. We then analyze the power consumption in clusters that have $I_k=0$ (in S4), $I_k=1$ (in S5) and $I_k>1$ (in S6). Finally in S7, we analyze the clustering efficiency in terms of $K$. For the analyses, the value of $\alpha$ defined in (5.13) is assumed to be 4, unless specified otherwise.

A tissue sample with skin, muscle and fat of dimensions $42 \times 25 \times 6$ cm$^3$, from the porcine shoulder is cleaned and moistened for electrode attachment. The experimental network is composed of a relay ($R$), 2 surface nodes ($N_1$ & $N_2$ that are 10 cm apart) and an implant ($N_3$, below $N_1$) by fixing the electrode pair from each node to the tissue as shown in Fig 5.3(a). We use a multi-channel signal generator and oscilloscope, along with the OEP PT4 1:1 pulse transformers to isolate the transmitter and receiver.

S1. Impact of shortened $\Lambda_{mR_k}$ on $P_t$: Here, we highlight the dramatic reduction in the transmission powers $P_{t1}$, $P_{t2}$ & $P_{t3}$ for the nodes $N_1$, $N_2$ and $N_3$, respectively, for communicating with $R$ (refer Fig 5.3(b)(top)) when $R$ is brought closer. When $R$ is moved from $p_1$ (14 cm from $N_1$ & $N_2$) to $p_2$
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Figure 5.5: Power consumed in \( N_1, N_2 \) and \( N_3 \) for relay positions in Fig. 5.3(b) (5 cm), \( P_{t1} \) and \( P_{t2} \) drops from 6.5 mW to 0.8 mW over the S-S path (refer Fig. 5.5(left)). Owing to the lower loss in M-S path, bringing \( R \) closer to \( N_3 \) by the same distance substantially cuts down \( P_{t3} \) from 4.6 mW to 0.2 mW.

![Graph](image.png)

Figure 5.6: (a) \( \Lambda_{1R} \) vs \( \alpha \) for varying \( \eta \); (b) \( \Lambda_{1R} \) & mean \( \Lambda_{mR} \) for varying \( \eta \)

S2. Moving relay closer to implant: When \( R \) is moved from \( p_2 \) to \( p_3 \) (refer Fig. 5.3(b)(bottom)), \( P_{t1} \) and \( P_{t3} \) drops even lower to 0.2 mW and 20 \( \mu \)W respectively, while \( N_2 \) is penalized by increasing \( P_{t2} \) from 0.8 mW to 3 mW (refer Fig. 5.5(right)). With this optimized M-S \( \Lambda_{mR_k} \), the life \(^1\) of an implanted blood glucose sensor that usually lasts for 254 days with RF links (2 mW [73]) will extend

\(^1\)By Peukert’s law, node life= \( \frac{\text{Battery capacity}}{\text{duty cycle (10%)xLoad}} \times \text{External factors. The load current is derived from } P_t \) and power consumption from other node functions (\( \approx 0.1 \) mW).

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FIGURE 5.7: Comparison of (left) link length, (right) $Pt(mW)$ and node life (in years) with relay position at $L_{Rk}$, $L_{Rk}^F$ & $L_{Rk}^E$ for S3

up to 300 days. The simulation and empirical results in Fig 5.5 verify the accurate matching of the simulation model tailored for the dimensions and properties of the porcine tissue. We next proceed with the simulation model for deeper analysis that includes a 3D tissue area of $100 \times 100 \text{ cm}^2$ with the depth including skin, fat and muscle tissues embedding a maximum of 50 iid nodes.

**S3. Optimized Link lengths:** In order to analyze the optimized inter-node distance obtained from fitted simulated results for both S-S & M-S paths in the NICO phase, we compare it with the expected $\Lambda_{mRk}$ in ICAP phase (derived in Appendix) in Fig 5.4. The ICAP distribution suggests longer $\Lambda_{mRk}$ per cluster (mean 6.9 cm) for $\Lambda_{th}^S = 15 \text{ cm}$. However, the fitted optimized distribution indicates shorter links with mean $\Lambda_{mRk} = 5.9 \text{ cm}$ for S-S path with $\Lambda_{S-S}^{th} = 15 \text{ cm}$. The distribution of $\Lambda_{mRk}$ for M-S path varies in accordance to its threshold. We note that for $\Lambda_{M-S}^{th} = 20 \text{ cm}$ that is higher than $\Lambda_{S-S}^{th}$, the mean M-S $\Lambda_{mRk}$ is 4.1 cm that is significantly lower than the S-S path. Thus the algorithm efficiently minimizes $\Lambda_{mRk}$ for M-S path even at higher thresholds.

**S4. All surface nodes cluster:** We compare our optimized relay position ($\hat{L}_{Rk}$) with conventional relay positions - at the ICAP cluster center ($L_{Rk}^F$) and at the center of extreme cluster node locations ($L_{Rk}^E$) [77] for 6 node clusters with $I_k = 0$. Considering the $\sum \Lambda_{mRk}$ over 50 simulations (refer Fig 5.7(left)), $\bar{\Lambda}_{mRk}$ obtained with $\hat{L}_{Rk}$ is the lowest ($\bar{\Lambda}_{mRk} = 33.5 \text{ cm}$, $\bar{\Lambda}_{mRk}^F = 41.4 \text{ cm}$ & $\bar{\Lambda}_{mRk}^E = 40.64 \text{ cm}$). Thus, $\hat{L}_{Rk}$ gives $\approx 40\%$ more energy savings than at other positions.

The average $Pt$ values (calculated for SNR of 5 and 10 KHz bandwidth) of six S-S nodes are listed in Fig 5.7(right)). As expected, $\hat{L}_{Rk}$ offers extended life of up to 992 days that is significantly higher than the conventional positions and hence becomes a critical component in GC-IBN topology design.
S5. **Single implant cluster**: We next consider a cluster of 6 iid nodes of uniform data rates with \( N_1 \) implanted in muscle (\( I_k=1 \) at depth \( z_1=0 \)) and relay on surface at \( \hat{L}_{R_k} \). The resulting \( \Lambda_{1R_k} \) is significantly shorter than the other links as depicted in Table 5.2. This ensures minimum \( Pt_1 \), as desired for an implant \( N_1 \) with a dramatic improvement of 88% battery life compared with the co-clustered nodes.

**Influence of \( \alpha \):** Now we analyze the influence of \( \alpha \) used in (5.13) on \( \Lambda_{mR_k} \). When \( \alpha \) is reduced from 4 to 2, decreasing the priority given to conserving implant energy, the average \( \Lambda_{1R_k} \) increases from 0.4 cm to 3.4 cm (refer Table 5.3). When \( \alpha \) is raised to 10, the average \( \Lambda_{1R_k} \) drops close to 0. Thus, \( Pt \) of implant can be controlled by varying \( \alpha \). e.g., for the blood glucose sensor mentioned in S1, the extension of life can be up to 425 days when \( \alpha=10 \) that is \( \approx 67\% \) more than a RF link and \( \approx 40\% \) more than the relay position at \( L_{R_k}^F \) with GC link. The influence of \( \alpha \) is plotted with respect to \( \eta \) in Fig 5.6(a).

**Table 5.2:** Average \( \Lambda_{mR_k} \) (cm), \( Pt_m \) (mW) & node life (years) for S4

<table>
<thead>
<tr>
<th></th>
<th>N1</th>
<th>N2</th>
<th>N3</th>
<th>N4</th>
<th>N5</th>
<th>N6</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda_{mR_k} )</td>
<td>0.4</td>
<td>9.1</td>
<td>9.0</td>
<td>8.5</td>
<td>7.8</td>
<td>9.3</td>
</tr>
<tr>
<td>( Pt )</td>
<td>0.29</td>
<td>2.73</td>
<td>2.72</td>
<td>2.59</td>
<td>2.39</td>
<td>2.79</td>
</tr>
<tr>
<td>Life</td>
<td>75.1</td>
<td>8.5</td>
<td>8.53</td>
<td>8.96</td>
<td>9.7</td>
<td>8.31</td>
</tr>
</tbody>
</table>

**Influence of \( \eta \):** Considering the S5 scenario with \( \alpha=4 \), when \( \eta_1 \) increases from 1 to 3 (units), while
CHAPTER 5. ENERGY EFFICIENT & ENERGY BALANCED IN-BODY TOPOLOGY

Table 5.3: Influence of $\alpha$ over $\Lambda_{mR_k}$ for S4

<table>
<thead>
<tr>
<th>$\alpha$</th>
<th>N1</th>
<th>N2</th>
<th>N3</th>
<th>N4</th>
<th>N5</th>
<th>N6</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>3.4</td>
<td>6.6</td>
<td>9.6</td>
<td>6.1</td>
<td>6.1</td>
<td>9.3</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
<td>9.1</td>
<td>8.9</td>
<td>8.5</td>
<td>7.8</td>
<td>9.3</td>
</tr>
<tr>
<td>10</td>
<td>1E-3</td>
<td>13.3</td>
<td>15.6</td>
<td>14.4</td>
<td>7.9</td>
<td>10.1</td>
</tr>
</tbody>
</table>

$\eta_m=1, \forall m=\{2,..,6\}$, $\Lambda_{1R_k}$ is further reduced in accordance with (5.12). Even when $N_1$ is on surface, when $\eta_1$ is increased from 1 to 5, the average $\Lambda_{1R_k}$ reduces from 8 cm to 0.3 cm, penalizing the other links $\Lambda_{mR_k}, m=\{2,..,6\}$ by $\approx 1.5$ cm (Fig.5.6(b)). Thus $P t_m$ is reduced for higher $\eta$ towards energy balance. Similarly, for $\eta_1=5$, when the mean $\eta_m, m=\{2,..,6\}$, increases from 1 to 2, $\Lambda_{1R_k}$ is penalized from 1.6 cm to 4.8 cm (Fig.5.8(a)), indicating a steeper decline with the rise in mean $\eta$, achieving equitable energy distribution.

S6. Energy distribution in cluster with $I_k>1$: When there are multiple implants in a cluster, the energy consumption must be balanced. With $\hat{U}$ in (5.14) set to 90% and the relay at $\hat{L}_{R_k}$, when the first implant is depleted of energy, the residual energy of other nodes in cluster varies between 4 and 7% ($<10\%$ is desired). However, with the conventional relay positions at $L_{R_k}^F$ and $L_{R_k}^E$, the residual energy ranges between 14% & 18%, much higher than the desired level.

Figure 5.9: Optimized GC-IBN planar clusters
CHAPTER 5. ENERGY EFFICIENT & ENERGY BALANCED IN-BODY TOPOLOGY

Table 5.4: Average $K$ for 50 iid nodes with $T_m = [1, 2]$, $\eta_m \in [1, 5]$, $\alpha=4$

<table>
<thead>
<tr>
<th>$\Lambda^{th}, \Lambda^{th}<em>{S-S}, \Lambda^{th}</em>{M-S}$</th>
<th>8 cm</th>
<th>10 cm</th>
<th>12 cm</th>
<th>14 cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICAP</td>
<td>59</td>
<td>51</td>
<td>47</td>
<td>45</td>
</tr>
<tr>
<td>NICO</td>
<td>31</td>
<td>25</td>
<td>22</td>
<td>18</td>
</tr>
</tbody>
</table>

**S7. Clustering efficiency:** Table 5.4 illustrates the reduction in mean $K$ obtained from simulated NICO phase from that of ICAP (obtained as mean by fitting the $K$ distribution in Lemma 5.3.1), for various threshold link lengths. $K$ intuitively increases with $n$ and also with $\hat{U}$ (Fig. 5.8(b)). For instance, for $\hat{U}=50\%$ and $n=50$, $K$ is only 15 (30% of relays required). However, when $\hat{U}=90\%$ for the same $n$, $K$ becomes 45, requiring 90% of relays. Thus enforcing more uniformity in $P_t$ results in higher $K$.

The proposed framework satisfies the clustering objectives mentioned in Sec 5.2 as shown below. In cluster of area $A$ (i.e., area under the dotted ellipse is $A$, as shown in Fig. 5.9), the relay is moved from $L_{RA}^F$ to $\hat{L}_{RA}$ towards the implant, reducing the link length by half. Clusters $D$ and $E$ demonstrate the shortening of the M-S links. Cluster $B$ reduces $K$ by merging multiple clusters. Cluster $C$ shows the assignment of dedicated relays for isolated nodes. The three nodes in the area enclosed by $F$ cannot be clustered together as the sum of their data rates ($\{4, 4, 5\}$) exceeds the cluster capacity ($Q_0=10$ units). Hence, another relay is assigned to the node with higher $\eta$ (5 units), based on the constraint in (5.10).
CHAPTER 5. ENERGY EFFICIENT & ENERGY BALANCED IN-BODY TOPOLOGY

Using 'Ref' points divide area into K cuboids; Set k=1

Add |Ck| nodes in cuboid k to cluster k. k=k+1

Is k=K & NL={}?

Update Ck, NL; flag=true; remove k if |Ck|=0;

ICAP clustering phase

Step 1
Optimize relay position

Step 2
Any node not obey (7)-(11), (14)?

Step 3
Assign closest relay to each node in NL using NN & Delaunay Triangulation

Step 4
Merge clusters for shorter links

Step 5
Assign cluster & relay for each node in NL

Is flag=false & terminate needs met?

NICO Clustering Phase

Remove node from Ck & add to NL

Terminate

Figure 5.10: Two Phase Clustering Algorithm
Chapter 6

Beamforming in the Body

Assistive technologies allow humans to augment their natural abilities and restore physiological functions lost due to illness or injury. An example of today’s closed loop communication with man-machines interfaces involves controller-driven artificial limb stimulation based on muscle exertion levels. An array of embedded sensors in the tissue detect the muscle stress and communicate their readings back to the controller for precisely computing the needed stimuli for limb movement [1]. Though the concurrent transmission by an interconnected array of implants is a sub-optimal communication technique, a synchronized form of array transmission can be leveraged to overcome several challenges posed by the galvanic coupled tissue link.

• Problem: The GC-IBN architecture is composed of multiple embedded implants that transmit their sensed data to an on-skin node, called as a relay. The muscle to muscle (M-M) path offers lowest pathloss ($\approx 19$ dB) and hence, is ideally suited for communication across different implants in the same muscle layer [70]. However, implant to the surface relay communication needs to traverse several different tissue boundaries that have higher path loss, for e.g, the muscle to skin (M-S) path has $\approx 38$ dB of loss. How to send these signals to the relay with the least overhead (even if the baseline GC performance is much more energy efficient than RF) with high SNR remains an open challenge [54, 8]. Further, existing standards like IEEE 802.15.6 designed for implant communication use contention-based medium access with the possibilities of collisions, backoff and packet loss. Such events incur energy costs of re-transmissions and idle-listening, which we wish to avoid in IBNs.

• Proposed Approach: We propose a light-weight cross-layer framework that combines code division multiple access (CDMA) with distributed beamforming in narrow band channels, while ensuring that computational costs are delegated to the relay. The implants themselves simply record
and forward data, with the relay being responsible for both the CDMA decoding (to extract the actual sensed value) and tuning of the beam-steering matrix (for directional communication with high SNR). Existing far-field beamforming techniques cannot be applied for GC-IBN, as the receiver is placed in the near-field of the low frequency transmitter, separated only by a few tens of centimeters (refer Fig. 6.1).

The complete procedure is described as follows: The relay assigns unique CDMA codes to the implants. The latter store the sensed values and create modulated codewords using these assigned orthogonal codes. Using the high-gain M-M channel, the implants inform a designated aggregator, placed in the same muscle tissue, of their individual codewords. Such aggregator records the received CDMA-coded data structure created by the simultaneous transmissions of multiple sensors on the same channel. Note that there is no decoding step at this point to save energy, and the aggregator simply broadcasts back this cumulatively received codeword to the implants. By using distributed beamforming, each implant then transmits this codeword to the relay. Through this process, the energy consumed per implant is reduced, greater directional transmission is obtained and the relay receives much higher SNR than what would have been possible via a single sensor transmission. The final CDMA decoding is then performed at the relay, and the individual sensor data is then
extracted. The entire 2-step process of (i) exchanging individual codewords among peer implants, and (ii) beamforming to the relay, is collision-free.

- **Contributions:** The main contributions of this work are:

1. We propose a CDMA-based cross-layer approach that allows implants in the muscle to communicate with surface relays using galvanic coupling, which is collision-free and has reduced complexity of decoding.

2. We present the first formulation of near-field distributed beamforming in the body that accounts for specific tissue paths, constraints of tissue safety \( \leq 25 \text{mA/m}^2 \) \([8]\) and increases SNR at the surface relays. We present tissue-phantom and Arduino-based proof-of-concept experimental study of how constructive phase addition is possible within the body.

3. We use empirically obtain data sets to model the body channel and evaluate the effectiveness of our approach using an extensive finite element based simulation using MATLAB-generated mathematical models.

The rest of the chapter is organized as follows: Sec.6.1 describes the related contributions, and the initial motivation using experiments is presented in Sec.6.2. The beamforming technique is explained in Sec.6.3 Sec.6.4 describes the modified CDMA scheme. We provide rigorous performance evaluation studies in Sec.6.5

### 6.1 Background & Motivation

Classical medium access techniques such as CSMA/CA, channel hopping and reservation impact definite time of delivery, energy efficiency, and is unable to handle sudden spikes in traffic. The frame-length and inter-frame spacing are designed for high frequency signal propagation in the air medium over long distances (>2 m), rather than the low frequency short range communication (<50 cm) inside the body. Other overheads such as handshakes, channel sensing, scheduling, transitions from frequent sleep and wake-up states, among others, increase the processing complexity.

The low rate and sparse traffic generated by implants under normal physiological conditions may become bursty when an abnormal event is observed, limiting utility of both contention-based and reservation-based access techniques. Hence, for contention and reservation-free access, we advocate enabling concurrent transmissions using CDMA. However, CDMA multiplies the energy costs by using a high rate code, which in turn contributes to the net energy consumed per unit of useful data. Hence, in this paper we design smart energy-focusing strategies. However, the problem of beamforming for near-field and narrow band signals in a heterogeneous tissue-like medium has
not been demonstrated so far, particularly for the low frequency signals (<1 MHz) used in GC-IBN. Coordinated beamforming using multiple separate antenna elements may be possible in many applications where implants are placed in close proximity of each other, such as for neuro-muscular stimulation or orthopedic sensors that merits further investigation on this topic.

6.2 Tissue Phantom Experiments

As a motivation for choosing beamforming, we use a tissue phantom-based testbed (see Fig. 6.3 for the block diagram describing the setup and Fig. 6.2 for a snapshot) to analyze the constructive and destructive combination of concurrently propagating signals through tissue. We use pulse width modulated (PWM) signals at 100 kHz and 0.5 V generated by a pair of Arduino Uno boards, whose phase is controlled by a common synchronization pulse generated by MATLAB. The PWM signals are passed through a safety circuit (Fig. 6.3b) in order to limit the signal within the safe bound (=1 mA) we set based on the suggestion by ICNIRP [8] and then coupled to the muscle phantom (mimicking implants) by two pairs of electrodes. The transmitters are separated by 16 cm
and the electrode pair in each transmitter is separated by 4 cm. A pair of receiving electrodes is positioned on the surface skin of the phantom at 15 cm from each transmitter, and connected to an oscilloscope to observe the output voltage. For each signal, the corresponding Thevenin-equivalent circuit is built to measure the output power level.

When only one Arduino is transmitting a power of 0.25 mW, the maximum average output power ($P_{r_{\text{max}}}$) we observed is 3 µW. When two transmitters are transmitting concurrently, and in perfect phase alignment (Fig 6.3(d)), $P_{r_{\text{max}}}$ is 6 µW, which is double than the case of a single transmitter. This shows that the constructive signal addition is beneficial. However, when the input signals are out of phase (Fig 6.3(e)), $P_{r_{\text{max}}}$ $\approx$ 2.6 µW, which is lower than the case of a single active transmitter, showing the impact of destructive signal combination. When the signals are partially out of phase (Fig. 6.3(f)), $P_{r_{\text{max}}}$ becomes $\approx$ 4.3 µW. The set-up includes the mutual coupling effect from multiple transmitters and thus mimics the real scenario. Our experiments motivate the potential benefits of phase-alignment based beamforming within heterogeneous tissues using GC-coupled links.
CHAPTER 6. BEAMFORMING IN THE BODY

6.3 Beamforming for implant to surface communication

In this section, we first develop the formulations for near-field beamforming that will be used within the tissues. We start the discussion assuming each implant has a common CDMA modulated codeword $D'$, created and disseminated by the aggregator back to the implants. The steps through which this vector $D'$ is constructed from the individual sensed values is explained in Sec. 6.4. The idea here is to let the set of implants collectively act as a distributed antenna array and improve the directional emission of the near-field transmission towards the relay, while minimizing its propagation in other undesired directions. Our method requires manipulating the signal transmitted from each implant in amplitude and phase using complex weights. Hence, we first explain the requirement for near-field analysis in Sec.6.3.2 and derive the electromagnetic field pattern of each implant in Sec. 6.3.3. We extend the near-field analysis to the array-structure resulting from multiple nearby implants, and then calculate the cumulative received power at the surface relay in Sec. 6.3.4. Then, we derive the complex weights to limit the beamformed signal within the safe power limit, focus the signal strength at the receiver and devise a method to steer the input signals from each node in the desired direction in Sec.6.3.5.

6.3.1 Network architecture and 3-D tissue channels

We assume a set of $M$ uniformly distributed co-planar implants $\{m_1, \ldots, m_M\}$ arranged in muscle tissue linearly, at locations $(r_m, \theta_m, \phi_m)$, where $r_m \in [0, r_{\text{max}}]$ is the maximum distance of separation in muscle. Let $\theta_m \in [0, 2\pi]$ be the azimuth angle measured from the X-axis, and $\phi_m \in [0, \pi)$ be the elevation angle measured from the Z-axis, respectively, with the origin at $(0, 0, 0)$ as shown in Fig. 6.4. The angle-units are in radians. The number of implants in a given body part can vary from 1 to $M$, for e.g., neural stimulation uses more than 50 implanted cuffs in one limb [1, 83].

The external relay node $R$ on the body surface controls the actions of the implant-group by issuing synchronization pulses, aggregating their information, providing receiver feedback for beamforming and decoding the sensed values [84]. It is located at $(r_R, \theta_R, \phi_R) = (T, 0, 0)$, where $T$ is the tissue thickness separating $R$ and the $(r, \theta)$ plane at $\phi=\pi/2$, in which the implants are embedded. We assume identical path loss for all the implants and the tissue channel has negligible signal reflection, scattering, or shadowing [70].

• **Implant-Implant channel:** The channel between a given muscle implant ($m_i$) and another peer implant ($m_j$) that communicates along the M-M path is specified by the gain $g_{x ij}^{M-M}$, and phase shift $\psi_{x ij}^{M-M}$ for a field $x \in [\vec{E}, \vec{H}]$. Here, $\vec{E}$ is the electric field and $\vec{H}$ is the magnetic field. The
Table 6.1: Variable definitions and ranges

<table>
<thead>
<tr>
<th>Variables</th>
<th>Definitions</th>
</tr>
</thead>
<tbody>
<tr>
<td>$M$</td>
<td>Total number of nodes</td>
</tr>
<tr>
<td>$R$</td>
<td>Relay</td>
</tr>
<tr>
<td>$m_i$</td>
<td>Implant $i$</td>
</tr>
<tr>
<td>M-M</td>
<td>Muscle to muscle path</td>
</tr>
<tr>
<td>S-M</td>
<td>Skin to muscle path (Transmitter on skin and receiver in muscle</td>
</tr>
<tr>
<td>M-S</td>
<td>Muscle to skin path</td>
</tr>
<tr>
<td>$\vec{H}, \vec{E}$</td>
<td>Instantaneous magnetic and electric fields</td>
</tr>
<tr>
<td>$\theta_i, \phi_i$</td>
<td>Azimuth and elevation angles</td>
</tr>
<tr>
<td>$r_i$</td>
<td>Distance between 2 points in spherical coordinates</td>
</tr>
<tr>
<td>$AF$</td>
<td>Array factor</td>
</tr>
<tr>
<td>$g^p$</td>
<td>Gain in path $p$: $g_e - \vec{E}$ gain; $g_h - \vec{H}$ gain</td>
</tr>
<tr>
<td>$\psi, \gamma$</td>
<td>Phase shift and Frequency offset</td>
</tr>
<tr>
<td>$f &amp; w$</td>
<td>Frequency and angular frequency</td>
</tr>
<tr>
<td>$\Delta f$</td>
<td>Bandwidth</td>
</tr>
<tr>
<td>$c &amp; c'$</td>
<td>Speed of EM signals in vacuum and tissue</td>
</tr>
<tr>
<td>$w^s, w^p, w^t$</td>
<td>Weights for safety, phase match &amp; steering</td>
</tr>
<tr>
<td>$c_{ik}$</td>
<td>$k^{th}$ bit of Walsh code for $m_i$</td>
</tr>
<tr>
<td>$b_{i,n}$</td>
<td>$n^{th}$ bit of $m_i$</td>
</tr>
<tr>
<td>$\eta_m$</td>
<td>Required data rate for $m_i$, $\forall i \in {1,..,M}$</td>
</tr>
<tr>
<td>$P_i$</td>
<td>Transmit power consumed in $m_i$, $\forall i \in {1,..,M}$</td>
</tr>
<tr>
<td>$P_{R}^R$</td>
<td>Received power in $R$</td>
</tr>
<tr>
<td>$P^S$</td>
<td>Safe transmit power</td>
</tr>
<tr>
<td>$\delta^{M-S}$</td>
<td>SNR in M-S path</td>
</tr>
<tr>
<td>$\delta^{M-M}$</td>
<td>SNR in M-M path</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>Noise variance</td>
</tr>
<tr>
<td>$N_0$</td>
<td>Gaussian distributed noise P.S.D $\in (0, \sigma^2)$</td>
</tr>
</tbody>
</table>

Channel gain and phase are obtained as $g_{x_{ij}^{M-M}}^{M-M} = f_1^{M-M} (||r_{ij}||, \theta_{ij})$ & $\psi_{x_{ij}^{M-M}}^{M-M} = f_2^{M-M} (||r_{ij}||, \theta_{ij})$ where, $\theta_{ij}$ is the relative azimuth angle between $m_i$ and $m_j$, and $||r_{ij}||$ is the separation between implants $(m_i)$ and $(m_j)$ through the M-M path estimated as $||r_{ij}|| = \sqrt{r_i^2 + r_j^2 - 2r_i r_j \cos(\theta_i - \theta_j)}$.

The relative elevation angle $\phi_{ij} = 0$ as the implants are assumed to be co-planar. Note the above formulation can be trivially extended for non co-planar muscle implants, though we leave out this case for space limitations.

• **Implant-Relay channel:** The channel between the implant $(m_i)$ to relay $R$ communication through the M-S path is given in terms of gain ($g_{x_{ij}^{M-S}}^{M-S}$) and phase shift introduced by the tissue path through
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Figure 6.4: Spherical coordinate system with an implant $m_i$ and a relay $R$

muscle-fat-skin interfaces ($\psi x_{iR}^{M-S}$) for a field $x$, written as,

$$g x_{iR}^{M-S} = f_1^{M-S}(|r_{iR}|, \theta_{iR}, \phi_{iR}) \& $$

(6.1)

$$\psi x_{iR}^{M-S} = f_2^{M-S}(|r_{iR}|, \theta_{iR}, \phi_{iR})$$

(6.2)

where $\theta_{iR}$ and $\phi_{iR}$ are angles defined similarly between $m_i$ and $R$. $|r_{iR}|$ is the separation between implant ($m_i$) and relay through the M-S path estimated as $|r_{iR}| = \sqrt{r_i^2 + r_R^2 - 2r_i r_R A + B}$, where $A = \sin(\theta_i) \sin(\theta_R) \cos(\phi_i - \phi_R)$ and $B = \cos(\theta_i) \cos(\theta_R)$, and $P \in \{M-M, M-S, S-M\}$ is the path of the signal. The functions $f_1^{M-M}, f_2^{M-M}, f_1^{M-S}$ and $f_2^{M-S}$ are obtained using the channel models for $\vec{E}$ and $\vec{H}$ fields in [70].
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6.3.2 Near-field signal propagation

Signals impinging on a receive antenna are typically assumed to have planar wavefront. This assumption is not valid in GC-IBN for the following reasons: First, GC-IBN uses the operating frequency of 100 kHz to 1 MHz, with a wavelength ($\lambda$) of $2E3$ to $3E3$ m. Second, the size of the electrodes used in implants range from few $\mu$m to mm. The far-field range of such small electrodes is given by $r \geq \frac{\lambda}{2\pi}$, i.e., $r \geq 3.1E2$ for 100 kHz and $4.7E2$ m for 1 MHz. However, the possible separation between the transmitter and receiver in GC-IBN can be at most 30 cm based on measurements in [70]. Beyond this range, the received SNR is too low for messages to be reliably decoded. Thus, GC-IBN communication is confined to the near-field range.

Consider the electric field ($\vec{E}_i$) that is proportional to the normal component of the voltage ($V_{in}$) applied to the input electrodes that couple the GC signal to muscle. The magnetic field ($\vec{H}_i$) is proportional to the applied current ($I_{in}$) in the same implant $m_i$. The instantaneous $\vec{E}_i$ and $\vec{H}_i$ field strengths in the far field decrease inversely with distance (inverse-square law) and carry a relatively uniform wave-pattern, where the received signal is assumed to have constant frequency and infinite plane of constant phase and constant peak-to-peak amplitude normal to the phase velocity vector. These fields are also orthogonal to each other. As opposed to this, in the near-field, $\vec{E}_i$ and $\vec{H}_i$ field strengths falls exponentially with increasing distance from the source, contrary to the inverse-square law. Moreover, they can exist independent of each other with their field distributions depending on the tissue structure complexity without a strictly defined decreasing relationship. The electric and magnetic components expand spherically from the electrodes and hence the received signal in the near-field can be modeled as spherical wavefront.

6.3.3 Electric field pattern based on tissue orientation

The current coupled to the input electrodes introduces an isotropic radiation pattern in the surrounding tissue. However, higher conductivity along the longitudinal axis of the muscle tissue results from the continuous muscle strands that are oriented similarly. Coupled with the layered structure of such tissues in the transverse direction, the electrical field is $\approx \sqrt{2}$ times stronger in the longitudinal direction of the muscle tissue [54] (refer Fig. 6.1). To incorporate this tissue anisotrophy,
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we model the spherical wave-front of electric field ($\vec{E}_i$) and magnetic field ($\vec{H}_i$) as follows.

$$\vec{E}_i = V_{in} \times \begin{cases} \sin\left(\frac{\pi}{2} - \frac{\phi}{4}\right), & \forall \phi \in [0, \frac{\pi}{2}] \\ \sin\left(\frac{\phi}{2} - \frac{\pi}{4}\right), & \forall \phi \in [\frac{\pi}{2}, \pi] \end{cases}$$ (6.3)

$$\vec{H}_i = I_{in} \times \begin{cases} 1.7 - \sin\left(\frac{\theta}{2} + \frac{\pi}{4}\right), & \forall \theta \in [0, \pi] \\ 1.7 - \sin\left(\frac{\theta - \pi}{2} + \frac{\pi}{4}\right), & \forall \theta \in [\pi, 2\pi] \end{cases}$$ (6.4)

For the instantaneous $\vec{E}_i$ and $\vec{H}_i$ fields emanating from $m_i$, the instantaneous energy flux density caused in the surrounding tissue is expressed as Poynting vector: $\vec{P}_T = \vec{E}_i \times \vec{H}_i$, where, the real part denotes the power flow and imaginary part represents the reactive near-field of antenna. The field pattern for the implant $m_i$ during transmission is shown in Fig. 6.8(a).

6.3.4 Received signal at the relay without beamforming

The received near-field signal at $R$ due to transmissions by source $m_i$ can be determined by modeling the propagation behavior through tissue channel independently for $\vec{E}$ & $\vec{H}$ as

$$\vec{P}_R = \vec{E}_i \times \vec{H}_i$$ (6.5)

where $E_i = E_i \cdot e^{i(M-S)} \cdot e^{i(\omega(M-S) + \gamma_{M-S})}$, $H_i = H_i \cdot g_{M-S} \cdot e^{i(\omega(M-S) + \gamma_{M-S})}$, $\gamma_{M-S}$ is the effect of drift in frequency and phase offset and $\omega/2\pi$ is the operating frequency. We consider $M$ co-planar implants transmitting simultaneously whose positions are uniformly distributed around the reference point with distribution $r_{max}$. The $ge$ and $gh$ values for different tissue path are obtained from the HFSS based finite element simulation model in [70]. We define the term array factor as the net received signal pattern at the receiver resulting from multiple concurrent transmissions from the array of implants. For the $\vec{E}$ and $\vec{H}$ fields in the uniformly distributed planar implant array, the respective array factors can be written as,

$$E[AF_E] = \frac{1}{M} \sum_{i=0}^{M-1} E_i \cdot e^{i(M-S)} \cdot e^{i(\omega(M-S) + \gamma_{M-S})}$$ (6.6)

$$E[AF_H] = \frac{1}{M} \sum_{i=0}^{M-1} H_i \cdot g_{M-S} \cdot e^{i(\omega(M-S) + \gamma_{M-S})}$$ (6.7)
where $E(.)$ is the expected value, as the parameters are uniformly distributed values depending on the uniformly distributed position of the implants. Recall that the $\overrightarrow{E}$ and $\overrightarrow{H}$ fields are mutually independent. Hence, the array factor can be written as,

$$E[\overrightarrow{AF}] = E[\overrightarrow{AF}_E \times \overrightarrow{AF}_H] \quad (6.8)$$

The resulting received signal power at the relay, due to the array effect in (6.8), is oriented along the muscle fiber with less energy propagating towards the relay. This pattern is plotted in polar form (azimuth and elevation planes) in Fig. 6.5(a)-(b) and the power for various number of implants is plotted in Fig. 6.8(a)-(c) using spherical coordinates.

### 6.3.5 Increased received signal at relay with beamforming

While beamforming focuses the signal energy to the relay, we must ensure that the maximum received power at any point in tissue surrounding the transmitting implant array should be less than the maximum limit ($P_S$). Using the motivation from our experimental study in Sec 6.2, we aim to minimize the phase differences among the transmitting implants and lower per-node power requirements.

We propose a conventional delay and sum beamforming method using three weights as explained below.

- **Safety weight ($w_s$):** Assuming the minimum required SNR for successful communication in the M-M and M-S paths to be $\delta^{M-M}$ and $\delta^{M-S}$, the minimum required transmission power by an implant ($m_i$) becomes:

$$P_{i}^{\text{min}} = \begin{cases} \frac{\delta^{M-M}_{ij}N_o \triangle f_j}{g_{ij}^{M-M}} & \forall i,j \in \{1, \ldots, M\} \\ \frac{\delta^{M-S}R N_o \triangle f_R}{g_{iR}^{M-S}} & \forall i \in \{1, \ldots, M\} \end{cases} \quad (6.9)$$

where $N_o^j$ is the Gaussian noise P.S.D received at the receiver $j$ with zero mean and variance $\sigma^2 = 1 \times 10^{-8} W/\sqrt{Hz}$, and $\triangle f_j$ is the receiver bandwidth. When the received power in the receiver exceeds the minimum requirement, the transmitting implant $m_i$ can suitably reduce $P_i$ to just meet the expected SNR threshold. The optimal amount of transmitted power by an implant $m_i$ to a receiver,
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Figure 6.5: Directivity of received signal before (a,b) & after (c,d) beamforming
be it either an implant or a relay, can be chosen as:

\[ P_i = \frac{P_{ic}}{w_{ij}}, \forall \in \{1, \ldots, M\} + \{R\} \] (6.10)

where \( w_{ij} = \frac{\delta_{jc}^{M-x}}{\delta_{j}^{M-x}}, \) \( P_{ic} \) is the current transmit power, \( \delta_{jc}^{M-x} \) is the current SNR, \( \delta_{j}^{M-x} \) is the expected SNR and \( x \in [M, S] \).

Note that the maximum transmission power \( P_{i}^{max} \) is limited by the permitted level of signal propagation through tissues as \( P_i \leq P_S \). If there are multiple concurrent transmissions, then the cumulative signal at any point should also meet the safety criteria \( \sum_{i=0}^{M-1} P_i \leq P_S \). Thus, for safe and energy efficient choice of transmit power, the safety weight is chosen as:

\[ w_{ij}^{s} = \max \left( \frac{\delta_{jc}^{M-x}}{\delta_{j}^{M-x}}, \frac{\sum_{i=0}^{M-1} P_i}{P_S} \right), \] (6.11)

\( \forall i \in \{1, \ldots, M\} \) & \( \forall j \in \{1, \ldots, M\} + \{R\} \). Using \( w_{ij}^{s} \), the magnitude of \( P_i \) can be estimated using (6.10).

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• **Phase-match weight** ($w^p_{ij}$): As seen in Fig. 6.8(c), the mismatch in phase among the signals results in destructive signal combination, and thus, reduces the net received power. To perfectly synchronize the uniformly distributed planar implant array, we first match the link-dependent phase shift of each implant obtained in (6.2) with respect to the reference position at (O). Then, using the excellent cross-correlation property of the Walsh codes that we use later in Sec. 6.4, we extract the phase differences from the frequency offsets iteratively as $\gamma^h_{M-S}$ and compute the overall phase lag of each implant in the form of Phase match weight as:

$$w^p_{ij} = \psi h^M_{R} - S + \gamma^h_{M-S} \quad (6.12)$$

• **Steering weight** ($w^s$): This weight allows steering the signal from the transmitter to the relay with the desired beam shape given in Fig. 6.5(c)-(d). In the desired beam, along the elevation plane in Fig. 6.5(d), the beam power is increased at $\phi=0$ towards the position of the relay and in the azimuth plane in Fig. 6.5(c), the propagation is steered away from the neighbors at $\theta=0, \pi$. The corresponding steering weight is given as:

$$w^s_{iR} = \sin(k\theta_{iR})\cos(k\phi_{iR}) + \sin(k\theta_{iR})\sin(k\phi_{iR}) \quad (6.13)$$

where, $\theta_{iR}$ and $\phi_{iR}$ are the respective relative azimuth and elevation angles, respectively, between...
the implant and relay (refer Fig.6.4), \( k = \frac{2\pi f}{c} \) is the wave number, \( c' \) is the propagation speed of signal through the tissue medium estimated using the permittivity of the medium as,

\[
c' = \frac{c}{\sqrt{\epsilon}} \text{ m/s}
\]

(6.14)

where \( c \) is propagation speed of light in vacuum and \( \epsilon \) is the permittivity of the medium. \( c' \) for muscle is around 9.5e6 m/s and that of skin is around 8.3e6 m/s.

We adjust the array factor of the \( \vec{H} \) field in (6.7) using the three weights derived above as,

\[
E[AF_H] = \frac{1}{w_{iR}M} \sum_{i=0}^{M-1} \vec{H}_{iR} g h_{iR}^{M-S} e^{i\omega (\psi_{iR}^{M-S} + \gamma_{iR}^{M-S})} e^{w_{iR} - w_{iR}^{P}}
\]

(6.15)

The average power pattern of the uniformly distributed planar array can be estimated as

\[
E[|AF|] = |AF|(1 - \frac{1}{M}) + \frac{1}{M}
\]

We use our simulation environment to study the maximum power level in the tissue area using

\[
P_{\text{max}} = \max_{\{r,\theta,\phi\}} E[|AF|]
\]

(6.16)

### 6.4 Beam Synchronization using CDMA

The orthogonal CDMA codes distributed by the relay to implants play a critical role in facilitating beamforming; it helps in creating a common data vector in each implant as well as solves the problem of instantaneous transmissions by multiple implants in case of sudden abnormalities. The beam formation and the process of end-to-end data communication is split into four phases, as described below (see Fig.6.7):

- **Stage I. Resource assignment by the relay:** Each communication cycle starts a parameter setting beacon by the relay that allows implants to synchronize, set duty cycles for peer-level and beamforming-based communication, use orthogonal CDMA Walsh codes to partition the collision domain, and compute feedback weights for the array factor given in (6.11), (6.12) and (6.13) for optimal beamshaping (refer stage.1 in Fig.6.7). Communication from implants are acknowledged in a successive round, enabling the implants to sleep immediately after they transmit the beam. Since we require each implant to have a common data vector prior to beamforming, the relay also appoints an aggregator (ID_A) for the next round of communication. The aggregator’s role is simply to collect the individual and simultaneously transmitted spreaded sequences that combine in the tissue channel, and save this as the common data vector. The aggregator is a peer-implant, and its role is rotated in
Stage II. Peer communication phase: The relay provides synchronized slots \((T_B)\) for all implants to combine their data using the Walsh codes and transmit them simultaneously. This transmission is intentionally set to very low power given that it traverses the high-gain M-M path to the aggregator node (refer stage.2 in Fig.6.7). The spreading factor of the code is chosen based on the number of implants and the prevailing SINR. A perfectly synchronized transmission by implants offers high orthogonality of Walsh codes and hence high SINR. Even with a small misalignment in the transmission schedules of implants, Walsh code offers excellent orthogonal properties ensuring high SINR. Further increase in SINR is possible by increasing the code length.

The implant ID is associated with a unique spreading code sequence \(c_i\) among all the CDMA codebook. For \(N\) data bits of the implant, each bit is directly multiplied by the Walsh code with \(L\) elements (refer Fig. 6.6) to form the spread sequence \(b_{i,n}c_i, \forall \ i \in \{1,..,M\}, \ n \in \{1,..,N\}\) of size \(L \times 1\). These orthogonal Walsh codes have excellent cross correlation properties that enable simultaneous non-interfering transmissions. After spreading at the sampling time instant corresponding to the index \(k, \forall k \in \{0,..,L-1\}\), the implants transmit the spreaded sequence \(b_ic_i\) through the M-M path. At this stage, neither other implants nor the aggregator performs any decoding. Note that an implant can opt out of transmission in a cycle and sleep for prolonged period if its sensing cycle is longer. Also, the M-M communication is not strictly synchronized that relieves the implants from complex scheduling and mutual phase offset computation for this first round of messaging. An implant can choose to transmit anytime between the allowed window of peer-level M-M communication.

The aggregator receives the sequence \(\mathbf{D}\) as a vector of size \(L \times 1\) from the \(M-1\) implants as,

\[
\mathbf{D} = \sum_{i=1}^{M-1} g_{iA}^{(M-M)}b_{i,n}\mathbf{e}_i + \mathbf{w}
\]  

(6.17)

where \(A\) represents the aggregator, \(b_{i,n}\) is the \(n\)-th bit sent by the implant \(m_i\), \(\mathbf{e}_i = [c_i(0), c_i(1), ..., c_i(L-1)]^T\) is the spreading code for \(m_i\), \(T\) denotes the transpose and \(\mathbf{w}\) is the iid additive white Gaussian noise vector with zero mean and variance \(\sigma^2\) of size \(L \times 1\) given by \([w(k-L+1), w(k-L+2), ..., w(k)]^T\). Since each implant transmits in a narrow band channel (100 kHz) the M-M channel can be represented as a single tap channel [70]. We assume that \(g_{ij}^{(M-M)}\) is constant during a transmission cycle.

Once the aggregator receives the overall CDMA vector containing the spread data, it sends back the common CDMA vector \((\mathbf{D})\) representing the aggregated value to the peer implants through
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a single broadcast, again using the high-gain M-M path. The spread data received back at the \( i \)-th implant can be expressed as

\[
D_i' = g_{M-M}^{(M-M)}D + w
\]  

(6.18)

Now, all the implants have the common CDMA vector \( D' \), which may only slightly differ from each other depending on the channel coefficient of the M-M path from the aggregator to the specific implant according to (6.18).

- **Stage III. M-S Beamforming phase:** In this phase, each implant acts as an independent antenna array element and attempts to form a beam sending the same overall CDMA data vector that has been
shared with all the implants at the instant $T_B$ predetermined by relay. The use of the same CDMA data during beamforming further improves the SNR and lowers the required M-S transmission power at the implants as shown in Sec.6.3. Although each implant acts as an element of a virtual antenna array sending the same information, the individual implant signals differ in amplitude and phase, as instructed by the beamforming weights. The implants tune their transmission based on the weights, such that all the transmissions from implants constructively amplify the received signal $y$ at the relay and maximize the received power.

Thus, each implant sends the information at lower power compared to individual transmissions in the M-S channel, enabling significant energy savings. The received vector at the relay is given by

$$y = \sum_{i=0}^{M-1} S_i D'_i + w$$

(6.19)

where $S_i = \frac{1}{w_{iR}^{M}} g h_i^{M-S} e^{j \omega (\psi_{iR}^{M-S} + \gamma_{iR}^{M-S})} e^{w_{iR}^p - w_{iR}^p}$ obtained from (6.15) that accounts for both the steering coefficient of the implant $m_i$ and the channel coefficient of the M-S path from the implant $m_i$ to the relay $R$ on surface. The implants enter into the sleep state immediately after sending the beam until the next transmission cycle (see stage.3 in Fig. 6.7).

- **Stage IV. Despreading and feedback at the relay:** Having received the common CDMA vector through the beamformed signal, the relay despreads the signals using a matrix with the same Walsh
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codes distributed to the individual implants. In this way, it recovers the sensed data and the associated ID of each implant, as shown in Fig. 6.6. The despreading uses the cross-correlation of the received signal $y$ with the known Walsh codes as follows

$$\hat{b}_{i,n} = y^T c_i$$  \hspace{1cm} (6.20)

At the relay, the signal-to-interference-plus-noise power ratio (SINR) is given by [85]:

$$\text{SINR} = \left( \frac{N_0}{E_s} + \frac{M - 1}{G} \right)^{-1}$$  \hspace{1cm} (6.21)

where $E_s$ is the energy per symbol, $G$ is the spreading factor, and $M$ is the number of concurrent transmissions, corresponding to the number of implants. The bit error rate (BER) can be computed from the SINR as

$$\text{BER} = \frac{1}{2} \text{erfc} (\sqrt{\text{SINR}})$$  \hspace{1cm} (6.22)

In terms of transmission and propagation time, the whole transmission cycle takes

$$\frac{L}{\eta} + 2 \frac{\psi x_i^{M-M} + \gamma x_i^{M-M}}{360f} + 2 \frac{\psi x_i^{M-S} + \gamma x_i^{M-S}}{360f} + 4T_R$$  \hspace{1cm} (6.23)

seconds, where $L$ is the frame (or chip) length and $T_R$ is the tissue relaxation time required between transmissions to assure normal tissue temperature under abnormal blood flow rates, calculated as $T_R = \sqrt{\frac{\epsilon}{\sigma}}$, $\epsilon$ & $\sigma$ being the tissue permittivity and conductivity.

Finally the relay computes the weights $w_{ir}^s$, $w_{ir}^p$ and $w_{ir}^t$ using (6.11), (6.12) and (6.13) for the successive transmission and transmits them to the implants at the predetermined interval as given in stage.1 in Fig. 6.7.

6.5 Performance Evaluation & Results

In this section, we evaluate the energy savings achieved by the proposed CDMA based beamforming framework by (i) comparing the energy consumption for the overall M-S path communication with/without our approach, (ii) analyzing the proportion of energy propagating in the direction of the relay to that leaking in the undesired directions, (iii) studying the influence of the number of array elements on the implant power consumption, and (iv) quantitatively measuring the improvement in implant lifetime. We develop a 3-D multi-layer, heterogeneous tissue channel.
model in MATLAB, operating at a narrow band of 100 kHz. The tissue area has the dimension of 20 × 20 cm, with \( r_{\text{max}} = 20 \) cm. The separation between the layer of implants in muscle and the surface is 2.2 cm. The maximum safe transmit power is 1 mW.

### 6.5.1 Energy consumption With and without beamforming:

In this section, we discuss a critical issue of whether the multiple rounds of communication between the sensors needed for beamforming results in overall energy savings, compared to each node directly communicating with the surface relay.

A relay-implant data exchange cycle comprises of a relay to implant transmission followed by the sequence of implants to relay transmission assuming reservation based medium access scheme. The relay transmits a packet of information to the implants consisting the request for
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data from individual implants, the individual beamforming weights, the time to transmit and the
acknowledgement for the previous transmission. The reception of this information by implants
through the S-M path consume a power denoted by $Pr$ in each implant. This is followed by the
transmission from each relay along the M-S path. We next compare the energy consumption for the
implant to relay communication through the M-S path with and without the beamforming procedure.
• **M-S communication without beamforming:** For the communication along the M-S path without
using beamforming, the implants take turns in transmitting the information to the relay in accordance
with the schedule dictated by the relay. For an expected link length of 5 cm, the average M-S pathloss
is 41.2 dB. The power consumption in implant for transmitting along M-S path is for a desired SNR
of $\hat{\delta}$ is estimated as $Pt_{M-S}$ using (6.9). For a given $\hat{\delta}=10$ and a noise factor $(N_0)$ of $1e-8$, $Pt_{M-S}$ becomes $6.62 \mu W$. Without following the beamforming procedure, each implant transmits and
receives once during a transmission cycle and disburses $Pr + Pt_{M-S}$ of Power. Assuming the data
rate $\eta$ of $10 \text{ kbps}$, bandwidth $\Delta f$ being $1e5 \text{ Hz}$ and $Pr = 0.7 \mu W$, the power consumption in each
implant without beamforming would allow battery of capacity of $240 \text{ mAh}$ to last for $2.68$ years
using the following relation:

$Battery \ Life = \frac{Battery \ Capacity(\text{mAH})}{LoadCurrent(\text{mA})} * 0.70$

, where the factor 0.7 makes allowances for external factors which can affect battery life.
• **M-S Communication with beamforming:** On the contrary to the transmission cycle briefed above,
the beamforming procedure uses $2+1/N$ transmissions and $2+1/N$ receptions per cycle as explained
in Fig[6.7] The reception of data from relay in each implant consumes the same power ($Pr$) as the
procedure without beamforming. As the next step in the cycle, the implants first broadcast their
individual codewords in the M-M path. This step requires $Pt_{M-M} = 0.68 J$ of power, considering
the same values as assumed above for all other parameters, other than the lower path loss through
M-M path (19 dB). The aggregator spends $Pr_A$ to receive the information. The aggregated codeword
is then transmitted from the aggregator as a broadcast to all implants that requires $Pt_A$ at aggregator
to transmit and $Pr_{M-M} \approx 0.7 J$ in each implant for reception. The implants assume the role of
aggregator on a round-robin basis and hence the corresponding transmission and reception energy
consumed in implants during a transmission cycle can be written as $\frac{Pt_A}{M}$ and $\frac{Pr_A}{M}$ respectively.
Finally, the beamforming towards the relay is undertaken in the M-S path, where each node spends
about $4e^{-5}M^{-1.03}$ times less energy than that actually required for the direct M-S path, given by
$PI_{BF} = 4e^{-5}M^{-1.03} * Pt_{M-M}$.
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The total power consumption in each implants for the complete transmission cycle using beamforming approach is given by

\[ P_{\text{Implant}} = P_r + Pt_{M-M} + \frac{Pt_A}{M} + \frac{Pr_A}{M} + 2 \times Pr_{M-M} + PI_{BF} \]

For a scenario with 4 implants, the total power consumption per implant becomes \( P_{\text{Implant}} \approx 3.12\ \mu W \), which is 57% lower than that required for M-S communication without beamforming. In this case, the proposed framework extends the life of implant from 2.68 years to 6.12 years assuming every other parameter remains the same. By increasing the length of Walsh codes, the number of nodes that can maximize the SNR of M-S communication can be increased further from the current number 6. This approach would make the beamforming approach even more energy efficient inspite of a lengthier walsh code. A sample calculation of power consumption per bit for varying number of participating nodes in beamforming is shown in Table.6.2 illustrating the dramatic decrease in power.

6.5.2 Effectiveness of beamforming

The pattern of received power at the relay resulting from the sum of the signals concurrently propagating through the tissue medium without beamforming is shown in Fig. 6.8(a)-(c). Here, the propagation is oriented towards the longitudinal muscle direction (at \( \phi = \frac{\pi}{2}, \theta = 0, \pi \)), where more energy flow occurs along the length of the arm. This causes minimal flux at the surface relay (at transverse direction at \( \phi = 0 \)). This pattern may also cause more interference to the potentially neighboring implants (refer Fig. 6.1 for the direction of neighbors). Before beamforming, the ratio of energy flow in the required direction to undesired direction is \( \approx 0.53 \). Fig. 6.8(d)-(f) shows the received signal at relay after beamforming, where more power is steered towards the relay (at \( \phi = 0 \)) and there is less power in the longitudinal direction (at \( \phi = \frac{\pi}{2}, \theta = 0, \pi \)) mitigating the interference to neighbors.

Fig. 6.8(b)-(c) shows power degradation when signals with different phases are combined together. After the phase mismatch is rectified using \( W^p \) in the beamforming process, the signals add up constructively (refer Fig. 6.8(e)-(f)) as demonstrated in Fig. 6.3(d) and improve the received power by an additional \( \approx 3\% \) as shown in Table 6.2 as \( Pr(W^p) \). Note that this is the received power obtained after the transmit power is reduced to sufficient level using \( w^s \) weight. We analyze the maximum induced power at every point in the given tissue area defined by \( \theta, \phi \) and \( r \) using (6.16) and verify that the cumulative received power at any point in the tissue with multiple concurrent transmissions does not exceed the restrictions posed by safety limits, confirming the tissue safety.
and normal thermal distributions. Using the simulation environment, we further ensure that
\[ \int_0^\theta \int_0^\phi \int_0^r P_r dr d\phi d\theta \leq 25 \frac{mA}{m^2}. \]

**Influence of number of implants:** The resulting proportion of power in the required to undesired
direction is plotted in Fig. 6.10(a) illustrating that more power is steered towards the relay when
there are more number of implants forming the array. Thus both the critical beamforming parameters
namely, the per implant power conservation and the directivity of beamforming, are improved with
the number of implants or array elements \((M)\). The actual SNR for individual transmission from
each implant through M-S path is compared with the exponential increase in SNR at the receiver
after beamforming with \(M\) implants in Fig. 6.10(b).

**Implant lifetime:** The transmission power of the implants is reduced to just meet the required SNR
by applying the safe weight \(w^s\) derived in (6.11). The resulting power consumed \((aPt)\) in each
implant vs \(M\) and the corresponding improvement in implant lifetime is shown in Table 6.2. We see
that the implant life dramatically extends from 10 weeks when used without beamforming, to \(\approx 138\)
weeks with beamforming for the scenario with 14 implants.

![Figure 6.10: (a) Directionality (b) SNR before & after beamforming](image)

**6.5.3 BER & energy analysis for CDMA-beamforming**

The proposed CDMA scheme allows concurrent simple transmissions for the implants.
There is, however, an additional overhead of (i) spreading the data using the Walsh codes, and (ii)
(albeit high gain M-M) communication between implants to the aggregator and back. We aim to
study whether this cost is offset by the energy savings achieved by beamforming.
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The CDMA performance without the beamforming is shown in terms of BER computed using (6.21)-(6.22) in Fig. 6.9(a). For $N_0=1e^{-8}$ and the receiver bandwidth of $1e3\ Hz$, the maximum SNR obtained is 0.92 for a single transmitter. This SNR gets worse with multiple CDMA transmissions resulting in poor BER. However, the CDMA solution performs better with dramatically improved BER when using beamforming contributed by the improved $SNR$ at the relay, i.e., $E_s/N_0$ in (6.21) reduces the $BER$ according to (6.21)-(6.22). This is illustrated in Fig. 6.9(b). For the given scenario, the BER improves from 0.18 for one implant upto $1.5e^{-4}$ for six implants. For $M>6$, the BER starts to decline with $M$, showing that the advantage of beamforming is more effective for a low number of implants in array. For a high number of concurrent implant transmissions, the interference effect $M-1$ in (6.21) becomes dominant, pulling down the performance.

**Network traffic and time:** With the proposed framework with $L=64$, the total traffic flow in a transmission cycle becomes 32 bytes, which is much lower than the existing IEEE 802.15.6 standard that requires a minimum of $13 \times 4$ bytes as frame header alone not including the data. The time required for the whole transmission cycle is estimated using (6.23) for $T_R=10\ \mu s$ and $\eta=100\\ kbps$ to be only $1.3\ ms$ that is mainly dominated by the transmission time.

The above discussion on performance evaluation proves that beamforming with CDMA is the suitable medium access procedure for transferring implant data to on-skin relay devices that offers energy efficiency, directivity and interference mitigation. This procedure of beamforming is also applicable for altering the signal propagation direction through tissues either towards or away from a target.
Chapter 7

Conclusion & future work

IBNs will lead to diverse health care applications that would benefit at health risk populations and patients at remote locations when the presence of a human caregiver or trained medical professional is not always possible. The ability to sense physiological changes within the body and take proactive monitoring steps will increase human longevity at reduced health care costs. We compare several technologies capable of building a communication network in body in chapter 2 and bring out that galvanic coupling based intra-body network (GC-IBN) would provide safe and reliable through tissue links.

As a first step towards building GC-IBN, we derived, verified and validated the equivalent electrical circuit model for human tissues in characterizing the physical layer in chapter 3. We conducted extensive studies regarding the gain and phase-change in the transmitted signal under varying operating frequencies, tissue dimensions, sensor placements, electrode separation distances and dimensions, among others, to comprehensively characterize the body channel, while respecting permissible safe current limits. We found that a maximum of $30 \text{ dB}$ in channel gain could result from variation in tissue properties from person to person. We identified the optimal frequency to lie between $100 \text{ kHz}$ to $1 \text{ MHz}$ for both on skin and in muscle paths, and determined that placing both the sender and receiver sensors within the muscle offered better channel propagation characteristics, as opposed to on the skin. In future, we would work on channel models for other parts of body and incorporate the temporal channel dynamics for realistic analyses.

In chapter 4, the galvanic coupled tissue safety is analyzed in terms of the induced $\vec{E}$ and $\vec{H}$ fields and suitable safe boundary conditions are derived. Further, with the objective of operating the GC communication technology within various safety thresholds, the tissue thermal analysis is presented demonstrating that GC-IBN provides safe operating conditions with the temperature
CHAPTER 7. CONCLUSION & FUTURE WORK

Elevation in normal tissue conditions restricted within the stipulated bound of $1 ^\circ C$. By appropriately connecting the tissue heating profiles with transmission sequence and duty cycles of concurrent transmissions, we have demonstrated the GC-IBN can operate with high reliability, better bandwidth efficiency, and is adaptable to the abnormal tissue conditions. A maximum of $1.6\%$ and $15.38\%$ improved in bandwidth efficiency is obtained for the poor blood perfusion and very high blood perfusion conditions respectively, while that of normal perfusion reaches up to $64.3\%$ for each device in GC-IBN.

Chapter 5 proposes an energy efficient and energy balanced clustering framework suitable for galvanic coupled intra-tissue communication. The proposed framework comprises of two-phases of clustering that adapts to the signal propagation paths within heterogeneous tissues, and accommodates varying data rate requirements of implants. We demonstrate that the clustering approach not only minimizes the quantity and size of clusters, but also optimally positions the external signal pick up relays. Results indicate that our approach extends the lifetime of implants up to $70\%$ longer than the existing RF-based techniques.

As our next effort, we propose an energy efficient implant to surface relay communication using galvanic coupling that uses beamforming in Chapter 6. The proposed communication technique is strongly focused on improving energy efficiency by: sharing sensed updates among peer-implants using CDMA codewords through the high-gain M-M path, avoiding unpredictable data delivery conditions caused by collisions and transmission back-offs. Then, through near-field beamforming performed by the implants organized into distributed transmitter arrays, communication through the vertical tissue layers is achieved with high SNR (or conversely, lower energy per implant). The proposed framework dramatically lowers the net energy required for end-to-end implant to relay communication that is $79\%$ more energy efficient than the direct case, and extends the lifetime of implants up to 13 years.

In summary, several unique challenges pertaining to signal propagation through human tissue is been addressed in this thesis with experimentally proven steps towards practical intra-body networking. The tissue channel models, algorithms, protocols and safety guidelines devised can be integrated to build continuous, safe and energy efficient intra-body wireless communication that connects multiple implants and wearables to external world, paving way for the emergence of revolutionary medical applications.
CHAPTER 7. CONCLUSION & FUTURE WORK

7.1 Future Work

GC-IBN is an emerging as well as fast developing technology. Albeit the simplicity of the galvanic coupling technology as such, the complex human tissue channel introduces several challenges that are need to be addressed to elevate GC-IBN to the matured status. In this section, we present a few of such potential future research directions in GC-IBN.

7.1.1 Transceivers and protocol stack for GC-IBN

The thesis presents the algorithms and protocols for topology definition, medium access and energy efficient communication in GC-IBN as well as the illustration of the technology using porcine tissue based and phantom based test-beds. However, further investigation is required in GC-IBN related to the following areas: (1) possible miniaturization of the transceiver hardware (2) comprehensive energy and body aware cross-layer protocols (3) data security assurance and quality of service for differential health care applications and (4) energy efficient as well as reliability guaranteeing policies on error handling among others.

7.1.2 GC-IBN to EBN Interface

We adopt GC links for connecting implants to relays, forming the IBN. The data collected by the relays is to be transferred to the network external to the body (EBN) for further processing. In contrast to the implanted devices, the relays are positioned on the body surface and hence the wireless communication link can be established over the air medium (rather than the tissue medium) using the RF links. Many communication standards exist to establish RF based EBN for connecting wearable devices such as BLE, IEEE 802.15.6 UWB and Zigbee as discussed in chapter 2. For the successful IBN to EBN data transfer, suitable interfaces are required to convert from GC-IBN specific format to that compatible with RF based wireless standards. The availability of multiple standards as choices for the EBN makes the design of an efficient GC to RF interface a challenging task. Further investigation is required to formulate the best strategy that tailors the IBN to EBN interface. The proposed solution should meet the body related communication constraints and be suitable to be programmed into the wearable device.
CHAPTER 7. CONCLUSION & FUTURE WORK

7.1.3 Body aware medium access and routing

Energy consumption for communication through tissues can be optimized through accurate estimation of transmission parameters for intra-body communication. However, temporal changes in the structural, chemical and biological properties of live tissue challenges the accuracy of channel estimates and transmission parameters. Direct real-time tracking of tissue properties is not feasible given that the implants are predominantly embedded in the hard-to-reach muscle tissues. Dynamic channel tracking algorithms are required to enable reliable communication and optimal resource allocation. Further, the choice of protocols across physical, link and network layers and the state machines of network operation are to be adapted based on the prevailing tissue conditions. This requirement can be satisfied using machine learning algorithms, making the intra-body network body aware.

Albeit the several interesting research problems, GC-IBN would soon find its place among the top technologies promising revolutionary medical applications.
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[63] D. O. Carpenter and Aİ, *Biological effects of electric and magnetic fields: sources and mechanisms*.


BIBLIOGRAPHY


Appendix A

Our Publications


M. Swaminathan, J. S. Pujol, G. Schirner and K. R Chowdhury, “Multi-path 2-Port Channel Charac-
APPENDIX A. OUR PUBLICATIONS


Appendix B

Dielectric Properties of Biological Tissue

Assuming classical macroscopic media, electromagnetic wave equation using Ampere’s
circuital law is,

\[ \nabla \times \mathbf{H} = J + jw \mathbf{D} \quad (B.1) \]

\[ = \sigma \mathbf{E} + jw \mathbf{D} \quad (B.2) \]

\[ \mathbf{D} = \varepsilon \mathbf{E} \quad (B.3) \]

The relative permittivity \( \varepsilon \) in complex form is written as

\[ \varepsilon_0 \varepsilon_r = \varepsilon' - j \varepsilon'' \quad (B.4) \]

where, \( \varepsilon' \) is dielectric constant and \( \varepsilon'' \) is the out of phase loss factor \[51\] such that

\[ \varepsilon'' = \sigma / \varepsilon_0 \omega \quad (B.5) \]

Neglecting impressed current density caused by outside source, and using Ohm’s law,
equation \[B.1\] can be written using equations \[B.3\] and \[B.4\] as

\[ \nabla \times \mathbf{H} = jw \varepsilon' (1 - j \frac{\sigma}{\omega \varepsilon'} - j \frac{\varepsilon''}{\varepsilon'}) \mathbf{E} \quad (B.6) \]

In the above equation, the first term with \( \sigma \) represents conduction current due to the
movement of charges defining the property of a conductor, while the second term with \( \varepsilon'' \) represents
displacement current from time-varying electric field defining the property of dielectric. Admittance
APPENDIX B. DIELECTRIC PROPERTIES OF BIOLOGICAL TISSUE

Y non-ideal of dielectric block is given by

\[ Y = G + j\omega C = (A/d)\left(\sigma' + j\omega\epsilon'\right) \]  

(B.7)

Using [B.4] and [B.5] the above equation can be written as

\[ Y = (A/d)(\omega\epsilon'' + j\omega\epsilon') \]  

(B.8)

Debye defines frequency dependent dielectric properties that characterize dielectric relaxation as

\[ \epsilon = \epsilon_\infty + \frac{\epsilon_s - \epsilon_\infty}{1 + j\omega\tau} \]  

(B.9)

while Cole and Cole describes the same with more flattened frequency dependence as

\[ \epsilon = \epsilon_\infty + \frac{\epsilon_s - \epsilon_\infty}{1 + (j\omega\tau)^{1-\alpha}} \]  

(B.10)

where \( \epsilon_\infty \) and \( \epsilon_s \) are dielectric constants at very high and very low frequencies, \( \alpha \) relaxation time distribution parameter mostly varying from 0.3 to 0.5 for biological tissues [51]. Using equation [B.9], tissue dielectric constant and polarization loss factor can be expressed as,

\[ \epsilon' = \epsilon_\infty + \frac{\epsilon_s - \epsilon_\infty}{1 + \omega^2\tau^2} \]  

(B.11)

\[ \epsilon'' = \frac{(\epsilon_s - \epsilon_\infty)\omega\tau}{1 + \omega^2\tau^2} \]  

(B.12)
Appendix C

Proof of Theorem 5.3.1

We assume that the coordinate distribution of nodes at a tissue $T$ follows two dimensional rectangular distribution over $[a\lambda, \lambda + a\lambda]$ horizontally and $[b\lambda, \lambda + b\lambda]$ vertically. Thus, the probability density functions of $X_m, X_{R_k}, Y_m, Y_{R_k}$ are given by

$$f_{X_m}(x_m) = f_{X_{R_k}}(x_{R_k}) = f_{Y_m}(y_m) = f_{Y_{R_k}}(y_{R_k}) =$$

$$\begin{cases} \frac{1}{\lambda} & x_m, x_{R_k} \in [a\lambda, \lambda + a\lambda], y_m, y_{R_k} \in [b\lambda, \lambda + b\lambda] \\ 0 & \text{otherwise} \end{cases} \quad (C.1)$$

while the cumulative distribution function is given by,

$$F(x_m) = F(x_{R_k}) = F(y_m) = F(y_{R_k}) =$$

$$\begin{cases} 0 & x_m, x_{R_k}, y_m, y_{R_k} < 0 \\ \frac{1}{\lambda} & \{x_m, x_{R_k}, y_m, y_{R_k}\} \\ 1 & x_m, x_{R_k} > \lambda + a\lambda, \quad y_m, y_{R_k} > \lambda + b\lambda \end{cases} \quad (C.2)$$

The expected value of $X$ in (5.1), $E[X]$, is given by

$$\int_{a\lambda}^{\lambda + a\lambda} \int_{a\lambda}^{\lambda + a\lambda} |x_m - x_{R_k}| f_X(x_m, x_{R_k}) dx_m dx_{R_k} \quad (C.3)$$

where $f_{X_m, X_{R_k}}(x_m, x_{R_k})$ is the joint probability density function of $X$, that can be written with
APPENDIX C. PROOF OF THEOREM 5.3.1

$L_m$ and $L_{Rk}$ having independent coordinates as $f_X(x) = f_{Xm}(x_m)f_{XRk}(x_{Rk})$. The corresponding CDF of $X$ can be obtained by integrating $f_X(x)$ \[86|Y\] also has the similar distribution as that of $X$. Now the CDF of $Z = \sqrt{\Lambda}$ is obtained as $F_Z(z) = \int \int f_X(x)f_Y(y)dx dy = \begin{cases} 0, & x<0 \\ \pi \frac{x}{\lambda^2} - \frac{8}{3}(\frac{x}{\lambda^2})^2 + \frac{1}{2} \frac{x^2}{\lambda^2}, & 0 \leq x<\lambda^2 \\ 1 - \left[ \frac{2}{3} + \frac{2}{\lambda^2} + \frac{1}{2} \frac{x^2}{\lambda^2} - \frac{2}{3} \sqrt{\frac{x^2}{\lambda^2} - 1}\right]^3 \\ -2\sqrt{\frac{x}{\lambda^2} - 1}(1 - \frac{x}{\lambda^2} - 2\frac{x}{\lambda^2}sin^{-1} - \frac{2 - \frac{x}{\lambda^2}}{\lambda^2}), & \lambda^2 \leq x<2\lambda^2 \\ 1, & X \geq 2\lambda^2 \end{cases}$ (C.4)

Assuming $\lambda' = \frac{\lambda}{\pi}$, the CDF of $\Lambda m_{Rk}$ in cluster $C_k$ becomes

$$F_{\Lambda}(r) = Pr(0<z<r^2) = \int_0^{r^2} f_Z(z)dz$$ \[C.5\]

$$= \begin{cases} 0, & r<0 \\ \lambda^2\pi - \frac{8}{3}\lambda^3 + \frac{1}{2}\lambda^4, & 0 \leq r<\lambda \\ 1 - \left[ \frac{2}{3} + \frac{3}{\lambda^2} - \frac{2}{3} \sqrt{\left(\frac{r^2}{\lambda^2} - 1\right)}\right]^3 \\ -(2\sqrt{r^2 - 1})(1 + \frac{r^2}{\lambda^2}) - 2\lambda^2^2 asin^{-1} - \frac{2 - \frac{r^2}{\lambda^2}}{\lambda^2}, & \lambda \leq r<\lambda\sqrt{2} \\ 1, & r \geq \lambda\sqrt{2} \end{cases}$$

$F_{\Lambda_{max}} = [F_{\Lambda_{max}}(s)]^N$ Assuming the left bottom corner in XY plane of cubical grid to be at origin and using (C.4) in (C.3),

$$E[X] = \frac{1}{s^2} \int_0^\lambda \int_0^\lambda |x_{m} - x_{Rk}| dx_m dx_{Rk}$$ \[C.6\]

and similarly,

$$E[Y] = \frac{1}{s^2} \int_0^\lambda \int_0^\lambda |y_{m} - y_{Rk}| dy_m dy_{Rk}$$ \[C.7\]

Assuming $u = \sqrt{\Lambda}$, the pdf of the event $(u \leq \tau)$ can be obtained as the convolution of $f_X$
APPENDIX C. PROOF OF THEOREM 5.3.1

in (C.4) and \(f_Y\) that is distributed similar to \(f_X\) using (2.4.9) in [87] as

\[
f_u(\tau) = \begin{cases} 
0 \leq \tau \leq \lambda^2 & \int_0^\tau f_X(\tau-x)f_Y(x)dx, \\
\lambda^2 \leq \tau \leq 2\lambda^2 & \int_{\tau-\lambda^2}^{\lambda^2} f_X(\tau-x)f_Y(x)dx,
\end{cases}
\]

(C.8)

\[
= \begin{cases} 
\frac{\pi}{\lambda^2} - \frac{4\tau}{\lambda^4} + \frac{\tau}{\lambda^4}, & 0 \leq \tau \leq \lambda^2 \\
\frac{4}{\lambda^2} \left[ \sin^{-1} \left( \frac{\lambda}{\sqrt{\tau}} \right) \right] - \frac{1}{2} + \frac{4}{\lambda^2} \sqrt{\tau - \lambda^2} \frac{\tau}{\lambda^4}, & \lambda^2 \leq \tau \leq 2\lambda^2
\end{cases}
\]

Now, the density of link length becomes,

\[
f_\Lambda(\Lambda) = f(u^2) \frac{du}{d\Lambda} = 2\Lambda f_u(u^2)
\]

(C.9)

The expected value of \(\Lambda_{mR_k}\) in each cluster \(E[\Lambda_{mR_k}]\) is

\[
= \int_0^{\sqrt{2\lambda^2}} \Lambda_{mR_k} f_\Lambda(\Lambda_{mR_k}) d\Lambda_{mR_k} = \frac{\lambda}{3} \ln(1 + \sqrt{2}) + \frac{\lambda\sqrt{2}}{15} (1 + \sqrt{2})
\]

(C.10)

\[
= \frac{\lambda}{3} \ln(1 + \sqrt{2}) + \frac{\lambda\sqrt{2}}{15} (1 + \sqrt{2})
\]

\(F(x) = P(x_m - x_{R_k} \leq x) = \) with distribution (follows a triangular distribution)

\[
E[\bar{\Lambda}] = \int_0^\infty P(\bar{\Lambda} > \Lambda_{mR_k}) d\Lambda_{mR_k}
\]

(C.11)