

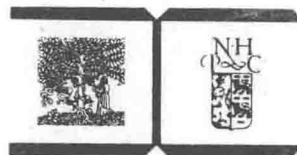
# EEG Primer



# EEG Primer

**R. SPEHLMANN**

*Professor of Neurology  
Northwestern University Medical School  
Director of EEG Laboratories  
VA Lakeside Medical Center, Chicago  
and Evanston Hospital, Evanston, Illinois, U.S.A.*



1981

ELSEVIER/NORTH-HOLLAND BIOMEDICAL PRESS — AMSTERDAM • NEW YORK • OXFORD



© 1981 Elsevier/North-Holland Biomedical Press

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without the prior permission of the copyright owner.

ISBN — hardbound: 0-444-80260-6  
— paperback: 0-444-80299-1

Published by:  
Elsevier/North-Holland Biomedical Press  
335 Jan van Galenstraat, P.O. Box 211  
Amsterdam, The Netherlands

Sole distributors for the U.S.A. and Canada:  
Elsevier North-Holland Inc.,  
52 Vanderbilt Avenue  
New York, N.Y. 10017

**Library of Congress Cataloging in Publication Data**

Main entry under title:

EEG primer.

Includes bibliographies and index.

1. Electroencephalography. I. Spehlmann, Rainer,

1931- CDNLM: 1. Electroencephalography. WL 150

E104J

RC386.6.E43E15 616.8'047547 80-26037

ISBN 0-444-80260-6 (Elsevier North-Holland)

PRINTED IN THE NETHERLANDS



## Preface

This book is written mainly for beginners in EEG. It may help several groups: Resident physicians who are in neurology training and need to learn EEG interpretation as part of this training; residents in other fields, interns and medical students who have become interested in EEG; neuroscientists who wish to understand the methods and clinical applications of EEG; EEG technicians who want to gain more insight into the test they perform; and anyone who wants to find out how clinical EEG is done and what it can do. On the other hand, even persons with some experience in EEG may find the book useful because it leads to fairly advanced levels. To make learning easier for the beginner, several methods have been adopted:

- (1) Explanations start with simple concepts and build up in steps following an order which generally corresponds with the steps of recording and reading an EEG.

- (2) The reader can choose the depth at which he wishes to master a topic. Each chapter is preceded by a summary. Reading all summaries takes from one to two hours and gives a rough overview of clinical EEG. Each part of the summary refers to numbered sections of the text of the text which expand on the summarized material. Each section presents the most important material at the start and then gives the details, allowing the reader to go as far as desired.

- (3) The book adopts widely accepted standards wherever they are available. The text is written in the terminology recommended by the International Federation of Societies for EEG and Clinical Neurophysiology. It incorporates the guidelines of the American EEG Society for proper EEG recording. The sections on epileptiform patterns conform with the international classification of epileptic seizures recently endorsed by federations of several societies.



(4) The clinical correlation of the EEG reverses the conventional order of listing diseases and describing the attendant EEG abnormalities. In this text, abnormal EEG patterns are described first and then correlated with clinical abnormalities. This method is chosen because it corresponds with the situation of the EEG reader who faces an EEG pattern and searches for its clinical correlates. Although this approach leads to repetition of some diseases under the heading of several EEG patterns, it causes much less repetition than the conventional method which repeats a few EEG abnormalities under the headings of many different diseases. The various EEG manifestations of a disease can be found through cross-references in the text and through the index.

(5) The main goal of this text is a didactic presentation of clinical EEG and its role in the diagnosis of cerebral disorders. Therefore, I have favored the established over the controversial and preferred modern insights to historical details. References for suggested reading are listed at the end of each chapter. They emphasize the more recent publications through which the reader may find the older ones.

It is a pleasure to acknowledge the many sources of help I have had in writing this book. I thank the residents, students and technicians who I taught EEG reading; they asked many of the questions which I tried to answer in this text. Dr. Donald W. Klass reviewed the manuscript and gave me his critical advice. Special thanks are due to Dr. Karyl Norcross who thoroughly revised several drafts of the text. Mr. Clifford C. Smathers helped me to design the technical illustrations. Mrs. Thelma Howell typed the manuscript without ever tiring.



# Contents

## Preface

## Part A: Technical background

### Introduction

#### 1 The source of the EEG

- 1.1 The generator of the EEG
- 1.2 Rhythmical EEG activity
- 1.3 Recording of electrical potentials with scalp electrodes

#### 2 Recording electrodes

- 2.1 Electrode shapes and application methods
- 2.2 Electrical properties of recording electrodes
- 2.3 Electrode placement

#### 3 The EEG machine: Parts and functions

- 3.1 The input board
- 3.2 Input selector switches
- 3.3 Calibration
- 3.4 The amplifiers
- 3.5 Filters
- 3.6 Writing units



4	<i>Recording strategy</i>	63
4.1	Multichannel recordings	63
4.2	Specific montages	72
4.3	Electrode combinations for monitoring of extracerebral activity	75
5	<i>The product of the recording: The clinical EEG record</i>	83
5.1	General technical standards	85
5.2	Standards for recordings from infants and small children	92
5.3	Standards for all-night sleep recordings	95
5.4	Standards for recordings in cases of suspected cerebral death	96
5.5	Telephone transmission	101
6	<i>Artifacts</i>	105
6.1	Artifacts from the patient	105
6.2	Interference	113
6.3	Artifacts from recording electrodes and equipment	115
7	<i>Other methods of recording and analysis</i>	119
7.1	Oscilloscope displays	119
7.2	Telemetry and portable magnetic tape recordings	120
7.3	Magnetic tape recording	121
7.4	Computer analysis	122
7.5	Computer analysis of spontaneous EEG	123
7.6	Computer averaging of evoked potentials: General techniques	128

## *Part B: The normal EEG*

8	<i>Definition of the normal EEG, relation to brain function</i>	143
8.1	Definition of the normal EEG	143
8.2	A normal EEG does not always mean normal brain function	145



8.3	An abnormal EEG does not necessarily mean abnormal brain function	145
9	<i>Descriptors of EEG activity</i>	147
9.1	Wave form	147
9.2	Repetition	150
9.3	Frequency	150
9.4	Amplitude	152
9.5	Distribution	153
9.6	Phase	154
9.7	Timing	155
9.8	Persistence	156
9.9	Reactivity	156
10	<i>The normal EEG from premature age to the age of 19 years</i>	159
10.1	Premature infants of 24 to 27 weeks of conceptional age	159
10.2	Premature infants of 28 to 31 weeks of conceptional age	161
10.3	Premature infants of 32 to 35 weeks of conceptional age	161
10.4	Premature infants of 36 to 40 weeks of conceptional age	163
10.5	Infants from full term to 3 months of age	165
10.6	Infants of 3 to 12 months of age	170
10.7	Infants, children and adolescents from 1 to 19 years of age	177
10.8	Major abnormalities	179
11	<i>The normal EEG of wakeful resting adults of 20 to 60 years of age</i>	183
11.1	Alpha rhythm	183
11.2	Beta rhythms	189
11.3	Mu rhythm	191
11.4	Lambda waves	193
11.5	Vertex sharp transients (V waves)	193
11.6	Kappa rhythm	194
11.7	Normal posterior theta rhythms	194
11.8	The low voltage EEG	195
11.9	Major abnormalities	195



## 12 *The normal sleep EEG of adults over 20 years*

12.1 Elements of normal sleep activity	201
12.2 Sleep stages	203
12.3 Sleep cycles	207
12.4 Major abnormalities	208

## 13 *The normal EEG of adults over 60 years of age*

13.1 Alpha rhythm	213
13.2 Beta rhythm	214
13.3 Sporadic generalized slow waves	214
13.4 Intermittent temporal slow waves	215
13.5 Sleep stages	217
13.6 Major abnormalities	217

## 14 *Activation procedures*

14.1 Hyperventilation	219
14.2 Sleep	222
14.3 Photoc stimulation	223
14.4 Other stimuli	227
14.5 Pentylenetetrazol, bemegride and other convulsant drugs	228

## *Part C: The abnormal EEG*

### 15 *Abnormal EEG patterns, correlation with underlying cerebral lesions and neurological diseases*

15.1 Definition of the abnormal EEG	235
15.2 Correlation between abnormal EEG patterns, general cerebral pathology and specific neurological diseases	236
15.3 The diagnostic value of the EEG	240



<b>16</b>	<b><i>Classification of seizures</i></b>	<b>245</b>
16.1	Definitions	245
16.2a	Classification of seizures—General	247
16.2b	Classification of seizures—Specific	250
<b>17</b>	<b><i>Localized epileptiform patterns</i></b>	<b>261</b>
17.1	Description of patterns	262
17.2	Clinical significance of focal epileptiform activity	268
17.3	Other EEG abnormalities associated with focal epileptiform activity	272
17.4	Grading of focal epileptiform activity	275
17.5	Mechanisms underlying focal epileptiform activity	275
17.6	Specific disorders causing focal epileptiform activity	277
<b>18</b>	<b><i>Generalized epileptiform patterns</i></b>	<b>285</b>
18.1	Description of patterns	286
18.2	Clinical significance of generalized epileptiform activity	298
18.3	Other EEG abnormalities associated with generalized epileptiform activity	301
18.4	Grading of generalized epileptiform activity	302
18.5	Mechanisms underlying generalized epileptiform activity	302
18.6	Specific disorders causing generalized epileptiform activity	304
<b>19</b>	<b><i>Special epileptiform patterns</i></b>	<b>309</b>
19.1	The infantile and juvenile patterns of hypsarrhythmia, slow spike-and-wave discharges and multifocal independent spikes	309
19.2	Periodic complexes	317
19.3	Ictal patterns without spikes and sharp waves	327
19.4	Epileptiform patterns without proven relation to seizures ('pseudo-epileptogenic patterns')	331
<b>20</b>	<b><i>Local slow waves</i></b>	<b>341</b>
20.1	Description of pattern	341



20.2	Clinical significance of local slow waves	344
20.3	Other EEG abnormalities associated with focal slow waves	347
20.4	Grading of focal slow waves	351
20.5	Mechanisms causing focal slow waves	351
20.6	Specific disorders causing local slow waves	352
21	<i>Generalized asynchronous slow waves</i>	359
21.1	Description of pattern	359
21.2	General clinical significance of generalized asynchronous slow waves	364
21.3	Other EEG abnormalities associated with generalized asynchronous slow waves	365
21.4	Grading of generalized asynchronous slow waves	365
21.5	Mechanisms causing generalized asynchronous slow waves	366
21.6	Specific disorders causing generalized asynchronous slow waves	366
22	<i>Bilaterally synchronous slow waves</i>	379
22.1	Description of pattern	381
22.2	Clinical significance of bisynchronous slow waves	386
22.3	Other EEG abnormalities associated with bisynchronous slow waves	387
22.4	Grading of bisynchronous slow waves	388
22.5	Mechanisms causing bisynchronous slow waves	388
22.6	Specific disorders causing bilaterally synchronous slow waves	389
23	<i>Localized and lateralized changes of amplitude: Asymmetries</i>	397
23.1	Description of pattern	397
23.2	Clinical significance of asymmetries	401
23.3	Other abnormalities associated with asymmetries	402
23.4	Grading of asymmetries	403
23.5	Mechanisms causing local changes of amplitude	403
23.6	Specific disorders causing asymmetries of amplitude	404
23.7	Asymmetries of alpha, beta, mu and other rhythms	410



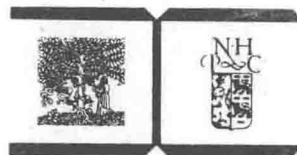
24	<i>Generalized changes of amplitude: Symmetrically high and low amplitude</i>	415
24.1	Description of patterns	418
24.2	Clinical significance of high and low amplitude	419
24.3	Other EEG abnormalities associated with high and low amplitude	420
24.4	Grading of amplitude changes	420
24.5	Mechanisms causing generalized changes of amplitude	420
24.6	Specific disorders causing a generalized decrease of amplitude of all types of activity	422
24.7	Generalized decrease or absence of alpha rhythm	427
24.8	Generalized increase of beta rhythm	428
24.9	Changes of amplitude of sleep patterns	429
25	<i>Deviations from normal patterns</i>	431
25.1	Abnormal frequency of alpha rhythm	431
25.2	Abnormal reactivity of alpha rhythm	437
25.3	Activity of alpha frequency in coma and seizures	438
25.4	Abnormal timing and incidence of sleep patterns	441
25.5	Abnormal photic responses	445
25.6	Immature patterns	449
26	<i>The EEG report</i>	451
26.1	Description of the record	452
26.2	EEG diagnosis	456
26.3	Clinical interpretation	457
	<i>Appendix</i>	461
	<i>Subject index</i>	463



# EEG Primer

**R. SPEHLMANN**

*Professor of Neurology  
Northwestern University Medical School  
Director of EEG Laboratories  
VA Lakeside Medical Center, Chicago  
and Evanston Hospital, Evanston, Illinois, U.S.A.*



1981

ELSEVIER/NORTH-HOLLAND BIOMEDICAL PRESS — AMSTERDAM • NEW YORK • OXFORD



**Part A**  
**Technical background**



## Introduction

The steps involved in recording an EEG are illustrated in Figure 0.1 and described in Chapters 1 to 7 comprising Part A of this text.

(1) *The Source* of the EEG are electrical potentials generated by nerve cells in the cerebral cortex in response to various kinds of input, including that from pace-makers of rhythmical activity in the depth of the brain. These fluctuating potentials summate and penetrate to the scalp where they can be recorded as the scalp EEG.

(2) *Recording electrodes* usually consist of small metal cups or discs which are attached to the scalp so that they make good mechanical and electrical contact. They cover the surface of the head at regular intervals.

(3) *The EEG machine* receives electrical input from the scalp electrodes which are connected to an input board. The cable of the input board terminates at the input selector switches which are used to select a pair of electrodes, or a calibration voltage, as the input of each recording channel. The input is connected to differential amplifiers which increase the size of the electrical potential differences between the two electrodes and reject interference simultaneously affecting both electrodes. High and low frequency filters are used to reduce the size of very slow and very fast potential changes and to emphasize clinically important electrical activity in the medium frequency range. A 60 Hz filter can eliminate the most common electrical interference in EEG recordings, namely that from power lines, if it cannot be eliminated by other means. The amplified electrical potentials are used to drive an ink pen, or other writing devices, up and down on chart paper which is pulled along at a constant speed.



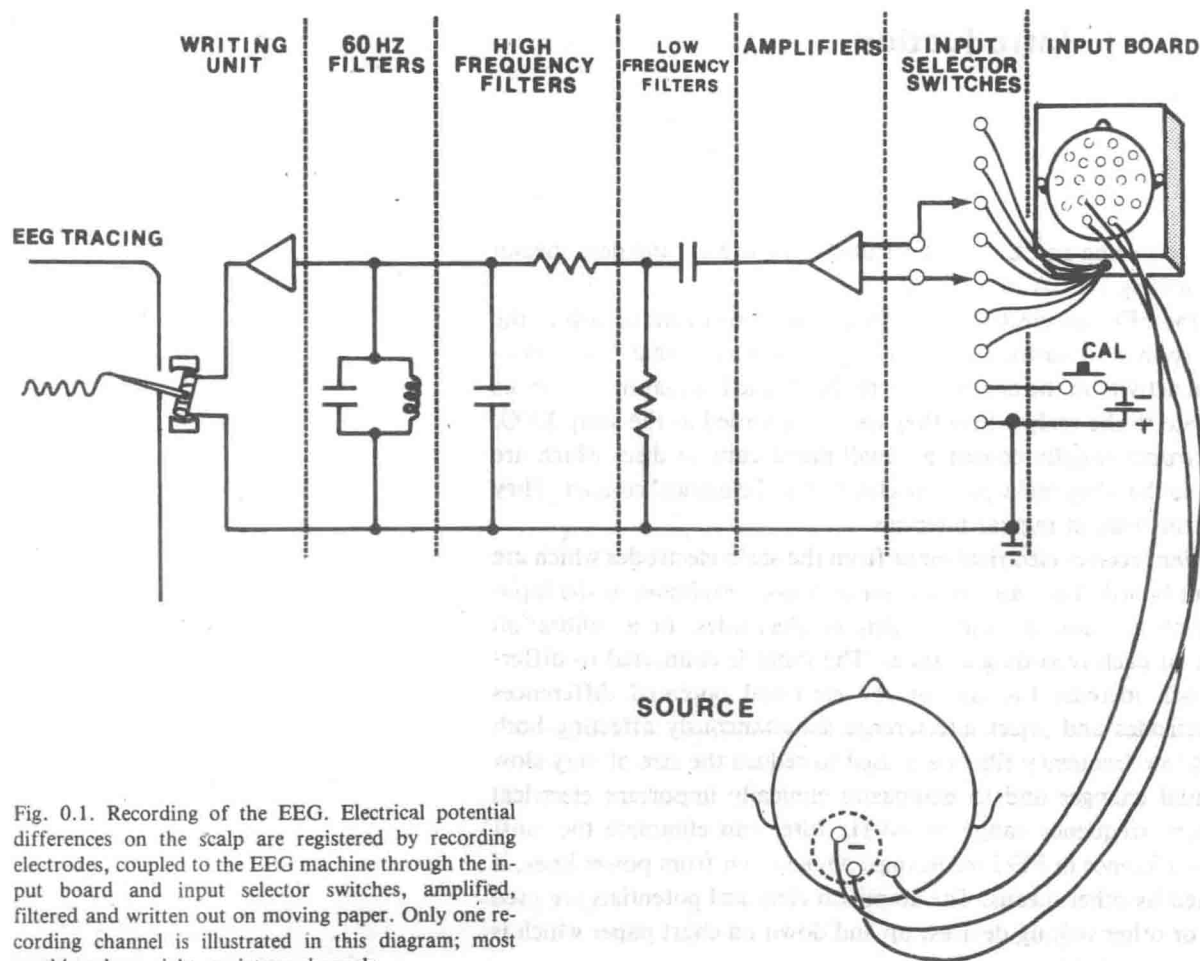


Fig. 0.1. Recording of the EEG. Electrical potential differences on the scalp are registered by recording electrodes, coupled to the EEG machine through the input board and input selector switches, amplified, filtered and written out on moving paper. Only one recording channel is illustrated in this diagram; most machines have eight or sixteen channels.



(4) *Recording strategy* uses several different combinations of electrodes, or montages, to display the potential changes from all parts of the head and to localize the origin of abnormal potential changes.

(5) *The product* of the recording, namely the clinical EEG record, must satisfy a number of technical requirements to be acceptable. Requirements for routine clinical recordings differ from those for recordings from infants and small children, for all-night sleep recordings, for recordings in cases of suspected cerebral death and for recordings transmitted by telephone.

(6) *Artifacts* are pen deflections that are not due to cerebral activity and may come from such extracerebral activity as eye movements, heart beat and muscle contraction or from electrical interference, malfunctioning recording electrodes, or defects of the EEG machine. They must be eliminated or clearly explained to avoid confusion with cerebral activity.

(7) *Other methods of recording and analyzing the EEG* use magnetic tape recorders, computers and other instruments to answer questions which cannot be answered by the conventional method of examining the pages of a paper record.