

Dipak Ghosh · Shukla Samanta
Sayantan Chakraborty

Multifractals and Chronic Diseases of the Central Nervous System



Springer

Multifractals and Chronic Diseases of the Central Nervous System

Dipak Ghosh • Shukla Samanta
Sayantan Chakraborty

Multifractals and Chronic Diseases of the Central Nervous System

Dipak Ghosh
Department of Physics
Sir C V Raman Centre for Physics and
Music, Jadavpur University
Kolkata, West Bengal, India

Shukla Samanta
Department for Physics
Seacom Engineering College
Howrah, West Bengal, India

Sayantan Chakraborty
Electrical and Electronics Engineering
ICFAI University
Agartala, Tripura, India

ISBN 978-981-13-3551-8 ISBN 978-981-13-3552-5 (eBook)
<https://doi.org/10.1007/978-981-13-3552-5>

Library of Congress Control Number: 2018964106

© Springer Nature Singapore Pte Ltd. 2019

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

The publisher, the authors, and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

This Springer imprint is published by the registered company Springer Nature Singapore Pte Ltd.
The registered company address is: 152 Beach Road, #21-01/04 Gateway East, Singapore 189721, Singapore

Preface

“Neurodegeneration” corresponds to any pathological condition which primarily affects neurons. Neurological disorders not only affect the brain but also the nerves that are found throughout the body and spinal cord. Neurodegenerative diseases (NDDs) are defined as disorders that affect the central nervous system causing progressive dysfunction of the nervous system. These incurable and exhausting conditions are characterized by loss of neuronal cell function and are often associated with the deterioration of structures of the affected nervous system. Diagnosis of neurological diseases is a growing concern and one of the most difficult challenges for modern medicine. According to the World Health Organization’s recent report, neurological disorders, such as epilepsy, Alzheimer’s disease, Parkinson’s disease, Huntington’s disease (HD), stroke, and headache, to name a few, affect up to one billion people worldwide. An estimated 6.8 million people die every year as a result of neurological disorders.

The book primarily focuses on the study of different neurological disorders like epilepsy, Alzheimer’s, Parkinson’s, HD, and motor neuron diseases (MNDs) from a new perspective by analyzing the physiological signals such as EEG, EMG, ECG, and gait rhythm associated with these diseases using nonlinear dynamics.

Physiological signals such as heart rate, blood pressure, respiration, and stride intervals fluctuate continuously over time reflecting the complex regulation of these signals by the central nervous system. Analyzing the dynamics of these human physiological signals is an important area of research to help control and to be able to predict the onset of pathological conditions. Since long, several researchers have used different techniques, mostly linear, for studying various diseases, but, of late, research on nerve-related diseases or disorders has gained much importance as the world suffers a lot of deaths due to these progressive neuron diseases which are a slow and silent killer due to the lack of proper knowledge and appropriate medication. Since we have been working on various neurological disorders for more than 10 years, we decided to summarize our works and also the works of other researchers in this field in the form of a book.

Like every other system found in nature, physiological signals are also of complex character, as they are composed of many subsystems which are strongly correlated to each other, but not in a linear fashion. Conventional linear techniques like amplitude, root mean square, or Fourier analysis cannot provide detailed information about these subsystems. The development of nonlinear methods has significantly helped in studying complex nonlinear systems in detail by providing accurate and precise information about them. Nonlinear time series analysis methods enable the determination of characteristic quantities of a particular system solely by analyzing the time course of one of its variables. Thus, from this viewpoint, nonlinear time series analysis methods are superior to mathematical modeling, since they enable the introduction of basic concepts directly from the experimental data. Since works of several researchers have established the complex nonlinear character of physiological signals like EEG, ECG, EMG, and human gait rhythm, we have been motivated to use nonlinear techniques in our work.

In a nutshell, the book provides a comprehensive study on most of the neurological disorders with special emphasis on the methods used which are not only new but also rigorous and robust. The findings provide simple parameters for the diagnosis and prognosis of different neurodegenerative disorders, and adequate software can be developed which can easily be coupled with machines. The overall premise of the book for analyzing bioelectrical signals using nonlinear techniques is easily achievable and need of the day. The book will be easily accessible and useful to a very large community working in biomedical sciences and engineering.

We hope that this compilation of the original research work on the analysis of brain through fractal analysis will definitely provide a platform and a direction for inquisitive students and researchers of biomedical engineering and neuroscientists to think objectively on the premises. We also feel that this work will stimulate more exhaustive research on different other neurological disorders which are not commonly studied.

We are really happy that the leading publisher Springer Nature has accepted to publish the book. We sincerely thank the editors and all the other staffs of Springer Nature for their continual help, support, and suggestions.

Kolkata, West Bengal, India
Howrah, West Bengal, India
Agartala, Tripura, India

Dipak Ghosh
Shukla Samanta
Sayantan Chakraborty

Acknowledgment

We begin this acknowledgment section with Albert Einstein's philosophical thought:

Many times a day I realize how much my own outer and inner life is built upon the labors of my fellow men, both living and dead, and how earnestly I must exert myself in order to give in return as much as I have received.

We would like to express our deepest appreciation to all brilliant authors, researchers, and doctors who have provided us with a wealth of wisdom in the domain of diseases of the central nervous system. We believe that the analysis of biomedical signal with a novel new approach yielding precise results has become possible due to a breakthrough idea of Mandelbrot introducing a new concept of "fractals." We take this opportunity to record our acknowledgment to Benoit Mandelbrot.

We would like to express our heartiest thanks to Springer Nature and the editors for their kind support and encouragement which have helped us in the completion of the book. We would also like to express our gratitude toward the reviewers for their kind cooperation and important suggestions which have helped to enrich the book. We also want to extend our thanks and appreciation to all the other staff members of Springer Nature for their continual support and suggestions.

Contents

1	Introduction	1
1.1	Central Nervous System and Its Diseases	1
1.2	Bioelectrical Signals	5
1.2.1	Electroencephalography	6
1.2.2	Electrocardiography	6
1.2.3	Electromyography	8
1.3	Linear Signal Processing Techniques	9
1.3.1	Root Mean Square (RMS) Method	10
1.3.2	Fast Fourier Transform (FFT) Method	10
1.3.3	Short-Time Fourier Transform (STFT) Method	10
1.3.4	Wavelet Transform (WT) Method	11
1.3.5	Discrete Wavelet Transform (DWT) Method	11
1.4	Limitations of Linear Analysis Techniques	11
1.5	Non-linear Techniques	12
1.5.1	Fractals and Multifractals	13
1.5.2	Non-linear Analysis of Biomedical Signals	22
1.6	Review of Studies on Neurological Disorders	26
1.6.1	Epilepsy	26
1.6.2	Dementia (Alzheimer's Disease)	27
1.6.3	Parkinson's Disease	28
1.6.4	Huntington's Disease	29
1.6.5	Motor Neuron Disease (MND)	30
	References	31
2	Multifractal Study of EEG Signal of Subjects with Epilepsy and Alzheimer's	47
2.1	Introduction	47
2.2	Neurological Disorder: Epilepsy, Alzheimer's, and EEG Data	48
2.2.1	Epilepsy	48
2.2.2	Alzheimer's Disease	52
2.2.3	EEG Data	54

2.3	Multifractal Detrended Fluctuation Analysis of EEG Signals	57
2.4	Multifractal Detrended Cross-Correlation Analysis of EEG Signals	64
2.5	Possible Application as Biomarker of Epilepsy	70
	References	71
3	Multifractal Approach for Quantification of Autonomic Deregulation Due to Epileptic Seizure with ECG Data	79
3.1	Introduction	79
3.2	Systematic Studies on Abnormalities in Cardiac Autonomic Status	80
3.3	Multifractal Detrended Fluctuation Analysis of ECG Signals	83
3.3.1	ECG Data	84
3.4	Results and Possible Biomarker	91
	References	91
4	Multifractal Analysis of Electromyography Data	97
4.1	Introduction	97
4.2	Motor Neuron and Musculoskeletal Disease: Neuropathy and Myopathy	98
4.3	Electromyography – A Tool to Detect Motor Neuron Disease . . .	98
4.4	Study of SEMG Signals	99
4.5	Electromyography Data	102
4.6	Multifractal Detrended Fluctuation Analysis of EMG Signals . . .	103
4.7	Results and Possible Advanced Level Biomarker	111
	References	112
5	Multifractal Study of Parkinson's and Huntington's Diseases with Human Gait Data	117
5.1	Introduction	117
5.2	Parkinson's Disease and Gait Data	120
5.2.1	Gait Data	121
5.3	Multifractal and Multifractal Cross-Correlation Analysis of Parkinson's Disease	124
5.4	Huntington Disease and Gait Data	139
5.5	Multifractal Analysis of Huntington's Data	140
5.6	Discussions on Possible Use of the Result for Biomarkers of Parkinson's and Huntington's	142
	References	143
6	Multifractal Correlation Study Between Posture and Autonomic Deregulation Using ECG and Blood Pressure Data	149
6.1	Introduction	149
6.1.1	Blood Pressure	151
6.1.2	Non-linear Heart Rate Variability Analysis	152
6.1.3	Correlation of Heart Rate and Blood Pressure Signals	154

6.2	Posture-Dependent ECG and Arterial Blood Pressure (ABP) Data	157
6.3	Multifractal Cross-Correlation Analysis Between ECG and ABP Data	158
6.4	Discussion of the Result and Possible Use as Biomarker of Neurological Disorder	166
	References	166
	Appendices	173

About the Authors

Prof. Dipak Ghosh a University Gold medalist and a Ph.D. in High Energy Physics, is currently an Emeritus Professor at Sir C.V. Raman Centre for Physics and Music, and Director (Hony.) of the Biren Roy Research Laboratory for Radioactivity and Earthquake Studies, Jadavpur University. Formerly, he was a Professor and Dean of the Faculty of Science, Jadavpur University. Prof. Ghosh has received several awards like the S.N. Bose Memorial Medal, C.V. Raman Award by the University Grants Commission, etc. in recognition of his in-depth research work. He was the Principal Investigator from India in projects with prestigious research institutes like CERN and FERMI Lab. He has over 500 publications in reputed international journals and several books to his credit. His research interests include nuclear and particle physics, nonlinear dynamics applied to biomedical problems, and neurocognition of music. He has also served as a reviewer for international journals like *Physical Review Letters*, *Physica A*, *EPL*, etc.

Dr. Shukla Samanta who holds a Ph.D. in Physics from Jadavpur University, is currently associated with Seacom Engineering College, Kolkata, as an Assistant Professor and Head of the Basic Science and Humanities Department. She holds a postgraduate degree in Physics from Presidency College, and an M.Tech. in Computer Science and Application from the University of Calcutta. She has published several papers on seismic activity, econophysics, and physiological systems in various peer-reviewed international journals. She has also served as a reviewer for international journals like *Physica A*, *IJAV* etc. Her current research interest is focused on the nonlinear study of different neurological disorders.

Mr. Sayantan Chakraborty is currently pursuing his Ph.D. in Electrical and Biomedical Signals at the Department of Instrumentation and Electronics Engineering, Jadavpur University. He completed his postgraduate degree in Electrical Power Engineering at the University of Calcutta. He has published several papers on electric bid prices, the Stock Exchange of India and biomedical signals in journals of international repute. He has also served as a reviewer for international journals

such as *Physica A*, *IET Science, Measurement & Technology*, etc. Mr. Chakraborty is also an Assistant Professor and Head of the Department of Electrical and Electronics Engineering at ICFAI University, Tripura. He was previously affiliated with Dr. Sudhir Chandra Sur Degree Engineering College and Seacom Engineering College, Kolkata, as an Assistant Professor and Head of the Department of Electrical Engineering.

List of Figures

Fig. 1.1	A schematic diagram of ECG. (Source: https://en.wikipedia.org/wiki/QRS_complex)	7
Fig. 1.2	A fractal fern: it repeats its pattern at various scales. (Source: https://commons.wikimedia.org/wiki/File:Sa-fern.jpg)	14
Fig. 1.3	Romanesco broccoli: showing self-similar form approximating a natural fractal. (Source: https://en.wikipedia.org/wiki/File:Fractal_Broccoli.jpg)	15
Fig. 1.4	A quartz stone. (https://en.wikipedia.org/wiki/File:Unknown_Quartz_crystal_66.JPG)	15
Fig. 1.5	Neurons in cerebral cortex. (Source: https://commons.wikimedia.org/wiki/File:Smi32neuron.jpg#file)	15
Fig. 1.6	The Cantor set. (Source: https://en.wikipedia.org/wiki/Cantor_set#/media/File:Cantor_set_in_seven_iterations.svg)	16
Fig. 1.7	The Sierpinski triangle. (Source: https://commons.wikimedia.org/wiki/File:Sierpinski_triangle.svg)	16
Fig. 1.8	The Von Koch flake. (Source: https://commons.wikimedia.org/wiki/File:KochFlake.svg)	17
Fig. 1.9	The Mandelbrot set. (Source: https://commons.wikimedia.org/wiki/File:Mandelbrot_set_with_coloured_environment.png)	17
Fig. 1.10	The Julia set. (Source: https://upload.wikimedia.org/wikipedia/commons/b/b1/Julia_set_%28ice%29.png)	18
Fig. 2.1a	Plot of a typical signal for set A for 2 s	55
Fig. 2.1b	Plot of a typical signal for set B for 2 s	55
Fig. 2.1c	Plot of a typical signal for set C for 2 s	56
Fig. 2.1d	Plot of a typical signal for set D for 2 s	56
Fig. 2.1e	Plot of a typical signal for set E for 2 s	57
Fig. 2.2	Plot of $\ln F_q$ vs. $\ln s$ for a particular signal (Dutta et al. 2014) . . .	59
Fig. 2.3a	Plot of $H(q)$ vs. q for a particular signal and its corresponding randomly shuffled series (Dutta et al. 2014)	60

Fig. 2.3b	Plot of $\tau(q)$ vs. q for a particular signal and its corresponding randomly shuffled series (Dutta et al. 2014)	60
Fig. 2.3c	Plot of $f(\alpha)$ vs. α for a particular signal and its corresponding randomly shuffled series (Dutta et al. 2014)	61
Fig. 2.4	Distribution of values of the multifractal width W for sets A–E (Dutta et al. 2014)	62
Fig. 2.5a	Plot of $\log F_q$ vs. $\log s$ ($q = 2$) of individual and cross-correlated signal for a particular set of C and E (Ghosh et al. 2014)	66
Fig. 2.5b	Plot of $\log F_q$ vs. $\log s$ ($q = 2$) of individual and cross-correlated signal for a particular set of D and E (Ghosh et al. 2014)	66
Fig. 2.6a	Plot of $\lambda(q)$, $h(q)$ vs. q for a particular signal of set C and E (Ghosh et al. 2014)	67
Fig. 2.6b	Plot of $\lambda(q)$, $h(q)$ vs. q for a particular signal of set D and E (Ghosh et al. 2014)	67
Fig. 2.7a	Plot of τ_q vs. q of individual and cross-correlated signal for a particular set of C and E (Ghosh et al. 2014)	68
Fig. 2.7b	Plot of τ_q vs. q of individual and cross-correlated signal for a particular set of D and E (Ghosh et al. 2014)	68
Fig. 2.8a	Plot of $f(\alpha)$ vs. α of individual and cross-correlated signal for a particular set of C and E (Ghosh et al. 2014)	69
Fig. 2.8b	Plot of $f(\alpha)$ vs. α of individual and cross-correlated signal for a particular set of D and E (Ghosh et al. 2014)	69
Fig. 3.1	Plot of $\ln F_q$ vs. $\ln s$ of a particular ECG signal. (Ghosh et al. 2017)	85
Fig. 3.2	Plot of $h(q)$ vs. q of seven postictal ECG signals. (Ghosh et al. 2017)	86
Fig. 3.3	Plot of $\tau(q)$ vs. q of seven postictal ECG signals. (Ghosh et al. 2017)	86
Fig. 3.4	Plot of $f(\alpha)$ vs. α of seven postictal ECG signals. (Ghosh et al. 2017)	87
Fig. 3.5	Plot of $h(q)$ vs. q of original and shuffled ECG signal of a particular subject. (Ghosh et al. 2017)	89
Fig. 3.6	Plot of $\tau(q)$ vs. q of original and shuffled ECG signal of a particular subject. (Ghosh et al. 2017)	90
Fig. 3.7	Plot of $f(\alpha)$ vs. α of original and shuffled ECG signal of a particular subject. (Ghosh et al. 2017)	91
Fig. 4.1a	Plot of EMG signal of a healthy subject for 1 s	104
Fig. 4.1b	Plot of EMG signal of a myopathy subject for 1 s	104
Fig. 4.1c	Plot of EMG signal of a neuropathy subject for 1 s	105
Fig. 4.2a	Plot of $\ln F_q$ vs. $\ln s$ for a particular set of EMG signal of healthy subject (Ghosh et al. 2017)	105
Fig. 4.2b	Plot of $\ln F_q$ vs. $\ln s$ for a particular set of EMG signal of myopathy subject (Ghosh et al. 2017)	106

Fig. 4.2c	Plot of $\ln F_q$ vs. $\ln s$ for a particular set of EMG signal of neuropathy subject (Ghosh et al. 2017)	106
Fig. 4.3	Plot of $h(q)$ vs. q for a particular set of EMG signals of healthy, myopathy, and neuropathy (Ghosh et al. 2017)	107
Fig. 4.4	Plot of $\tau(q)$ vs. q for a particular set of EMG signals of healthy, myopathy, and neuropathy (Ghosh et al. 2017)	108
Fig. 4.5	Plot of $f(\alpha)$ vs. α for a particular set of EMG signals of healthy, myopathy, and neuropathy (Ghosh et al. 2017)	110
Fig. 5.1a	Plot of the signal (force under each foot) for a particular Control subject for Experiment 1 for 3 s	122
Fig. 5.1b	Plot of the signal (force under each foot) for a particular Parkinson subject for Experiment 1 for 3 s	123
Fig. 5.1c	Plot of the signal (force under each foot) for a particular Control subject for Experiment 2 for 3 s	123
Fig. 5.1d	Plot of the signal (force under each foot) for a particular Parkinson subject for Experiment 1 for 3 s	124
Fig. 5.2a	Plot of $\log F_q$ vs. $\log s$ ($q = -5, 0, +5$) of Left Foot for a particular subject of Control group for Experiment 2 (Dutta et al. 2016)	125
Fig. 5.2b	Plot of $\log F_q$ vs. $\log s$ ($q = -5, 0, +5$) of Right Foot for a particular subject of Control group for Experiment 2 (Dutta et al. 2016)	126
Fig. 5.2c	Plot of $\log F_q$ vs. $\log s$ ($q = -5, 0, +5$) of Cross between Left and Right Foot for a particular subject of Control group for Experiment 2 (Dutta et al. 2016)	126
Fig. 5.2d	Plot of $\log F_q$ vs. $\log s$ ($q = -5, 0, +5$) of Left Foot for a particular subject of Parkinson's group for Experiment 2 (Dutta et al. 2016)	127
Fig. 5.2e	Plot of $\log F_q$ vs. $\log s$ ($q = -5, 0, +5$) of Right Foot for a particular subject of Parkinson's group for Experiment 2 (Dutta et al. 2016)	127
Fig. 5.2f	Plot of $\log F_q$ vs. $\log s$ ($q = -5, 0, +5$) of Cross between Left and Right Foot for a particular subject of Parkinson's group for Experiment 2 (Dutta et al. 2016)	128
Fig. 5.3a	Plot of $h(q)$ and $\lambda(q)$ vs. q for a particular subject of Control Group for Experiment 1 (Dutta et al. 2016)	129
Fig. 5.3b	Plot of $h(q)$ and $\lambda(q)$ vs. q for a particular subject of Parkinson's Group for Experiment 1 (Dutta et al. 2016)	129
Fig. 5.4a	Plot of $h(q)$ and $\lambda(q)$ vs. q for a particular subject of Control Group for Experiment 2 (Dutta et al. 2016)	130
Fig. 5.4b	Plot of $h(q)$ and $\lambda(q)$ vs. q for a particular subject of Parkinson's Group for Experiment 2 (Dutta et al. 2016)	130
Fig. 5.5a	Plot of $f(\alpha)$ vs. α for a particular subject of Control Group for Experiment 1 (Dutta et al. 2016)	131

Fig. 5.5b	Plot of $f(\alpha)$ vs. α for a particular subject of Parkinson's Group for Experiment 1 (Dutta et al. 2016)	131
Fig. 5.6a	Plot of $f(\alpha)$ vs. α for a particular subject of Control Group for Experiment 2 (Dutta et al. 2016)	132
Fig. 5.6b	Plot of $f(\alpha)$ vs. α for a particular subject of Parkinson's Group for Experiment 2 (Dutta et al. 2016)	132
Fig. 5.7a	Distribution of values of auto-correlation coefficients for Left foot for Experiment 1 (Dutta et al. 2016)	134
Fig. 5.7b	Distribution of values of auto-correlation coefficients for Right foot for Experiment 1 (Dutta et al. 2016)	134
Fig. 5.8a	Distribution of values of auto-correlation coefficients for Left foot for Experiment 2 (Dutta et al. 2016)	135
Fig. 5.8b	Distribution of values of auto-correlation coefficients for Right foot for Experiment 2 (Dutta et al. 2016)	135
Fig. 5.9a	Values of multifractal width W for $q = -10$ to $+10$ and $q = -5$ to $+5$ for Experiment 1 (Dutta et al. 2016)	138
Fig. 5.9b	Values of multifractal width W for $q = -10$ to $+10$ and $q = -5$ to $+5$ for Experiment 2 (Dutta et al. 2016)	139
Fig. 6.1	Plot of (a) ECG and (b) ABP signal for a particular subject for 2 s (Ghosh et al. 2018)	157
Fig. 6.2	Plot of $\ln F_q$ vs. $\ln s$ ($q = 2$) for a particular subject (Ghosh et al. 2018)	159
Fig. 6.3	Plot of $h(q)$ and $\lambda(q)$ vs. $q(=2)$ of original and shuffled series for a particular subject (Ghosh et al. 2018)	161
Fig. 6.4	Plot of $f(\alpha)$ vs. α of original and shuffled series for a particular subject (Ghosh et al. 2018)	162
Fig. 6.5	Plot of $\tau(q)$ vs. q for eight subjects (Ghosh et al. 2018)	165

List of Tables

Table 2.1	Mean values of multifractal width and variance for the sets A–E (Dutta et al. 2014)	61
Table 2.2	Mean values of h , γ , and W for sets C, D, and E (Ghosh et al. 2014)	70
Table 2.3	Mean values of λ , γ_x , and W for sets CE and DE (Ghosh et al. 2014)	70
Table 3.1	Values of multifractal width (w) and auto-correlation exponent (γ) of seven postictal ECG signals for original and shuffled series (Ghosh et al. 2017)	87
Table 3.2	Values of multifractal width (w) of ECG signals of normal healthy people and CHF patients (Channel I) (Ghosh et al. 2017)	88
Table 4.1	Values of $h(q)$ corresponding to q for a particular set of EMG signals of healthy, myopathy, and neuropathy subjects (Ghosh et al. 2017)	109
Table 4.2	Values of w for all the five sets of EMG signals of healthy, myopathy, and neuropathy subjects (Ghosh et al. 2017)	110
Table 4.3	Values of γ for all the five sets of EMG signals of healthy, myopathy, and neuropathy subjects (Ghosh et al. 2017)	110
Table 5.1	Mean values of the multifractal width W and asymmetry parameter B , auto-correlation coefficients for the left foot and right foot and cross-correlation coefficients for control and Parkinson's group along with variance and ANOVA parameters F and P , and confidence level for Experiment 1 (Dutta et al. 2016)	136
Table 5.2	Mean values of the multifractal width W and asymmetry parameter B , auto-correlation coefficients for the left foot and right foot and cross-correlation coefficients for control and Parkinson's group along with variance and ANOVA parameters F and P , and confidence level for Experiment 2 (Dutta et al. 2016)	137

Table 5.3	Mean values, variance of multifractal width W , and ANOVA parameters F and p values for all three groups (Dutta et al. 2013)	141
Table 5.4	Values of multifractal width W and auto-correlation coefficient γ for (i) healthy subjects (control group), (ii) subjects with Parkinson’s disease, and (iii) subjects persons with Huntington’s disease for both the original series and the shuffled series (Dutta et al. 2013)	141
Table 6.1	Values of $h(q = 2)$ for ECG and ABP, cross-correlation scaling exponent (λ), auto-correlation (γ), and cross-correlation (γ_x) coefficients along with mean and standard deviation for ECG and ABP (Ghosh et al. 2018)	160
Table 6.2	Values of multifractal width (w) of ECG, ABP, and cross-correlation (w_x) of original and shuffled series (Ghosh et al. 2018)	164
Table 6.3	Values of auto-correlation (γ) and cross-correlation (γ_x) coefficients for ECG and ABP of shuffled series	165

Chapter 1

Introduction



Abstract Disease of the central nervous system has been described in the literature as a group of neurological disorders for which the function of the brain or spinal cord is affected. This chapter outlines the general description of the diseases like epilepsy, Parkinson's, Huntington's, Alzheimer's, and motor neuron diseases. Also a discussion on the diagnostic tools and the methodologies adapted is reviewed in detail.

1.1 Central Nervous System and Its Diseases

The nervous system controls all activities of human beings. The nervous system consists of the brain and the spinal cord, as well as all the nerves throughout the body. Compared to other living organisms, humans are considered to be superior as the anatomy and physiology of the nervous system of humans are unique. The brain and spinal cord form the central nervous system (CNS), and all other nerves throughout the body are referred to as the peripheral nervous system (PNS). As the central nervous system (CNS) can determine the consciousness of us humans, it has been attributed to be the most complex organ in the human body. All aspects of our behavior from breathing to supporting our thoughts and feelings (Kandel and Squire 2000) are controlled by the nervous system. The human brain is the most sophisticated organ in the human body. The brain regulates vital body functions such as emotion, memory, cognition, motor activities, heart rate, respiration, and digestion. The human brain is a complex network of millions of neurons packed in a matrix of glial cells (Benson et al. 2017).

Diseases of the brain may be caused either due to inherent dysfunction of the brain or due to complex interactions of the brain with the environment (Hyman et al. 2006). Brain diseases range widely from common neurological to psychiatric disorders. Throughout the life span, brain diseases affect a very significant portion of the population and are widely spread both across the developed and developing nations. Compared to other diseases, brain diseases account for the highest burden in terms of health, economy, and social capital globally (Nathan et al. 2001). More than 1.5 billion people are affected due to brain disorders worldwide, and with the passage of time, it is feared that this population will increase. Thus there is an urgent need of not

only producing more drugs to treat CNS disorders but rigorous research so that early prognosis and diagnosis can be made and is the need of the day to help control this epidemic.

Across the life span of human, the nature of the brain disorders changes. In young there is a high prevalence of psychiatric disorders, like depression, anxiety, schizophrenia, and substance abuse, whereas the elderly suffer markedly from neurodegenerative disorders such as dementia or stroke (Wittchen et al. 2011). More widely appreciated are the neurodegenerative disorders, namely, Parkinson's (PD) and Alzheimer's disease (AD), which are on the rise due to an older population (von Campenhausen et al. 2005). Huntington's disease (HD) and amyotrophic lateral sclerosis (ALS) are other neurodegenerative diseases. The neurodegenerative diseases are characterized by inevitable gradual decline in cognitive ability and also the potential to self-sustain (Prince et al. 2013). The neurodegeneration produces a clinical syndrome called dementia, whose symptoms include inability to recollect, sudden and unexpected changes in one's mood, and problems to communicate and reason (Devous 2002).

Next to stroke, epilepsy is the second most common neurological disorder affecting approximately 50 million people worldwide. "Epilepsy" is derived from the Greek term *epilambanein* which means to seize, and it denotes the predisposition to have recurrent, unprovoked seizures (Quintero-Rincon et al. 2016). In epilepsy, the nerve cells of the brain transmit exorbitant electrical impulses that cause seizures. An epileptic seizure is defined as "a transient symptom of excessive or synchronous neuronal activity in the brain" (Fisher et al. 2005). Epilepsy is defined by two or more such unprovoked seizures. Seizures may be either focal or generalized. In focal seizures, only a specific segment of the brain is affected, while in generalized seizures the whole brain is affected (Acharya et al. 2012a). Epileptic seizures may lead to impairment or unconsciousness and psychic, autonomic, sensory, or motor problems (Lehnertz 2008).

Electroencephalography (EEG) is an important clinical tool for monitoring and diagnosing neurological changes in epilepsy. Compared with other methods such as magnetoencephalography (MEG) and functional magnetic resonance imaging (fMRI), EEG is an affordable and safe technique for inspecting brain activity. Drugs and surgical treatment options are not sufficient to treat epilepsy. Among new therapies developed, implantable devices that deliver direct electrical stimulation to affected areas of the brain have shown promising results. The effectiveness of these treatments depends mainly on robust algorithms for seizure detection. As seizure onset cannot be predicted, a continuous recording of the EEG is essential to ascertain epilepsy. But since visual assessment of long EEG recordings is tedious and time-consuming (Song 2011), automated detection methods of epilepsy have gained importance. With a view to study the changes that occur in the brain in seizure and seizure-free status, we analyzed EEG signals using a latest state-of-the-art methodology. The observations made are very interesting and are described in Chap. 2.

Alzheimer's disease another disease of the central nervous system is the major reason of dementia. This disease is described by an intensifying reduction in brain