OSNABRÜCK UNIVERSITY

MASTER THESIS

Optical Flow, Helmholtz Decomposition and Autoencoders for the characterization of slow-waves

A new approach for temporospatial pattern detection in neuroimaging

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Declaration of Authorship

I, Michael GERSTENBERGER, declare that this thesis titled, "Optical Flow, Helmholtz Decomposition and Autoencoders for the characterization of slow-waves" and the work presented in it are my own. I confirm that:

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- Where I have consulted the published work of others, this is always clearly attributed.
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Abstract

Optical Flow, Helmholtz Decomposition and Autoencoders for the characterization of slow-waves

Neural signal transduction at different levels of alertness occurs in distinct shapes (Gemignani et al., 2015, p. 137f.). One may observe stimulus dependent activity during wakefulness but also spontaneous patterns of activation that dominate in stages of deep sleep (Dang-Vu et. al. 2011). Similarly, fast neuronal firing is replaced by slow, traveling waves of activation during anaesthesia. Slow-waves can be captured by high-speed fluorescence microscopy of GCamp channel activity in transgenic mice at up to 100Hz (Celotto et al. 2018).
Different types of slow-waves exist which can incorporate rather localized or more widespread regions of cortex. In principle, they are thought to occur spontaneously in various regions, however it was also noted that synchrony with somatosensory states and more importantly signals in subcortical structures exists during sleep. This synchrony is related to memory formation. Recently important differences between sleep slow-waves and their counterpart during anaesthesia were identified. This shows that slow-waves represent an important yet highly variable neural phenomenom and indicates that methods are required that allow to systematically dinstinguish slow-waves based on their properites.
Townsend and Gong (2018) suggest to characterize temporospatial properties of neural recordings by the detection of patterns in Dense Optical Flow. It shows, however, that the use is accompanied by two challenges: First it shows that focal brightness changes result in sources and sinks in the vector fields. Second successful estimation of Optical Flow is challenged by the quality of the contrast it is applied on. Here it is demonstrated how both challenges can be addressed and Dense Optical Flow used to characterize slow-waves. The presented procedure allows for a scale independent split of optogenetic recordings into several segments that relate to slow-waves. This allows for an event related analysis and the computation of a contrast that is independent from anatomical structures and meets the smoothness requirements of Dense Optical Flow. It is further demonstrated that Helmholtz-Decomposition can be used to distinguish between the effect of local sources of neural activation and global patterns of neural flow. Finally, it is shown how the measured high-level features can be analysed using variational autoencoders. The approach reveals a complex topology of the distribution of different features in latent slow-wave-shape space. These features include the direction of flow and the distribution of sources, phase and amplitude as well as the correlation with breathing. It is further suitable to investigate how individual waves and the combination of events differs between stages of anaesthesia.
In summary a new technique to characterize slow-waves is presented that can help to improve our understanding of neural processing in the brain under anaesthesia. The use of Optical Flow, Helmholtz-Decomposition and autoencoders to characterize and distinguish different types of slow-waves appears promising. It bears potentials to study the relationship between distributions of different slow-waves and other features including anaesthetics, experimental stimuli or animal behavior.

Acknowledgements

I would like to thank my supervisors for their support of my Master Thesis. Especially I want to thank Silviu Bodea who initiated the project, recorded all the data and made my contribution possible. Special thanks go to him and Prof. Gil Westermeyer of the Institute of Biological and Medical Imaging (IBMI) for the continuous feedback and advice regarding the results and further direction of my work. Besides I want to genuinly thank my first supervisor, Dr. Dominik Juestel for his input regarding the methodological questions and especially for pointing out the benefits of Helmholtz-Decomposition. I truly enjoyed taking part in the meetings of his group at the Institute for Computational Biology (ICI) and appreciate that I gained interesting insights in the work of the other members which gave variety to my work during the global pandemics. Finally I want to thank my second supervisor Prof. Peter König of Osnabrück University especially for his stuctured guidance regarding a cooperation with external partners during the Master Thesis.

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List of Abbreviations

LAH	**L**ist **A**bbreviations **H**ere
WSF	**W**hat (it) **S**tands **F**or

Physical Constants

Speed of Light $\quad c_0 = 2.997\,924\,58 \times 10^8\,\mathrm{m\,s^{-1}}$ (exact)

List of Symbols

a	distance	m
P	power	W ($\mathrm{J\,s^{-1}}$)
ω	angular frequency	rad

For/Dedicated to/To my…

Chapter 1

Introduction

Anaesthesia inhibits neural activity and alters the signals in the brain. Depending on the dosage of the anaesthetic in use different patterns can be observed. At intermediate concentrations many agents cause propagating low-frequency waves that occur in close succession and manifest as delta oscillations in EEG. Similar patterns can be observed during deep sleep. Slow-waves differ in shape and size and with respect to specific temporo-spatial features: Most importantly the source location of recurrent activation, the direction and speed of spread and different sets of functionally connected regions can be observed. These features can be measured using modern imaging techniques that capture the fluorescence of genetically modified GCamp channels.
The development of new experimental technologies to capture GCamp fluorescence with an increasing temporal resolution of up to 100Hz offers new opportunities for the study of the neural circuitry and dynamic state changes of the brain under anaesthesia. At the same time, they also impose new challenges that result from the complexity of the recorded data. These challenges must be addressed by a well-controlled experiment or suitable approaches for data analysis.
Strict experimental control simplifies data processing. Under certain experimental conditions, high amplitude slow-waves with a good signal to noise ratio and contrast to the hemodynamic autofluorescence occur in a regular pattern. For ketamine induced anaesthesia (100mg/kg), regular patterns of slow-waves with highly similar shapes, durations and amplitudes occur. This simplifies the separation of events and the subsequent analysis (Celotto et al. 2020). However, such strict control means that the variance of the captured slow-waves is highly reduced.
More general findings regarding the structure of slow-waves are only possible in less controlled settings. This means, however, that suitable methods for data processing and analysis must be established that are both robust and suitable to capture the dynamics of slow-waves of different shapes and signal strengths.
Hence a new approach was developed that allows to distinguish and characterize slow-waves of various shapes and forms. It reveals a complex topology of events and shows how different levels of isoflurane change the way signals travel in the brain. The development was accompanied by two major questions: (1) What are slow-waves and (2) how can their temporospatial-properties be measured and characterized. To answer the earlier question and provide a working-definition for neural slow-waves that allows to destinguish different events, relevant slow-wave literature was systematically reviewed. To tackle the latter question it was investigated how Dense Optical Flow can be used to measure the strengths and direction of neural flow and how Helmholtz-Decomposition can help to distinguish it from neural sources and areas of extended recurrent activity. As not only features but characteristic patterns are of interest it was further assessed how autoencoders can be used for dimensionality reduction of the measured properties.

In the following the results of named literature review are presented first (section 2). It shows that anasthetics change important properties of neurons most prominantly their excitability which arguably explains why bistable states of network level activity can be observed empirically (section 21). While important differences exist between anaesthesia and sleep, slow-waves can be observed in both states (section 2.2). Here slow-waves are understood as extended periods of recurrent neural activation that manifests as above baseline activity (section 23).
The methods to analyze slow-waves are expained in the second part. First a new approach is presented that allows to track hemoglobin rich blood in vessels of various size. It is shown that high amplitude oscillations result from intracranial bloodflow and band-stop filtering can be used to reduce this effect. Second an overview of the processing steps is provided. The procedure of splitting events is reported, then Dense Optical Flow is introduced and it is demonstrated how Helmholtz Decomposition can be used to distinguish between the effect of local brightness changes and global flow. Subsequently the use of autoencoders for unsupervised characterization of slow-waves is discussed and the hyperparameters of the models used are presented.
The results of the approach are presented in the third part. First simple statistics regarding the dataset, selected features and the experimental conditions are reported. Then the relationship between properties of waves is summarized for representative events. Finally the results of the autoencoders are shown.
Finally the findings regarding the methods and the achieved results are discussed. Optical-flow, Helmholtz decomposition and autoencoders are a suitable tool to study slow-waves. Feature engineering helps to select relevant and robust features. Optical flow can be used in combination with Helmholts-Decomposition to distinguish between local sources and global flow of neural activity. Autoencoders represent an easy to use tool for the analysis of high level features. The results confirms the existence of fundamentally different types of slow waves. However, the approach allows presented here for more fine grained distinctions and reveals a complex topology static and dynamic properties in latent slow-wave space.

Chapter 2

Slow-waves

2.1 Overview of proposed types and functions

There is a large body of literature that addresses slow oscillations in the brain. Slow waves in the delta range (1-4 Hz) can been identified in electroencephalograms (EEGs). How evern more recently slow-waves have also been studied using wide-field flourescence microscopy. Simultaneous imaging and electrophysiological recordings indicate how both signals relate to each other. As a new approach to analyze the resulting data is presented here it is crucial to summarize our knowledge about slow-waves. A clear understanding is not only necessary to discern events and measure slow-wave properties but it is also important for the cross-validation and interpretation of the results achieved. Hence relevant literature on slow-waves, sleep and the effect of anaesthetics is briefly reviewed in this chapter.
The effects of anaesthesia on the properties and the spiking behavior of individual cells has been studied extensively. It shows that anaesthetics change several properties of neurons which modify the temporal and spatial integration of signals and cause bistable states in distributed neural networks. This can be demonstrated using spiking network models which provide an explanation for the occurence of slow-waves under anaesthesia that is coherent with named changes of neural properties (see ???). Stronger inhibition of neural activity also exists during sleep. However important characteristics of slow-wave dynamics differ between NREM sleep and anaesthesia. Hence it was argued that two different types of slow-waves occur during sleep and anaesthesia.
Evidence from several decades of research highlights the role thalamic circuits play for the generation of rhythmical activity and sleep. While slow-wave sleep is arguably promoted by several mechanisms including the cardian cycle that alters neural excitabilty, findings suggest that deep sleep is triggered by sleep spindles of thalamic origin. This contrasts with with the idea of slow-waves that form spontaneously in different regions of cortex if the excitabilty is reduced. Not all mechanisms of sleep are fully understood. It must be mentioned, however, that sleep is a vegetative state and thalamic nuclei play a crucial role in the generation of neural oscillations including slow-waves.
Sleep slow-waves are known to be involved in processes of memory consolidation. The assumed modes of action have been described in the hippocampal neocortical dialog model. Many results support the hypothesis that memory replay occurs during sleep and that it includes both hippocampus and neocortex. One may assume that sleep-slow waves represent the neural signature of an important part of this process. Memory consoldation is arguably the most prominent function of slow-waves (see ??).
First steps to distinguish slow-waves in a data driven approach were carried out by Bernardi et al. (2018). Two types of sleep slow-waves may be distinguished in

EEG. This distinction is based on a metric that includes a shape parameter and a term that indicates whether slow-waves are rather local or widespread. The distibution of these types of slow-waves differs during different stages of deep sleep. It highlights the importance of means to discern slow-waves based on their shape and spatial pattern of activation.
A working definition that allows to detect and separate slow-waves is derived from the developed understanding of slow-waves.

2.2 Effects of anaesthesia on single cells and neural networks

Anaesthetics alter the spiking behavior of neurons. Under deep anaesthesia the activity of cells is highly reduced and quiescence can be observed for most units. However, the modes of action that cause the respective changes of neural properties differ between anaesthetic agents. In addition, the effects can depend on the exact concentration of the drug in use and low dosages may, in some cases, even have opposing effects. As isoflourane was used to aquire the dataset at hand its effects on individual cells is shortly summarized highlighting important differences to other anaesthetics when necessary. It is known that alterations of the properties of individual cells change the dynamics of neural interaction on a population level. By adjusting the properties of cells in simple models of neural networks bistable states can be reproduced that resemble slow-waves under anaesthesia.
The exact pharmacological mechanisms of action of inhalant anaesthetics such as isoflourane remain uncertain (Miller et al. 2020). Effects on several ion channels have been reported including both chemically gated CL- channels (GABA receptors and glycine receptors) and K+ channels (Glutamate receptors). For example isoflourane is known to reduce the hyperpotentiation that results from CL- influx as a consequence of in vitro GABA administration (Jenkins 1999). Hence it can be assumed to have exitatory effects in the nervous system as it decreases the inhibition due to GABA. However isoflourane also has inhibitory effects as it supresses K+ channel currents resulting in smaller electrically triggered peak amplitudes of action potentials (Buljubasic 1992). Besides a potentiation of glycine receptors is assumed alongside other neurochemical mechanisms that alter the excitabilty of neurons (National Center for Biotechnology 2021). In vivo studies indicate that the net-effect of isoflourane appears to be inhibitory for all relevant dosages. In this respect isoflourane contrasts with many other anaesthetics (including e.g. halothane and ketamine) that show concentration dependance (Mogdahan 2019). It shall be noted however that differences in the density of different types of receptors may exist in different brain regions. While isoflourane can be assumed to decrease the excitability of neurons and inhibit neural signaling at all concentrations the strengths of decrease could differ between brain areas.
Single cell recordings reveal decisive effects of Isoflourane on the properties and the spontaneous bahavior of neurons. Moghadam et. al (2019) performed a comparative study of systemic and volatile general anesthetics in single cell cultures and the isolated brain of lymnaea stagnalis. The substances tested include the volatile agents sodium pentobarbital, sodium thiopentone, ketamine on one side as well as halothane, enflurane and isoflurane on the other. It was found that isoflourane causes a gradual decline of both amplitude and frequency of spontaneous action potentials. Differences between the six types of neurons studies were found to be marginal. Upon stimulation neurons remained silent at all studied concentrations of isoflourane. In contrast a gradual decline of evoked action potentials showed for

inceasing levels of enflourane. Using either halothane or barbiturates the authors were also able to produce bistable states in vitro during which periods of rapid spiking and quiesence alternate spontaneously.
Besides named alternations in spiking, effects on subthreshold properties of neurons have been identified. Increasing levels of isofourane can cause a decrease in the membrane time constant i.e. the duration between stumulus onset and 63% potential change of the cell membrane. Under normal conditions it takes longer for the neuron to reach maximal voltage as compared to anaesthesia with isoflourane. This is reflected in the estimated membrane capacitance that analogously decreases. The membrane capacitance is especially interesting because of the role it plays in the integration of electrical inputs (Golowasch & Nadim 2014). The most likely explanation for the apperent reduction is however an increase in the leakage current. This is because it is (1) rather unplausible for the capcitance of the constant size membrane to change significantly and (2) because of the abovementioned interaction between membrane proteins and anaesthetics (Quazzaz & Winlow 2017). Neurons act as temporal integrators and fire if the combined voltage of input spikes that occur simultanously or in short succession exceeds threshold. If the leakage current is stronger the timing of input spikes becomes more critical as the membrane potential goes back to baseline more quickly which may pohibit charge accumulation. Hence it can be hypothsized that anaesthetics affect temporal integration. [1]
Arguably the abovementioned effects explain important features of population level dynamics. Marshall (1981) systematically studied the EEG patterns in the awake state and during anaesthesia with isoflourane for five different dosages at up to 2.9% in humans. For very light anaesthesia (iso = .56%) low voltage fast activity can be observed. At a light surgical level (isp = .56% and 1.78%) slow oscillations are present that change from more regular to irregular patterns and alternating patterns with high amplitude oscillations at a moderate surgical level (iso = 2.2%). For deep anaesthesia only occasional low voltage activity shows (iso = 2.9%). leads to a gradual shift from stable up state to bistable states with distinct patterns and finally deep anaesthesia where quiesence dominates.
The effects of anesthesia presented here provide the background for a mechanistic interpretation of slow-wave activity. The overall dynamics that arise from changes of neural properties can be modeled using a simple model that employs Adaptive Exponential Integrate And Fire cells as shown by Nghiem et al. (2018): Simulations of a network that consists of 80% excitatory cells and 20% fast spiking inhibitory neurons produce sequences of continuous activity (up-states) and widespread absence of action potentials (down-states). A shift from down-states to and up state can be triggered by background noise while "spike-frequency adaptation on excitatory cells produces a self-inhibition that, destabilizing the up state, causes a reset to the down state" (Nghiem et. al 2018, p. 2). Spiking network models can reproduce alternating sequences of up states and down states, an important characteristic of population level activity under anaesthesia.
While simple mechanistic models explain some of the features of anaesthesia on a population level they represent a coarse generalization. It was highlighted that the exact mechanisms of action of anaesthetics remain uncertain. Generalizing over different agents and dosages is not necessarily justified and the dynamic of neural signalling might be affected by differences in the distribution of receptors for different neurotransmitters. Besides the complex anatomy of the brain including the various pathways that connect different regions uni- or bidirectionally is typically

[1] Note that other anaesthetics were found to increase the membrane capacitance

not reflected in simple mechanistic models. More precise measurements of the pathways and dynamics of neural signal transduction during slow-wave anaesthesia are necessary to understand bistable states of the brain. This is especially important also because it was argued that slow-wave sleep differs significantly from anaesthesia.

2.3 Two types of slow-waves in anaesthesia and deep sleep

Recently it was argued that the dynamics of slow-wave sleep and slow-wave anaesthesia differ substantially (Ngiehm et. al 2018). Jercog et al. (2017) found that the length of up states and the subsequent down state is correlated during urethane induced anaesthesia in rats for clearly synchronized periods where high-amplitude, slow fluctuations are present in local field potentials. Coefficients indicate a very weak relationship (r = .2). However, the correlation was found to be consistantly positive across experiments whearas the correlation with later down-state periods (time-lag > 2) is close to zero (Jercog et al. 2017). This pattern in the dynamic of neural firing can be reproduced using simple mechanistic models (see 2.2). During human non-REM sleep, however, no such relationship holds. As a tendancy, long down states are followed by short up states instead as indicated by a very weak but significant negative correlation (r = -.04). A negative peak in the temporal corsscorrelation exists around zero (Ngiehm et. al 2018). This gave rise to the argument that sleep-slow waves are fundamentally different from their counterpart during anaesthesia (Ngiehm et. al 2018). It allows for the interpretation that sleep slow-waves are elicited by a process that is not explained by spontaneous firing due to random cortical noise.
Sleep can be described as a physiological process that includes two seperate effects. First a decrease in neural excitability due to changes in the concentrations of disperse neurotransmitters and neurohormones and second a processes that repeatatively toggles down and up-states.
Serotonin and melatonin. Pineal gland. The vast majority of cells are pinealocytes (95%) that produce melatonin (Aulinas 2019). Inhibitory. It was found that melatonin interacts with voltage-sensitive Ca2+ channel and inhibits the release of neurotransmitters as well as synaptic transmission (Choi 2014). in vitro. The scn projects to the pineal gland. The SCN is assumed to be capable not only of creating a light-dependant circadian rhythm, but also of maintaining an entrained rhythm (Koella 1984).
Sleep can be triggered by electrical stimulation of thalamus. Cats dogs. Delayed. Electrostimulation of the reticular core was also found to be followed by an increased theta output in the hippocampus. In this respect spindles. They are considered a switch for the transition between light an deep sleep(Montagna 2005). Rats. Electrical stimulation of the raphe nuclei induces sedation and sleep in rats. Raphe nuclei are the area of origin of the ascending serotonergic pathways.

Thalamus gateway to consciousness. In awake. Relay. However it was found that the thalamus can also act as neural oscillator. In sleep. Also under anaesthesia with barbiturates. "During these states, the behavior of thalamic cells is characterized by long-lasting hyperpolarizations and phasic burst discharges recurring rhythmically" (Steriade 1984, p. 21). Thalamic -> Athalamic cat: non-REM sleep 38% -> 11.8% of total observation time. Diencephalic cats (ablation of the entire neocortex and striatum): Still Sleep spindles (after Montagna 2005).
• lesioning the reticular nucleus also decreased delta waves and led the animals to

sudden death.36
Findings from fatal familial insomnia indicate congruently that thalamic atrophy is "associated with lack of sleep spindles and delta sleep implicate the thalamus in the origin of slow wave sleep"(Montagna 2005). Preferential thalamo-olivary degeneration. "paramedian thalamus acts as 'final common pathway'"??

Rhythmical stimulation of cortex evokes periodic oscillations in thalamic neurons that sustain over extended periods of time after stimulation offset. cortical area 7. Once in a bistable state it can self-enhance synchronization (Steriade 1984).

sleep as an instinctive behaviour. medial thalamo-limbic structures as mechanistically involved in SWS production and autonomic balance

that include the cardian cycle sleep spindles are seen as a crucial signal that triggers a between up and down-states.

This gives rise to the question: What is sleep and how is it different from anaesthesia? what is sleep. Explains dynamics during anaesthesia not sleep. Sleep.... hippocampus. Long up states –> long down states. during anaesthesia.

Functions of sleep. short. Ending with the role of memory formation. Question wheather it is different is unclear. The circumstance that it is debated whether sleep-slow waves and slow-waves under anaesthesia are comparable highlights the importance of an approach that allows to automatically characterize these patterns of neural signals.

Methodological triangulation.

2.4 Sleep slow-waves and memory consolidation

According to the hippocampal-neocortical dialogue model of slow-waves, the interaction of hippocampal sharp wave ripples and cortical slow-waves fosters memory consolidation (Buzsáki 1989, Walker et al. 2009): Recently it was shown that generating cortical slow-waves in prefrontal networks such that they are coupled to the occurrence of short wave ripples in the hippocampus increases the performance of rats in a memory task (Maingret 2018). Coherently, a correlation of slow-wave activity and the brain-derived neurotrophic factor was found for humans (Duncan, 2013). Spontaneously occurring slow-waves arguably play a similar role for memory. They predominantly occur phase locked to sleep spindles (Demanuele et. al. 2017). However, the exact mechanisms that orchestrate this synchrony are unknown (Sanda et. al 2020). Moreover, it was found that at least two different types of slow-waves exist that potentially relate to distinct synchronization processes (Bernardi 2018). Because of its high spatial resolution fluorescence microscopy can provide more fine-grained distinctions and may potentially reveal trajectories of neural signal transduction during slow wave anesthesia. This highlights the importance of methods that allow to capture the variance of temporospatial patterns of neural slow-waves.

2.5 A working definiton

A distinction between slower and shorter waves has already been made in the pioneering electroencephalographic study almost a century ago (Berger, 1929, p. 550). Shortly later the term delta wave was coined to describe oscillations in the recorded voltage in the respective frequency band of around 1 to 4 Hz. The term slow-wave is typically also used to describe oscillatory patterns in EEGs. "Slow-wave activity

(SWA) is defined as the EEG power in the slow wave frequency band" (Furrer et al., 2019, p. 1). This frequency band relates to the delta band. Hence the term delta wave which is oftenly used in EEG research can be considered a synonym for slow waves. However, when speaking of slow-waves one oftenly referrs to slow wave sleep or anaesthesia where delta waves occur largely independant from higher frequency components.
As explained above slow waves are however more then an oscillation in the scalp potential relative to a reference electrode. The term is also used in relation to the neural phenomenom, its functions and its signatures that can be measured using various methods. Slow-waves are short periods of above baseline activity that occur in bistable states such as deep sleep and anaesthesia which are characterized by reduced neural excitabilty. Slow-waves are dynamic patterns of recurrent acticity of large potentially disctinct populations of neurons. They can differ with respect to the pathway of neural flow and the set of recruited cells. Sleep slow waves represent an important mechanism for memory consolidation. Evidence suggests that memory replay occurs both for sparse hippocampal codes and the associated dense cortical manifestations of the previously encoded learning experience. If thalamic short wave rippels correlate with cortical up-states one may speak of a thalamocortical slow wave event. Analogously one could form subcategorizations for slow-waves that incorporate deviant sets of temporally connected neurons. One may hypothesize that slow waves show neural associations in temporal isolation.
If slow-waves are understood as short periods of above baseline activity they can hence incorporate multiple peaks or slow oscillations in the strict sense. This is especially important for the detection and separation of events. Instead of relying on local minima alone this definition requires to detect in addition wheather activity went back to baseline. While conceptualizing slow waves as slow oscillations in the strict sense this approach chosen. It bears potentials of revealing the temporal dynamics of slow waves that incorporate several stages.

Chapter 3

Methods

3.1 Dense Optical Flow

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3.2 Helmholtz-Decomposition

3.3 Helmholtz-Decomposition of Dense Optical Flow

Chapter 4

Results

4.1 Main Section 1

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4.1.1 Subsection 1

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Chapter 5

Discussion

5.1 Main Section 1

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5.1.2 Subsection 2

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5.2 Main Section 2

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Appendix A

Frequently Asked Questions

A.1 How do I change the colors of links?

The color of links can be changed to your liking using:

`\hypersetup{urlcolor=red}`, or
`\hypersetup{citecolor=green}`, or
`\hypersetup{allcolor=blue}`.

If you want to completely hide the links, you can use:

`\hypersetup{allcolors=.}`, or even better:
`\hypersetup{hidelinks}`.

If you want to have obvious links in the PDF but not the printed text, use:

`\hypersetup{colorlinks=false}`.

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