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TEXT-BOOK OF NERVOUS DISEASES

TEXT-BOOK
OF
NERVOUS DISEASES
FOR
PHYSICIANS AND STUDENTS
BY
PROFESSOR H. OPPENHEIM
OF BERLIN

FIFTH ENLARGED AND IMPROVED EDITION
WITH 432 ILLUSTRATIONS IN THE TEXT AND 8 PLATES

AUTHORISED TRANSLATION BY
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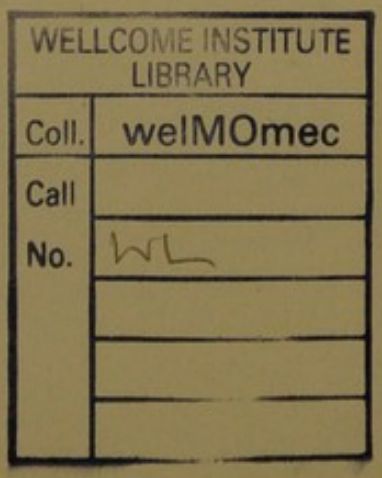

ERRATUM

Page 346, line 14.—*For* hill-climbers *read* miners.

TRANSLATOR'S PREFACE

THIS translation of the fifth edition of Oppenheim's "Text-book of Nervous Diseases" has been undertaken with the sanction and by agreement with its distinguished author, in consequence of suggestions made from various quarters that a work which holds such a high position in the estimation of those who can read it in its original form should be made more easily accessible to English and American readers. Every endeavour has been made to keep the translation as close as possible to the German text, and to reproduce the meaning of the text exactly and clearly. In conformity with the desire of the author, the work has not been in any way edited. The translator feels sure that the bibliographical references given in this edition have greatly enhanced the value of the work, and that their addition has more than justified the labour that they must have entailed.

The translator has to express his appreciation of the most valuable assistance and collaboration in the work of translation and in the reading of proofs given by Mrs H. M. Crowe, Dr Marian Erskine, Dr J. W. Dawson, Dr W. Kelman Macdonald, Dr J. H. Harvey Pirie, and Mr D. P. D. Wilkie, and also his thanks to Mr Otto Schulze for the great personal interest with which he has supervised the publication of the work.

EDINBURGH, *Dec.* 1910.

PREFACE TO THE FIFTH EDITION

The main change which this "Textbook of Nervous Diseases" has undergone in this new edition is that I have yielded to the urgent persuasion of the friends of the work and have determined, after much doubt and hesitation, to introduce references to the literature. In order that this should really enrich the matter and increase the value of the book, it has been necessary, in view of the enormous scope of our literature, to limit and select these references and to economise space in such a way as to make them at once concise, clear, and comprehensive. I have sought to attain this by quoting the *most recent* in addition to the most important and valuable articles, as well as the *monographs* and *résumés*, from which the reader can easily and conveniently trace his way back to the older literature. These references are indicated merely by brief and simple abbreviations. It is obvious in the nature of things that this bibliography can be neither complete nor of uniform value, and that I have had to reserve to myself absolute freedom of selection, without feeling bound to include a reference to every source. Many valuable papers have no doubt been passed over, but I have certainly erred rather in the direction of excess, and many of the references might have been omitted had it been in my power to read in the original those thousands of papers written in every language of the world.

The reader must therefore take the defects of this change in the work along with its advantages. I trust that as a whole it will prove to be a gain and to be worth the trouble which I have expended upon it.

I have also sought to enrich the subject-matter of the book by taking into consideration every advance that has been made in the study of nervous diseases and their treatment within the last four years. Although much that now seems to us to be well established may come to be disproved, it is well that we should ascertain the present state of our knowledge and science, and adapt our teaching to it.

In the meantime the Society of German Neurologists has been founded, and if the signs are not deceptive, the time is not far off when the hopes and desires expressed in the Preface to the earlier editions will be fulfilled.

H. Oppenheim.

Berlin, *July* 1908.

PREFACE TO THE FIRST EDITION

In submitting this "Textbook of Nervous Diseases" to my colleagues, present and future, I am deeply conscious of the responsibility which the step involves. I have tried as far as possible to choose from the great store of literature at my disposal only that which seems to be definitely established by investigation. But in addition to the inadequacy and imperfection of much of the matter which I have had to admit into the work, the limitations of my own experience have restricted me in regard to many points.

I have made it my chief aim to consider the requirements of the practitioner. For this reason I have devoted most of the space to symptomatology, diagnosis, prognosis, and treatment, giving to pathological anatomy only so much as is necessary to throw light upon the nature of the symptoms and to afford an aid to diagnosis. The account of the normal anatomy and physiology of the nervous system is perhaps somewhat concise, but at the same time it covers all the essential points and is illustrated by descriptive figures. I have decided to omit references to the literature, as had I given these the work would have extended beyond the desired limits. As a rule I have mentioned only the authors of the more important investigations, but I am well aware that I have not carried this out with absolute consistency.

The specialist will at once perceive that I have freely availed myself of the text-books and handbooks on neurology already published, and especially of the monographs which form the most valuable source of our knowledge, and that I have made use of many of the illustrations which they contain, but at the same time he will not fail to recognize the modest service rendered by my own experience and observation.

The most difficult point has been to deal with the therapy of nervous diseases and to find a firm footing on this uncertain ground. I have endeavoured to include in the sections on treatment all that has been recommended by our recognised specialists, as well as the methods which I have tried myself, and I hope I have succeeded in avoiding both excessive scepticism and the much more dangerous error of want of discrimination.

I cannot bring my work before the public without expressing my gratitude to those whom I must, in a certain sense, regard as its collaborators. When shortly after the death of my master, Westphal, I had to leave the congenial sphere of many years' work and to confine myself to the

duties of a polyclinic, it was the directing physicians of some of our hospitals who put me in a position to continue my clinical and anatomical studies. I am therefore deeply indebted to Professors Ewald and Langenbuch, to Dr Rotter, and above all to Dr Moses, Member of the Board of Health, who placed at my disposal the abundant sources of observation afforded by the Municipal Hospital.

I am also indebted to Fräulein v. Mayer, to Herr Krause, and to Dr Kroug, who amongst others have assisted me with their skill in the preparation of photographs and illustrations.

H. Oppenheim.

April 1894.

TABLE OF CONTENTS

FIRST VOLUME

I. GENERAL PART

Mode of Examination. General Symptomatology

	PAGE
Anamnesis	1
OBJECTIVE EXAMINATION	2
Examination of the Mental Condition	3
Examination of the Skull	4
Condition of the Muscles. Examination of Motility	5
Muscle Tonus	6
Examination of the Tendon-Reflexes	7
Examination of the Active Movements	12
The Function of the Muscles. Muscles of the Shoulder and Arm	13
The Muscles of the Pelvis and Lower Extremities	20
The Muscles which move the Head and Spinal Column	23
Disturbances of Co-ordination	26
Tremor	29
Electrical Examination	31
Morbid Changes of Electrical Excitability	38
Reaction of Degeneration	41
Examination of the Mechanical Excitability of the Muscles and Nerves	43
Examination of the Gait. Disturbances in Walking	44
Examination of the Sensibility	46
Affections of Sensibility	51
Cutaneous Reflexes	57
Spasm (Hyperkinesis)	62
Vasomotor, Trophic, and Secretory Disturbances	64
Examination of the Functions of the Special Sense Organs: Functional Disturbances of the Sensory Organs	69
Examinations relating to the Function of the other Cranial Nerves	76
Muscles which move the Eyeball	76
The Function of the Internal Ocular Muscles	80
The Sensibility of the Face	85
The Masseter Muscles	86
The Muscles of the Tongue	86
The Muscles of the Palate and Pharynx. The Act of Swallowing and its Impairment	88
The Muscles of the Larynx. Their Functional Disturbance	89
Disturbances of Speech	91

II. SPECIAL PART

First Section

Diseases of the Spinal Cord

PAGE

On the Anatomy, Physiology, and General Pathology of the Spinal Cord . . . 95
Secondary Degeneration 121
Localisation in the Spinal Cord 125
Unilateral Lesion of the Spinal Cord, Brown-Séquard Paralysis 136
The Vessels of the Spinal Cord 141
Classification of Spinal Cord Diseases 142

The Systemic Diseases of the Spinal Cord

Tabes Dorsalis (Locomotor Ataxia) 142
Spastic Spinal Paralysis (Lateral Sclerosis) 176
Congenital or early acquired Spastic Paraparesis, Little's Disease 181
Combined Diseases of the Posterior and Lateral Columns of the Spinal Cord . . 187
Hereditary Ataxia (Friedreich's Disease) 194
Acute Anterior Poliomyelitis; Acute Atrophic Spinal Paralysis; Spinal Infantile Paralysis 199
Acute Anterior Poliomyelitis of Adults 218
Subacute and Chronic Anterior Poliomyelitis 220
Amyotrophic Lateral Sclerosis 225
Progressive Muscular Atrophy 231
Spinal Form of Progressive Muscular Atrophy, Amyotrophia Spinalis Progressiva . 231
Appendix: Hereditary or Familial Form of Progressive Muscular Atrophy of Spinal Origin 237
Primary Myopathy, Progressive Muscular Dystrophy 238
Neurotic or Neural Form of Progressive Muscular Atrophy (Peroneal Type, Type of Charcot-Marie and Tooth) 252
Appendix: Thomsen's Disease (Myotonia Congenita) 255

Diffuse Diseases of the Spinal Cord

A. Spinal Cord Affections caused by Diseases of the Vertebræ

Dislocation and Fracture of the Spinal Column 261
Appendix: Symptoms caused by Shooting and Stabbing Injuries of the Spinal Column 273
Caries of the Spinal Column, Tubercular Spondylitis (and other Forms of Spondylitis) 273
Carcinoma and other Tumours of the Vertebræ 289
Syphilitic Diseases of the Vertebræ 293
Arthritis Deformans and Allied Chronic Articular Affections of the Spinal Column . 294

B. Diseases of the Spinal Cord due to Meningeal Affections

Spinal Meningitis 298
Chronic Spinal Meningitis 300
Hypertrophic Cervical Pachymeningitis 301
Chronic Syphilitic Meningitis (Arachnitis Gummosa, Syphilitic Meningo-myelitis, etc.), and other Spinal Cord Diseases of Syphilitic Origin 304

C. Primary Diffuse Diseases of the Spinal Cord

Myelitis 315
Appendix: Senile Paraplegia 331
Disseminated Sclerosis, Sclerose Multiplex Cerebro-Spinalis 332
Abscess of the Spinal Cord 350
Hæmatomyelia; Apoplexia Medullæ Spinalis 352

PAGE

Diseases of the Spinal Cord produced by Great Variations of Atmospheric Pressure (Diver's Disease, Caisson Disease) 357
Tumours of the Spinal Cord 358
Spinal Gliosis and Syringomyelia 376
Appendix: Diseases of the Cauda Equina (and Conus Medullaris) . . 387

Diseases of the Spinal Cord without any known Pathological Lesion 395

Concussion of the Spinal Cord 395

Appendix

I. Congenital Malformations of the Spinal Cord and its Membranes 396
II. Reflex Paralyses; Exhaustion Paralyses; Periodic Paralysis of the Extremities; Osteomalacic Paralysis 399

Second Section

Diseases of the Peripheral Nerves

Anatomy of the Peripheral Nerves 402

Peripheral Paralysis

Traumatic Paralysis of the Peripheral Nerves 404
Neuritis; Inflammation of the Nerves 417

Peripheral Paralysis of Spinal Nerves

Paralysis of the Phrenic Nerve 423

Paralysis of the Brachial Plexus. Combined Peripheral Paralysis of the Nerves of the Shoulder and Arm 425

The (Duchenne) Erb Paralysis of the Shoulder 427
Paralysis of the Inferior Plexus (Klumpke) 431
Total Plexus Paralysis 432
Obstetrical Paralysis 435

Paralysis of Individual Nerves of the Shoulder and Arm . . 439

Paralysis of the Long Thoracic Nerve 439
,, ,, Circumflex Nerve 440
,, ,, Musculo-Cutaneous Nerve 442
,, ,, Suprascapular Nerve 443
,, ,, Dorsalis Scapulæ Nerve 443
,, ,, Musculo-Spiral Nerve 444
,, ,, Median Nerve 450
,, ,, Ulnar Nerve 453

Peripheral Paralysis of the Intercostal Nerves 457

Peripheral Paralysis of the Nerves of the Lower Extremities . 457

Paralysis of the Crural and Obturator Nerves 458
,, ,, External Cutaneous Femoris Nerve 459
,, ,, Sciatic Nerve 460
,, ,, Peroneal Nerve 462
,, ,, Posterior Tibial Nerve 464
,, ,, Gluteal Nerve 464

Peripheral Paralysis of the Cranial Nerves 465

Paralysis of the Nerves of the Ocular Muscles 465
Periodic Paralysis of the Oculo-motor Nerve 472

PAGE
Paralysis of the Trigeminal Nerve 474
Facial Paralysis (Prosopoplegia) 481
Primary and Isolated Peripheral Diseases of the Auditory Nerve 493
Paralysis of the Glosso-Pharyngeal Nerve 495
Paralysis of the Vagus Nerve 497
Paralysis of the Spinal Accessory Nerve 505
Paralysis of the Hypoglossal Nerve 507

MULTIPLE NEURITIS (POLYNEURITIS) 509

Alcoholic Neuritis 511
Lead Paralysis 517
Arsenical Paralysis 520
Diphtheritic Paralysis 522
Typhoid Paralysis 526
Puerperal Neuritis 527
Gonorrhœal Neuritis 527
Tubercular Neuritis 527
Diabetic Neuritis 528
Senile Neuritis 528

APPENDIX

I. Landry's Paralysis; Acute Ascending Paralysis 536
II. Acute and Chronic Polymyositis 542
III. Occupation Pareses; Professional Pareses 548

THE NEURALGIAS 550

Neuralgia of the Trigeminal Nerve (Tic douloureux, Prosopalgia etc.) . . . 561
Occipital Neuralgia 567
Neuralgia of the Phrenic Nerve 569
Brachial Neuralgia 569
Intercostal Neuralgia 572
APPENDIX: Herpes Zoster 574
Neuralgia of the Mammæ; Mastodynia; Irritable Breast 577
Lumbar Neuralgia 578
Sciatic Neuralgia; Sciatica 580
Intermittent Claudication 586
Pudendo-Hæmorrhoidal Neuralgia 594
Neuralgia of the Coccygeal Plexus; Coccygodynia 595
Visceral Neuralgia 596

TUMOURS OF THE NERVES 596

SECOND VOLUME

THIRD SECTION

Diseases of the Brain

ANATOMY AND PHYSIOLOGY OF THE BRAIN. CEREBRAL LOCALISATION

The Cerebral Membranes 605
The Convulsions and Fissures of the Cerebral Surface 608
The Relation of the Skull to the Cerebral Surface 611
Histological Structure of the Cerebral Cortex 614
Localisation in the Cerebral Cortex 618
Motor Tracts 632
Optic Tract 647
Central Ganglia 650

PAGE

Origin of the Cranial Nerves 652
Vascular Supply of the Brain 667
Secondary Degeneration in the Brain 670

General Symptomatology of Brain Diseases

I. General Symptoms 671
II. Focal Symptoms 677
Motor Localising Symptoms 677
A. Symptoms of Irritation 677
B. Symptoms of Paralysis 680
Mind Paralysis; Apraxia 681
Hemiplegia 684
Contracture 686
Post-hemiplegic Motor Disturbances (Hemichorea, Hemiathetosis) 690
Associated Movements 695
Hemiplegia Alternans 697
Conjugate Deviation, etc. 698
Nystagmus 701
Disturbances of Sensibility 703
Visual Disturbances 711
Affections of Speech 725

Diseases of the Cerebral Meninges

Inflammation of the Dura Mater (Internal Hæmorrhagic Pachymeningitis, Hæmatoma of the Dura Mater) 743
Acute Inflammation of the Lepto-Meninges. Meningitis or Acute (Purulent) Cerebral Leptomeningitis 752
Lumbar Puncture 769
Epidemic Cerebro-Spinal Meningitis 770
Tubercular Meningitis 778
Appendix: Chronic Meningitis 786

Disturbances of the Cerebral Circulation 787

Cerebral Anæmia 787
Cerebral Hyperæmia 789

Diseases of the Brain Substance

Cerebral Hæmorrhage 792
Cerebral Softening (Encephalomalacia) 813
Chronic Progressive Cerebral Softening 824
Encephalitis (acute, non-purulent) 825
1. Acute, Hæmorrhagic Encephalitis 825
2. Acute, Hæmorrhagic Superior Poliencephalitis 833
Infantile Cerebral Paralysis 837
Appendix: Familial Spastic Paralysis and Amaurotic Idiocy . . . 854
Cerebral Abscess 856
Thrombosis of the Cerebral Sinuses 877
Cerebral Tumour 887
Aneurism of the Cerebral Arteries 934
Parasites of the Brain 938
Hydrocephalus 947
Acquired Hydrocephalus (Serous Meningitis) 953
Syphilitic Diseases of the Brain 960
Progressive Paralysis of the Insane (Paralytic Dementia) 978
Appendix 995

Diseases of the Pons and Medulla Oblongata

PAGE
Anatomy and Physiology 997
Progressive Bulbar Paralysis (Progressive Glosso-Pharyngo-Gabial Paralysis) . . 1004
The Infantile (Hereditary, Family) Form of Progressive Bulbar Paralysis . . . 1010
Acute (Apoplectic) Bulbar Paralysis 1011
Acute Bulbar Neuritis 1019
Bulbar Paralysis from Compression 1019
Pseudo-Bulbar Paralysis and Cerebro-Bulbar Glosso-Pharyngo-Labial Paralysis . 1021
Infantile Form of Pseudo-Bulbar Paralysis 1028
Bulbar Paralysis without Anatomical Lesions, or Myasthenic Paralysis . . . 1029
Ophthalmoplegia 1038

Diseases of the Cerebellum 1043

Fourth Section

The Neuroses

Hysteria 1053
Appendix: Hypnotism and Hypnosis 1109
Neurasthenia or Nervous Weakness 1111

Appendix

Morbid Conditions which, as a Rule, accompany Hysteria and Neurasthenia, but which occasionally occur as an Independent Disease:
Conditions of Anxiety: Phobias 1147
Imperative Ideas 1151
Astasia-Abasia 1159
Akinesia Algera 1160
Traumatic Neuroses 1162
Hemicrania (Migraine) 1180
Headache (Cephalalgia) 1189
Vertigo 1193
Glossodynia 1200
Epilepsy 1201
Eclampsia 1233
1. Eclampsia 1233
2. Eclampsia Gravidarum 1235

Localised Muscular Spasms

Facial Spasm, Convulsive Tic 1237
Spasm of the Muscles of Mastication 1244
,, ,, Tongue; Glossospasm 1245
Spasm in the Glosso-Pharyngeus; Pharyngismus 1246
,, ,, Muscles of the Neck 1246
,, ,, Muscles of the Trunk and Extremities 1256
Respiratory Spasm 1257
Saltatory Reflex Spasm 1258
Localised and General Tic (Maladie des Tics) 1259
Paramyoclonus Multiplex (Friedreich), Myoclonia, Polyclonia 1264
Occupation Spasms 1268
Writer's Cramp (Graphospasmus, Mogigraphia) 1268
Other Occupation Neuroses 1272
Tetany 1273
Chorea Minor (St Vitus's Dance) 1284
Other Forms of Chorea. Hereditary Chorea (Chronic Progressive Chorea, Huntington's Disease, Choreic Dementia) 1295
Electric Chorea 1297
Paralysis Agitans (Parkinson's Disease) 1298

Fifth Section

Diseases of the Sympathetic. Angioneuroses and Trophoneuroses

PAGE

Anatomy and Physiology 1311
Paralysis of the Sympathetic, etc. 1316

Angioneuroses (and Trophoneuroses) 1319
Vasomotor Neurosis of the Extremities (Nothnagel). Acroparæsthesia (Schultze) . 1320
Acute Circumscribed Œdema of the Skin: Hydrops Hypostrophos . . . 1322
Intermittent Articular Dropsy 1325
Symmetrical Gangrene (Symmetrical Local Asphyxia, Raynaud's Disease) . . 1326
Erythromelalgia 1332
Sclerodermia 1335

Progressive Facial Hemiatrophy 1338
Exophthalmic Goitre (Basedow's Disease, Graves' Disease) 1342
Myxœdema, Pachydermic Cachexia 1360
Acromegaly, Pachyakria 1371

Sixth Section

Conditions of Intoxication in which the Nervous System is chiefly affected

Alcoholism 1381
Morphinomania 1388

Appendix

Tetanus 1392
Cephalic Tetanus; Hydrophobic, Paralytic, and Bulbar Tetanus 1397

Index 1399

LIST OF ABBREVIATIONS

A. f. P. . .	Archiv für Psychiatrie und Nervenkrankheiten.
M. f. P. . .	Monatsschrift für Psychiatrie und Neurologie.
Z. f. N. . .	Zeitschrift für Nervenheilkunde.
Z. f. P. . .	Zeitschrift für Psychiatrie.
N. C. . . .	Neurologisches Zentralblatt.
C. f. N. . .	Zentralblatt für Nervenheilkunde.
R. n. . . .	Revue neurologique.
R. of N. . .	Review of Neurology and Psychiatry.
C. f. Gr. . .	Zentralblatt für die Grenzgebiete der Medizin und Chirurgie.
M. aus d. Gr. .	Mitteilungen aus den Grenzgebieten, etc.
B. k. W. . .	Berliner klinische Wochenschrift.
D. m. W. . .	Deutsche medizinische Wochenschrift.
M. m. W. . .	Münchener medizinische Wochenschrift.
W. m. W. . .	Wiener medizinische Wochenschrift.
W. kl. R. . .	Wiener klinischer Rundschau.
W. m. Pr. . .	Wiener medizinische Presse.
J. f. P. . .	Journal für Psychologie und Neurologie.
Jahrb. f. P. . .	Jahrbücher für Psychiatrie, etc.
V. A. . . .	Virchows Archiv.
Br.	Brain.
Jour. Nerv. and Ment. Dis. .	Journal of Nervous and Mental Disease.
Z. f. kl. M. . .	Zeitschrift für klinische Medizin.
A. f. A. . .	Archiv für Anatomie und Physiologie.
A. f. kl. M. . .	Archiv für klinische Medizin.
A. f. kl. Chir. .	Archiv für klinische Chirurgie.
Z. f. kl. Ch. .	Zeitschrift für klinische Chirurgie.
Obersteiner. .	Arbeiten aus dem Institut für Anatomie, etc.
M. f. Ohr. . .	Monatsschrift für Ohrenheilkunde.
Z. f. Ohr. . .	Zeitschrift für Ohrenheilkunde.
Kl. M. f. Aug. .	Klinische Monatsblätter für Augenheilkunde.
M. f. U. . .	Monatsschrift für Unfallheilkunde.

The other abbreviations are in general use, or quite clear in their meaning.

I. GENERAL PART

METHOD OF EXAMINATION. GENERAL SYMPTOMATOLOGY

Anamnesis (History of the case).—Great care must be devoted to the anamnesis. Many facts which are of great service in the diagnosis of a nervous disease are of no importance in the patient's eyes, and would be disregarded by him if his attention were not specially directed to them. The first question should be in regard to *heredity*. A person has a neuropathic heredity when his ancestors and relations have suffered or suffer from nervous diseases. The existence of psychoses, of epilepsy, hysteria, neurasthenia, and hemicrania in the family should specially be considered. The neuropathic family disposition may be revealed by morbid tendencies and impulses (suicidal impulse, alcoholism, etc.) in several of its members. Besides alcoholism, chronic lead poisoning, gout, and tuberculosis in the ancestors may cause a disposition to nervous diseases in the descendants. Finally, blood-relationship of the parents is a very important factor.[1]

Questions as to the origin of the disease should be preceded by close investigation into the *previous history*. It should be first ascertained whether a disposition to nervous disease had become evident in youth, or whether spasms, attacks of fainting, mental disturbances, headache (especially migraine), attacks of vertigo, or gastric disturbances have been present at an earlier period of life. Further important questions are :

Have there been any *infective diseases* ? Acute as well as chronic infective disease may lay the foundation for nervous diseases, which may follow them directly or after an interval of weeks, months, or years. Amongst infective diseases, typhoid, smallpox, diphtheria, scarlatina, measles, and influenza are specially apt to affect the nervous system. The relations between tuberculosis and nervous diseases are manifold. But the greatest difficulties of all are presented by the investigation with regard to previous venereal diseases, and especially with regard to *syphilis*. Then, further, it has to be discovered whether the patient is now or has at any time been given to the abuse of alcohol. The misuse of other poisons (morphia, cocaine, chloral hydrate) must also be borne in mind in the examination. The patient's occupation or trade may give occasion to

[1] The heredity is *similar* when the descendants are affected with the same disease as their parents ; in other cases it is *dissimilar*, or polymorphous.

The *neuropathic disposition* does not always coincide with the *neuropathic heredity*, in so far that an abnormal constitution of the nervous system may exist from birth, although there is no hereditary transmission.

absorption of poisons, and it is of great importance to ascertain whether the patient has in this or any other way been working for a time with lead, arsenic, mercury, copper, brass, bisulphide of carbon, etc.

Amongst possible anomalies of the *sexual life*, the physician must direct his attention to masturbation, perverse impulses, etc.

As an important part is played in the etiology of nervous diseases by *injuries*, *mental over-strain*, and especially by *emotional excitement*, great care must be devoted to these factors. Consideration of the psychical life and of everything that has acted upon the mind of the patient is in very many cases the most certain way by which to determine the nature of the existing disease.

These questions disposed of, the origin of the disease and its *subjective symptoms* have to be most thoroughly investigated. In this relation it should be remembered that many nervous diseases are characterised by a development in successive stages, the various phases being separated from each other by intervals of time which are frequently of long duration. The patient knows nothing of the connection ; he may know that he once suffered from some ocular or abdominal trouble ; but he has no idea that these conditions have a close relation to the present nervous disease, so that he only gives the information in response to special inquiries.

One should make it a rule to devote the fullest attention to the subjective symptoms. A lung disease, a heart affection can usually be diagnosed by objective examination, sometimes without any regard to the subjective sensations. The neurologist has often enough to deal exclusively with morbid sensations and thoughts, whilst the methods of physical examination may fail to give him any assistance. Therefore he must very closely determine what the patient feels, what sensations are troubling him, what symptoms he himself is conscious of. For this, time and patience are often necessary—but they are absolutely essential.

Objective Examination

It is obvious that an examination which is directed exclusively to the functions of the nervous system is incomplete. The danger of falling into this error is particularly great as regards the specialist, and he should therefore make it a rule to precede or to follow the investigation of the nervous system by a general physical examination. Neglect of this principle may lead to serious mistakes.

A study of the patient himself whilst the anamnesis is being ascertained may often lead to valuable results. The facial expression not infrequently reveals any morbid mental condition, or diminution of intelligence, that may be present. The bearing during the description of his disease, the tendency to emotional outbreaks, the violent gesticulation, the general motor restlessness, a tremor in the facial muscles, in the extremities, a rapid change of colour in the face, a frightened start—all these are signs which must be considered and which may confirm the diagnosis.

It is not necessary to follow a definite method of examination, but the beginner is advised to do so. One should first ascertain the general condition of nutrition and the state of the blood, so far as is possible from the colour of the skin and mucous membranes.

Examination of the Mental Condition.—Disturbances of the mental life play so prominent a part in the symptomatology of nervous diseases that the condition of the mind must be taken into account in every case. The study of psychiatry is a necessary preparation for the understanding of the majority of nervous diseases. There is room here for only a few general indications.

In order to form an opinion as to anomalies of perception of ideas and of will, a prolonged observation is, as a rule, necessary. The more marked disturbances may, of course, often be recognised during the first examination. The expression of the face sometimes reveals the morbid state of mind as well as the abrupt change of mood. Sensory hallucinations and delusions may give a characteristic impression to the features, the attitude, and the whole bearing. The examining physician will have an opportunity in his conversation with the patient of gaining information as to his ideas and thoughts, the power of his memory, his ability to reason and to draw conclusions. It requires both experience and tact to make him responsive, and not to confuse and put him out of humour by an awkward, rough, or inconsiderate examination. Great care is especially requisite where it is a case of discovering fixed hallucinations. One should not go direct to the goal; but one must endeavour, while talking on apparently indifferent matters, to lead the conversation as if by accident to the desired subject. The patient must have every confidence. Otherwise a question touching his sore point may at once make him abnormally excitable or excited. The description of the illness, the life history usually afford an opportunity of learning to know the patient from his emotional side. When this has not been the case, enquiry as to his previous life and its vicissitudes, his own conception of his illness and its outcome, will quickly lead to the object. In *testing the intelligence*, the degree of education, the measure of acquired knowledge should be taken into consideration. The more marked symptoms are revealed in ordinary conversation. One can generally infer from the patient's account of the history of his illness whether his memory is enfeebled or not. In other cases one should inquire whether the patient remembers events and the order in which they occurred, which should have made a strong impression upon his memory, according to his degree of education and his social position. Historical and political facts should be employed, but above all the patient's own personal experiences. It is important to distinguish between the memory for long past events and that for the most recent occurrences. Thus questions as to the events of the day before will reveal severe disturbances, whilst the patient's memories reach back to his childhood. It is also advisable to ascertain whether the patient receives fresh impressions into his memory and can reproduce them, whether he can repeat a number of figures, names, and so on (Wernicke's *Merkfähigkeit*—power of retention). The definition of simple ideas, the attempt to explain them by examples: "what is brave, modest, etc.? what is the opposite of brave, modest, sober, etc.?"—such questions and tests are very well adapted to reveal the intellectual capacity, at least in cases where there are marked defects to be demonstrated. It is specially useful to test whether he can work with figures as well as formerly. Ask him to add or to multiply one or more figures mentally, and notice not only whether the solution is correct, but also whether the mental work is accomplished with the usual rapidity. Naturally one must

know how far the patient could manipulate figures in his healthy days. When dealing with a patient of a low degree of education, let him give a description of his occupation, his mode of life. The answers relating to day and date, to age, year of marriage, to the number and names of his children, etc., will often quickly reveal marked defects of intelligence and memory.

As to the other disturbances of the thought-processes (inhibition of thought, flight of ideas, incoherence, confusion, etc.) they do not require to be discussed here, but should be thoroughly studied in the text-books on psychiatry.[1]

We have frequently to rely upon the information of the relatives in order to draw conclusions as to alteration of character, morbid desires and actions.

It is specially valuable to procure the patient's history in writing. Many a one who, from his emotional state or his shyness, gives but an incomplete history, can easily furnish a detailed description of his disease and his sensations in writing. Comparison of his writing at the time of his illness with that of an earlier period may give valuable results, and it is not only the alteration of the thought-content, but that also of the style and the character of the writing which have a diagnostic significance.

Examination of the Skull.—Inspection and palpation of the skull may supply information as to the presence of scars, exostoses, etc. An abnormal circumference of the skull as well as other anomalies of formation quickly reveal themselves to the experienced eye, but it is to be recommended that these conditions be ascertained by measurement. The greatest circumference of the skull at the level of the external occipital protuberance and glabella amounts in adults of the male sex to about 56 (about 22 in.), in women 50 cm. (20 in.), in the new-born to between 35 and 40 (or about 14-16 in.), and in the course of the first year to about 45 (18 in.), and in the twelfth year to about 50 cm. (20 in.). Longitudinal measurement from the root of the nose to the occipital portuberance is in man about 35 cm. (14 in.). Measurement with callipers is not usually necessary for our purpose.

Great asymmetry of the skull, unusual size (macrocephaly) or smallness (microcephaly), marked disproportion between the facial and the cerebral portion of the skull, excessive protuberance of the jaws, especially of the under-jaw, so that the lower row of teeth protrude beyond the upper (prognathism)—these symptoms are classified as *signs of degeneration* (Stigmata hereditatis). Amongst the signs may be also mentioned developmental anomalies of the ear : absence or adherence of the lobule, anomalies of the helix, absence of helix or antihelix, large projecting ears with low margins and flat fossa (Morel's ear), the Darwin ear (characterised by the marked development of the so-called Darwin's tubercle), the Wildermuth ear with salient antihelix, the handle-like ear, etc. Hare-lip, cleft-palate, narrow, scaphoid arched palate, obliquely projecting teeth, retinitis pigmentosa, medullated nerve-fibres on the papilla of the optic nerves, albinism, and many other anomalies of formation (polydactyly, syndactyly, polymastia, hypospadias, etc.) are all regarded as anatomical

[1] We cannot here describe the fine methods of examination which have been instituted by Kraepelin and his school, by Sommer and others. We must also forego the description of the apparatus for psychological measurements.

signs of neuropathic and psychopathic diatheses. Too much weight should not, however, be laid upon these, as almost any one of them may occasionally occur in otherwise thoroughly healthy individuals.

Percussion of the skull may give important indications in local disease of the brain and its membranes. Neither should *auscultation* be neglected, since vascular murmurs are more frequent in brain diseases than is generally recognised.

The use of Röntgen rays may be of value in examination of the skull, but hitherto it has given positive results only in a few diseases (see specially the section on cerebral tumour and acromegaly).

Condition of the Muscles. Examination of Motility

A pronounced increase or decrease in the size of the muscles when confined to one side of the body, to one extremity or segment of a limb, is recognised at the first glance, but the conclusion formed from comparison of the corresponding muscles and muscular groups of the two sides may be extremely difficult when the differences are but slight. In making this examination care should be taken that the two extremities are in exactly the same position and that the muscles are equally contracted or relaxed. It should also be remembered that slight differences in the size of the muscles of the two sides of the body, especially an excess in those of the limbs of the right side of about 1 cm. ($\frac{1}{2}$ in.), occur even under normal conditions.

In order to determine the degree of atrophy, careful *measurements* with a tape are necessary. The position of the limbs must, of course, be similar. In the forearm and leg the thickest part of the limb should be selected. In the upper arm the middle part should be chosen ; in the thigh one should start from a fixed point and determine the size at about 12 to 15 cm. (or 5 to 6 in.) above the patella. Measurements are best made when the extremities are extended, but they should be held out, as differences may be marked by unequal pressure when they are supported. Even measurement is no exact evidence. Small faults creep in which may even amount to $\frac{1}{2}$ to 1 cm. Metal tapes should be preferred. Muscular atrophy is also revealed by the formation of grooves, hollows, cavities at places which were formerly filled by muscular tissue, whilst hypertrophy is sometimes characterised by an equal increase of the circumference of the whole muscle, sometimes by the formation of swelling in certain segments. It should always be remembered that changes in the size of the muscle by no means reveal every condition of degeneration, and that a muscle which has a normal appearance may be as severely affected as a hypertrophied muscle.

Palpation does not furnish certain evidence with regard to the conditions of the muscular substance. The degenerated muscle is felt to be soft and even " pulpy," but when the change is a fibrous one, it appears to the palpating finger to be hard and firm. If the muscle is much overgrown by fat or wholly changed into fatty tissue, it is felt to be doughy. But much experience is necessary to enable one to discern such anomalies by palpation. On the other hand, one is liable to many mistakes in this examination. *Excision* of small particles of muscle cannot be employed in practice for diagnostic purposes, and even as regards scientific investigation this method has somewhat gone out of vogue, since it has

been found that excision in itself may cause changes in the muscular tissue.

The most important and exact method of testing the nutritive condition of the muscles is *electrical examination*, which, without doubt, forms one of the most valuable aids to diagnosis (see below).

Muscle Tonus.—Before passing on to test the power of active movement, it is well to form an opinion as to the condition of tonus of the muscles, since changes in it materially influence active motor power.

Muscle tonus may be exaggerated or diminished. We find this from *examination of the power of passive movement* and of the *tendon phenomena.*

In order to carry out passive movements, we take hold of the extremities of the patient and endeavour to move them at each separate joint in every direction and to the maximum extent, where the mechanical condition of the joints permit of movement. This offers no difficulty when the muscle tonus is normal and there is no muscular tension to be overcome. Exaggeration of muscle tonus does indeed, even under physiological conditions, follow passive movement, *i.e.* there is a reflex increase of tension from the associated stretching of the muscle (Westphal, Hering, Foerster), but this is so slight that a palpable resistance only sets in when the passive movement is at its ultimate limit. Further, pathological tension may be simulated when the patient stretches the muscle actively, either intentionally or, as is usually the case, unconsciously—from awkwardness caused by ignorance of what he should do, or from anxious expectation and excitement. He should be instructed to leave his limb, entirely to the examiner and to avoid all tension. He should be taught that relaxing of the tension subjects the passively raised limb to the force of gravity, so that it falls down, etc. When this does not succeed, his attention should be engaged in other directions, by occupying his mind, setting him a mental sum, etc. It is only when the passively lifted limb falls by its own gravity back on to the bed that this disturbing factor can be assumed to be set aside and the further results of the examination duly taken into consideration.

Pathological Conditions of Tension, Hypertonus of the muscles is, firstly, revealed by *difficulty of passive movements.* When the hypertonus is great, the difficulty is felt in every attempt at movement. If we try to abduct the leg at the hip-joint, tension of the adductors at once becomes visible and palpable—it is not the leg that is moved outwards, but the whole pelvis. A certain, often a great, effort of strength is required on the part of the patient to overcome this tension, and as soon as he relaxes it, the extremity returns to its original position. The same resistance caused by the examination makes itself felt in the other joints, and opposes a more or less serious obstacle to passive movement.

As a rule we have not to deal with these most marked degrees, but with a slighter increase of muscle tonus, which becomes manifested by a reflex increase during the passive movement. In such cases passive movements if executed *slowly* can be carried to their full extent and without special resistance. But as soon as the attempt is made more abruptly, and the limb moved rapidly and forcibly, muscular tension appears. This tension becomes marked, especially in abduction of the leg at the hip or its extension at the knee-joint. Attention should be specially paid to this at the commencement of the examination, as it usually diminishes and there is no further opposition to the movement.

Exaggeration of muscular tonus (stiffness, rigidity or spastic condition of the muscles) can be recognised, secondly, from *exaggeration of the tendon phenomena*. On account of the close, though not absolute and constant, relation of the tendon phenomena to the muscle tonus, it is advisable to examine them directly after examination of the passive mobility of the limb.

Examination of the Tendon Phenomena.—By this symptom, which Westphal [1] and Erb [2] described, independently of each other, in 1875, we understand muscular contractions which are induced by a mechanical stimulation of a tendon. The most important of these is the knee jerk (also called the patellar phenomena or reflex). This is always present in healthy persons, and its absence has great diagnostic singificance.

Erb regards this symptom as a simple reflex. Westphal held that the knee jerk depends on a direct excitation of the muscle which is maintained in a state of tonus through reflex influences.

Results of later investigation favour the reflex-theory (Sternberg, Jendrássik, Scheven and others). Recently several writers have given their support to Westphal's view (Muskens, Sherrington).

In order to produce the knee jerk, the following method is the best : the leg of the patient, who lies on his back, is bared, placed at an obtuse angle and supported by the left hand of the examiner at the popliteal space, the heel resting on the bed. The right hand first feels for the patellar tendon, if it does not at once stand out distinctly, and then gives it a sharp tap with the percussion hammer. The thigh should first be watched to ascertain whether the extensor cruris muscle becomes tense. The effect of this tension is, when it is sufficiently strong, an extension of the leg. But it is advisable to place less weight on this factor (as it does not always appear) than on the muscular contraction. This should not be confused with a simple vibration of the skin and muscle substance, which is directly communicated to them from the tendon.

If the tap on the tendon has not elicited the contraction, then we should discover whether there is not an active or unconscious voluntary muscular tension. This will be rapidly recognised if the supported leg does not fall back to the bed immediately on the supporting hand being taken away. Relaxation of the tension should then be induced according to the rules given above, the examination being modified by allowing the patient to cross one leg over the other. If this has not the desired effect, then Jendrássik's method may be used : the patient is asked to fold his hands and at a word to make a forcible effort as if to pull them asunder, without, however, really doing so. At that moment the tendon is tapped. If this does not succeed in eliciting the knee jerk then we should examine the patient in a sitting position, which is best done if he sits on a table and allows his legs to hang free.

It has also been proposed that during examination of the knee jerks other stimulations should be applied, such as the prick of a needle, the throwing of a bright light on the retina. These act not only by distracting the attention of the patient, but apparently also by increasing the nerve conduction. It is further recommended that the patient should breathe deeply, read aloud, etc., during the examination. I have, however, always been able to dispense with these and similar procedures.

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[1] *A. f. P.*, Bd. iv. and Bd. v.

[2] *A. f. P.*, Bd. v. See also specially the monograph of Sternberg, " Die Sehnenreflexe, etc.," Leipzig u. Wien, 1893 ; also Scheven, *A. f. d. g. Phys.*, 117.

If one is making a rapid examination he may begin with the patient seated on a chair, the leg being so placed as to form a right or obtuse angle with the thigh.[1] But absence of the contraction in examination of this kind is never a proof that the knee jerk is really abolished, and a further examination with the patient in the recumbent position is always requisite.

Exaggeration of the knee jerk is shown by the fact that a very feeble blow, even a tap with the finger, elicits a marked, or a very strong contraction, or by the fact—and this is a more reliable sign—that the single contraction is replaced by a number of contractions or clonus of the quadriceps muscle. Much less frequently there is a tonic contraction of the muscle. Exaggeration may occasionally be demonstrated in another way: one may grasp the patella from above with the thumb and forefinger, then push it downwards with a sudden jerk. If one then endeavours to keep it in this position, but with a slightly reduced pressure, clonic contractions will appear in the muscle, which will only cease when the patellar tendon is allowed to return to its position of rest. This sign, known as *patellar clonus*, is, however, very inconstant.

A muscular contraction can also, as a rule, be elicited from the Achilles tendon. The leg is moderately flexed at the knee-joint, the ball of the foot is grasped and then pressed gently upwards (the patient not actively assisting this dorsal flexion). Then a slight blow is given with the percussion hammer upon the Achilles tendon. The result is a plantar flexion of the foot.

Modifications of this method of examination are described by Schultze, Strassburger, and others. I consider far the best method to be that of Babinski, in which the Achilles tendon is tapped while the patient kneels upon a chair. The feet must hang free of the edge of the chair.

The Achilles tendon phenomenon is not an absolutely constant sign, even in healthy people, or, at any rate, it is not always distinctly elicited. But by using this method of Babinski's, however, I have seldom failed to elicit it in healthy subjects. In the Achilles tendon and its neighbourhood, however, changes appear more frequently than in the region of the patellar tendon which do not arise from the nervous system (see below), which have a mechanical influence upon the condition of this reflex and make it sometimes very difficult or impossible to elicit it. Where, however, such changes are not present, absence of the Achilles tendon jerk (or heel phenomenon, as I call this symptom for short) is a symptom which I, along with Ziehen,[2] Babinski, Sarbó,[3] and others regard as evidence of a pathological condition.[4] Exaggeration of the Achilles jerk is shown by the fact that a tap on the tendon elicits not merely a contraction, but clonic tremors, the foot tremor,[5] ankle clonus (foot-phenomenon). This symptom may be easily produced by putting tension upon the Achilles tendon. The leg is kept slightly flexed at the knee-joint and is supported by one hand, whilst with the other hand the ball of the foot is grasped and

[1] It is incomprehensible to me why, even in otherwise good text-books on diagnosis, the sitting position has always been recommended as the best for examination.

[2] *D. m. W.*, 1894

[3] "Der Achillessehnenreflex und seine klinische Bedeutung," Berlin, 1902. For further literature, see Schönborn, *Z. f. N.*, xxix.

[4] E. Bramwell has often missed the reflex in old persons, but he regards it as constant in healthy subjects under fifty years old. Walton and Paul also found it regularly in healthy persons.

[5] The symptom had already been observed by Charcot and Vulpian, but was first closely studied by Erb and Westphal.

pressed upwards. It has then to be ascertained how much strength is required to call forth this clonus. Beginners usually make the mistake of fixing the foot so strongly in the position of dorsal flexion that a contraction (plantar flexion) cannot take place. On the other hand one must not relax the pressure too much, as the tension on the foot must always be maintained, since as soon as the ball of the foot is pressed downwards, the tremor ceases. In severe exaggeration, even active dorsi-flexion of the foot will occasionally give rise to a clonus.

A real foot clonus is sometimes simulated by a *false* clonus. In this there appear merely a few tremors at unequal intervals, which are not dependent on the tension upon the Achilles tendon, but give the impression of being voluntary movements. This symptom has been noted specially in hysteria. The graphic method has been employed for more exact registration of the movements by Eshner, and specially by Claude and Rose (*R. n.*, 1906), and Ettore Levi (*Obersteiner*, 1907). I have observed another kind of false ankle clonus in paralysis agitans (*loc. cit.*). In painful rheumatism of the joints also I have found a foot tremor starting from the dorsal flexors, the Achilles jerk remaining normal. Babinski describes a method by which a kind of ankle clonus may be elicited even in healthy persons.

Exaggeration of muscle tonus is sometimes shown by the fact that percussion of the patellar tendon *of one side* produces contractions in the extensor muscles of the leg *on both sides*, and even in other muscles of the thigh. Not infrequently percussion of the tibia elicits a contraction of the quadriceps and of the adductors, or the internal rotators of the thigh.

Later on we shall more fully discuss another important sign, which has been studied in recent years, and one which is generally associated with or develops upon the same basis as a spastic condition of the muscles, —the so-called "Babinski sign," and also a leg phenomenon which I have myself described.

The intensity of the tendon phenomena varies within wide limits, even in normal individuals. We cannot lay very great weight upon an exaggeration of the single contractions, as there are a number of conditions which fall within physiological limits in which the tendon phenomena may be temporarily exaggerated. Amongst these is the mental excitement which may arise from anxiety as to the examination, or exhaustion after a long march. Then, further, any pain, especially if it be situated in the legs—sciatica for instance—may give rise to exaggeration of the knee jerk. In periostitis and articular rheumatism, in phthisis, in convalescence from feverish illnesses, and in exanthemata accompanied by severe itching, a simple accentuation of the tendon phenomena is not at all uncommon.

This accentuation, therefore, is not in itself sufficient to characterise the condition of the muscles as spastic. Rigidity of the muscles, revealing itself in the prevention of passive movements, is the most essential factor, exaggeration of the tendon phenomena being merely an almost constant accessory symptom. This "almost" is a necessary limitation, as the degree of tonus in the muscles may be so high that it is impossible to elicit a contraction in a muscle which is already contracted to the maximum.

Exaggeration of the tendon phenomena very frequently occurs without any trace of muscular rigidity. We shall find that this is a sign of increased excitability in the various neuroses (neurasthenia, hysteria, etc.).

Increase of muscle tonus—muscular rigidity—also occurs in the

upper extremities, and reveals itself in the increased resistance to passive movements and the accentuation of the tendon phenomena. The increased resistance to passive movement is in our experience most early and most markedly revealed in the effort to supinate the pronated hand abruptly, and in the rapid extension of the flexed fore-arm. With regard to the tendon phenomena we should note that if the region of the styloid process of the radius is tapped with the percussion hammer, there appears as a rule a contraction of the supinator longus, and sometimes also a slight contraction of the biceps muscle. It is advisable during this examination to have the elbow flexed and the forearm midway between the position of pronation and supination. In this position, a blow upon the triceps tendon will elicit a contraction of this muscle.

The tendon reflexes in the arms are, however, not always so apparent, even in the normal condition. I am convinced, in opposition to Kollarits and others, that they may be absent or very feeble, uncertain and variable in healthy persons. It is therefore only their exaggeration which has a diagnostic importance, and also the constant absence on one side of a reflex which is marked on the other. Mohr, indeed, in an examination of healthy soldiers found unilateral absence in about 13 per cent. of the cases, so that too much weight must not be laid even upon this symptom.

Exaggeration is recognised in this way, that a slight tap elicits a very powerful contraction, in which as a rule other muscles are involved as well as the muscle to which the tendon belongs. This is still more pronounced if clonic contractions take place. Occasionally also there appear in the hand phenomena analogous to ankle clonus. If the fingers which are in a flexed position are suddenly pressed upwards, and thus forced into an extended position, and kept there with slightly diminished pressure, the response will be clonic contractions in the flexors (hand clonus).

Under these conditions marked muscular contractions may be elicited from different bony points, such as the ulnar styloid process, the carpal-metacarpal bones, etc. ; but as to the reflex nature of these there is still doubt.

In the *muscles of the jaw* also tendon reflexes may be produced. If we lay a finger, the handle of a spoon, etc., on the lower row of teeth, and give a tap upon this with the percussion hammer, a slight contraction takes place in most persons, which raises the jaw (de Watteville, Beevor [1]). Under pathological conditions, especially in exaggeration of the muscle tonus, this phenomenon may be so exaggerated that even pulling the lower jaw down elicits a clonus (lower-jaw clonus, masseter-clonus). Ballet first described this symptom and compared it to ankle clonus.

Decrease of the muscle tonus (*atony*, *hypotonia*) is not uncommon. It is true that it is frequently associated with atrophy of the muscles, but it is also found along with a quite normal condition of the muscular substance. The atonic muscle is softer to the feel than the normal muscle. This diminution of muscle tonus, with which there is usually a relaxation of the joint and capsular ligaments, is shown (1) by an *increased facility in the passive movements*, so that they can be carried out without any muscular resistance to a degree far in excess of the

[1] *Br.* 1886.

physiological limit (Fig. 1); and (2) usually, though not constantly, by a *diminution* or *absence of tendon reflexes.*

If the knee jerks cannot be elicited under any of the conditions described above, in any of the leg positions, with the attention distracted, and even with the aid of Jendrássik's method, then we are justified in regarding them as abolished, as absent. But before this result has any diagnostic value, we must determine whether there is not a *mechanical* cause. Disease of the knee-joint, dislocation of the patellar tendon, an ununited fracture of the patella may be present. Indeed a great accumulation of fatty tissue, in which the tendon is embedded, or a severe œdema may be the cause of the absence of the contraction. There are also some individuals in whom the tendon is so short and so deeply placed, that it can hardly be affected by the hammer. If it is very flaccid and lies very deep, the reflex is sometimes only elicited after the leg has been flexed to an acute angle. I have sometimes had to resort to this artifice in cachectic individuals.

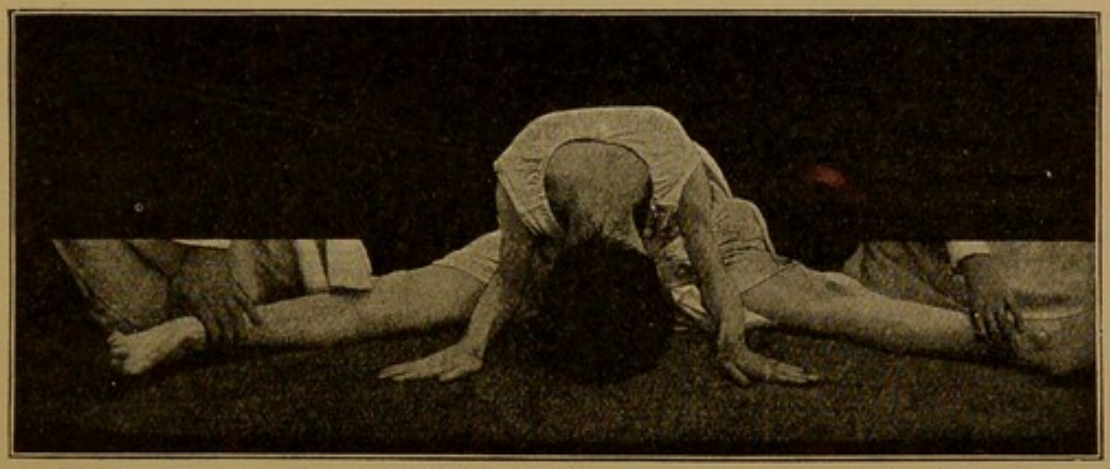


FIG. 1.—Muscular hypotonia and its influence on passive motility (after Dejerine).

Absence of the knee jerk is a sign of the greatest value in diagnosis. Although Westphal's discovery relates only to the condition of this symptom in a certain disease (tabes dorsalis), yet it is so firmly associated with his name, that loss of the knee jerk is generally known as "Westphal's sign." It should always be remembered that the condition of the tendon reflexes is influenced not only by diseases of the nervous system, but also by other factors. Thus the knee jerk cannot as a rule be elicited in deep narcosis, or in complete unconsciousness. Great rise of temperature may have the same effect (Petitclerc, Sternberg). Severe exhaustion from excessive physical exertion may also lead to temporary loss of the tendon reflexes (Auerbach,[1] Edinger, Knapp and Thomas, Oeconomakis [2]).

It is very difficult to recognise and to estimate diminution of the knee jerk. When it can be elicited only to a slight degree, even by means of Jendrássik's method, and when the contraction is limited to single segments of the quadriceps, to the vastus internus, for instance, then there is a pathological modification. In unilateral diseases, comparison with the unaffected side facilitates the decision.

It has already been shown that the same factors determine the

[1] *N. C.*, 1905. [2] *N. C.*, 1907.

condition of the Achilles jerk, and that the absence or diminution of this reflex is to be estimated with even greater care.

The examination which reveals the condition of the muscle tonus is followed by

Examination of the Active Movements.—If this investigation is to be a systematic one, it is advisable to pass from the testing of single to that of complicated movements, to examine, for instance, the motility of the legs first in the dorsal position, then in standing, walking, running, etc. In practice this is not the usual procedure. We watch the patient come into the room, and from the alteration in his gait we form our opinion as to the condition of his motor power.

Before we turn to examination of the simple active movements, we must determine whether factors exist which would cause a mechanical difficulty of movement. From this point of view diseases of the joints resulting in rigidity, atrophy of the fasciæ, cicatricial shortening of tendons and muscles, etc., deserve special consideration. Muscular tension may also be the cause of motor disorders. The fact that *pain* very frequently leads to limitation of the motility and may simulate a paralytic condition should also be borne in mind. With individuals who cannot give information themselves, especially with children, the neglect of this circumstance may give rise to serious errors in diagnosis and treatment.

The power of active movement of the legs is first tested in the dorsal position :

The patient is requested to move the extremities in all the joints, in every direction, to the maximum extent and with the maximum rapidity ; flexion, extension, abduction, adduction, internal and external rotation of the hip-joint are first examined. In this way limitation in the *extent*, as well as *slowing* of movement are easily recognised. But it is still more important to test the *strength* with which each movement is carried out. The so-called dynamometers, such as Duchenne and Charrière introduced, are not indispensable. It is better to measure the strength of the patient by one's own, opposing a resistance to the movement which he is to carry out. If for instance we are to determine the force with which the thigh is flexed upon the pelvis, then we place both hands against the thigh and endeavour to prevent the movement. Or on the other hand, the patient keeps the thigh firmly flexed, and the examiner tries to extend the leg. Some experience is required to determine in this way the " gross strength " or the " motor power " of the muscles. The examination should extend to all the movements of all the joints and groups of muscles. This investigation reveals any material loss of power. Slight weakness can usually be discovered by comparison with the strength exerted by the corresponding muscles of the other side of the body. It should be remembered, however, that the left thigh is normally somewhat weaker than the right (the proportion is about 4 to 5). It must be determined also whether all the muscles which under normal conditions combine to carry out the movement participate in it.

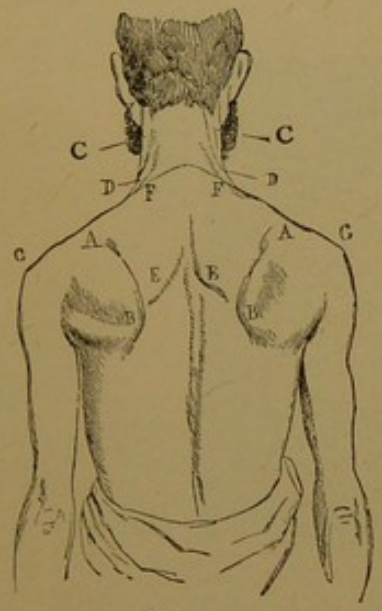
In order to estimate these conditions correctly, it is necessary to know precisely the *functions* of the various muscles, as well as the alterations of movement and position which will arise when the activity of any muscle or muscles becomes lost.

We owe to Duchenne [1] the fundamental work on this subject. Since then other clinicians have elaborated the teaching on several points. Of recent years Richet, Mollier, Fischer, Braune, Beevor,[2] and Du Bois-Reymond [3] have added experimental contributions to the question.

Action of the Muscles. Muscles of the Shoulder and Arm

The *trapezius* muscle raises the shoulders in bilateral action, and adducts the shoulder blades towards the middle line. In unilateral action, it elevates the shoulder of the corresponding side, draws the head backwards and rotates it at the same time somewhat towards the opposite side. (By contraction of the right trapezius the chin is rotated slightly towards the left.)

It is the upper or clavicular portion (from the occiput to the outer third of the clavicle) which moves the head in the direction described above when the shoulders are fixed. It is known as the "respiratory portion" of the trapezius, because it is used in deep breathing. The middle portion (from the lig. nuchæ and the three upper thoracic vertebræ to the acromion and the outer segment of the spine of the scapula) is the real elevator of the shoulder blade. If it is strongly



(Fig. 2.)

(Fig. 3.)

Fig. 2.—"See-saw" position of the scapula in paralysis of the trapezius (after Duchenne). The levator anguli scapulæ is not paralysed. The lower angles of the scapulæ (B, B) are approximated to the spine, the inner and upper angles (A, A) are displaced outwards. The acromions (G, G) are lowered. D, D, Levator anguli scapulæ. C, C, Sternomastoid.

Fig. 3.—Abnormal position of the clavicles following paralysis of both trapezius muscles (Author's case).

developed, the neck is short. The lowest portion passes from the fourth and following thoracic vertebræ to the inner half of the spine of the scapula. This moves the shoulder blade towards the middle line, and draws it to the spinal column.

If the clavicular portion is paralysed, the shoulders do not move during respiration.

As regards backward movement of the head, paralysis of this part of the muscle is not of much importance, as the deeper neck muscles can carry out this movement. The upper bundle of the trapezius is spared in many affections of the muscles, and it is therefore known as the "*ultimum moriens*."

Paralysis of the middle portion gives rise to marked symptoms: the acromion falls, being weighed down by the upper extremity because it is no longer fixed by the trapezius: it lies at a deeper level than the inner and upper angle of the scapula, which is still raised by the levator anguli scapulæ, the inner and lower angle being consequently approximated to the middle line. The shoulder is drawn forwards and downwards, and cannot be raised in the usual way. Elevation

[1] "Physiologie des mouvements," Paris, 1867.
[2] "The Croonian Lectures in Muscular Movements," etc., London, 1904.
[3] "Spezielle Muskelphysiologie," etc., Berlin, 1903. Also Jendrássik, *Z. f. N.*, xxv.

of the arm is particularly affected by the low position of the acromion, and the downward dragging of the shoulder causes pain. But most characteristic of all is the see-saw position (Schaukelstellung, Fig. 2). Mollier lays great weight on the forward displacement of the clavicle. Movement of the arms at the shoulder-joint, especially elevation, is to a certain degree restricted on account of the defective fixation of the acromion.

In paralysis of the lower portion the internal margin of the scapula, which normally lies parallel to the middle line and about 5-6 cm. (2-2½ in.) from it, is displaced outwards (some 10-12 cm.) (4-5 in.). The back is broadened, the clavicle projects forwards, *i.e.* its acromial portion describes a forward arch, and lies in a line with the sternal portion (Fig. 3). If the shoulder blades are approached to the middle line (as when the patient is told to throw out his chest) then the rhomboids come into operation. If these and the middle portion of the trapezius are intact, paralysis of the lower third causes no material disturbance of function. But if the middle trapezius is also atrophied, then contraction of the rhomboids brings the lower angle of the shoulder blade nearer to the spinal column and thus exaggerates the deformity caused by the depression of the acromion.

The *levator anguli scapulæ* muscle raises the inner upper angle of the scapula. In paralysis

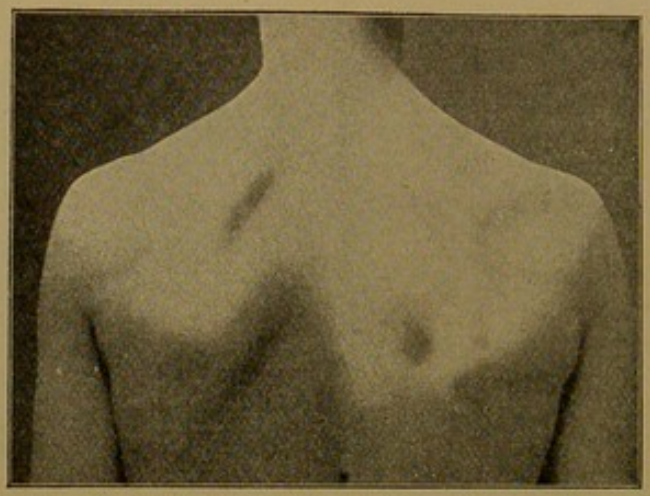


FIG. 4.—Displacement of right scapula in paralysis of the right levator anguli scapulæ (with weakness of rhomboids).

of the trapezius it may help to effect the movement of shrugging of the shoulders. Isolated paralysis of the muscle does not necessarily cause any marked disturbance, but I have seen in one case of paralysis of this muscle (and paresis of the rhomboids) an abnormal position of the shoulder blade, its internal upper angle being lower down and further apart from the spinal column than on the unaffected side (Fig. 4).

The *rhomboid* muscles move the scapula upwards and inwards, and approximate the lower angle to the middle line. According to Mollier, they come specially into play in active lowering of the arm. When this muscle is paralysed, the lower angle of the shoulder blade becomes somewhat removed from the spinal column, and the inner margin is somewhat raised from the chest wall (Jorns). Hirschlaff gives a detailed account of the movements affected in paralysis of the rhomboids (*N. C.*, 1904.)

The *serratus magnus* muscle rotates the shoulder blade round its sagittal axis, so that the lower angle is brought outwards and the acromion is raised. It also fixes the scapula to the thorax, and keeps the inner margin united with the ribs. Along with the rotation of the shoulder blade there is also slight elevation.

Paralysis: When at rest the scapula stands higher and its inner margins are approximated to the spinal column, the lower angle more so than the upper, so that the inner margin of the shoulder blade has an oblique direction (Fig. 5) from below inwards to above outwards, and the lower angle is slightly raised from the thorax. This oblique position is, however, not very prominent, and as a symptom of isolated serratus paralysis, it is quite ignored by many writers, who refer it to an involvement of the trapezius.

The paralysis is, however, made very evident by the loss of certain movements and the anomalies of position thus produced.

1. In abduction of the arm to the horizontal, the shoulder blade is approximated to the spinal column, the inner border is raised from the thorax and pushes the trapezius and the rhomboids before it (Fig. 6).

2. The arm cannot be raised beyond the horizontal, owing to the absence of the external rotation of the shoulder blade, which is necessary for the completion of the movement. When the examiner carries out this movement by pressing the lower angle of the scapula outwards, the arm can be raised to the vertical position.

3. In the attempt to extend the arms forwards, the scapula is raised, especially in its inner border, away from the thorax like a wing, so markedly in some cases that a hand can be placed between the scapula and the thorax (Fig. 7).

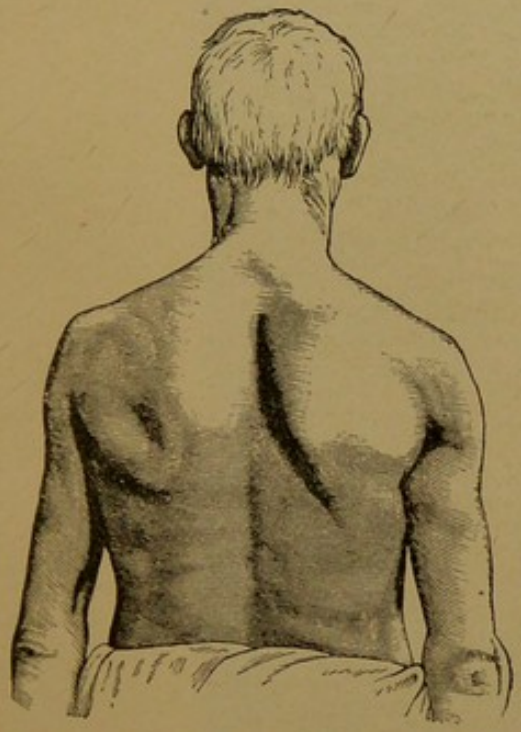


FIG. 5.—Position of scapula with arms dependent in paralysis of right senatus magnus (after Bäumler).

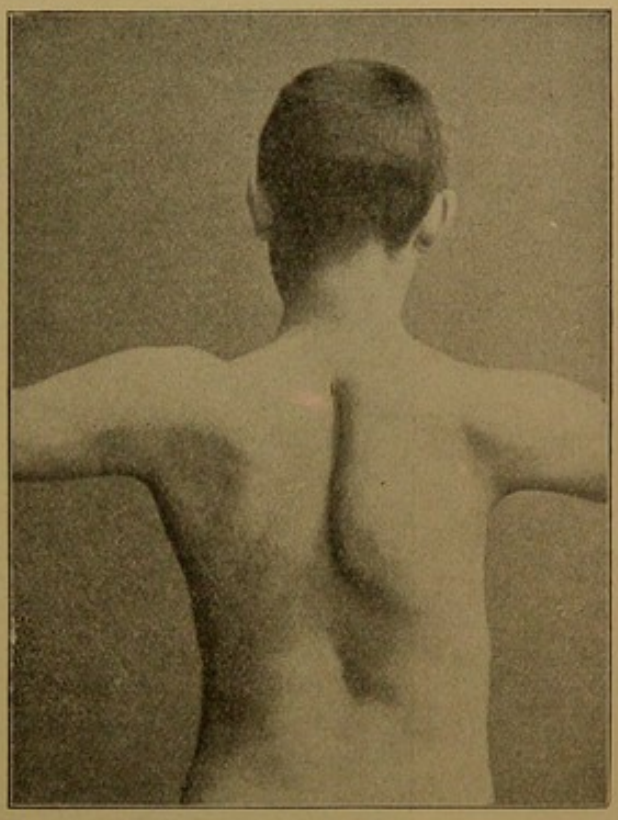


FIG. 6.—Position of scapula in senatus paralysis during abduction of the arm (after Bruns.)

In some cases of serratus paralysis the arms can, however, be forcibly raised as far as the vertical position (Bäumler, Jolly, Bruns). It is thought that in such cases the middle portion of the trapezius effects the external rotation of the shoulder blade, either because it is specially well developed from birth (Souques), or because it has gradually become compensatory for the serratus. Some writers (Steinhausen [1] and others) have recently laid it down as the rule that even in complete serratus paralysis the arm can be raised slightly beyond the horizontal (even to an angle of 120-140°), as they attribute to the deltoid muscle a greater range of action than has hitherto been ascribed to it, or regard the usual form of this paralysis as a partial paralysis with integrity of its uppermost bundles. The latter view is, however, opposed by Bramwell and Struthers.[2] Visible and palpable absence of the digitations of the muscle may also be a symptom of serratus paralysis, as it is usually associated with atrophy.

The *deltoid* muscle moves the arm outwards, forwards, or backwards, according as its middle, anterior, or posterior bundle is contracted. The arm cannot, however, be elevated above the horizontal plane. The posterior bundle does not carry it by any means so far. The view that the deltoid abducts the arm to an angle of 90° and that further elevation is then produced

[1] *Z. f. N.*, xvi. [2] *R. of N.*, 1903.

by the action of the serratus, has been modified by recent research (Steinhausen, Du Bois-Reymond and others) to this, that the abducting action of the deltoid extends further than the horizontal, whilst the function of the serratus (rotation of the shoulder blade) commences not at this point, but before it. A preliminary condition for the action of the deltoid is the fixation of the scapula by the trapezius, as when the latter is paralysed the deltoid loses its support on the acromion and drags it down instead of raising the arm. In paralysis of the deltoid the arm cannot be abducted nor moved backwards or forwards (the latiss. dorsi does not raise the hand beyond the gluteal region). In the effort to raise the arms, the shoulders are elevated as a whole, whilst the arm remains close to the thorax. It can be moved slightly in the forward and outward direction by means of the supraspinatus. In long existing paralysis of the deltoid, a subluxation of the head of the humerus occurs and the shoulder hangs loose. This looseness and subluxation of the shoulder appears more readily when there is also paralysis of the supraspinatus.

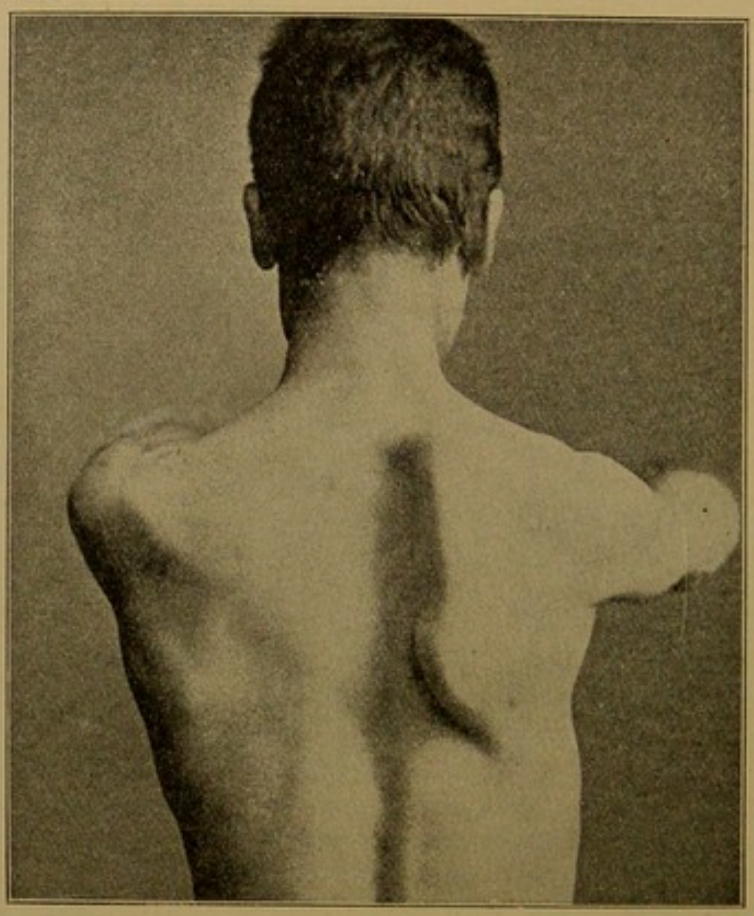


FIG. 7.—Position of scapula in serratus paralysis, during forward elevation of arm. Wing-like projection from thorax (Oppenheim).

Cases have been observed by Duchenne, and more recently by Kennedy, Kron, Hoffmann, Rothmann, Wendenburg and myself, in which with complete paralysis of the deltoid, abduction of the arm was more or less entirely retained, other muscles (serratus magnus, trapezius, pectoralis major, supraspinatus, infraspinatus) having replaced its function.

The *infraspinatus* and *teres minor* are external rotators; the *subscapularis* rotates the arm inwards. Atrophy of the subscapularis is indicated by a grating sound which comes from the rubbing of the scapula against the ribs when the shoulder blade is moved. Even in healthy individuals, however, a slight grating may be produced by this movement. Paralysis of the infraspinatus affects the power of writing.

The *pectoralis major* adducts the arm to the thorax. The clavicular portion brings the raised arm down to the horizontal and then inwards. When the arm is hanging down, it pulls the acromion forwards and upwards, as in carrying a weight. The sternal portion pulls down the raised arm, and when the arm hangs down it draws the acromion forwards and downwards.

In paralysis of the pectoralis major none of the movements are quite lost, but adduction is carried out with diminished force. (It is possible that the anterior portion of the deltoid, the teres major and the rhomboid, may compensate for part of the functions of the pectoralis major.) In order to demonstrate the paralysis, the patient is asked to extend both arms forwards and then to press the palms of the hands together. This can either not be done at all, or but very feebly.

The *latissimus dorsi* brings the uplifted arm backwards and downwards, the hanging arm inwards and backwards, and with unilateral action bends the trunk to one side, with bilateral action extends it.

The *teres major* adducts the arm to the trunk when the shoulder blade is fixed by the rhom-

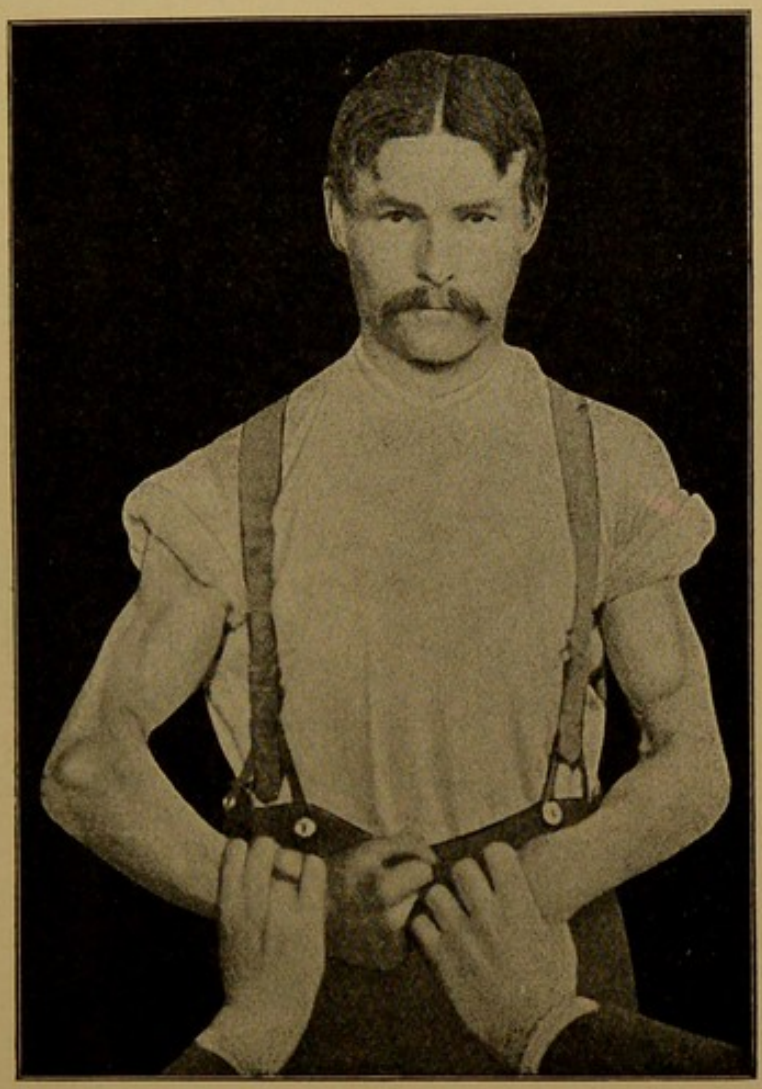


Fig. 8.—Position of paralysed left supinator, contrasted with normal right supinator. (Oppenheim.)

boids, etc.; when the arm is fixed and hanging down, it draws the scapula outwards and rotates it so that the acromion and the shoulder are raised. Its paralysis causes no material disturbance.

Amongst the muscles which maintain the head of the humerus in its socket and prevent its dislocation (which would otherwise result from the action of the latissimus dorsi and pectoralis major) are the long head of the triceps and the coraco-brachialis. As these are strongly contracted when the arm is dragged down, they maintain the head of the humerus firmly in the joint. If they are atrophied, then forcible pulling of the arm down will cause downward dislocation of the head of the humerus, especially if the deltoid is paralysed at the same time.

The *triceps brachii* is the extensor of the forearm. If this muscle is paralysed, the forearm can assume the position of extension only under the influence of gravity. If resistance be opposed to it, or if extension be attempted when the arm is raised upright, it is impossible.

The *brachialis anticus* flexes, but does not pronate or supinate the forearm. The *biceps* flexes and supinates the forearm, whilst the *supinator longus* brings it into a slightly pronated position and then flexes it.

In forcible movements these muscles are equally contracted. Paralysis of one of them is indicated by rotation (pronation or supination) of the hand, which appears when the elbow is flexed. If all three are paralysed, slight flexion can still be carried out by the flexors of the hand and fingers, since they arise from the internal condyle of the humerus, and by the pronator teres becoming excessively tense or contracted, whilst the hand is held firm by the antagonists. The effect then is flexion of the elbow. In the same way the extensors of the hand and fingers can produce flexion of the elbow, after the forearm is pronated and the wrist-joint is extended or fixed in the position of flexion.

This special form of flexion, which was first described by Duchenne, is easily recognised by the fact that it is impossible in the ordinary position of the hand and fingers, and is always a very weak movement.

If the biceps alone is paralysed, the forearm can still be strongly flexed, but the patient becomes easily fatigued and complains of pain in the shoulder. Paralysis of the supinator longus is easily recognised from the circumstance that in forcible flexion of the forearm—carried out against resistance—the muscle fails to stand out (Fig. 8). Atrophy of this muscle gives the forearm a spindle-shape.

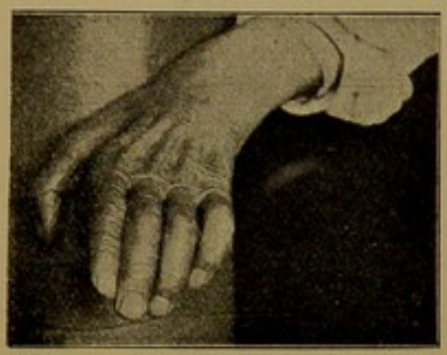


FIG. 9.—Incompletely developed claw-hand in atrophic paresis of interossei and lumbricates. (Oppenheim.)

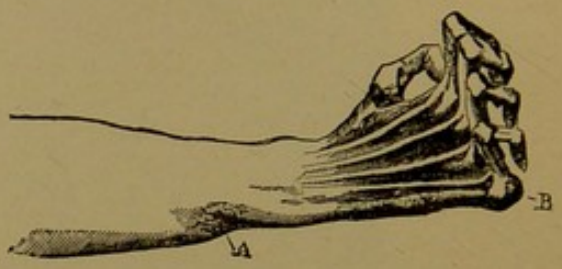


FIG. 10.—Completely developed claw-hand after old-standing ulnar paralysis. Cicatrix at A. (After Duchenne.)

The *supinator brevis* supinates the hand when the forearm is extended. The *pronator teres* and *pronator quadratus* are true pronators.

The *extensor carpi radialis longior* extends the hand and at the same time draws it towards the radial side. The *extensor carpi ulnaris* extends it and moves it towards the ulnar side. The *extensor carpi radialis brevior* is simply an extensor.

If all the extensors are paralysed, the hand hangs down, and when passively raised it falls back into this position. The grasp is feeble, as it is only by extension of the hand that the flexors of the fingers can develop their full strength. If the hand is passively brought into the extended position, the force of the grasp can be increased.

The *extensor communis digitorum*, the *extensor indicis* and *minimi digiti* are powerful extensors of the proximal phalanges of the four fingers and separate the fingers somewhat from each other, abducting them from the middle finger. The *extensor indicis* when acting alone extends the first phalanx of the index finger and approaches it to the middle finger.

Forcible contraction of the ext. comm. dig. slightly extends the hand at the wrist-joint. This muscle has nothing to do with extension of the second and third phalanges.

The *flexor carpi radialis* flexes the hand and slightly pronates it, so that the palm of the hand is turned slightly to the ulnar side. The *palmaris longus* simply flexes the hand, while the *flexor carpi ulnaris* flexes especially the ulnar part of the hand, and supinates it, so that the palm is turned somewhat towards the radial side. The fifth metacarpal bone is also flexed upon the carpus by this muscle. Paralysis of the flexors of the wrist does not give rise to any marked deformity, as the force of gravity enables the hand to assume a flexed position. If the flexors

of the fingers are intact, they can, to a certain extent, undertake the function of the paralysed flexors of the wrist.

The *flexor sublimis digitorum* flexes the second, the *flexor profundus digitorum* the terminal, or the middle and terminal phalanges. These muscles are not concerned in flexion of the proximal phalanges. In extreme contraction, however, or if the terminal phalanges are kept extended in spite of their contraction, they also produce a flexion of the proximal phalanges. The more the hand is kept extended the more powerfully do the flexors of the finger act.

In paralysis of the *flexor sublimis digitorum* the second phalanx may be gradually over extended against the first, on account of the preponderance of the extensors (interossei). In paralysis of the *flexor profundus* this displacement may occur between the second and third phalanges, but on the whole this is very uncommon, as isolated paralysis of this muscle is rare.

It is of the greatest importance to make oneself familiar with the function of the interossei and the lumbricales muscles, because these are very frequently affected, and in diseases of very different kinds. The dorsal and palmar interossei (which in pathology we may regard as being always affected together) adduct and abduct the fingers. These movements are only complete when the fingers are extended at the metacarpo-phalangeal joints. If therefore we wish to test this function when there is paralysis of the ext. dig. comm., we must first passively extend the fingers, and resting them on some support, such as the hand of the examiner, watch the adduction and abduction. Another important function of these muscles is the *simultaneous flexion of the proximal phalanges and the extension of the second and third phalanges of the fingers.* In this action they are supported by the lumbricales.

In incomplete paralysis (or atrophy) of these muscles it is the lateral movement which suffers first, especially adduction of the fingers, so that, for instance, the patient cannot hold an object between them. As the paresis progresses, however, extension in both interphalangeal joints becomes diminished, and a very characteristic deformity of the hand develops. Whilst normally, when the hand is at rest, the fingers are slightly flexed at all the joints, in paralysis of the interossei (and lumbricales) the proximal phalanx is in a position of extension, and the other phalanges are flexed, the middle more so than the last. Finally the antagonists (the ext. dig. comm. on one side, the long finger flexors on the other) attain the ascendency and place the first phalanx in a position of extreme extension—over-extension—whilst the second and third phalanges are kept strongly flexed (claw-hand, *main en griffe*) (Fig. 9 and Fig. 10). This position may gradually become completely fixed by secondary changes in the joints.

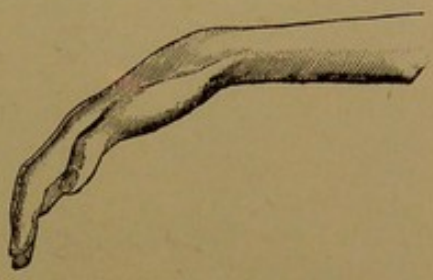


FIG. 11.—Ape hand due to atrophic paralysis of the thenar muscles. (After Duchenne.)

Thumb-muscles.—The *extensor longus pollicis* extends both phalanges of the thumb and directs the whole thumb backwards. If it is paralysed, the metacarpal bone of the thumb is bent slightly forwards, and the second phalanx is flexed against the first. Extension can then be carried out only by the abductor and the flexor brevis, if the metacarpal is kept flexed and adducted and the first phalanx flexed. Simultaneous extension of the first and second phalanx is impossible.

The *extensor brevis pollicis* is an abductor of the thumb. It turns the first metacarpal directly outwards, extends the first phalanx, but has no influence upon the second. Its paralysis, however, only becomes important when the *abductor longus pollicis* (*extensor ossis metacarpi pollicis*) is also paralysed. This muscle moves the first metacarpal outwards, but also forwards, flexes it against the wrist-joint and in maximum contraction is not only an abductor, but also a flexor and pronator of the hand. If the *abductor longus pollicis* and *extensor brevis pollicis* are paralysed, the thumb is adducted and falls into the palm of the hand.

The *flexor longus pollicis* flexes the second phalanx of the thumb. Its paralysis suspends this movement, and produces difficulty in writing, etc.

Of the muscles of the ball of the thumb, those which are situated at the radial side of the first phalanx and of the metacarpal have the function of moving the first metacarpal forwards and inwards, of flexing the first phalanx and rotating it so that it stands in opposition to the fingers. The adductor and the internal head of the flexor adduct the first metacarpal on the second, the first phalanx being slightly flexed whilst the second is extended.

The *opponens pollicis* does not act upon the phalanges, but merely moves the first metacarpal

forwards and inwards, so that it is in direct opposition to the second. For complete opposition the co-operation of the abductor brevis and the outer portion of the flexor brevis is necessary.

In paralysis of all the muscles of the ball of the thumb the metacarpal of the thumb is brought by means of the action of the ext. longus pollic. into the same line as the other metacarpals (Ape-hand) (Fig. 11). In paralysis of the abductor brevis and opponens pollicis, slight opposition is still possible by means of the flexor brevis, but flexion of the first metacarpal is so incomplete that the thumbs can only touch the tips of the other fingers when these are flexed in the interphalangeal joints.

If the *adductor* is paralysed, then the first metacarpal stands further apart from the second than normally, and cannot be approximated to it in the position of flexion, so that, for instance, the patient cannot hold a stick.

The Muscles of the Pelvis and the Lower Extremities

The *gluteus maximus* extends the thigh at the hip-joint and rotates it slightly outwards. If the thigh is fixed, it extends the trunk.

This muscle is specially active in climbing steps, jumping, and in rising from a chair, and its paralysis makes these movements difficult. If in paralysis of the glutei the patient attempts to get upon a chair, his pelvis is strongly bent forwards.

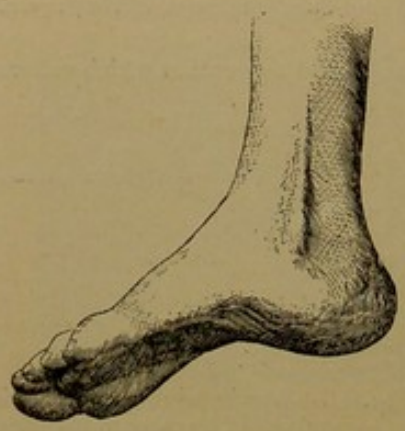


Fig. 12.—Pes valgus produced by secondary contracture of the peroneus longus muscle. Foot seen from outer side. (After Duchenne.)

The *gluteus medius* is mainly an abductor. If its anterior portion alone is contracted, the leg is moved forwards and outwards and rotated slightly inwards; its posterior portion moves the leg backwards and outwards, and at the same time rotates it outwards. When the thigh is fixed this muscle bends the trunk sideways. The *gluteus minimus* has a similar action.

In paralysis of these muscles the leg cannot be abducted; the adductors have the ascendency. In walking the leg swings too far inwards; the excessive raising and lowering of the pelvis is specially noticeable—the waddling gait. The pelvis bends towards the opposite side, especially in unilateral paralysis (the trunk towards the side of the paralysis). In bilateral paralysis it bends, during walking, towards the side of the swinging leg.

The *pyriformis*, *gemelli*, *obturator internus* and *externus*, along with the *quadratus femoris* rotate the thigh outwards. If they are paralysed the leg is turned permanently inwards.

The *ileo-psoas* muscle flexes the thigh at the hip and rotates it slightly outwards, whilst the *tensor fasciæ latæ* combines a slight internal rotation with the flexion. If both these flexors are paralysed, walking is impossible; if there is merely paresis, walking is impeded and in the recumbent position the leg cannot be raised when the knee is kept straight. The patient also has difficulty in raising the trunk from the recumbent position.

The *pectineus*, *adductores* and *gracilis* adduct the thigh. The pectineus both flexes and adducts it.

The *adductor longus* and *brevis* flex it less strongly. All three also produce a slight externa rotation of the thigh (?). The *adductor magnus* moves the thigh directly inwards, and its lower portion also causes slight inward rotation.

In paralysis of the adductors, adduction of the leg is impossible, and the leg deviates outwards when it is raised from its support, on account of the preponderance of the abductors. In paralysis which is confined to the lower portion of the adductor magnus, adduction is accompanied by external rotation.

The *quadriceps femoris* extends the knee. The *rectus femoris* is the only part which also flexes the hip; its extension of the knee is inversely proportional to the degree of flexion of the hip-joint. When the knee is bent, it is a powerful flexor of the hip-joint.

In paralysis of the extensors, standing with the knee extended is possible (owing to the nature

of the joint and its ligaments). Walking also is possible, with difficulty, but the knee has to be kept straight, as the upright position would become impossible if its flexors came into action. Flexion in the knee is avoided by making the steps short, since at the end of a full swing of the limb the leg is left in the flexed position. The pelvis, however, is pushed forwards on the affected side (in unilateral paralysis). Immediately the patient lets the knee bend, he is in danger of falling.

An individual suffering from bilateral paresis of the quadriceps can walk, however, with the aid of a stick. Besides the difficulty in walking, the excessive flexion of the knee of the advancing limb attracts attention. The cause of this may be, however, that the patient is forced to flex the hip-joint strongly on account of an accompanying weakness in the extensors of the foot.

In order to demonstrate a quadriceps paralysis, the patient should be placed upon his back, asked to flex the hip-joint and then to straighten the limb at the knee. The foot must not rest upon the bed, as it could be mechanically pushed forward by the movement of extension and rotation at the hip-joint. In the sitting position, the paralysis prevents the leg from being extended, and when it is passively raised, it falls back again. Rising from the kneeling position is impossible; when the paralysis is incomplete it can only be carried out by the patient laying his hands on his knees and pressing them backwards.

Fig. 13.—Position of the feet in paralysis of the extensors of the foot and toes. (Peroneus paralysis. Pendulum position of feet.) (Oppenheim.)

If the *vastus internus* alone is paralysed, then in extension the patella is pushed outwards by the *vastus externus*, and it may even become dislocated. In rupture of the lig. patellæ, the knee can still be feebly extended by means of the muscle fibres which go from the vasti laterally to the tibia.

The *sartorius* muscle flexes the hip- and knee-joints and rotates the thigh slightly outwards. Its action is a feeble one. The *gracilis* muscle is a feeble flexor of the knee; its main action is to adduct the thigh and rotate it somewhat inwards.

The *biceps*, *semitendinosus* and *semimembranosus* are flexors of the knee and extensors of the hip. They extend the hip-joint in ordinary walking (as the gluteus maximus in climbing, etc.).

If these muscles are paralysed, the pelvis would bend and fall in front of the trunk did the patient not instinctively throw the centre of gravity backwards by bending his body backwards. As the leg can no longer be actively flexed, the knee is bent by an excessive flexion of the thigh, so that the leg, following the centre of gravity, passes into a position of flexion. In the standing position the quadriceps is unduly strained, and extension of the knee becomes so forced that there may be a slight retroflexion. Jumping, running, and dancing are impossible.

The *popliteus* muscle causes internal rotation of the flexed leg and even flexes the knee slightly.

The *triceps suræ* (gastrocnemius, plantaris and soleus) cause plantar flexion of the ankle [1] and adduction of the foot. The foot is also rotated so that the dorsal surface looks outwards and the tip inwards. In simple plantar flexion the *peroneus longus* co-operates. The action is stronger when the knee is extended (the gastrocnemius, which is attached to the femur, can slightly flex the knee). If the leg is flexed on the thigh, the soleus alone is active.

In paralysis of the calf muscles flexion of the foot is almost impossible; it does not surpass a right angle. The *peroneus longus* moves the head of the first metatarsal downwards, and causes

[1] I shall refer to *plantar flexion* as *flexion*, and to *dorsal flexion* as *extension*.

valgus position of the foot (Fig. 12). A *talipes calcaneus* gradually develops, on account of the preponderance of the extensors (dorsal flexors). There is never, however, contracture of the antagonists. The patient cannot raise himself on the point of his foot, and walking is impeded. There is secondary retraction of the muscles and fasciæ of the sole of the foot, which therefore shows marked arching or hollowing.

The *peroneus longus* muscle is not much concerned in plantar flexion ; it is mainly an abductor of the foot. It lowers the internal and raises the external margin, moves the head of the metatarsus downwards and outwards, and thus narrows the front part of the foot whilst it increases the arching of the sole.

In paralysis of the peroneus longus, extension of the foot is associated with adduction ; the internal half of the front part of the foot is no longer supported and it succumbs to the pressure forcing it upwards. In walking only the external margin of the foot touches the floor, the head of the first metatarsus is raised off the ground and the great toe is strongly flexed. The arching

FIG. 14.—Position of foot in paralysis of the tibialis anticus muscle. The extensor communis digitorum produces a slight abduction during the attempt to extend the foot. (After Duchenne.)

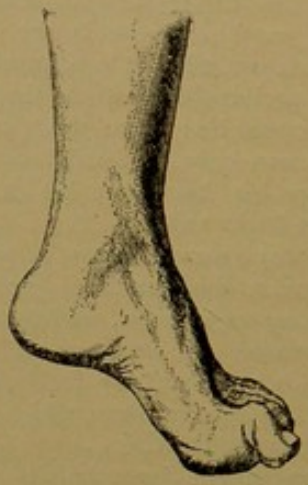


FIG. 15.—Position of the foot in paralysis of the tibialis anticus muscle. Pes equinus, marked projection of the tendon of the extensor longus hallucis. (After Duchenne.)

of the sole is diminished in standing, and flat foot develops. Walking is very fatiguing and standing on the point of the foot is very unsteady or altogether impossible.

The pressure which in walking affects the nerves of the sole of the foot produces paræsthesiæ and pain.

The *tibialis anticus*, *extensor communis digitorum* and the *extensor longus hallucis* dorsi-flex the foot. The *tibialis anticus* is also an adductor ; it moves the head of the first metatarsus upwards and inwards and raises the internal margin of the front part of the foot (whilst the toes, especially the great toe, are flexed). The *extensor communis digitorum* extends the four toes feebly ; it is mainly an extensor of the foot, and also raises the external margin and abducts the foot. The *extensor hallucis longus* causes dorsi-flexion of the second phalanx of the great toe, and also aids extension and adduction of the foot.

In paralysis of these muscles the foot cannot be raised ; it hangs limp as soon as it is lifted off the ground (Fig. 13). In walking the point of the foot tends to drag along the ground. To avoid this, the limb is in walking excessively flexed at the hip- and knee-joints. This produces a characteristic gait (*steppage*) (the tyro at once suspects ataxia).

In paralysis of long duration, contracture of the foot flexors develops and *pes equinus* is produced (if the peroneus longus is also paralysed there is *pes equinovarus*) ; or on account of the paralysis the equinus position is not corrected during lying or walking, so that the deformity gradually becomes constant.

If the tib. ant. only is paralysed, extension of the foot is accompanied by abduction (Fig. 14).

The long toe-extensors, especially the extensor halluc. long. become excessively extended and the first phalanx of the great toe is permanently extended (Fig. 15).

In isolated paralysis of the ext. dig. comm., dorsal flexion of the foot is always accompanied by adduction.

The *peroneus brevis* muscle adducts the foot and slightly raises its external margin, without extending or flexing it.

The *tibialis posticus* muscle adducts the foot without extending or flexing it, whereby it is so curved that the outward margin becomes convex and the head of the astragalus stands out on the back of the foot. Paralysis of these muscles (the peroneus brevis and the tibialis posticus) abolishes simple adduction and abduction without affecting flexion and extension, and in course of time produces corresponding deformities.

Function is as a rule more severely affected by paralysis of single muscles or muscle groups in the foot than by paralysis of all the foot muscles, since in the latter case no material deformity develops, but merely a slight valgus position, the os calcis being pressed somewhat outwards by the weight of the body. This description, however, does not apply to individuals who are permanently confined to bed, as static conditions play so great a part in determining the development of secondary contractures. If the foot is fixed at a right angle to the leg by a suitable apparatus, walking becomes possible.

The *extensor brevis digitorum* moves the toes more forcibly in the dorsal direction than the long extensor.

The *interossei* and *lumbricales* abduct and adduct the toes, and also flex the first phalanx and extend the second and third.

The *flexor longus digitorum* and *flexor brevis digitorum*, as well as the *flexor longus hallucis* flex the terminal phalanges strongly towards the sole

The *abductor, flexor brevis* and *adductor hallucis* muscles flex the first phalanx of the great toe and extend the second. The abductor and the inner head of the flexor brevis move the great toe inwards, the adductor outwards. These muscles are contracted in the movements of the foot when it is raised from the ground.

If the flexors of the toes are paralysed, the interossei are permanently contracted ; the first phalanges are flexed, the last extended, and the normal position of the toes is lost.

In paralysis of the *interossei*, the first phalanges are over-extended, their heads are partially dislocated, and the second and third are flexed (claw-foot). Walking is not actually impossible, but it is painful, and running and jumping are very greatly affected.

The Muscles which move the Head and the Spinal Column

The functions of the following muscles deserve special attention.

The *sternomastoid* turns the face towards the opposite side and raises the chin, whilst the head itself inclines towards the corresponding side and the opposite ear is raised. If both muscles act together upon the head when it is bent back, they bring it forward, at the same time raising the chin. To test their function the patient, while lying on his back, is asked to raise his head against pressure made upon the chin.

The form of the muscles stands out so distinctly under the skin that their contraction is evident from this alone; care is required in this connection, however, as the muscle may be normally developed even although it does not become prominent in this way. Unilateral paralysis does not necessarily give rise to an abnormal position of the head ; it usually assumes that due to the action of the opposite muscle, and may be finally kept permanently in this attitude by the contracture of the latter. In bilateral paralysis, if the head is excessively bent backwards, it can only be inclined forward by a great effort. I have seen one case of this kind, however, in which the head could be flexed with full force.

The *recti capit. ant.* (*maj.* and *min.*) flex the head at the atlanto-occipital joint.

The *rect. capit. lat.* muscle bends the head to the side.

The *long. colli* muscle is a flexor of the neck.

The *rect. cap. post.* muscle moves the head backwards at the atlanto-occipital joint.

The *obliqu. cap. infer. s. maj.* muscle rotates the head.

The *bivent. cervic.* and *complex maj.* muscle move the head backwards.

The *splenius capit.* and *colli* move the head backwards and thereby rotate it towards the side of the contracted muscle.

The *sacrolumbalis* and *longissimus dorsi* muscles extend the lumbar and lower thoracic spinal column. In unilateral action the spine is drawn backwards and to the same side, so that the lower part of the spine as far as the eighth thoracic vertebra is curved, with the convexity towards the opposite side. In walking the muscle on the side of the advancing leg contracts (Lamy).

The *semispinalis dorsi* and *multifidus spinæ* muscles rotate the spine.

The *quadratus lumborum* bends the lower part of the spine sidewards.

The spine is bent forwards as well as forwards and laterally by the abdominal muscles.

If the *erector trunci* are paralysed on both sides, then in standing and walking the trunk is thrown backwards, so that a weight dropped from the most prominent thoracic vertebra falls behind the sacrum (Fig. 16). The pelvis is tilted (by the action of the abdominal muscles). There is a moderate degree of lordosis of the spine, which disappears when the patient lies down. In sitting the spinal column has a convex posterior curve, and the patient prevents himself from falling forwards by supporting himself with his hands.

In paralysis of the *abdominal muscles* there is also lordosis of the lumbar region of the spine, but here a weight let down from the thoracic vertebra falls on the middle of the os sacrum, because the pelvis is strongly bent forwards. The abdomen and nates are both prominent (Fig. 17).

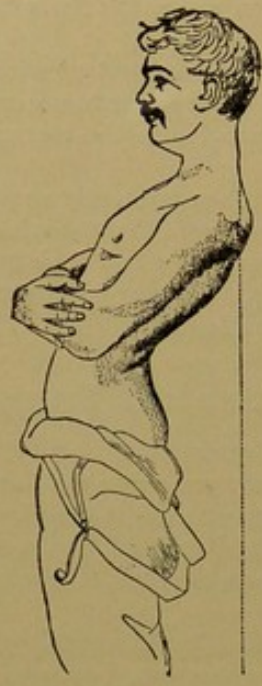


Fig. 16. — Lordosis of the vertebral column and abnormal position of the trunk in paralysis of the extensors of the spine. (After Duchenne.)

Fig. 17. — Lordosis and abnormal position of the trunk in paralysis of the abdominal muscles. (Oppenheim.)

The patient can raise himself from the dorsal position only by the support of his arms.

Paralysis of the abdominal muscles also affects expiration, and forced movements of expiration as coughing, singing, screaming are no longer possible. Abdominal pressure is absent, so that emptying the bladder and bowel become difficult. "As the viscera which push the flaccid abdominal wall forward do not afford a sufficient point of support to the diaphragm, the latter cannot raise the ribs, but narrows the lower part of the thorax."

Strassburger (*Z. f. N.* xxxi.) states that in rising from the horizontal position the vertical abdominal muscles come into action, in contraction of the abdomen the horizontal muscles act. In *unilateral* paralysis of the abdominal muscles, the umbilicus is drawn towards the sound side, and this deviation is increased in coughing, pressure, etc. In forced expiratory movements

also the abdominal parietes on the paralysed side are driven forwards by the pressure of the viscera and form a marked projection.

Palpation is a valuable aid in all these examinations, as by its means the contracted can be easily distinguished from the relaxed muscle.

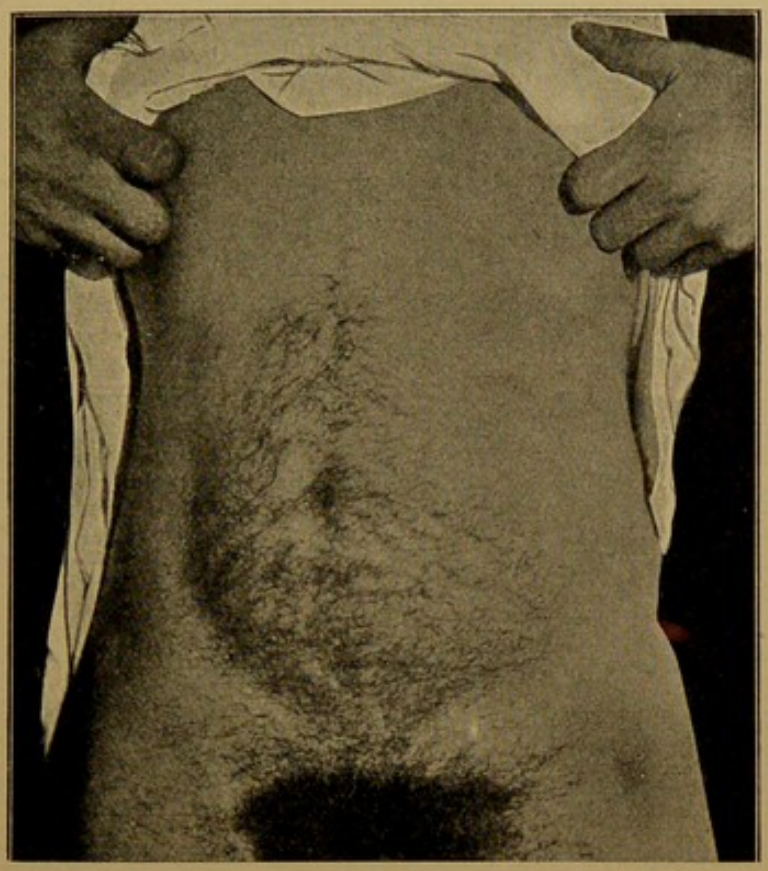


FIG. 18.—Lift-sided paralysis of the abdominal muscles. Displacement of the umbilicus to the right and increase of the visible area of the left half of the abdomen in the attempt to contract the abdominal muscles. (Oppenheim.)

Complete paralysis is easily diagnosed by the entire absence of the corresponding movements. Certain difficulties may arise when one cannot come to an understanding with the patient (childhood, deafness, unconsciousness, aphasia, mental anomalies). If a whole extremity is beyond the control of the will, it is usually quickly discovered, as when it is passively raised it falls back from the force of gravity. We must remember, however, that many people, children especially, simply let the limbs fall back in this manipulation, even when they are not paralysed. But this can almost always be determined by repeated testing. If individual muscles only are paralysed, and if the patient cannot respond to direct requests, then the extremity should be placed in a position from which it can only be moved by contraction of the muscles which are to be tested.

Incomplete paralysis (paresis) is much more difficult to diagnose, and the slighter it is, the less we can be sure of it. If one side only is affected, then comparison with the healthy side will usually reveal the slight condition of weakness. With children we must make use of various devices. If, for instance, we wish to examine the power of movement of the extensors of the foot, then we should prick the sole

with a pin. The reflex movement which results must not of course be confused with active movement. But when motor power is conserved, an active movement at once follows the reflex movement, or it is sufficient to bring the pin close to the sole of the foot—the child watching it—to produce energetic withdrawal of the foot. At this moment we can determine the force with which extension can be carried out. It is more difficult under these conditions to estimate the strength exerted in plantar flexion. By exciting the back of the foot as just described, however, the child can as a rule be induced to contract the calf muscles energetically. At first he attempts to withdraw the whole leg, but if the thigh is held, plantar flexion will take place. Paralysis of the foot flexors can also be rapidly recognised by the fact that when the leg is raised vertically from the bed, the foot hangs down.

Motor weakness or paralysis may be simple, or may be associated

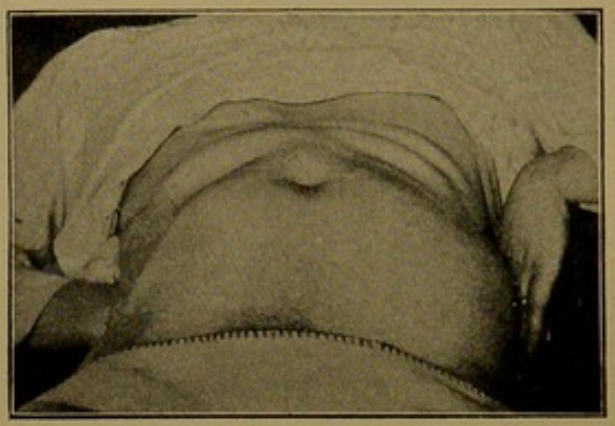


Fig. 19.—Left-sided paralysis of the abdominal muscles. Dorsal position. (Oppenheim.)

with diminution of muscle tonus (flaccid paralysis) or with its increase (spastic paralysis).

The criteria of the spastic condition have already been described. It only remains to add that spastic conditions are usually associated with motor weakness, although the relations between these two factors are very inconstant.

Spastic paresis may affect all four extremities, but is usually confined to the lower limbs, or to the arm and leg of one side, and sometimes even to one limb. The more severe degrees of this condition, in which the muscular tension leads to persistent alterations of position in the joints, are known as *active* or *spastic contracture* in opposition to *paralytic* or *passive contracture*, i.e. the secondary contracture, shortening and shrinking of the muscles of which the antagonists are paralysed. This passive contracture is completely fixed and mechanical, and cannot be even transiently overcome by pulling or pressure (even in narcosis). It therefore offers not an elastic, but a dead resistance and is not associated with exaggeration of the tendon reflexes.

Disturbances of Co-ordination

Although the strength is conserved, the active movements may be severely affected, on account of disturbances of co-ordination.

In order that a movement be co-ordinated, it is necessary that a certain number of muscles should enter into action in a definite grouping and succession and with a regulated distribution of strength. When this regulated correlation of movements—which is controlled by determined centres—is disturbed, they may still be carried out with energy, but they are inco-ordinate, ataxic. This disturbance is shown by the fact that the movements do not reach their object by the shortest way, or with precisely the requisite degree of force, but with a dissipation of muscular strength which not only sets into action the muscles which are normally concerned in the carrying out of this movement, but radiates motor impulses to muscles which not only are not required for the desired movement, but which have an inhibiting effect upon it or stand in no relation whatever to it.

Every movement which has a determined object sets a number of muscles in action. Thus raising the leg from the bed requires in the first place contraction of the flexors of the hip—the chief *agonists*, as O. Foerster [1] calls them—but other muscles of the thigh—the adductors and abductors, the rotators to a less degree, and the *synergists* which are associated with each of these muscles in finely graduated movements,—are also contracted in order to maintain the direction of the movement, etc. With regard to the condition of the antagonists in active movements, the views of investigators differ. Duchenne has taught that they become active in collective movements in order to give to these precision and certainty. Sherrington, Hering and Mann say, on the contrary, that contraction of the agonists is associated with relaxation of the antagonists. O. Foerster thinks that there is no constancy in these relations, but that the antagonists frequently take part in the movement in order to equalise it, to moderate it, and so on. It is comprehensible that any modification in the proportion of force with which each of the muscles concerned takes part in the action will disharmonise the movement, and that the result of this disturbance in the distribution of the action will result in the motor anomaly which we call ataxia.

In order to determine inco-ordination in the lower extremities, we ask the patient, who is lying on his back, simply to raise the extremity. Ataxia is revealed if the limb is not lifted straight and steadily into the vertical position. It becomes adducted or abducted, rotated outwards or inwards, is jerked from one position to another, and after it is lifted up, it oscillates constantly, especially in the lateral direction. In laying it down again it is not simply lowered, but is thrown down heavily, not beside the leg which is at rest, but across it, apart from it, or in some other position from that desired. We see clearly that the object is not attained by the shortest way, nor with the exact output of strength required, and that unnecessary muscles take part in the movement.

The fact that this inco-ordination is as a rule aggravated when the eyes are closed requires careful consideration. In every case in which it is not pronounced when the patient's eyes are open, examination with the eyes shut should not be omitted.

It may be very difficult to recognise slight degrees of ataxia. For this test complicated movements are required, the attempt, for example, to touch the knee of the resting leg with the heel of the other. If ataxia is present, the knee will not at once be exactly touched, but some part near it, or the effort succeeds only after several trials. If the inco-ordination is not at once evident, this examination must be repeated several times, as even healthy people may sometimes miss the object. The patient may also be requested to describe a circle in the air with the

[1] "Die Physiologie und Pathologie der Koordination." Jena, 1902.

lifted leg. If ataxia exists, a very irregular figure will be made, especially when the eyes are shut. It is always necessary, however, to compare with a healthy person. I have sometimes found that the ataxia is markedly evident at the first examination. If I requested the patient to raise the leg whilst I pressed against it, the movement was not steady but there was continuous swerving of the leg. The symptom that the elevated extremity cannot be kept still, but oscillates, that the trunk sways in sitting and the body in standing, has been called *static ataxia*. In order to determine if there is ataxia in the upper extremities, the patient is asked to touch with the index-finger the nose, the ear, or some other fixed point, or to grasp an object held before him. If the ataxia is severe, he does not succeed even with the control of his eyesight; the finger goes too far, instead of the nose for instance it touches the cheek or the brow. An incompletely developed ataxia may only become apparent when the eyes are shut during the examination.

Ataxia has no connection with motor weakness. The movements are strong in spite of the inco-ordination, and may even be carried out with unnecessary force. Close investigation always shows that in severe ataxia the full strength is not *steadily* exerted; if the leg is raised against resistance, we feel that the movement is at times forcibly carried out, that the power comes and goes as it is diverted to one or other group of muscles. Ataxia may of course be associated with motor weakness, but this must be regarded as an independent and unconnected phenomenon. Slight ataxia may be simulated by weakness in so far that in this condition the raised leg may sway on account of fatigue, but it then swerves up and down and not in the lateral direction. Nor does the disturbance increase when the eyes are closed.

Ataxia is very frequently, though not always, accompanied by sensory disturbances, especially diminution of sensibility of the deep parts (bathyanæsthesia), mainly in the joints, and a consequent absence of the sensations which are connected with movement of the limbs. The absence of this centripetal impulse has been considered by Bell and Longet, and specially by Leyden to be the cause of ataxia. It should, however, be remembered that even centripetal stimulations which do not reach the consciousness, but which influence the muscle tonus, are necessary for the regulated executions of the movements, and that their absence and the hypotonia or atony of the muscles thus produced plays a part in the causation of the ataxia (Jaccoud, Strümpell, Sherrington, Hering, Bickel and others). We assume, therefore, that the co-ordination of muscular function is regulated mainly by centripetal impulses which do not become conscious sensations, and we describe ataxia in this sense as *sensory*.

Cerebellar ataxia occupies a position of its own. It is a disturbance of the balance which becomes evident in standing or walking. The patient stands with his legs apart, and in walking he is unsteady and lurches from side to side like a drunken man. Or he may in walking move the leg forwards whilst the trunk is pushed backwards, etc., a symptom—described by Babinski[1] as "cerebellar asynergy"—which reveals with special clearness the disturbance in the organised co-operation of the muscles. The designation of cerebellar ataxia indicates that this symptom appears mostly in diseases of the cerebellum, which forms the main

[1] *R. n.*, 1899.

centre of co-ordination. But affections of this region may also cause a motor ataxia which is in every way similar to the sensory disturbance of co-ordination already described. The former is as a rule, however, accompanied by a diminution of sensibility, which is not present in cerebellar ataxia.

Active movements may also be affected by another disturbance :

Tremor

This term is used for the more or less rhythmic, rapid contractions, of inconsiderable range, which appear in a certain muscle group (in opposition to contractions which pass irregularly from one muscle group into another).

According to the nature of the disease, the tremor appears sometimes during rest, sometimes only during movement, and in other cases, particularly under the influence of mental excitement ; all these conditions therefore must be taken into consideration in the examination. First we must note whether the tremor occurs during rest, in a relaxed, well-supported limb. If we wish to ascertain whether the head trembles during rest, the patient must be placed on his back with his head upon a pillow. The tremor is almost always recognised at sight. It is only when the oscillations are very fine that palpation is necessary, and that the tremor is recognised from vibration of the body. A hand laid upon the head, the shoulder, etc., distinctly feels even this gentle tremor. Examination with the myograph is not necessary to the expert. In order to estimate the influence of active movement upon the tremor, the extremity should first be raised or extended in the air. This simple act is quite sufficient to show that active movement has a sedative or stimulating influence upon the tremor. Where however it only occurs on movement, it is usually necessary to test it by a complicated muscular act. The patient should be asked to raise his hand to his nose, to carry a spoon or a glass of water to his mouth, to make some forcible or fine movement for which a " steady hand " is necessary, such as threading a needle or writing. The tremor which accompanies active movement will be perceptible also in walking. In order to analyse it, the patient should, while lying on his back, raise, flex and extend the limb.

We can distinguish between a static and a motor tremor according as it appears during the active maintenance of a special position, in the outstretched hand for instance, or during the act of movement itself.

The influence of the mind is to be inferred from the fact that the tremor either increases or becomes noticeable only during conversation with the patient about his trouble. Attention and self-consciousness are also important factors. Under their influence the tremor may either increase or diminish.

Special consideration should be given to the range of individual tremors, to the rapidity with which these movements succeed each other, and to their rhythm and regularity. With regard to the first point, we can distinguish between a *rapid* and a *slow* tremor. It is rapid when there are 8 to 10 oscillations per second, and slow if about 3 to 5. There is also a tremor which stands midway between the rapid and the slow forms. With regard to the amplitude of the oscillations, we speak of a *fine* and a *coarse* tremor. The rapid tremor is usually

fine in its oscillations, and this form is known as "vibratory" tremor.

Examination with Marey's apparatus has shown that there is a tremor which exhibits an increase and a decrease of the amplitude of the oscillations repeated with a certain regularity in each tremor-movement (*allorhythmic* tremor), but this fact has not so far proved of diagnostic value.

When the tremor does not appear in simple acts of movement, it is advisable to let the hand follow an object which is being moved away from it.

The tremor which arises from exaggeration of the tendon-reflexes or of the muscle tonus, such as foot-clonus, is known as *spastic* when it is produced by the act of movement—as in active elevation of the point of the foot.

A special kind of tremor is that which affects fascicles of muscles—*fibrillary* tremor. The tremor appears in a single fasciculus, or there may be a contraction and undulation which rapidly pass from one bundle to another of the same muscle, so that the whole muscle vibrates as if a wave had passed over it. In very spare and in excitable persons, cold or the exposure of the skin may excite this tremor. It may also arise from physical overstrain. In severe degree, however, it is only observed in pathological conditions, and the excitation of cold is not sufficient to produce, although it may exaggerate the tremor. A form of tremor in which there is a persistent, marked undulation (Wogen) of the muscle substance, known as *myokymia* (Kny, Schultze) has been described. Fibrillary tremor may sometimes be produced if the nerves of the affected muscle are stimulated by electricity for some time; the muscular contraction is followed by an undulation which persists for a considerable time (Rumpf).

In estimating the significance of tremor, the fact should never be overlooked that even healthy people tremble under certain conditions,[1] in strain, excitement, and in chill, in lifting a heavy or unaccustomed burden, or after similar efforts, in violent emotion or after some excess; in convalescence from exhausting illnesses this symptom is frequently observed. In trembling from cold, it is very obvious that the will can exercise an inhibiting effect on the tremor. The chattering of the teeth in chill comes on more easily and more violently the more one gives in to the sensation and the less one endeavours to suppress the symptom. In some forms of pathological tremor, especially in hysteria, the case is somewhat similar.

Smoking and drinking may produce a transient or a persistent tremor. It is an almost constant symptom of chronic alcoholism.

The tremors which are produced by other poisons and which occur during certain diseases will be referred to in the special part (see specially the chapters on disseminated sclerosis, paralysis agitans, and hysteria). There is also a *hereditary* tremor, which is transmitted through generations and is not accompanied by symptoms of any other disease. Although senile tremor is specially characterised by a tremor of the head resembling the motion of negation, there are also other forms of essential, hereditary,

[1] Pitres speaks of physiological tremor, and Bloch and Busquet especially (*R. N.*, 1905) have succeeded in establishing this tremor by means of measuring with finer instruments. See also Steinhausen's remarks (*N. C.*, 1907) with regard to physiological and artificial tremor.

family tremor which manifest themselves in the same way. We can hardly point out any other characteristic of the latter tremors, as they vary in their nature.[1]

When we have formed an opinion as to the condition of active motor power, we have further to discover whether we are dealing with a *simple* or a *degenerative* paralysis. On this point electrical examination gives the most important evidence.

Electrical Examination

For the examination of electrical excitability the following apparatus is required: (1) an induction apparatus, which is worked by one or two cells; (2) a galvanic battery, which supplies a current up to 30 milliampères, and is furnished with an absolute galvanometer and a transformer; (3) a couple of good (not defective) conduction wires; (4) a number of electrodes of different sizes—(the largest have a surface of 50-70 sq. cm. (20-28 sq. in.), the smallest an average of 1-3 cm. ($\frac{3}{8}$-$1\frac{1}{3}$ sq. in.), the ordinary electrode measures 10 sq. cm. (4 in.). A metal brush is also necessary for some examinations.

For an electrical examination one requires in the first place to know the so-called *motor* points or sites of election, *i.e.* those points at which stimulation of the nerves and muscles gives the maximum effect. These are cutaneous areas at which the motor nerve lies very near the surface, or which correspond to the points at which the nerve enters into the muscle.

The figures (Figs. 20 *et seq.*) show the position of these points. The most important, however, should be mentioned.

That for the *facial nerve* lies directly below the external ear; it can be directly excited either below or in front of the external auditory meatus. The effect is contraction of all the muscles which it innervates.

With regard to the position of the various branches (for electro-diagnosis we distinguish a superior, middle, and inferior), see Fig. 20.

For direct stimulation of the muscle, the electrodes are applied to the skin on points corresponding to the position of the muscle; palpation is usually necessary before the most suitable point is discovered.

Of the *masseter muscles* the temporal and masseter can be directly stimulated.

The *spinal accessory nerve* is found between the trapezius and the sternomastoid, on a line which divides the angle formed by these muscles, and somewhat nearer the margin of its upper and middle third. In order to stimulate the sterno-mastoid directly, the electrode is placed about the middle of the belly of the muscle.

The *hypoglossal nerve* is deeply situated above the cornu of hyoid bone. It is difficult to stimulate and strong currents are necessary.

The *dorsalis scapulæ*, about 2 to 3 cm. (1 in.) below the point for the spinal accessory. The point for the *axillary nerve* lies somewhat lower down; it cannot always be stimulated by itself.

Erb's point lies about two fingers' breadth above the clavicle and one to the outer side of the sterno-mastoid. Stimulation of it produces contraction of the *deltoid, brachialis anticus, biceps* and *supinator longus* muscles.

Phrenic nerve. The electrode is passed deeply between the sternomastoid and scalenus anticus downwards and is also pressed inwards below the sternomastoid. When a strong current is used there is audible inspiration and protrusion of the abdomen. Stimulation can be exactly and purely carried out by means of the galvanic current.

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[1] See the recent Memoir by Gemanus Flatau (*A. f. P.*, Bd. 44).

The *long thoracic* is best reached in the axillary line. The effect of stimulation is shown in the external rotation of the shoulder blade. The muscular digitations cannot always be directly reached, and it is not always possible to say whether nerve or muscle has been stimulated.

The *musculo-cutaneous nerve* is found at the anterior margin of the axilla between the two heads of the biceps.

The *median nerve* can be excited in its whole course in the sulcus bicipitalis internus, but best so at the elbow, where it lies superficially. Flexion of the hand and fingers, especially if there is also pronation, shows that the nerve is reached.

In order to stimulate the hand muscles that are innervated by the median nerve, the electrode is applied over the wrist-joint, but with fine electrodes there must be deep pressure between the tendons of the flex. carp. radial, and the palmaris longus muscles.

Ulnar nerve.—Upper point: about 1½ to 2 cm. (1 in.) above the internal condyle of the humerus

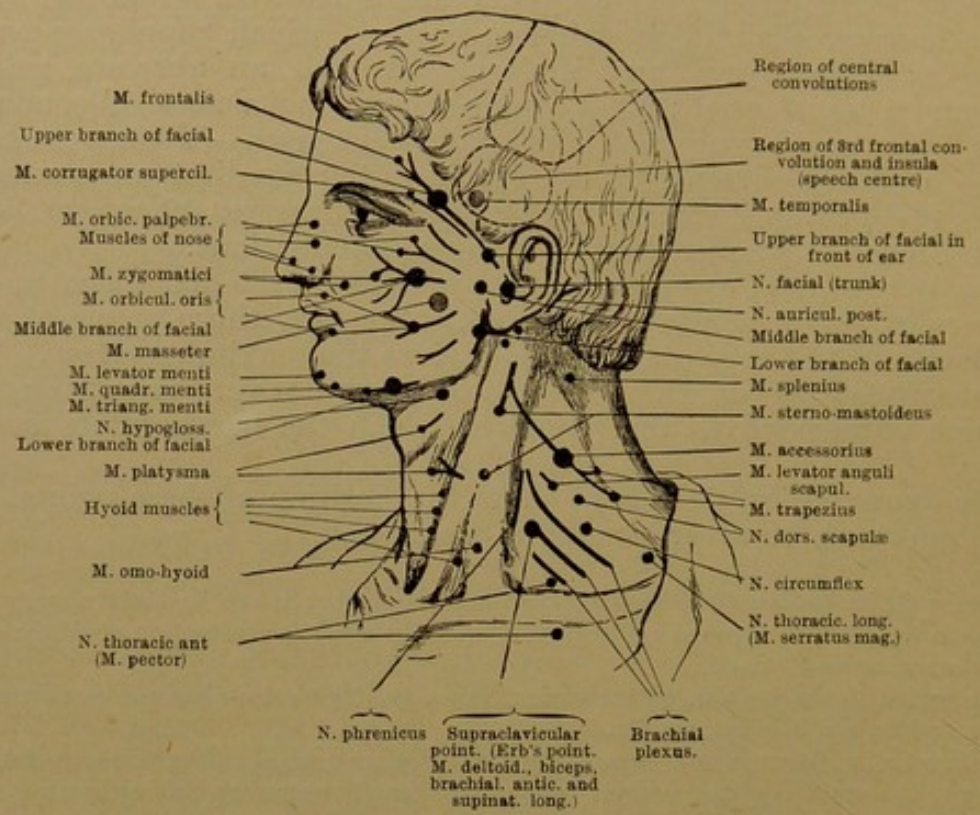

FIG. 20.—(After Erb.)

or between this and the olecranon in the fossa. The hand is flexed in the ulnar direction, the fingers flexed in the metacarpophalangeal joints, extended in the interphalangeal, but the end phalanges of the fourth and fifth fingers may be flexed (flexor. prof.) and the thumb adducted.

If the effect is limited to the small hand muscles controlled by the ulnar nerve, then the electrode is applied over the wrist-joint close to the tendon of the flexor carpi ulnaris; the position of the "accoucheur hand" is then produced (flexion of the proximal, extension of the other phalanges, adduction of the thumb), perhaps associated with an abduction or adduction movement.

The *musculo-spiral nerve* is found a little above the elbow, where a tourniquet is usually applied; it is not always easy to reach, as it lies comparatively near the surface for a short space only, and when it is stimulated neighbouring muscles (especially the triceps) may be contracted and thus lie above it. It is sometimes necessary to fix the triceps with the fingers and to raise it up in order to reach the nerve. Extension of the hand and the proximal phalanges, extension and abduction of the thumb are produced. The figure gives sufficient information as to the points to be sought in the various muscles.

With regard to the *interossei* and *lumbricales*, it should be noted that they are stimulated

jointly in the interosseous space of the dorsal surface of the hand. Feeble currents induce only lateral movements, stronger currents movements of flexion and extension.

The *brachialis anticus* muscle can only be directly stimulated if the biceps is raised with the hand and fixed, and a fine electrode pushed below this muscle.

Of the shoulder and spinal muscles, the *infraspinatus* can in many cases be directly stimulated when the trapezius is atrophied. We can usually, however, succeed in producing a contraction (external rotation of the upper arm) by employing a strong current and applying the electrode in the intraspinous fossa.

The *rhomboid muscles* are also covered by the trapezius. The *latissimus dorsi* can be directly reached. For stimulation of the *erector spinæ* a very strong current is necessary.

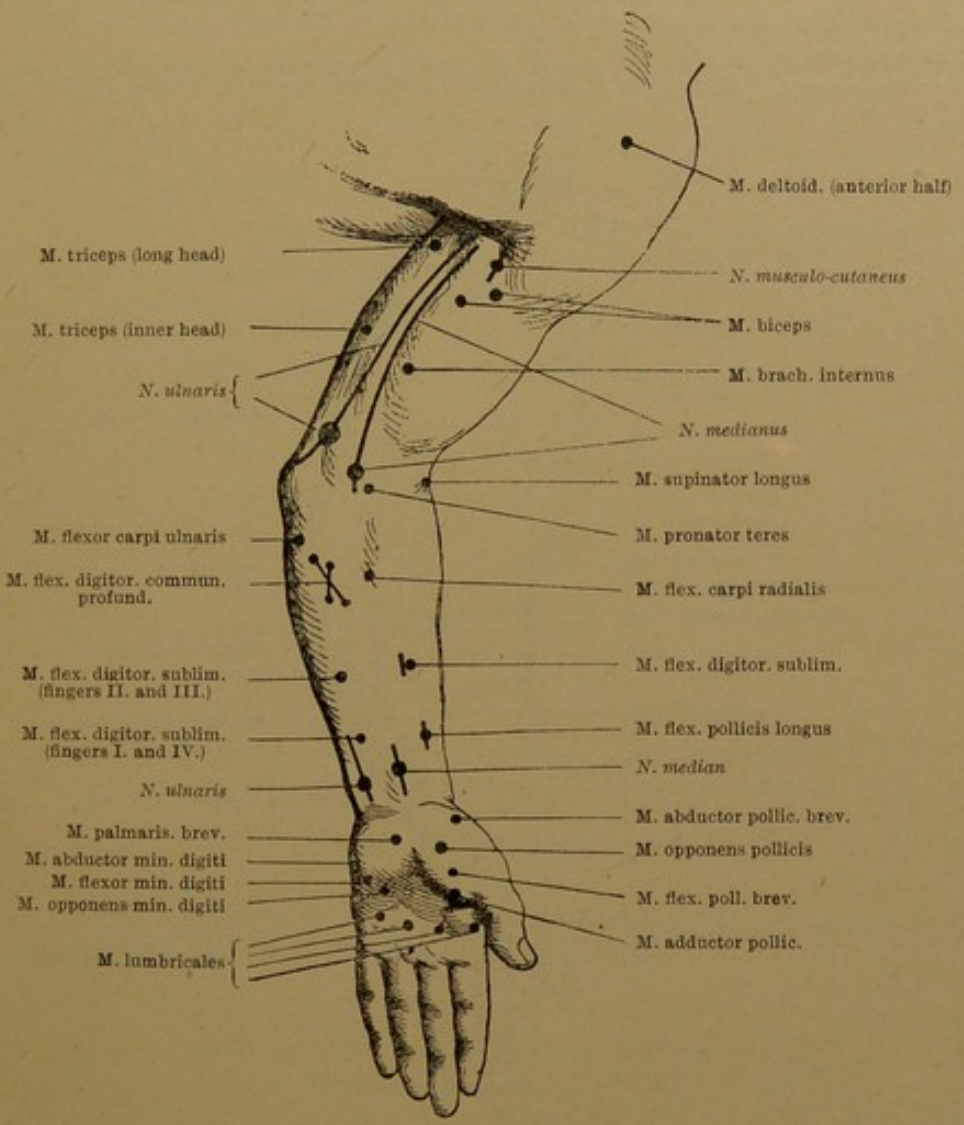

FIG. 21.—(After Erb.)

There are several points for stimulation of the *rectus abdominis*, corresponding to the outer margin of the various segments of this muscle.

The *obliquus abdominis* can be excited from the lower intercostal spaces, almost midway between the nipple line and the axillary line, and especially from the free ends of the two last ribs.

Lower Extremities.—Crural nerve. The electrode is applied somewhat external to the femoral artery in the flexure of the groin below Poupart's ligament and is pressed deeply. Stimulation produces contraction of the *quadriceps* and *sartorius muscles*. Both muscles should stand out prominently. If the sartorius alone contracts, the nerve has not been reached, and the electrode must be moved. The *ileopsoas* cannot be stimulated from the nerve (nor is it accessible to direct excitation).

Obturator nerve. Below the horizontal ramus of the os pubis (which must be felt for) at the

upper margin of the obturator foramen. The electrode should be firmly pressed directly down on the underlying tissues. If the nerve is reached, the thigh will be strongly adducted.

In stimulation of this region it may easily happen that the os pubis interrupts the contact and prevents the closing of the current. Care must be taken on this point.

Sciatic Nerve (Fig. 24). At the lower margin of the gluteus maximus between the trochanter major and the tuber ischii. This can only be well reached in spare persons and a strong current is required.

The points for the muscles (biceps, semitendinosus, semimembranosus) lie about three fingers' breadth below this point.

Peroneal nerve. Towards the outside of the popliteal space, at the inner margin of the biceps tendon; the electrode must be deeply pressed. It can also be excited at the point where it skirts round the head of the fibula, somewhat posterior to the lower margin. (Effect: dorsal flexion of foot and toes.)

Tibialis posticus nerve. About the middle of the popliteal space, or rather slightly outwards

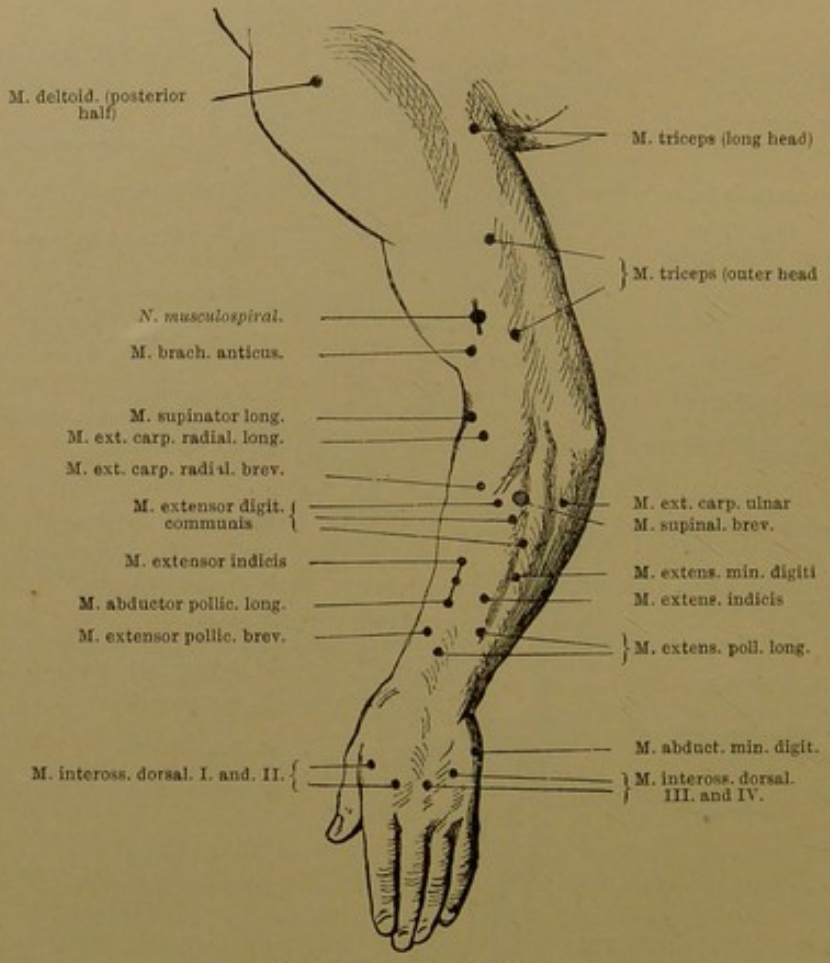

FIG. 22.—(After Erb.)

(so that it easily happens that the peroneus is involved in the excitation). The electrode must be very deeply pressed.

If we desire to produce contractions from the nerves in the muscles of the sole of the foot, the electrode must be applied behind the internal malleolus, between it and the Achilles tendon.

The muscles should be touched at the points marked on the illustration. Some difficulty is frequently afforded by the extensor longus digitorum, but with a strong current it can always be made to contract.

Rules: The cloth covering the electrodes should be thoroughly soaked. A single immersion in warm water is not sufficient. The covering should be wet through and through. It becomes so dry, especially when it has not been in use for a considerable time, that it opposes an almost insuperable resistance to conduction.

The large (50 to 70 sq. cm.) (20 in. to 28 in. sq.) indifferent electrode should be firmly applied to the lower part of the sternum or in the region of the neck. The patient himself may hold it, but it should not be moved during the examination, or it may be fixed.

The stimulating electrode, which should always be small (about 10 sq. cm.) (4 in.), is taken by the examiner in the right hand ; the thumb is laid on the interrupter, and the current being open, the electrode is placed firmly with its whole surface upon the point of contact ; the current is closed by lifting the thumb for a moment, and is then immediately re-opened. If there is no effect from the stimulation, a slight movement of the electrode is usually sufficient to produce it. If this is not the case, the strength of the current should be gradually increased.

The examination should be commenced with the faradic (secondary) current, and the minimal strength of current necessary for excitation should be ascertained.

If the muscles of the arm are to be examined, the procedure is as

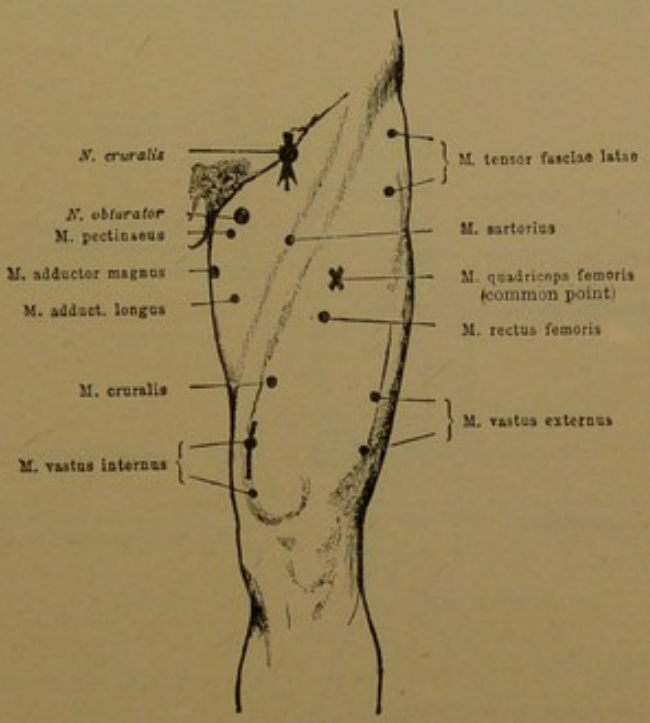

FIG. 23.—(After Erb.)

follows : First ascertain the lowest threshold of stimulation for excitation of the nerves somewhat in this order :—

Erb's point.
Musculocutaneous nerve.
Median nerve.
    Upper point.
    Lower point.
Ulnar nerve.
    Upper point.
    Lower point.
Radial nerve.

As soon as the first visible contraction appears, the necessary intensity of current has been reached, and should be noted. The figures on the scale are noted, as for instance :—

Erb's point—120 mm. R.A.
Median nerve—125 mm. R.A., etc.

Then follows direct muscular stimulation, in which a somewhat larger electrode is used as a rule. The diameter of the small electrode should always be noted, as the effect of the stimulation depends on the density as well as on the intensity of the current. ($D=\frac{I}{D}$, *i.e.* the density of the current is in proportion to its intensity and inversely in proportion to the diameter.)

The extremity should always be placed so that the effect of the stimulation may be seen at its maximum; for instance, in stimulation of the extensores carpi the hand should be flexed; in stimulation of the extensor communis digitorum the proximal phalanges of the fingers

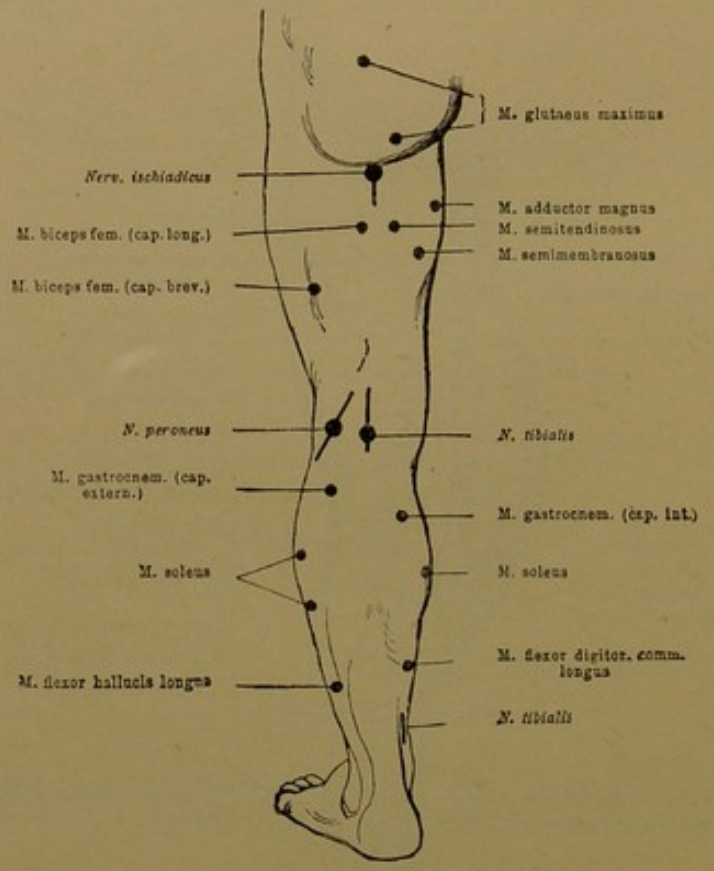

FIG. 24.—(After Erb.)

should be in the position of flexion, etc. The patient should avoid any active contraction, as he would in this way diminish the effect of the stimulus or entirely conceal it. Electrical examination is very difficult in small children, as they cannot keep the limbs still, and it is difficult to distinguish the electrical contractions from the voluntary and reflex movements. In such cases the limbs must be held firmly by another person. If there is any doubt as to whether the muscular contraction is caused by the electrical stimulation, the faradic current should be kept closed for a time. The muscles then remain in a condition of tetanic contraction.

In small children strong development of the subcutaneous fat adds to the difficulty of electrical examination. According to the researches

of C. and A. Westphal,[1] the nerves and muscles of new-born children react only to strong currents and the contractions are always sluggish. This arises not so much from great cutaneous resistance to conduction as from the incomplete development of the peripheral nerves, especially of their medullary sheaths, and of the muscles. It is only from the fifth, according to Mann [2] from the eighth week of life, that the conditions resemble those of the adult.

Narbut has recently made some further contributions to this question (*M. f. P.*, xiv.).

Galvanic Examination.—The cathode is first placed upon the nerves in the same order. Should there be any doubt as to the polarity, it can rapidly be discovered by dipping the ends of the wire in water and turning on a galvanic current of medium strength. A number of gas bubbles

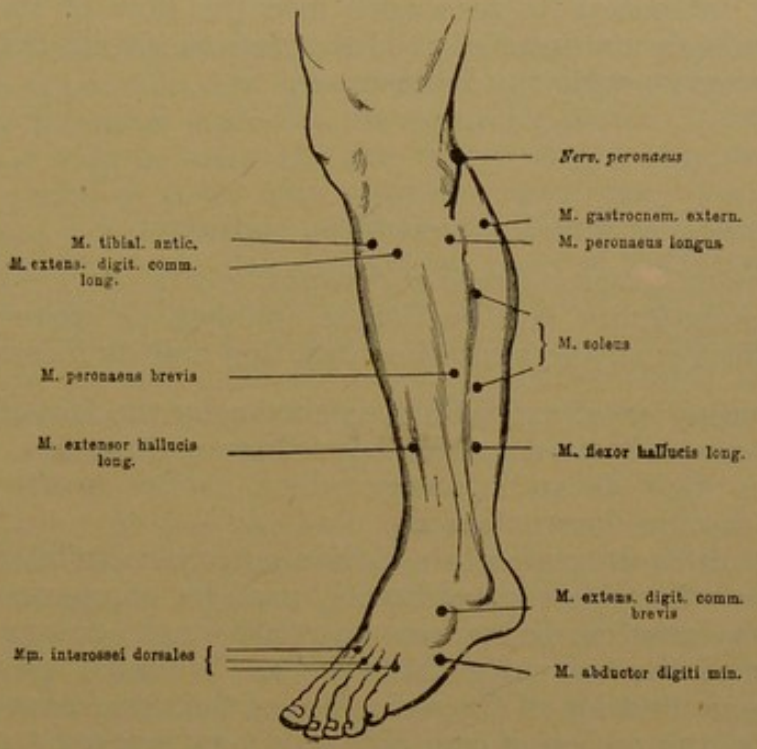

Fig. 25.—(After Erb.)

at once appear at the cathode. A weak current is then turned on and is gradually increased until when it is closed one begins to see contractions, just sufficiently strong to show whether all the muscles innervated by the nerve participate in it or not. At the moment of closure the galvanometer is thrown into the circuit and the number of milliampères indicated by the needle. The number of elements applied need not be recorded.

The result of such an examination will be somewhat as follows :—

Erb's Point, C.C.C. (cathodal closure contraction), 2·0 M.A. (milliampère).
Median Nerve, C.C.C., 0·8 M.A., etc., etc.

The contraction is normally short, like lightning.

[1] *A. f. P.*, xxvi. [2] *M. f. P.*, vii

Now follows direct galvanic muscular excitation, in which it must always be determined whether the C.C.C. proves stronger than the A.C.C. For this the commutator must be used. The condition of the opening contractions is not as a rule of much importance. In this stage of the examination it is the kind, the character of the contraction which should specially be considered. Under normal conditions it is lightning-like, and momentary, but in disease this character may be altered.

It should be remembered that the resistance of the skin to conduction diminishes under the influence of the galvanic current. Under long manipulation, when the current is kept long closed, the contraction goes on increasing in force, so that in order to calculate the minimal contraction the intensity of current must again be diminished (by reducing the current or inserting resistances). On the other hand the insertion of the galvanometer for the purpose of estimating the minimal contraction will suddenly introduce its resistance into the flow of the current and will thus diminish its intensity. In order to avoid that, a suitable galvanometer may previously be inserted.

Morbid changes of electrical excitability consist—

1. In *quantitative increase or diminution*,
2. In *quantitative* and *qualitative* changes, *i.e.* disturbances which affect even the kind of contraction and its formula.

Simple diminution of excitability is shown by the fact that a stronger current than normally is required to produce contraction, or that when the current is used at an intensity which excites healthy nerves and muscles, contraction does not occur.

This is not difficult to ascertain if the changes are unilateral, so that the muscles of the other side can be directly used for comparison. But even then errors may arise, as differences may exist in the cutaneous resistance to conduction of the two sides of the body. We must therefore make a comparative examination of the resistance of the skin to conduction. It is only where the diminution of excitability is very marked and no apparent changes in the skin are present (scars, cyanosis, œdema, etc.) that this examination may be dispensed with. If the changes affect both sides of the body, the excitability may be compared with that of healthy persons, but this is always an uncertain method and the results are not absolutely exact. Examinations have been published of the excitability of the nerves and muscles in healthy individuals (Stintzing). To apply these, however, in so far as the faradic current is concerned, we should have to make use of the same apparatus. Stintzing found that the degree of excitability of a nerve (or muscle) does not greatly differ in different persons, and that the maximum of difference for the same nerve in different individuals is about 21 mm. R.A. This always implies, of course, that Stintzing's electrode of 3 sq. cm. area is employed.

From Stintzing's tables we take the following data as regards the faradic excitability of the nerves :—

Nerve	Lowest reading	Middle reading	Highest reading	Maximal difference in the two sides of the body.
Facial	145 mm. R.A.	132–110	102	10
Accessories	..	145–130	125	10
Median (in sulc. bic. int.)	141 ,, ,,	135–110	100	12
Ulnar I. (above olecranon)	145 ,, ,,	140–120	110	6
Ulnar II. (groove between olecranon and int. condyle) . . .	..	130–107	..	11
Radial	125 ,, ,,	120–90	..	16
Cruralis	..	120–103	..	8
Peroneus	138 ,, ,,	127–103	95	13
Tibialis post	125 ,, ,,	127–95	93	10

In practice it usually happens that each examiner gradually learns to trust his own apparatus and knows from his own experience at what average on the indicator scale the nerves and muscles of healthy persons react. But it should be made the rule to lay weight only on great differences, and to judge of these only after the resistance to conduction has been taken into consideration. No faradimeter has yet been devised to determine with accuracy the absolute intensity of the faradic current.

The excitability of the muscles varies within wide limits.

Quantitative diminution of faradic excitability is shown by this, that a smaller figure on the indicator scale (Rollenabstand) is required to provoke the first obvious contraction. This may be exaggerated to such a degree that even at 0 on the indicator the contractions are absent or very feeble.

In examination of the galvanic irritability the use of the absolute galvanometer enables us to make an exact measurement and to turn to account in diagnosis even an inconsiderable quantitative diminution.

Before we go into this matter, however, it is necessary that we should learn to recognise Pfluger's law in the living. The physiological conception of the ascending and descending current is quite disregarded in this connection. We employ as a rule only one electrode for the stimulation, the other being held firmly upon the sternum. It will then appear that the nerve and muscle react only to variations of the current, specially to closing and opening of the current, and when a feeble current is used, only at the moment of closure by means of the negative pole (cathodal closing contraction or C.C.C.). When the current is increased in strength the positive pole also provokes a contraction at the moment of closing as well as of opening (A.C.C. and A.O.C.)—the former more frequently than the latter), and on further increase in the strength of the current, at the moment of closing by means of the cathode, a tetanus takes place, cathodal-closure tetanus (C.C.Te.), *i.e.* the muscles persist in a condition of tetanic spasm so long as the current remains closed. Finally there occurs a C.O.C. and A.C.Te., but with a current of such intensity that it can usually be disregarded; on the other hand an A.O.Te. (anode-opening tetanus) cannot be provoked in healthy subjects. These symptoms can be simply demonstrated on any healthy person. The ulnar nerve is chosen for example; the opened interrupting electrode is firmly placed on the nerve, without displacing it; a feeble current is applied by means of the cathode,

the minimal C.C.C. is determined, the current is reversed, and the other phases of Pfluger's law similarly obtained. In order to produce C.O.C., we must wait until the tetanus caused by the closure has passed, or endeavour to evade it by gradual introduction of the current.

In direct stimulation of the muscles, Pfluger's law remains as a whole unaltered, except that the muscle reacts less to the opening stimulus, and it sometimes also happens that the normal muscle responds to A.C. with as strong, or an even stronger contraction than to C.C.

Dubois' view that the effect of the stimulation depends essentially on the voltage can no longer be maintained (Mann, Hoorweg).

Quantitative diminution of the galvanic excitability is shown by this, that the C.C.C. appears only when the intensity of the current is greater than normal, *i.e.* with a greater number of milliampères, and that the higher phases of Pfluger's law (C.C.Te., etc.), which require a strong current even in normal cases, cannot be provoked.

Erb has shown that, in the most superficially placed nerves, the first C.C.C. appears at 0·5–2·4 M.A.

We take the following data with regard to normal galvanic excitability from Stintzing's table.

Excitability (C.C.C.)

Nerve	Lowest reading in milliampères	Middle reading	Highest reading	Maximum difference between two sides
Facial	0·8	1·0–2·5	2·8	1·3
Accessory . . .	..	0·01–0·44	0·6	0·15
Median	0·27	0·3–1·5	2·0	0·6
Ulnar I.	..	0·2–0·9	1·3	0·6
Ulnar II. . . .	..	0·6–2·6	..	0·7
Radial	0·7	0·9–2·7	3·0	1·1
Crural	0·3	0·4–1·7	2·6	0·6
Peroneus . . .	..	0·2–2·0	2·7	0·5
Tibialis post . .	..	0·4–2·5	..	1·1

If we use the same order of examination, Stintzing's table may serve as a standard for the condition of excitability, but here also only the marked deviations should be taken into consideration and regarded as pathological. Even the circumstance that the thickness of the skin varies greatly in different individuals and that the nerves lie nearer the surface in some than in others gives rise to certain differences. If, for example, the facial on the healthy side first shows C.C.C. at 1 M.A., and that of the affected side at 1·5–2·0, I should lay no practical weight upon this fact, and would attach significance only to more considerable differences. It is always necessary also first to make sure whether no other circumstances arising from the method of examination are playing a part and thus simulating changes in excitability.

Example of quantitative diminution of excitability :—

Atrophy of the right arm from want of use.
Large electrode of 70 sq. cm. (28 in.) upon the sternum.
Electrode of 3 sq. cm. for stimulation of the nerves, one of 10 sq. cm. (4 in.) for stimulation of the muscles.

	Right arm				Left arm			
Erb. P. Nerve	90	R.A	5·0	M.A (C.C.C.)	130	R.A	3·0	M.A. (C.C.C.)
Median ,,	98	,,	6·0	,, ,,	120	,,	1·5	,, ,,
Ulnar ,,	90	,,	4·0	,, ,,	140	,,	1·0	,, ,,
Radial ,,	80	,,	6·5	,, ,,	110	,,	2·5	,, ,,
Deltoid Muscle	85	,,	14·0	,, (C.C.C., A.C.C.)	100	,,	8·0	,, ,,
Biceps ,,	100	,,	7·5	,, ,, ,,	130	,,	3·0	,, ,,
Sup. long. ,,	90	,,	8·0	,, ,, ,,	120	,,	5·0	,, ,,
Extens. carpi rad. Muscle	100	,,	7·0	,, ,, ,,	110	,,	4·5	,, ,,

etc.

Simple diminution of excitability takes place in atrophy from disuse, hysterical muscular atrophy, and in primary muscular affections, *e.g.* progressive muscular dystrophy, myositic atrophy, atrophy resulting from compression of the muscle from injury, tumour, etc., in muscular atrophy accompanied by affections of the joints, and finally in slight peripheral neuritis. In chronic spinal diseases with muscular atrophy there has been observed in rare cases a diminution of the excitability which is not the result of reaction of degeneration, but is a real quantitative decrease. I have found this unusual condition in some cases of gliosis and tumour of the spinal cord.

Quantitative increase of the electrical excitability is but rarely observed. It manifests itself by the first marked contraction appearing while the strength of the current is still below normal, at 0·05–0·1 M.A., for example, by an increase of the intensity of the contraction with a current of normal strength. This is pronounced only on galvanic examination, and is specially characterised by the fact that the higher phases of the contraction (Pfluger's law) (C.C.Te., C.O.C.) occur even when a current of comparatively feeble strength is used, and that a reaction can be provoked which we never observe in normal nerves, namely the anodal-opening contraction.

This exaggeration is markedly present only in one disease (tetany), and will be described in that connection.

Reaction of Degeneration (R.D.)

From a diagnostic point of view this is the most important form of alteration of irritability.

Complete reaction of degeneration is characterised by the following symptoms :

(1) The excitability of the nerves by the faradic current is abolished ;
(2) The excitability of the muscle by the faradic current is abolished ;
(3) The excitability of the nerves by the galvanic current is abolished ;
(4) The excitability of the muscles by the galvanic current is (*a*) increased, or (*b*) modified in such a way that the contraction is slow and that the A.C.C. exceeds the C.C.C. in force (A.C.C. > C.C.C.). (The C.O.C. is relatively increased more than the A.O.C., may equal or even exceed it—a fact which may as a rule be disregarded).

Of these characteristic symptoms of R.D. the exaggeration of the galvanic irritability is found only in the first stages and it gradually diminishes, so that eventually (though often even after the lapse of years) a feeble and very sluggish A.C.C. remains, with strong currents only, as the sole residue of the disturbance. This circumstance is frequently overlooked by beginners. When the contraction does not appear, a very strong current should be used, the commutator should be turned, or the electrode may be passed slowly over the skin, close observation being directed to the muscle,—not to the segment of limb which is to be moved, as the locomotor effect of this contraction is often absent or very slight,—in order to detect the minimum contraction which passes gently over the muscle like a scarcely visible wave. Even the preponderance of the A.C.C. over the C.C.C. should not be taken as an axiom, as the C.C.C. occasionally prevails in reaction of degeneration, and *vice versâ*, the C.C.C. may preponderate in normal muscles. We may therefore have reaction of degeneration even when the C.C.C. equals or exceeds the A.C.C. The *most important factor is the slow contraction.*

Besides the complete, there is also a *partial reaction of degeneration* characterised by slight diminution of the irritability of the nerves, the faradic muscular excitability being also only diminished or abolished, whilst in direct galvanic stimulation the sluggishness of the contraction (and the reversal of the contraction formula) become evident.

Between partial and complete R.D. there are all possible stages of transition. Stintzing, for instance, distinguishes thirteen varieties of R.D. Amongst others the muscular contraction which follows stimulation of the nerve may be sluggish (partial R.D. with indirect slowing of contraction), but such peculiarities have no real diagnostic value.

Slowness of contraction in faradic excitation of the nerves and muscles has also been observed (Remak), but this may be the effect of cold, for instance, and has no practical interest.

It has several times happened to me that in the examination of a patient at the Polyclinique, I have found a slow contraction which at my lecture a short time later I could not demonstrate to my students, because in the meantime the disturbance which had been caused by cold only, had disappeared in the warm room.

It is easy to understand that the diagnosis will be all the more difficult the less complete the reaction of degeneration, and that these forms of partial R.D. are particularly easy to overlook. Special attention should be given to the slowness of the contraction in direct galvanic stimulation, so that this condition may be recognised by comparison with the healthy muscles.

The reaction of degeneration is the most certain and constant sign of degenerative processes in the motor nerves and muscles. It can be experimentally produced by solution of the continuity of the nerves (by section, etc.). The processes of degeneration in the nerves and muscles proceed parallel with these changes of electrical excitability, and as these develop in their full extent only at the end of the first or the beginning of the second week, the R.D. should not be expected before the end of a week, other conditions being equal.

It is present in all severe diseases of the peripheral (motor or mixed) nerves, and in affections of the anterior horns and anterior roots, wherever, therefore, the trophic centres of the muscles are affected or the

conduction tracts between them and the muscles (anterior root, peripheral nerve) are affected by a deep-reaching morbid process.[1]

Such diseases are—

I. Diseases of the *Anterior Horns :*
   1. Acute anterior poliomyelitis.
   2. Subacute and chronic poliomyelitis.
   3. Amyotrophic lateral sclerosis.
   4. Spinal form of progressive muscular atrophy.
   5. Spinal gliosis.
   6. Diffuse cervical and lumbo-sacral myelitis (with involvement of the grey matter).

I.a. Diseases of the *nuclei of the bulbar nerves* corresponding to those of the anterior horns (progressive bulbar paralysis, acute inferior poliencephalitis, etc.).

II. Diseases of the *Anterior Roots :*
   1. Compression from tumours and the tumour-like thickening of the meninges ; as in syphilis, cervical hypertrophic pachymeningitis.
   2. Compression in the intervertebral foramina in diseases of the spinal column (caries, tumour, fracture, dislocation).

III. *Severe Affections of the Peripheral Nerves :*
   1. Traumatic (section, severe bruising, pressure by tumour).
   2. Rheumatic (facial paralysis, for example).
   3. Toxic and infections.
      (*a*) Lead paralysis.
      (*b*) Alcoholic paralysis.
      (*c*) Arsenical paralysis.
      (*d*) Infective forms of multiple neuritis, etc.

Wherever incomplete R.D. is present, we must remember that regeneration and involution may already take part in the changes. It is also important to know that in recovery of function of a muscle it frequently cannot be stimulated electrically for a considerable time after the reappearance of voluntary contractions.

Other more uncommon forms of changes of irritability, such as the *myotonic* and *myasthenic* reaction will be considered in the special part of this work.

Franklinisation has hitherto acquired no value in electro-diagnosis. With regard to the value of the Leyden jar discharge in electro-diagnosis, we have the experiments of Zanietowski, Ziehen-Hoorweg, Mann, T. Cohn, and especially of Bernhardt (*Z. f. E.*, vii., and *N. C.*, 1906), who ascribe some value to the method.

Other points which aid the diagnosis are afforded by—

Examination of the Mechanical Excitability of the Muscles and Nerves

If the muscles of a healthy person are tapped with a percussion hammer, there follows either no sign of contraction or merely a feeble, short contraction of the muscle that has been struck. Here and there, especially in

[1] An incomplete R.D. is to be found only in isolated cases of primary muscular diseases (dystrophy, trichinosis).

the biceps, we may succeed by strong stimulation in producing a local swelling, an "idiomuscular" contraction (Schiff, Auerbach). In emaciated individuals, especially in consumptives, this mechanical irritability is markedly aggravated; contractions can be provoked at any point of the muscle. If, for instance, we draw the handle of the percussion hammer over the pectoralis major, the muscle bundles will contract one after the other as visibly as if we had run over the strings of a harp. *Exaggeration of the mechanical excitability* is also not infrequently present in those nervous diseases which are accompanied by a general increase of irritability (neurasthenia, traumatic neuroses, etc.). Chronic alcoholism may also give rise to this symptom. I have sometimes found it specially marked in chronic muscular rheumatism.

In pathological conditions this idio-muscular contraction may follow the slightest stimulation, and the muscular swelling produced by the tap may even persist for many seconds. In some cases I have also seen the swelling pass over the whole belly of the muscle. Not much is as yet known as to the value of this symptom (see the recent work of H. Curschmann, *Z. f. N.*, xxviii.).

The mechanical irritability of the muscles undergoes a special modification in Thomsen's disease (see chapter on this subject).

In conditions of degenerative atrophy the contraction produced by mechanical stimulation is, in the stage of galvanic over-excitability, sometimes markedly prolonged (mechanical R.D.).

The peripheral nerves may also in part be excited by mechanical stimulation. This examination must of course be carefully carried out (Dejerine). If the ulnar nerve is tapped with the percussion hammer, or if it is rolled by the finger against the bone, a slight contraction of the eorresponding muscle occurs in most people. Under pathological conditions this excitability may be greatly increased, most constantly and markedly so in tetany.

There is also an exaggeration of the mechanical excitability of the sensory nerves.

Examination of the Gait

Affections of the Gait.—Examination of the simple active movements while the patient is in the dorsal position is not sufficient, and should be supplemented by that of the more complex motor functions of standing and walking. Although the condition of the motor power is by no means the only thing that is concerned in these, since the gait is sometimes affected by other causes as well, yet the most important of them may be considered here. It is well from the first to remember that a number of peculiarities of gait are really physiological. No one person walks exactly like another. The individual variability is so great that we can almost speak of "a physiognomy of gait." We not infrequently see, for instance, swaying of the trunk, great raising and lowering of the pelvis in walking (in corpulent women, for example). Such peculiarities must always be taken into consideration before we assume anything morbid.[1]

[1] Precise examination as to the mechanics of normal and pathological gait by exact measurement of the length of step by graphic, chronophotographic, and kinematographic methods, such as used by Vierordt after Weber, Richer, De la Tourette, Marey, Marinesco, Mönkemöller-Kaplan,

Gait in Simple Paresis.—The affection of the gait which is caused by simple muscular weakness, is indicated by slowness of movement in walking and by a shortening of the step. The active movements of the legs are also less free, whilst the muscular weakness may cause an excessive, but purely mechanical flexion at the knee-joints ("Einknicken"). When this condition is marked, the patient shuffles laboriously along, and requires a support for the upper limbs. When these are strongly supported he can still move about even although the legs are completely paralysed. We might then say that the patient walks with his arms.

Gait in Partial Paresis.—This gait will be materially modified if only a few muscles or muscle groups are affected (see chapter on Function of Muscles). A specially typical and common form is that caused by bilateral peroneal paralysis. The foot of the advancing leg hangs down with the toes pointing to the ground. The leg is thus lengthened, and the patient has, in order to compensate for this, to over-flex the hip- and knee-joints. A double sound is made as the foot comes to the ground. The gait resembles that of a horse (*steppage gait*).

Gait in Spastic Paresis.—If the muscular weakness is associated with muscular rigidity, a further impediment to walking is added. The rigidity is manifested by slowness of movement and limitation of excursion. The leg is moved forwards as a whole, like a rigid column. The toes remain firmly in contact with the ground in a specially characteristic manner, and cause a shuffling noise. This is caused by the fact that the contracture of the gastrocnemius muscle can only be overcome slowly and with difficulty by the elevating of the foot and the toes. The defective movement in the various joints of the limb is usually compensated by elevation of the pelvis on the side of the leg which is advancing. When this condition is very marked the toes are to a certain extent adherent to the ground, remain constantly in contact with it, whilst the patient works himself forward with short steps. If, as is frequently the case, the contracture of the adductors of the thighs is very great, the knees will rub against each other and the limbs are crossed in walking.

Ataxic Gait.—In pure (spinal) ataxia the gait is typical and is specially characterised by the excess in excursion of the movements. The swinging leg is over-flexed in the hip-joint and is rotated outwards. This movement is abrupt, flail-like; the front part of the foot is at the same time strongly raised and then the leg is thrown heavily down, so that the heel stamps on the ground, and the knee of the other leg is thus subjected to abnormal pressure. The gait is uncertain, the legs kept widely apart, and the patient gazes fixedly at the ground and is in dagner of falling as soon as he lifts his eyes.

The *cerebellar-ataxic gait* is a modification of the ataxic. Two forms, which may sometimes be combined, may be distinguished: (1) In one form arising from (vertigo and) disturbance of equilibrium, the gait has a great resemblance to that of a drunken person with falling, stumbling, and swaying from side to side. (2) The second form is caused by the ataxia of movement. The patient walks with the legs widely apart and stamps his feet, but does not sway excessively. He stands with his legs far apart, and as he does so one can detect a constant undulation, a

Jendrássik (*A. f. A.*, 1904), and others have not proved to be of any great value for practical diagnosis, and need not be further described.

momentary tension of the extensors of the foot and toes steadily repeated. A sharp distinction between this variety and the spinal-ataxic form is only possible when the form first described is not associated with it.

Modification of gait *caused by Tremor*.—Tremor may affect the legs and become so exaggerated during walking that each simple action of the muscles is accompanied by a tremor. We observe suggestions of this in the spastic gait, where the placing of the point of the foot on the ground gives rise to an ankle clonus, and may thus cause a spastic tremor, consisting usually of only a few jerks which may shake the whole body. Under other conditions (multiple sclerosis) the whole extremity may shake or show (as in hysteria) a complete clonic spasm.

If the tremor affects mainly the trunk and head and consists in large oscillations (as is the case in multiple sclerosis) marked uncertainty of gait may appear. The patient may fall to the ground after a few steps.

A combination of the various forms is not unusual, especially of the spastic-paretic with the cerebellar-ataxic or with the simple ataxic gait.

Modifications of the gait arising from *pain* are very numerous. It is impossible to describe the various forms as there is often a subjective element in them. If the pain comes on when the toes are used, the patient avoids this movement and walks mainly on the heels, and *vice versâ*. Frequently the whole sole is sensitive in walking, so that the patient either avoids walking or walks only with the greatest care, seeks to avoid every energetic movement, and distorts the face with pain at every step. The gait is modified according to the site at which the pain is felt, and this possibility should be remembered whenever walking is visibly affected.

The gait may also be affected by the *idea* that walking is impossible. The loss of the power to walk, although the motility of the legs is unaffected when patient is in the recumbent position, is known as *abasia*.

The tendency to walk or run backwards occurs in paralysis agitans, and much less frequently in hysteria and traumatic neuroses. I have observed it in one unusual case of hereditary chorea.

Further peculiarities, such as the gait in chorea, paralysis agitans, etc., are treated in the special part.

Examination of the Sensibility

The simplest methods of examination are the best. All the æsthesiometers may be dispensed with. The exact measurements employed by physiologists are as a rule unsuited for clinical examination. For testing the various forms of sensibility we use a brush or a pad of cotton wool, a needle with a good point, a test-tube filled with hot and another with cold (or iced) water.

It is necessary to know that a healthy person distinctly feels a slight touch with a soft object, a brush for instance, or a light tap with the finger on every part of the surface of the body. It is only where there are scars or callosities, as for instance in the region of the balls of the toes in many individuals, that such feeble cutaneous excitement is unperceived. Pressure with a hard substance (with the handle of the brush or the finger) can be distinguished from the touch of a soft object all over the skin, except perhaps where there is callous, thickened epidermis.

Pressure affects the deep parts as well as the skin, and is therefore a combination of the senses of pressure and of contact (Strümpell, Head).

In making the examination we first close the eyes of the patient (preferably with the thumb and fore-finger) and then lightly touch the skin on the various parts of the affected region ; the patient should say "now" the moment he is touched. In order to discover whether his failure to respond arises from affection of sensation or from inattention, some other part of the body which is not included in the zone of the anæsthesia should also be tested. Thus errors are avoided. The differences in sensitiveness of the various cutaneous areas as tested by fine methods (Frey and others) may be practically disregarded in ordinary diagnosis.

When the skin is touched, and especially when it is pricked by a sharp needle, the sensation of pain is produced in all healthy persons at any site. The intensity of this sensation varies in each individual. All parts of the body do not possess the same degree of sensitiveness to pain, the scalp, the back of the tongue, and the dorsal surface of the fore-arm being less sensitive than other parts. The sensitiveness to painful stimulations is also less developed in early childhood.

When the sensation of pain on the prick of a needle is diminished on account of morbid conditions, it can sometimes be aroused by a scratch, the point of the needle being passed over a long strip of the skin. Here, however, the result is produced not by a single stimulation, but by a summation of stimuli.[1] The same may almost be said with regard to the *faradic brush*, which may also be used for testing the sense of pain. A current is used of such an intensity as would produce a sensation of pain in one's own skin or in the unaffected parts of the patient, and a comparison is made with the sensitiveness of the areas of the skin involved in the disease. The sudden closure of a strong galvanic current while the brush is used as a cathode, may cause a very severe pain which is absent only in marked sensory lesions.

The electro-cutaneous measuring of the sensibility by determining the figure on the indicator scale of the faradic coil at which perceptible feeling of pricking is produced in the skin has no advantage over the simpler methods.

If the point of a needle is distinguished from its head, this will show the quality not of one sense only, but of several : touch, pressure, pain, as well as the capacity to recognise impressions according to their extent (sense of space), as the point of the needle stimulates a smaller area of skin than its head. If we use an ordinary pin, then the stimulation with the point must be very distinctly marked in order to produce a definite difference at every site. Otherwise, even in healthy persons, the test will not be an exact one, especially in the dorsal region.

We do not possess exact methods for the measurement of the sense of pain. Pain meters (algesimeters) have been used in the form of pincers by which a graduated pressure can be exerted, but they have not proved useful in practice. Moczutowsky has recently recommended such an apparatus and he has used it to investigate the degree of sensibility to pain in the various cutaneous areas of normal individuals. He finds that the sensitiveness is least on the skin of the pelvic and gluteal regions, whilst it increases gradually from here upwards to the head and fingers

[1] The symptom that rhythmically repeated, moderate or feeble stimulation produces in some diseases periodic sensations of pain, may also be ascribed to summation (Nauyn).

and downwards to the toes. The skin of the forehead is the most sensitive. He gives exact numerical data as to the sensitiveness to pain of the various areas of the skin, but further tests will have to be made before these data can be employed. Other algesimeters are recommended by Bechterew and Thunberg. Ziehen also (*B. K. W.*, 1904) advises finer methods for examination of the sensibility.

The endeavours of Sticker to find other objective means of representing the sensibility have led to nothing. We can only draw attention here to the interesting observation of Veraguth (*M. f. P.*, xxi. and xxiii.), with regard to the so-called psycho-galvanic reflex phenomena.

In order to test the sense of temperature a vessel filled with cold or hot water is brought into contact with the skin. It is advisable not to use very high degrees of temperature which may cause pain and make it difficult to distinguish the qualities of sensation. It should be remembered also that warmth penetrates through the skin gradually, and therefore the contact must be maintained for several seconds. If the skin is very cold, from frost, etc., the sensitiveness to temperature is thereby diminished.

Goldscheider's (*A. f. O.*, xvii.) more exact method of testing the sense of temperature is not usually of value at the sick-bed on account of its minuteness, and the demands which it makes upon the attention of the patient. But the facts upon which it is based are so important that they must be referred to. The skin possesses special sensory nerves for pressure, cold and heat (Blix, Goldscheider, Alrutz). These terminate at distinct points in the skin, the points for pressure, cold and warmth. At the pressure-points the sensitiveness for mechanical stimulations is particularly fine. At these points as well as on the skin between them, every stimulation of a certain intensity produces pain. There are no special pain-points. Pin-pricks are certainly felt with special intensity at some points, but this is probably on account of the nerve-ending being specially exposed (Goldscheider). We must not, however, from these facts infer the existence of specific nerves for pain. Frey thinks, however, that there is pain conduction in connection with some of the end-apparatus. We do not know for certain which nerve-endings are related to the various kinds of sensation.

The points for cold and heat are not equally distributed all over the skin. The sensitiveness to temperature is therefore unequal in the different cutaneous areas. According to Goldscheider these differences are almost constant, and he has marked out on the surface of the skin the topographical differences which he divides for sensitiveness to cold into twelve, and to warmth into eight gradations. His method of examination consists in comparing the sense of temperature in the cutaneous areas in question with a normal area of the same gradation, and where the sensitiveness is unequal he tests the lower gradations which correspond to the sensitiveness of the cutaneous area to be tested. For this examination he uses a solid metal cylinder with a basal surface of about 1 sq. cm., and a handle of ebony. The cylinder is brought to the required temperature by plunging into cold water or heating over a lamp.

Head (*Br.*, 1905) has done special service in showing that affections of the temperature sense are often revealed only by the inability to distinguish medium temperatures (see special part).

Examination of the *sense of locality* (Ortssinnes) may be limited to asking the patient to indicate the site affected by the stimulation. We should remember that even normal persons cannot always give accurate answers. An error of 1 cm. is not pathological in the hands, and in the arms and legs the error may amount to 2 to 4 cm. (according to Ziehen and Loewy it may even reach 6 to 7 cm. in the upper arm and thigh). Precision of localisation also depends somewhat on the intensity, duration, and after-duration of the stimulus. Localisation is exact in the face of healthy subjects. The sense of locality is specially slightly developed in the female genitals (Calman) and in the conjunctiva and cornea (Frey.[1])

[1] On the question of the sensibility of the cornea, see also Cabannes et Robineau (*R. n.*, 1904).

To determine the sense of locality, another method may be followed, that of examining the capacity to distinguish two points applied to the skin at some distance from each other. This capacity varies greatly at different points of the skin. On the tip of the tongue, for example, the two points of a compass placed 1 mm. apart from each other can be distinguished, whilst on the back they must be 65 mm. apart.

The use of this method, which is a very uncertain one, can usually be dispensed with, but Head has again recently advocated it (*Br.*, 1905 and 1907). The more elaborate methods of examination recommended by Frey can hardly be used with patients.

Although the sensibility of mucous membranes differs in some respects from that of the skin (Frey, Kiesow and Hahn, etc.), the methods of examination are the same, and they enable us to discover any affection of it, at least if it be well marked.

It is absolutely necessary to include in the examination the condition of the sensibility of the deep-lying parts (joints, fasciæ, muscles). For this purpose we test the *sensation of passive movement* and of *position, i.e.* we endeavour to ascertain whether the patient recognises slight movements which we make with his limbs and appreciates the attitude into which we place them. It is necessary to make use of movements of very slight extent, such as bringing the great toe out of its flexed position and slightly extending it, avoiding in so doing any pressure upon the skin which might give the patient any information. If, for example, in extending the side which is flexed, or in flexing the side which is extended, the skin is pressed, he can draw his conclusion as to the direction of the movement. We should therefore take the great toe (the end phalanx) between the thumb and fore-finger and exercise upon it in every movement an equal pressure from above and below. The sense of movement should be tested in the other joints in the same way. The patient should not accompany the passive by active movements, as he often tends to do.

With regard to the sense of movement of the various joints, as shown in the recognition of passive movements at a certain degree of the angle of rotation, Goldscheider has given the following data :—

Second interphalangeal joint of the fore-finger	1·0°–2·0°
First ,, ,, ,, ,,	0·7°–1·0°
Wrist-joint	0·3°–0·4°
Shoulder-joint	0·2°–0·4°
Hip-joint	0·5°–0·8°
Knee-joint	0·5°–0·7°
Foot	1·0°–1·03°
Metatarsophalangeal-joint of great toe	2·0°– —

He has also constructed an apparatus (movement-meter) for the examination of this sense, which, however, we can quite well dispense with at the bed-side, for the sense of movement is so fine in normal persons that " rotation which is hardly visible or palpable to the examiner will produce a sensation."

The patient should also be asked to shut his eyes and to describe, either verbally or by a motion of his hand, the position and site into which his limbs are placed. For example, we raise the leg from the bed, rotate it inwards and ask the patient to indicate with his fore-finger the position of the great toe in space. If one side only is affected, the other being

healthy, he should imitate the position given to the limb with the limb of the other side.

It is usually possible to dispense with examination of the sense of weight (Kraftsinnes) [1]—the power to estimate the weight of a lifted object. The mode of examination is as follows: A towel is attached to the extremity so that a weight can be placed in the hanging loop. For the lower limb, a stocking with a pocket sewn to it is recommended. We then try to ascertain what differences of weight can be appreciated by the patient. Healthy persons differ so much in their power of estimating weights that it is not always easy to assess the results of the examination of a patient. One succeeds best where the corresponding extremity of the other side can be used for comparison. The sense of weight is finer in the upper than in the lower extremities. In the upper differences of weight of one-tenth can be distinguished with certainty. Thus 90 grms. can be distinguished from 100 grms. In the legs Hitzig could always correctly distinguish 0 from 100, but not from 90 grms., 200 from 250, and differences of weight from 100 up to 1000 grms. Chavet states that by the upper extremities 1 grm. can be felt as a weight, but by the lower nothing less than 30 to 40 grms. As, however, the power of estimation varies within wide limits even in healthy persons, only marked deviations should be regarded as pathological. Hitzig [2] uses for this examination balls of the same size and of weight varying according to the amount of lead they contain (Kinesiaesthesiometer).

The *stereognostic sense* should be examined in many cases. To do this methodically geometrical objects should be used; these are best made of wood, spheres, hemispheres, cones, cubes, octagons, etc., 3 to 6 cm. in diameter. A normal individual will recognise the object so soon as it is placed in his hand, though his eyes are shut.

We may also use any small, easily recognised object, such as a piece of money, watch-key, a button, etc. Here however we have to do not with one special sensation, but with a combination of several—we might almost say of most qualities of sensation, specially with the pressure-sense, the sense of passive movement and the sense of the position of the limbs. Stereognostic perception may be abolished whilst the sense of touch, pain, and temperature are retained. On the other hand I have very frequently found it affected along with disturbance of the sense of localisation. In the recognition of objects through touch, associative and other mental processes (reproduction of memory-pictures) play a part along with the elementary sensation, and we can therefore understand that this complicated act may be affected not only by the absence of simple sensation, but in other ways (see chapter on brain diseases). It should be further remembered that conditions of paralysis make the examination difficult, as much more accurate ideas of an object are obtained from active than from merely passive palpation (Markora).

Some years ago Egger carried out under Dejerine's direction investigations from which he concluded that sensibility of the bones (or the periosteum) may be examined by means of a tuning-fork, and used as an aid to diagnosis. Rumpf [3] had studied this question even earlier, and Treitel as well as Bonnier had recognised in the "sensation of vibration" a special quality of sensation. These data had received little

[1] It is better to avoid the expression "muscle-sense." According to Goldscheider's definition a number of sensory-sensations are included under this idea: 1. The sense of passive movement (Bewegungsempfindlichkeit). 2. The sense of active movement. 3. The sense of weight and resistance (Kraftsinn). 4. The sense of localisation. According to Curschmann (*N. C.*, 1905) the sensations which arise from contraction of the muscle, the muscle sensation in the strict sense, can be produced and measured by galvanic stimulation, as the lower stimulation threshold of this sensation corresponds to the minimal contraction in this excitation.

[2] *N. C.*, 1888.

[3] *N. C.*, 1889.

consideration (compare third edition, p. 51) until they were confirmed and completed by Seiffer and Rydel.[1] They bring *pallæsthesia*, as they name this feeling of vibration, into relation to the deep sensibility, but they do not attribute it to the bones alone. It shows no constant relation to the other qualities of sensation, but is found to be specially often affected or abolished where the sense of position is destroyed and where ataxia is present, whilst in other cases it is associated with analgesia and thermanæsthesia. Seiffer and Rydel recommend Gradenigo's tuning-fork of 64 oscillations for the accurate testing of the vibration sense. I have made similar examinations with a tuning-fork of 128 oscillations and obtained similar results.

During the last few years Minor (*N. C.*, 1904), Goldscheider (*B. k. W.*, 1904), Neutra, Pelnar, Stcherbak, Williamson, Herzog, and Stern have studied this method of examination and have found that it is not a matter of any specific sensation, that is to say of one exclusive sensation, but rather of preponderating bone conduction. The sense of vibration should not be confused with osteoakusis.

In all cases where the sensibility is being examined it should be remembered that the individual has to be attentive to what is being done. If he is unintelligent or inattentive, or if there is any disease which affects the sensorium, special skill and perseverance are required to form an opinion which is to be of any value as to the condition of the sensibility. During the examination a stimulus should be now and again applied to healthy points on the skin in order to discover whether the patient is attentive. To keep his attention alert, he may be asked to indicate with his finger the places which have been touched or pricked. Even with small children this method is advisable, as it interests them far more to point out the part which is excited than simply to tell the moment they are touched. The factor of fatigue has also to be taken into consideration, and the examination should not extend over too long a time. Pain and excitement especially cause such a distraction of the attention that as a rule we have to be satisfied with a superficial examination.

On the other hand it cannot be too strongly impressed that too much weight should not be laid upon the first test by pricking. It frequently happens, even in healthy people, that the first prick—especially in the leg—produces no pain, whilst all the successive pricks are painful, or that the application of " heat " and " cold " to the lower extremities at first gives rise to confusion. There are certain affections of sensibility which, on the contrary, may disappear in the course and on account of the examination : amongst these are double sensation, slowness of the sensory conduction, and impairment of sense of location.

The disturbing influence of paræsthesiæ frequently makes itself felt during the examination, so that the patient cannot distinguish sharply between subjective and objective stimulations, and says " now " before he has been touched.

The fact that the patient perceives all the stimulations is not a proof of normal sensibility. Care must be taken to ascertain whether he feels the stimulus as strongly as at the sites where the sensibility is normal. It is specially advisable that in morbid conditions which affect one side of the body only, comparison should be made with the healthy side.

In certain diseases of the brain which lead to unilateral sensory disturbances I have sometimes made use of the following method of examination (*N. C.*, 1885) : two symmetrical sites on the two sides of the body are stimulated at the same time (by contact with a brush, pin-prick, etc.). The patient will then perceive only the stimulation on the healthy side, whilst on unilateral examination he will feel every stimulation on the affected side. We shall refer to this mode of examination as the *method of double-stimulation*.

Affections of Sensibility

These are subjective and objective. Amongst the subjective, we include *pain* and *paræsthesiæ*.

It would be superfluous to discuss here the nature of pain. A few

[1] *A. f. Ps.*, xxxvii.

points only, which are of importance in diagnosis, will be considered. The physician must never be satisfied with the mere statement: that the patient suffers pain. It is very important to learn the character, the extent, the time of onset, the duration, the accessory symptoms, and the influence of the pain upon the general condition.

In considering the intensity of the pain it must of course always be remembered that the subjective element comes mostly into play. The same stimulation produces in one individual a slight, in another a very acute pain, and further sensations of pain may arise in the centres themselves although the end organs or the conduction tracts have not been subjected to any stimulation. Severe pain not infrequently causes certain accessory symptoms in the motor, vaso-motor, and secretory systems which are not dependent on the will (muscular tremors, pallor or flushing of the skin, secretion of tears (not due to weeping), rapidity or slowness of the pulse). Delirium may occur at the height of an attack of pain. As to the extent of the pain we should discover whether it follows certain nerve tracts, has a girdlelike distribution, is limited to one point, etc. A permanent pain limited to one site, and having no obvious cause, is known as *Topoalgia* (Blocq), and a painful sensation of burning heat in the skin as *causalgia* (Weir Mitchell). It should be mentioned that patients are apt to describe as "pain" very various sensations which may produce only a feeling of discomfort.

The localisation of the pain that occurs in diseases of the internal organs appears to stand in a certain relation to the spinal innervation of these organs. As the sympathetic nerves of each organ belong to certain segments of the spinal cord, disease of these nerves produces pain and hyperæsthesia in those cutaneous areas which derive their sensory fibres from the same segments (Head).[1]

There are many varieties of *paræsthesiæ*. The most common are formication, tingling, the feeling of numbness or deadness. There are also paræsthesiæ of the temperature-sense (*e.g.* painful sensation of cold —psychro-æsthesia). The study of paræsthesia is specially important as it usually gives information as to the objective affection of sensation. When the patient complains of formication, numbness, a dulling of sensation is very frequently also present. The paræsthesiæ are not infrequently located in the area of distribution of a certain nerve, and its limitations may be described with anatomical sharpness, especially in lesions of the peripheral nerves. They are often also described as painful, as painful tingling, as dead pain, etc. The way in which they are represented sometimes reveals the psychogenic, hypochondriacal origin of the sensations. Simple sensations are described in terms of imagination and interpretation: "I feel as if a ball were rising from stomach to my throat, as if worms were crawling under my scalp," etc.

We speak of *hyperæsthesia*, *hypæsthesia*, and *anæsthesia*.

Hyperæsthesia is rare on the whole and has less clinical interest than the conditions arising from diminution or loss of sensibility.

Hyperæsthesia is present when painful stimulations cause more acute pain than they would do in healthy persons, or when painful stimuli of slight or medium intensity cause a pain which was formerly caused only by very strong stimuli. Thus hyperæsthesia is characterised

1 Disturbances of Sensation in Visceral Disease, *Br.*, vols. xvi., xvii., and xix. German by W. Seiffer. Berlin, 1898.

by the fact that a stimulus which would in health arouse a sensation of contact, pressure, or temperature, gives rise to a feeling of pain. This disturbance is specially noticeable when even a light touch on the skin, a gentle stroke produces pain. We speak of relative hyperæsthesia when stimulations below a certain degree are at first not felt, but later are suddenly felt as painful (Leyden).

Frequently, though not always, the hyperæsthesia finds an objective expression in a corresponding exaggeration of the reflexes. Pressure on the painful part often causes a marked increase in the frequency of the pulse (Mannkopf's symptom). Hypæsthesia is much more common than anæsthesia, but there is a tendency to use the term anæsthesia for a loss of sensation which is not quite complete. Hypæsthesia may extend to all the qualities of sensation (total anæsthesia) or to some only (partial). We speak of *partial sensory paralysis* when the sense of pain or of temperature, or both these qualities, are diminished or absent. It seldom happens that the sense of cold only, or of warmth is abolished.

Loss of the sense of pain = analgesia,
,, ,, ,, of contact = tactile anæsthesia,
,, ,, ,, of temperature = thermanæsthesia.

We speak of analgesia dolorosa when an anæsthetic area is the seat of spontaneous pains. This is a very frequent occurrence.

Confusion between the qualities of stimulation (*e.g.* perceiving warmth as cold) is known as *perverted sensation.* It occasionally happens in health that an intensely cold stimulus will cause a momentary sensation of heat. Contact of the skin with a very hot object will sometimes be felt by a normal person like a stab, whilst a pin-prick not infrequently provokes the feeling of burning.

The degree of thermanæsthesia may be estimated by the fact that the patient bears contact with a burning hot object for a considerable time. The pain produced by heat is of course tested. It must naturally not be forgotten that individual differences occur in normal persons. Those who work in great heat, whose hands are brought by their work into contact with hot objects, can often bear considerable degrees of heat on their hands. There are even healthy people who can put their fingers for a time into the flames without experiencing any particular pain. Scars abolish the sense of temperature. In compression of a nerve the sense of cold is first abolished.

Anæsthesia of the sense of movement and position—which I call *bathyanæsthesia* because the loss of sensation affects the deep parts—often occurs as an isolated symptom. In its lesser degrees it is only slight movements or changes of position (in the toes or fingers) that are unrecognised or wrongly perceived. When it is severe the patient has no idea of the position of his limbs, and when he attempts to grasp the affected side with the healthy hand, his eyes being closed, he wanders far beyond it.

The pressure-sense may be retained in anæsthesia of the skin and *vice versâ* (Strümpell [1]). In diseases of the peripheral sensory nerves, it follows other laws, since the fibres which conduct deep pressure do not pass along with the cutaneous nerves (Head and Thomson [2]).

Osteoanæsthesia or "*pallanæsthesia*" (loss or diminution of the vibration-sense) is a variety of bathyanæsthesia, but it may occur alone

[1] *D. m. W.*, 1904. [2] *Br.*, 1907.

also, for I have found it in individuals who showed no sign of disturbance of the sense of position.

Anæsthesia for one kind of sensation may be combined with hyperæsthesia for another. Thus it happens that painful stimuli are not felt to be painful, whilst a slight touch will arouse severe pain. The reverse —tactile anæsthesia combined with hyperalgesia—is frequently observed. Paradoxical as it may sound, there may be a kind of mixed anæsthesia and hyperæsthesia in one and the same quality of sensation. Thus under certain conditions a pin-prick may produce a " dumb " pain which nevertheless is more severely felt than on the healthy skin.

In addition to the quantitative changes of sensibility we have also to consider the *rapidity of conduction*. In some pathological conditions conduction, especially of painful impression, may be delayed. This is indicated when a pin-prick is not at once felt to be painful, but only after an interval of two to five seconds. There may also be a *double sensation*, the pin-prick producing two sensations, one felt immediately as touch, the other later as pain. The delayed pain sensation may be exaggerated. The double sensation rarely manifests itself by the pin-prick causing two sensations of the same strength with an interval between them, or by the second being less painful.

Polyæsthesia, the condition in which a simple touch is felt as double or multiple, is rare. When the patient is touched by one point he feels as if two or three touched him at the same time. *Macroæsthesia*, in which the object palpated appears to be materiallly larger, is still rarer.

Disturbances of the sense of locality (Ortssin) are as a rule found only in combination with other sensory changes—especially with bathyanæsthesia (according to Foerster and our own experience, whilst Schittenhelm regards its relation to cutaneous sensation as closer), but it may also occur as an isolated symptom. It may be so severe that a stimulation applied to the hand, for instance, will be localised in the upper arm.

Allocheiria or allæsthesia (Obersteiner) is the term used when a stimulus applied to one extremity is felt at a corresponding point on the other. It is a rare phenomenon, and is often erroneously assumed to exist from the fact that the patient, during the stimulation of the left leg, is conscious of a paræsthesia on the right, which outweighs the objective stimulation on the other side. I have observed a real allocheiria in a very few cases, and even then the symptoms could be detected for a short time only. There are, however, a number of unimpeachable observations of the condition (Weiss, Fischer, Hoffmann, Jully, Determann,[1] Jones,[2] and others). The allocheiria affected in these cases sometimes one, sometimes several qualities of sensation. A corresponding condition of the reflex movements was noted in one case. In another described by Stewart the stimulation which affected the ulnar side of the extremity, was referred to the radial side. In another case I have found a somewhat analogous condition in the lower extremity.

As in morbid conditions we have much more frequently to do with a diminution than with a loss of sensation, the establishment of this and especially the determination of its limits may be very difficult. Where the loss of sensation is complete, it is easy to demarcate the boundaries between sensitive and insensitive areas; but in practice one must not

[1] *Z. f. N.*, xviii. [2] *Br.*, 1907. See literature here.

expect to find that these are always sharply defined and unchangeable. According to the mode of testing and the attention given by the patient, the results of examination may vary at different times. We may proceed by drawing the brush or needle from the sensitive area over the skin towards the hypæsthetic or anæsthetic region, the patient being asked

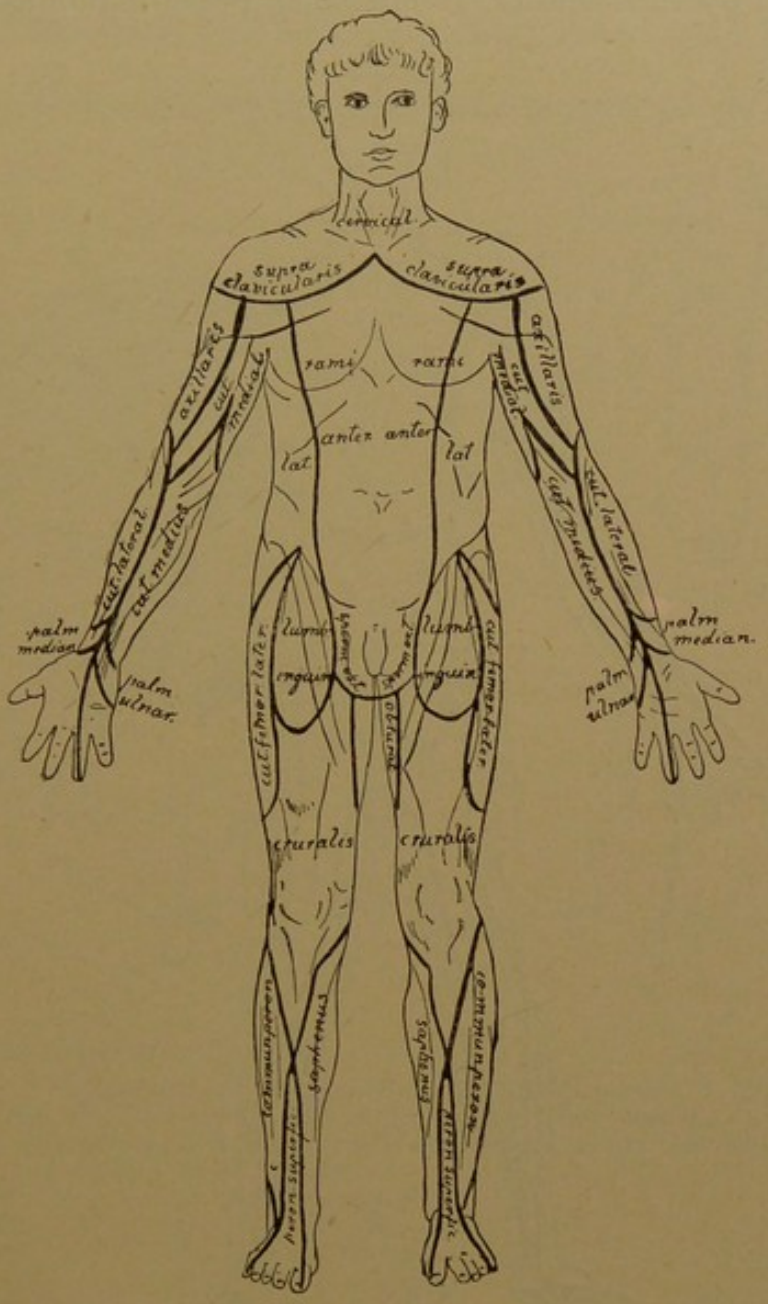

Fig. 26.

Figs. 26-33.—Areas of Cutaneous Innervation (after Freund).

to indicate where the sensation ceases or begins to grow weaker. The reverse way may also be followed.

In order to estimate correctly the anatomical distribution of the sensory disturbance an exact knowledge of the cutaneous innervation is first required. This is well illustrated by Freund's tables (Figs. 26–33, Fig. 28 after Frohse).

It should not however be forgotten that the area of distribution of the various cutaneous nerves is subject to many variations, and that the

degree of overlapping of adjacent areas is very inconstant. This has been specially demonstrated by Frohse and Zander in regard to the cranial nerves. The innervation is here constant only in the median areas, whilst at the lateral parts of the head the distribution of the sensory branches is extremely variable, so that there is hardly a site here which is innervated by the same nerve branch in every individual. A portion

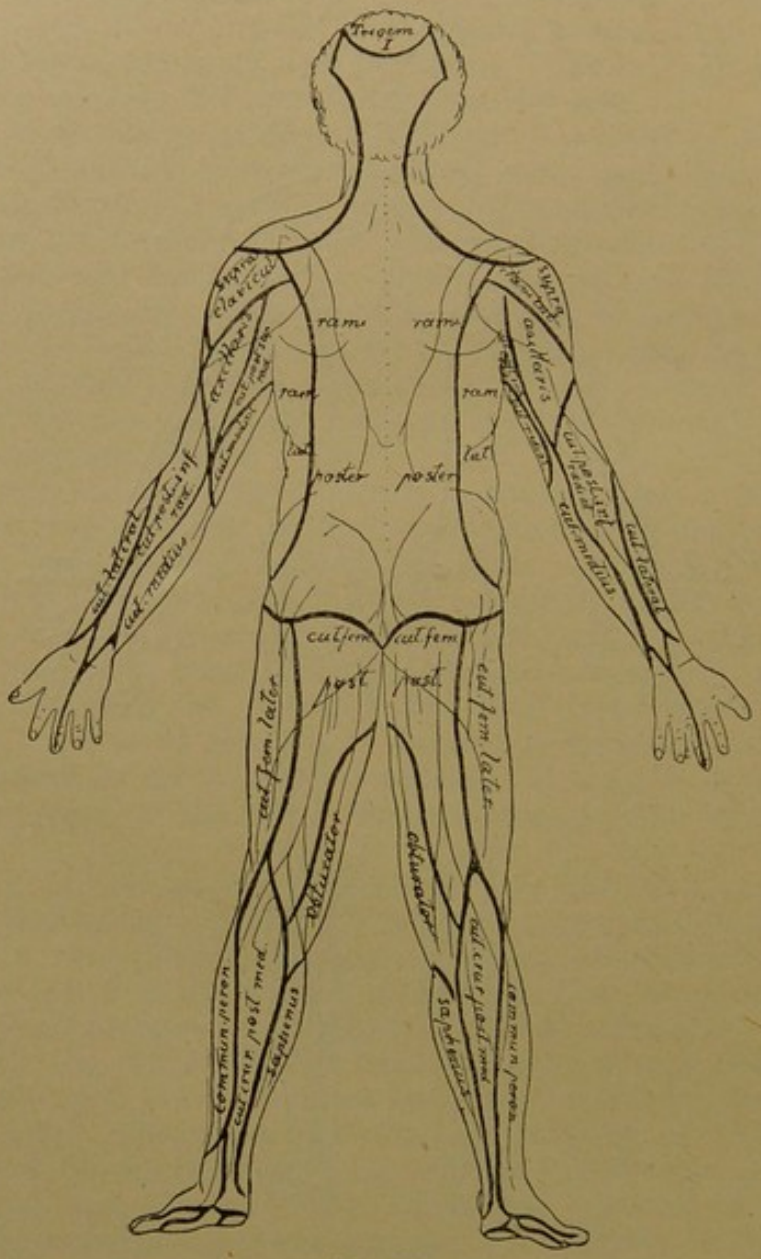

Fig. 27.

of skin, for instance, which lies about 2 cm. above the right margin of the orbit will be in one case innervated by the first trigeminal branch, in a second by the first and second, in a third by the third or by the third and the first. In the same way the areas of the great auricular nerve, the auriculo-temporal and the auricular branch of the vagus, merge into each other in the most varied manner. At other sites also the regions of sensory innervation (for instance that of the musculo-cutaneous nerve, the external saphenus and the anterior tibial nerves at the back of the

foot) are subject to individual fluctuations. Frohse found in one case that the anterior tibial nerve participated in the innervation of the opposed dorsal margins of the 2nd and 3rd toes. It is also worthy of note that in the face the cutaneous nerves of each side pass beyond the middle line, so that the median area is innervated from both sides. According to Zander this also is true of the other median areas of the body. Fig. 28 illustrates the areas of sensory innervation in the head after Frohse.

The relation of the skin to the roots and segments of the spinal cord,

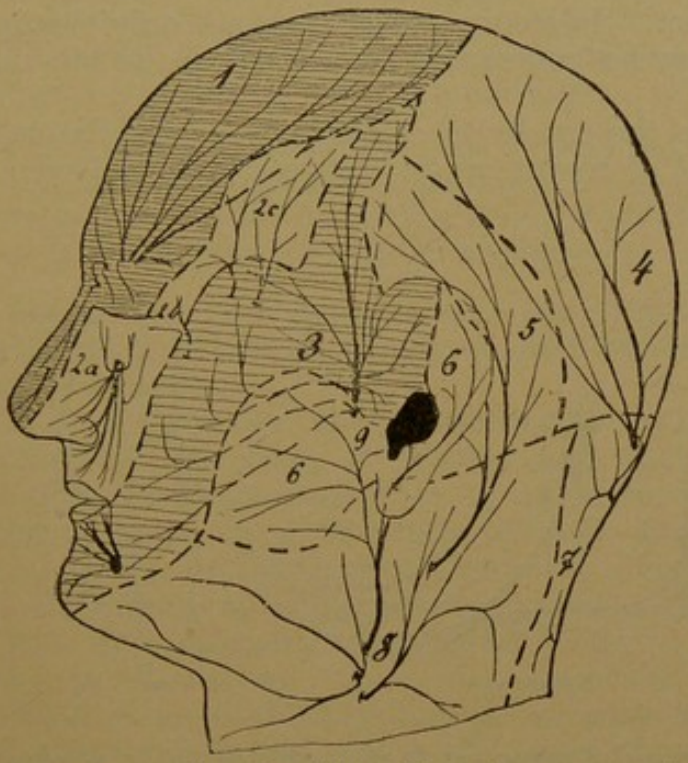

Fig. 28.—(After Frohse.)

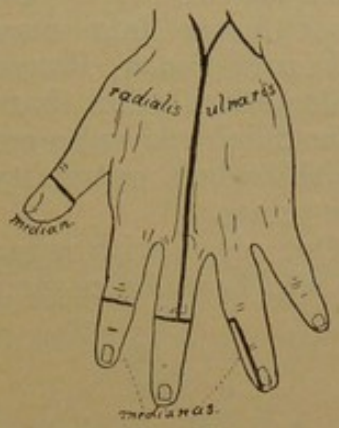

Fig. 29.

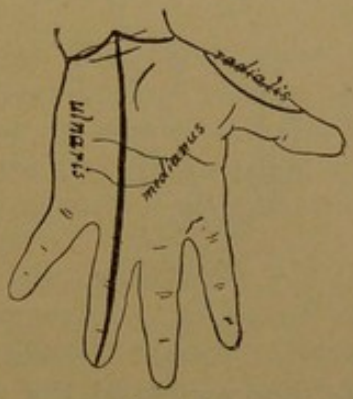

Fig. 30.

1. First Branch of Trigeminus. 2. Second Branch of Trigeminus. 3. Third Branch of Trigeminus. The cross lines indicate the area of the first and third branches of the trigeminus, the black shows the area of the auricular branch of the vagus nerve in the outer ear.

2a N. infraorb.
2b N. zygomatico-fac.
2c. N. zygomatico-temp.
3. N. auriculo-temp.
4. N. occip. magnus.
5. N. occip. minor.
6. N. auricul. magnus.
7. N. cervic. post. (dorsalis).
8. N. cervic. lateral. (ventral).
9. N. auricul. vagi.

the areas of innervation of the sensory spinal roots, will be described in other parts of this work.

Cutaneous Reflexes

Although reflexes may be elicited from every part of the body, the following only are of special importance for diagnostic purposes: 1.

the plantar reflex; 2. the abdominal reflex; 3. the cremaster reflex.

By the term *plantar reflex* we indicate that a stimulation applied to the sole of the foot provokes a movement of the leg which depends on the involuntary contraction of a part of its muscles and which is essentially momentary in character. In healthy persons any stimulation will elicit this movement, a touch, a prick, the application of a hot or cold object, or tickling. The reflex on the whole corresponds in force to the strength of the stimulation employed. The usual form of the reflex movement is dorsal flexion of the foot. A contraction of the tensor fasciæ latæ may even be elicited by stimulation of the sole (Brissaud, Crocq, Renault); indeed it occurs more frequently and on feebler stimulation than does dorsal flexion of the foot. Other muscles of the thigh may also contract. In strong stimulation not only the foot, but the whole extremity may be contracted by flexion of the hip- and knee-joints.

Our views with regard to the condition of the toes in stimulation of the sole have been modified of recent years. It was Babinski who in

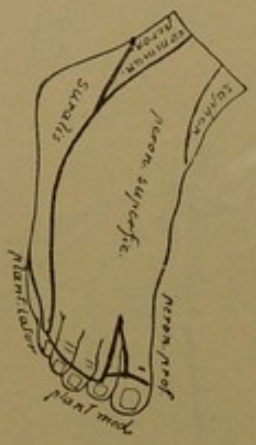

Fig. 31.

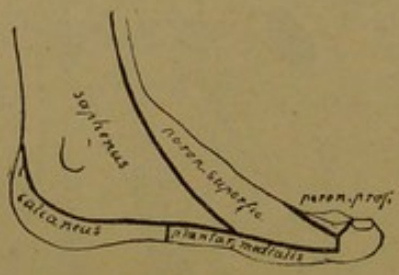

Fig. 32.

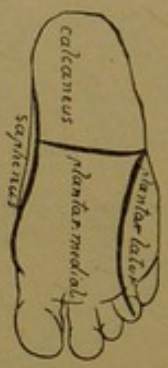

Fig. 33.

1898 demonstrated the previously unnoted fact that normally a *plantar flexion of the toes*[1] followed stimulation of the sole of the foot. He further proved that in organic nervous diseases (particularly of the spinal cord), which are accompanied by the spastic symptoms described on p. 6 *et seq.*, with a lesion of the pyramidal tracts, or which at least create the conditions for these, a dorsal flexion as a rule takes place[2] instead of the plantar flexion. This dorsal reflex, which occurs most prominently, indeed usually exclusively at the great toe, and is as a rule

[1] *Sur le réflexe cutané plantaire. Comptes rendus de la Soc. de Biol.*, 1897. Also: *Du phénomène des orteils, etc., Semaine méd.*, 1898.

[2] Babinski's observations have been practically confirmed by Van Gehuchten, Brissaud, O. Kalischer, Koenig, Collier, Pastrovich, Crocq, Guidicandra, Sano, Ganault, Prince, Homburger, Walton-Paul, Levi, Böttiger, Goldflam, Rossolimo, Marinesco, Lévi, Kornilow, Specht, Richter, Muggia, Medea, Stanley Barnes, Kutner, Knapp and others. My own experience entirely accords with them. Objections raised against and exceptions to the universality of the rule will be discussed in the special part.

The fan-movement (signe de l'éventail) of the toes, which Babinski found in testing for his reflex mainly under pathological conditions, is a sign of very uncertain value, as he himself admits.

With regard to the toe reflex during sleep, the results of observation are not unanimous (Bickel, Goldflam, Kutner, *D. m. W.* (1907), and others); Babinski's reflex may be elicited in healthy individuals by subcutaneous injection of scopolamin (Link, *D. m. W.*, 1905).

slower in its response than the normal plantar reflex, is known by the name of " Babinski's sign." It is always an indication of a pathological condition, except in children during the first months of life, in whom dorsal flexion is the normal reflex. The significance of this sign will be further discussed in the corresponding chapter of the special part. In examining the toe reflex, the stimulus should be at first too weak to produce a dorsal flexion of the foot. We may use, for example, the handle of the percussion hammer and stroke the sole of the foot with it—from the heel towards the toe—or we may employ a pin in the same way, as Babinski himself does. He draws attention to the fact that the pathological reflex, the dorsi flexion of the great toe, is usually best elicited from the outer margin of the foot. It should also be noted that in many individuals, the toe reflex can be either not elicited or only so vaguely that we cannot determine with certainty whether there is flexion or extension of the toe. It is usually necessary to divert the attention of the patient during this examination.

It is generally sufficient to talk to him, or to ask him to answer a question which seems important to him. Where there is difficulty in distracting his attention I find the best plan is to let an assistant form a number or a letter in the hand of the patient during the examination, so that he has to take some trouble to recognise it.

The intensity of the plantar reflex varies greatly. There are a few individuals in whom it is only elicited by strong stimulation (the deep prick of a needle).

It is well known that considerable individual differences exist as regards the sensation of tickling and the corresponding reflex. By prolonging the stimulation, or by rapid repetition of single stimuli, by a kind of summation therefore, we can exaggerate the intensity of the reflex. The investigations of Kronecker and Stirling show that a summation of stimuli is specially adapted to elicit reflexes.

It is not necessary to test the condition of the reflexes by all the modes of stimulation. We may assume an exaggeration of reflex excitability when slight stimulations produce not merely a simple dorsal flexion of the foot and plantar flexion of the toes, but an energetic withdrawal of the whole leg. One thing however has to be remembered: if the dorsi-flexion of the foot has become impossible from a mechanical impediment or from paralysis of the extensors of the foot, then the plantar reflex is always modified so that the extremity is withdrawn by flexion at the hip and knee.

I[1] have established the following facts: If in a healthy person we draw a heavy stroke with the handle of the percussion hammer, or better still with the pulp of the thumb, from above downwards along the internal surface of the leg, a *plantar flexion* of the toes (less often of the foot) as a rule follows, or there is no reflex movement at all. Under pathological conditions—in spastic conditions or the affections which cause them—there is instead of this movement, a *dorsal flexion* of the foot and toes, sometimes the tibialis anticus and the extensor hallucis longus only, sometimes all the extensors contracting. Frequently the pinching of a fold of skin on the inner surface of the leg has the same effect. The pressure exercised by the thumbs has often to be strong, and the lower parts of the leg right down the ankle are reflexly the most sensitive.

[1] *M. f. P.*, xii.

My results have been confirmed by Cassirer,[1] Pfeifer [2] and others, and this pathological leg-phenomenon is called by them "Oppenheim's sign." Its diagnostic value, which is not to be regarded as slight, will be pointed out in the special part.

With regard to Strümpell's tibialis sign, which as an associated and not a reflex movement does not come under this heading, compare the special part.

Bechterew and K. Mendel (*N. C.*, 1904) describe independently of each other the following symptom: if the outer side of the back of the foot is tapped in its proximal half, corresponding to the base of middle of the 4th and 3rd metatarsal bone, the cuboid and the 2nd cuneiform bones, a dorsal flexion of the toes appears in healthy persons, whilst under the conditions which lead to Babinski's sign there is a plantar flexion or a spreading out of the toes. I can give this a general confirmation, but I should advise that the symptom be made use of with caution and restraint. See further Osann (*M. m. W.*, 1907) and O. B. Meyer (*B. k. W.*, 1907).

The *abdominal reflex* (Rosenbach) is thus tested: the finger or some blunt instrument is rapidly passed over the skin of the abdominal or hypochondriac region; and there follows a retraction of the abdomen from contraction of the abdominal muscles. This reflex is inconstant in healthy persons, and specially tends to be absent when the abdominal wall is flaccid or where is a thick layer of subcutaneous fat. It may also be present at one time and absent at another in the same individual. On the whole therefore this reflex is not of great value in pathology, though its absence on one side only is always of morbid origin.

Its disappearance under the eyes of the observer may be of great diagnostic significance.

Strümpell and his pupils Müller and Seidelmann [3] have, however, come to another conclusion as regards this reflex, as they have found it to be almost constant in healthy, especially in young people.

Investigations as to the condition of this reflex in affections of the abdominal cavity have been made by Jamin (*N.C.*, 1904), Müller-Siedelmann, Sicard, Rolleston, and others. In perityphlitis there is usually diminution or absence of the abdominal reflex on the right side, etc.

A few writers (Gowers, Dinkler) distinguish between a superior (epigastric), middle (mesogastric) and inferior (hypogastric) abdominal reflex. For practical objects the distinction of a supra-umbilical and an infra-umbilical abdominal reflex is sufficient (Oppenheim [4]). We shall again refer to this reflex in the special part.

Under pathological conditions other reflexes which are not normally present may appear. Remak describes as a "femoral reflex" the plantar flexion of the toes, of the foot and the contraction of the quadriceps which follows stimulation of the skin on the inner surface of the thigh. Westphal had already noted the extensor reflex from contraction of the quadriceps in paraplegics. I have seen in some cases of spastic spinal paralysis a reflex movement, on stimulation of the sole of the foot, by which one leg was crossed upon the other. One of my patients had discovered that he could provoke this movement by pinching the skin on the inner surface of the thigh and he made use of this device to change the position of the paralysed leg.

In isolated cases I have seen, under conditions which will be described later, a *pronation reflex* on the arm; that is to say that by pinching a fold of skin on the ulnar side of the forearm or by a prick at the corresponding part, a pronation of the hand was elicited, which was sometimes associated with an internal rotation of the arm.

Other pathological cutaneous reflexes have been described by Raymond, Jamin, Oppenheim and others, but they are only of secondary importance.

A *diaphragmatic reflex* is described by O. Hess (*B. k. W.*, 1906): "By slight percussion or by simple touching of the nipple in young individuals, the diaphragm contracts and there is a drawing in of the epigastrium and the ensiform process as the result of a shortening of its sternal part.

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[1] *M. f. P.*, xiv.
[2] *M. f. P.*, xiv. and xvi.
[3] *M. m. W.*, 1905.
[4] *Z. f. N.*, xxiv.

The *Cremaster reflex* (Jastrowitz) is elicited by passing a finger or a pointed instrument over the inner surface of the thigh (in the adductor region), or by pinching a fold of skin in this region ; the response as a rule is a contraction of the cremaster, which elevates the testicle. This reflex is not however constant, as the cremaster is frequently in a condition of tension and keeps the testicle raised. And also a good many local processes, especially varicocele, will cause a disturbance of this reflex activity. On the whole, therefore, absence of the cremaster reflex must be used very cautiously in diagnosis.

An equivalent of the cremasteric reflex is to be found in women, according to Geigel (Dinkler, Bechterew) : if the skin on the inner surface of the thigh is pinched, there follows on this side a contraction of the lowest bundle of the internal oblique muscle (inguinal reflex). This reflex is sometimes found in men also (Crocq, Kornilow).

The hypochondriac, gluteal, scapular and hypothenar reflexes have not so far proved to be of any real diagnostic importance. We shall not discuss here the number of other reflexes, of doubtful nature, which have been described during recent years.

The cremasteric reflex should not be confused with the scrotal reflex contraction of the tunica dartos, with wrinkling of the skin of the scrotum in response to touching or stroking the latter or the skin of its neighbourhood.

The scrotal reflex may sometimes be elicited from the skin of the anus and exceptionally even from the sole of the foot (Finkelnburg).

With regard to the so-called anal reflex, see the special part.

The pilomotor or goose-skin reflex has not proved to be of any diagnostic value. It follows cutaneous stimulation corresponding to the distribution of the spinal roots (Mackenzie).

In determining the diagnostic value of the cutaneous reflexes we must remember that some of them—including the plantar reflex and the movements of recoil from painful stimuli at all other parts of the skin of the body—may be more or less entirely suppressed by a strong effort of the will. So much concentration of the attention is however necessary, that the deception is usually recognised on repeated examination. Artificial inhibition of the plantar reflex can generally be recognised by the fact that the extensor movement of the foot is prevented by a prolonged active tension of the calf muscles. This voluntary suppression is of course only possible when there is no paralysis of the corresponding muscles.

Strümpell (*Z. f. N.*, xv.) states that there are "reflex sensitive" and "reflex insensitive cutaneous points," and that reflexes can often only be elicited from the latter by a rapid repetition of local stimuli. This reflex insensibility is particularly noticeable in the upper extremities as compared with the lower. The sites from which reflexes can be very readily elicited, such as the sole of the foot, are known as reflexogenous zones.

The condition of the cutaneous reflexes is entirely independent of that of the tendon reflexes, and should not be confused with it.

The relations between sensibility and reflex excitability are not constant : reflex excitability may be absent though sensibility is retained, or it may be absent, present or even exaggerated although sensibility is lost. This will be discussed later, but it may be here stated that the

cutaneous reflexes are absent in anæsthesia and paralysis of peripheral origin. In spinal diseases the condition varies and depends upon whether the disease has destroyed the reflex arc or not. In the first case the reflexes are absent, in the latter they are conserved and usually exaggerated if the morbid process has developed at a site which lies above the reflex arc. Recent observations (Bastian, Bruns, etc.) have led to the view that when the interruption of conduction is a total one, even the reflex movements which are controlled from the inferior segments of the spinal cord disappear. This question, however, to which I shall return later, is still undecided.

With regard to the cutaneous reflexes in brain diseases, we cannot make any definite statements, but we may note the fact that in unilateral diseases of the brain which lead to hemiplegia, the abdominal and cremasteric reflexes are as a rule absent on the paralysed side. This symptom may be ascribed to an exaggeration of the reflex inhibition.

According to many writers (Jendrássik, Pandi, Sherrington, Crocq, Munch-Petersen,[1] Rothmann [2]) the cutaneous reflexes have a cortical origin. This theory however, as Leyden and Goldscheider think, is by no means sufficiently established.

In sleep and in narcosis, and also in conditions of complete loss of consciousness, the cutaneous reflexes are absent (and usually also the tendon phenomena).

The fact that the reflexes may be abolished by injection of stovain, novocain and other chemicals into the subarachnoid space of the spinal cord is of great interest. I have myself found this to be so in some cases treated by Sonnenburg. The condition has been closely studied by Finkelnburg (*M. m. W.*, 1906) and others. See also Dönitz (*Arch. f. kl. Chir.*, Bd. 77), Lazarus (*B. k. W.*, 1906) and others.

The term *paradoxical contraction* has been given by Westphal to the contraction which is produced by the passive approximation of the origin and insertion of any muscle (*A. f. P.*, x.). If for instance we press the patient's foot upwards, a tonic contraction appears in the extensors of the foot, especially in the tibialis anticus muscle, which persists for a considerable time and maintains the foot in the position of dorsi-flexion (and adduction). In tickling the sole of the foot also, the dorsi-flexion may pass into this paradoxical contraction. This symptom does not seem to have any practical diagnostic value. It must not of course be diagnosed when the patient, in the belief that he should keep the foot in this position, actively contracts the extensor muscles. In the cases which I have seen, the sensation of fatigue was usually absent in such paradoxical contractions.

Spasm (Hyperkinesis)

By spasm in the widest sense of the word we understand (1) muscular contractions which are provoked by non-physiological stimuli, (2) muscular contractions which are caused by physiological stimuli of abnormal intensity. We distinguish between *tonic* and *clonic* spasms. An involuntary muscular contraction of great intensity and long duration is known as a tonic spasm. The spasm is clonic when muscular contraction and relaxation alternate in rapid succession. With regard to distribution, we differentiate between *general*, *partial* or *localised* spasm. The spasm may be limited to a single muscle, to one innervated by one nerve, to a group of muscles which are synergic in their function, or it may involve a whole extremity, one-half of the body or even all the

[1] *Z. f. N.*, xxii. [2] *A. f. A.*, 1904.

muscles of the body. Clonic contractions, which lead to severe, shaking movements of one extremity or of the whole body are known as *convulsions*. *Cramp* is a tonic and painful spasm confined to one muscle or to a circumscribed muscular area. We speak of *tetanic* spasm when the tonic spasm extends to the whole of the muscles or to the greater part of them.

Tonic and clonic spasms are not infrequently combined.

Spasms take place either from direct or from reflex causes, *i.e.* the stimulation which produces the abnormal muscular movements arises from the motor centres or the nerve tracts themselves, or it originates in the sensory sphere and is transmitted by means of the sensory conduction tracts to the motor centres. Spasms are also very frequently of psychogenic origin (see below).

It is doubtful whether stimulation of the motor nerves can produce spasms in the muscles under their control. This mode of origin is certainly unknown with regard to clonic spasms. When in lesions and diseases of the peripheral nerves spasms are observed in the muscles which belong to them, the possibility of a reflex origin through stimulation of the sensory branches should always be kept in mind.

Spasms of *reflex* origin are exceedingly common. Every painful affection and every condition of excitation in the distribution of a sensory nerve may provoke spasms, and here Pflüger's law with regard to the extension of reflex movements comes into action in so far that the stimulus as a rule involves the motor nerves which arise on the same side and at the same level as the sensory nerves. Thus a painful affection of the eyes, injury of one branch of the trigeminus, or trigeminal neuralgia, often cause a spasm in the facial nerve of the same side. Spasms are also observed which may be traced to a condition of excitation in the sensory nerves of distant areas, as in facial spasm resulting from uterine disease, etc. The mode of this connection is still unknown. The same is true of the so-called traumatic reflex epilepsy. Reflex spasms may also be caused by the reflex centres themselves being in a condition of abnormal excitability, as in strychnine poisoning (also in tetanus and rabies), or because the influence of the reflex-inhibition centres is suppressed. Strychnine and certain other poisons put the grey matter of the spinal cord into a condition of exaggerated excitability, which is evidenced by the occurrence of spasms; the slightest stimulation will produce reflex spasms instead of simple reflex movements.

The muscular tension which appears in organic diseases of the spinal cord is not usually termed a spasm in the narrow sense of the word; nor are the tremors caused by exaggerated reflex excitability and which are provoked by touching the skin, tapping the tendons, etc. The distinction is, however, an artificial one.

Certain forms of spasm, such as myoclonia, have been ascribed to a condition of excitement of the ganglion cells in the anterior horn of the spinal cord. This view is entirely hypothetical as regards myoclonia, but in tetanus and strychnine intoxication recent investigations have shown that there are organic changes in the motor cells.

It is probable that spasms in the region of the motor cranial nerves may have their origin in a condition of excitability of the nuclei of the nerves, that, for instance, facial spasm may be caused by a fine change in the cells of its nucleus. The pons and medulla oblongata contain

tracts and centres, stimulation of which gives rise to spasmodic movements, but not to real epilepsy.

The principal birthplace of spasms is the cerebral cortex. Spasms in single muscles as well as those which are unilateral or general may take their origin from the cortex. It is put into a condition of excitement by organic diseases and poisons, and by disturbances of the circulation which lead to motor discharge. But it is functional disturbances in especial, fine changes evading anatomical investigation, which create and keep alive in the cortex of the motor zone the excitability which gives rise to the spasms. The forms of spasm which originate in this way are partly characterised by the fact that they may be provoked and exaggerated by mental excitement, the motor manifestations which normally accompany emotion being so exaggerated as to become spasmodic. Self-attention, exaggerated self-observation is perhaps also capable of producing these spasms. Wundt has shown that the nervous apparatus of the muscles of one part of the body is directly influenced by the attention directed to it.

Abnormal mental conditions may not only directly produce spasms, but may also so influence the reflex centres that the usual stimuli, instead of producing simple reflex movements, provoke spasms.

Vasomotor, Trophic and Secretory Disturbances

The cerebral cortex contains a vasomotor centre, the site of which has been shown by animal experiments to be in the neighbourhood of the motor centre (Lepine,[1] Eulenburg-Landois,[2] Bechterw), and which has also been supported by clinical observations (Rossolimo, Oppenheim). Stimulation of this centre causes lowering of temperature of the skin of the opposite limbs. The nerve tract coming from this centre appears to pass downwards through the internal capsule. Vasomotor centres are also to be found, according to the well-known observations of Cl. Bernard, Ludwig, Dittmar, etc., in the medulla oblongata and the spinal cord. The principal centre is the one in the medulla oblongata. In animals its stimulation causes general contraction of the vessels. Nothing is definitely known as to its site in the human medulla oblongata. It has been suggested that the lower central nucleus represents this centre. Reinhold has marked off a large area on the floor of the fourth ventricle as the vasomotor centre, but Cassirer[3] has rightly contested his conclusions. Other theories will be discussed in the special part. In the spinal cord the vasomotor centres are probably contained in the grey matter of the lateral horns and the area which lies between the anterior and the posterior horns. The tracts which pass from the centres in the oblongata to these spinal centres take their way through the antero-lateral column ; we do not, however, know anything definitely as to their course. The impulses leave the spinal cord through the anterior roots, in order to reach the sympathetic by way, either, wholly or for the most part, of the rami communicantes. The entrance of such fibres into the posterior roots, shown by some experimental observations (see below) has not been proved with regard to man. That vasomotor fibres also pass *directly* into the peripheral nerves is not improbable.

[1] *Revue de méd.*, 1896. [2] *V. A.*, Bd. 68.
[3] *Die vasomotorisch-trophischen Neurosen.* Berlin, 1901, S. Karger.

Vasomotor disturbances may occur in diseases of almost every part of the nervous system.

In lesions of the grey matter of the cord, vasomotor phenomena are often observed. This is true also of affections of the peripheral nerves—the influence of which upon the vascular system has long been known and has recently been specially studied by Lapinsky [1]—and more particularly as regards the sympathetic, which contains the greater part or the whole of the vascular nerves.

There are vasoconstrictor and vasodilator nerves, but the latter have hitherto been found only at certain sites, as in the chorda tympani, the glosso-pharyngeal and vagus (?), the erigentes and the sciatic nerves, and also in the anterior roots of the spinal cord (Dastre-Morat). Whether vasodilator fibres pass also through the posterior roots, as Stricker and others think, has not been definitely proved. This view has recently been accepted by Bayliss and Kohnstamm, whilst Lewandowsky still remains doubtful as to the interpretation of the experimental data.

Fibres to the viscera were found by Steinach and Weiner in the posterior roots of the frog. Roux and Heitz affirm the same with regard to the posterior roots of mammals.

A number of morbid conditions come under the designation of *trophic disturbances*. The chief of these is *muscular atrophy* ; then come *disturbances of the nutrition of the skin*, the *mucous membranes*, the *soft parts*, the *bones* and *joints*. Amongst the trophic changes in the skin the following are especially to be noted : glossy skin (in which it becomes smooth, thin and shining), loss and greyness of hair (canities), and also the falling out of the hair at circumscribed sites (alopecia), or greyness of the hair on certain areas (the eyelashes for instance have been observed to turn grey after a severe mental shock), cracking and brittleness of the nails, or thickening (onychorrhexis, onychogryphosis), atrophy of the nails, leukopathia unguium, falling out of the nails (idiopathic disease of nails or alopecia unguium), the development of ulcers which show little tendency to heal (perforating disease, neuroparalytic keratitis, etc.) ; and also many cutaneous affections the origin of which is not yet fully explained (anomalies of pigmentation, nævi, for instance, which may be confined to the area of innervation of certain nerves, herpes, urticaria, sclerodermia, pemphigus, etc.). Charcot thinks that decubitus (bed-sores) also should be attributed to trophic disturbances, a view which other writers dispute.

Amongst the trophic affections of the bones and joints there is abnormal brittleness of the bones leading to spontaneous fractures (Weir-Mitchell), intermittent hydrops articulorum, arthropathy, etc. etc. Abnormal fragility of the bones is specially seen in certain diseases of the spinal cord, and also in certain forms of arrested development of the central nervous system. They may also, however, appear as isolated symptoms in otherwise healthy individuals (osteopsathyrosis or "idiopathic atrophy of the bones with periostal dysplasia"). Simple atrophy of the bones [2] may accompany atrophic paralysis, trauma and other conditions. Recent radiographic studies in particular

[1] *A. f. A.*, Suppl.-Bd., 1899 ; *Arch. d. méd. expér.*, 1899 ; *Z. f. N.*, xvi.

[2] Compare for references to literature Gayet et Bonnet : Les altérations osseuses d'origine nerveuse, *Arch. gén. de Méd.*, 1901.

have made us acquainted with this symptom (Sudeck, Nonne[1] and others.)

There are of course conditions of quite a different kind which are designated as trophic disturbances. These are classified by Samuel into three categories, *atrophy*, *hypertrophy*, and *dystrophy*. The significance of the corresponding physiological processes—the doctrine as to *trophic centres* and *trophic nerves*—has given rise to a great number of theories. Some writers, Samuel at their head, teach that there are special centres for the innervation of the tissue and also specific nerve fibres for trophic purposes and for these only. Others do not ascribe special nervous apparatus to this function but regard disturbances of innervation rather as a secondary process, arising from the failure of the nerve cells to fulfil their physiological functions on account of direct injury or because the impulses do not flow to them in sufficient number and force. Trophic disturbances are also considered by many, by Schiff in particular, as a simple result of the affection of the vasomotor system.

Let us consider the various processes involved. The relation between the nervous system and the muscular apparatus is very simple. The nerve cells in the anterior horns of the spinal cord control the innervation of the muscles of the body. The nerve cells with the axons which penetrate into the anterior root and through the peripheral nerve into the muscle, where they divide into branches, form an anatomical unit (neurone).[2] If the cell becomes affected, then the whole neurone degenerates. If the nerve fibre is destroyed at any point, then that part of the neurone which is no longer in connection with the cell first degenerates. Degeneration of the neurone is accompanied by degeneration of the muscle. We have no definite knowledge of the process by which the nerve cell controls the nutrition of the muscle. The processes of nutrition do not coincide with those of functional stimulation, but probably take place through the same nerve fibres, so that the view of the existence of specific trophic nerves appears not to be justified in this case. According to recent opinions (Kopp, Marinesco, Goldscheider) it is not sufficient for the function of the trophic centre that it should itself be intact and its connection with the muscles uninterrupted ; the stimulations which influence its activity must also flow from the periphery and the higher centres if it is to fully and completely carry out its function. The integrity of the "trophic" centre itself and its connection with the muscle is, however, the most important factor.

The doctrine of trophic centres for the *sensory nerves* rests only partly upon sure foundations. The nerve cells of the posterior root ganglia are, according to Waller's famous investigations, the trophic centres of the sensory cutaneous nerves and their terminal ramifications, and also of the posterior roots and their direct continuations. These cells and fibres form an anatomical unit and thus it is explained why lesion of these cells should involve disturbances of the nutrition of their processes, and that the part of the nerve which is no longer in connection with its cell of origin should degenerate. Marinesco's teaching also seems to apply here in so far that the cells of the spinal ganglion require to be stimulated to a certain degree by impulses from the periphery in order to execute their trophic function in a complete and continuous manner.

However, this doctrine by no means explains all the pathological changes. Difficulties are offered even by the fact that along with the disappearance, atrophy or degeneration, *productive* processes—new formation and even exuberant proliferation of the tissue—play a great part. Are the spinal ganglia cells also the trophic centres for the skin, the soft parts, the bones and joints, or is the innervation of these areas dependent upon certain segments of the spinal cord ? Is the trophic function, as Samuel attempts to show, a specific one, or are the so-called trophic affections of the skin, etc., merely a product of altered circulation, of the diminished sensibility and of injuries of the skin ? Are we dealing here always with symptoms of loss of function or do conditions of excitement in the nervous system also play a part ?

These questions cannot all be answered with complete certainty. The cessation of sensory

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[1] *Fortschr. aus d. Geb. d. Röntgen.*, 1901.

[2] The neurone theory itself and the objections which are brought forward against it will be discussed in another part of this work.

innervation does not appear, especially according to recent investigations (Turner, Krause [1]) to explain the trophic disturbances. Although this may make the skin and mucous membrane less capable of resisting the effect of any irritant, so that for instance wounds heal badly, yet it does not explain the nutritive disturbances in the narrow sense.

It is evident that the disturbance of the circulation of blood which accompanies injury of the vasomotor nerves will also influence the nutrition in the corresponding areas, but even this factor cannot be the only cause of the numerous trophic disturbances. It is a fact of great importance with regard to this question, that nutrition is regulated by the vasomotor nerves (Nothnagel) and that this process may be to a certain degree under the reflex influence of the sensory nerves. Vasomotor and trophic changes very often accompany each other. We can thus understand that trophic disturbances occur in diseases of the peripheral nerves, spinal ganglia, spinal cord and sympathetic. Specific trophic fibres are attributed to the latter (Arloing, Brissaud, Durdufi), and the experiments of Lewaschew, Frenkel, and Lapinsky appear to favour this theory.

Joseph (*V. A.*, Bd. 107) shows that extirpation of the 2nd spinal ganglion along with the adjacent portion of anterior and posterior roots in the cat results in a circumscribed loss of hair without any other vasomotor symptoms. His experiments, however, are not confirmed by Behrend, and Jaquet, Bikeles-Jasinski, etc., have had other results from similar experiments, whilst Köster (*Zur Physiol. der Spinalganglien*, etc., Leipzig, 1904) and Trendelenburg (*N. C.*, 1906) have recently reported experimental observations which are similar to those of Joseph.

Kohnstamm (*Fortschr. d. Med.*, 1905) agrees with Bayliss in regarding the sensory nerves as also conducting in a centrifugal direction, and in this way influencing the vasomotor action of the trophic nerves of the skin.

On the whole we may summarise the facts and opinions known to us somewhat as follows :

Degeneration of the muscles and nerves and its dependance upon diseases of the spinal cord require no further discussion.

Trophic disturbances of the skin, soft parts, bones and joints occur in diseases of the peripheral nerves, spinal ganglia, sympathetic and spinal cord.

In the production of these anæsthesia plays some part, making the body less capable of resistance against injury and setting at nought the pain which is "the watchman of the organism." Vasomotor influences also are of an importance which should not be underrated, and affections of the sensory nerves, especially when they are of an *irritative* nature, are capable of influencing the vasomotor functions.

It seems to me, however, that we require a still further hypothesis to explain the pathological changes of trophic nature.

I assume, partly relying on Charcot, Vulpian and Mitchell, that the *function of the nerve cells in the cord may undergo a pathological change*, a morbid *exaggeration* ; that these can then only function in a normal way if the impulses flowing to them from the periphery can be conducted undisturbed to the centre. Diseases of the spinal cord, which prevent conduction of sensory impulses and their transmission to the 2nd process (axone) of the nerve cell (compare the anatomical-physiological remarks in the chapter on diseases of the spinal cord), lead to an accumulation of the stimuli in the nerve cells, which morbidly affect their trophic function, so that a pathological exaggeration of the nutritive processes is produced at the periphery.

In this way also affections of the peripheral nerves, which do not bring about a complete interruption of conduction, may cause conditions of irritation which are transmitted to the trophic centre and so alter its function that nutritive disturbances appear in the corresponding nervous area.

G. Koester comes to similar conclusions from his experiments. The whole question has been recently reviewed by Goldscheider (*Z. f. k. M.*, Bd. 60). He concludes from the teaching of Weigert and of Verworn on the theory of dissimilation and assimilation of the tissue under the influence of irritants, that the course of these processes is disturbed by the absence of physiological stimulations as well as by their excess. Here the intermediary rôle is played by the sensory and vasomotor nerves, and it is not necessary to assume the existence of specific trophic nerves and centres for the production of so-called trophic disturbances, *i.e.* tissue changes caused by defective assimilation and excessively exaggerated dissimilation. The combination of disturbances of conduction in the nerves with irritation is the condition which is most favourable for the causation of so-called trophic disorders.

[1] *Die Neuralgie des Trigeminus*, Leipzig, 1896.

Gowers has recently expressed himself as opposed to the view of specific trophic nerves (*Brit. Med. Journ.*, 1906).

Other valuable data are given by Head and Sherren (*Br.*, 1905).

Among *affections of secretion*, disorders of the sweat secretion are particularly common. We know very little definitely as to the sweat centres and corresponding tracts. It is assumed and shown by some well-known experiments to be probable that the cerebral cortex contains sweat centres. The tracts which come from these go to the medulla oblongata and must cross before they reach the spinal cord (Schlesinger [1]) in order to pass downwards in the antero-lateral column and to enter into relation with the spinal sweat centres at the various levels of the cord. A sweat centre for all four extremities has been found in the medulla oblongata (Nawrocki, Luchsinger, Vulpian). The spinal sweat centres are localised in the intermediate region between the anterior and posterior horns, in the lateral horn and (by Adamkiewicz) in the anterior grey matter. From these the sudoral fibres pass, wholly or partly, into the sympathetic (Langley) and through it into the peripheral nerves. Thus, for example, the sudoral fibres for the face come from the medulla oblongata, pass through the whole cervical cord, reach the level of the 2nd dorsal root and by means of it leave the spinal cord and enter the sympathetic, passing from it into the trigeminal and facial nerves (Higier). It has been shown by Goltz, Luchsinger, Levy-Dorn and others that a secretion of sweat may be provoked by irritation of the peripheral nerves. Ostroumoff has demonstrated experimentally that the secretion is not associated with the blood circulation, although this may have a considerable influence upon the production of sweat. The sympathetic is said also to contain fibres for the inhibition of the secretion of sweat (frenosudoral fibres). Disturbances of sweat secretion may occur in diseases of the brain, spinal cord, peripheral nerves and of the sympathetic.

A general hyperidrosis occurs in the neuroses (hysteria, neurasthenia) and also in exophthalmic goitre. Poisons (pilocarpin) may also produce it. In diseases of the spinal cord either excessive sweating or drying up of the sweat secretion (anidrosis) is frequently observed. In lesions of the spinal sweat centres of the homolateral or of both sides of the body, the affection had a segmentary distribution somewhat corresponding to the sensory innervation of the skin by the posterior roots, or segments of the spinal cord [2] (Schlesinger). We do not, however, know on what factor the occurrence of a hyperidrosis or an anidrosis depends. Hemiplegics sometimes perspire on the paralysed side. A *unilateral hyperidrosis*, or *hemihyperidrosis*, *i.e.* sweating which extends to one-half of the face, or to this and parts of the corresponding half of the trunk, less frequently to the whole of one side of the body, may occur in healthy people—especially after the use of mustard, acid foods, etc.—and also in affections of the sympathetic, in hemicrania, exophthalmic goitre, erythromelalgia, tabes dorsalis, gliosis and other diseases of the nervous system, as well as appearing as an isolated symptom in mental degeneration, and after acute infective diseases.

[1] *Spinale Schweissbahnen und Schweisszentren beim Menschen. Festschrift Kaposi.* Wien, 1900.

[2] See corresponding chapter in the section: Diseases of the Spinal Cord.

An unusual symptom is a congenital incapacity to sweat—a *congenital anidrosis* from absence of the sweat glands (Tendlau), a condition which is not negligible, since these individuals when attempting to work in heat cannot regulate the temperature of their bodies and therefore react with fever (Zuntz : *Höhenklima und Bergwanderungen*, Berlin, 1906, p. 391). Zuntz observed a thermic polypnœa, such as occurs in dogs, which do not sweat. This defect increases the tendency to heat stroke (Hiller).

" Hemihyperidrosis cruciata " is a very rare symptom. We speak of a paradoxical sweat secretion (Schlesinger, Kaposi) when it appears under conditions which usually have the opposite effect, for example in cold stimulation.

A frequent form of local hyperidrosis is that confined to the distal parts of the extremities (acrohyperidrosis of Kaposi).

Hyperidrosis in the region of the peripheral nerves appears as an irritative phenomenon in neuritis, especially in the traumatic form (Weir Mitchell).

Examination of the Special Senses : Functional Affections of the Sensory Organs

The Sense of Smell.[1]—In estimating this we are almost entirely dependent upon the indications given by the patient. Substances, with a strong odour that is likely to be readily recognized, and which do not irritate the sensory nerves of the mucous membrane (specially the essential oils : oil of peppermint, oil of lavender, oil of cloves, spirit of turpentine, etc.), are held under one nostril, the other being kept closed. The patient should then say whether he has any sensation of smell, and if so he should try to describe it. If he cannot do so, it is frequently not because of a diminution of the sense of smell but because he does not know the substance, or at least not sufficiently to enable him to identify it. It is necessary to compare the intensity of the sensation of smell on both sides. Here we are dependent upon the statements of the individual under examination, which are often unsatisfactory. When the odour is disagreeable the sensation of smell is accompanied by a kind of reflex movement, the muscles which narrow the nostrils being contracted and the head thrown back ; or the mouth is distorted as if in an expression of disgust. There are instruments for measuring the sense of smell (olfactometer), such as Zwaardemaker recommends, but the attempt of Toulouse, who used camphor solutions of various degrees of concentration, to introduce a more exact quantitative determination of the sense of smell into practice has not been successful.

Electrical examination of the olfactory nerves cannot be used clinically.

We speak of *anosmia respiratoria* when the odorous substance is prevented from reaching the mucous membrane of the nose, as in swelling of the nasal mucous membrane, polypi, etc., or from displacement of the posterior nares (anosmia gustatoria). Local diseases may also cause anosmia from destruction of the nerve endings. Finally, disease of the olfactory nerves in their whole course may be the cause—especially when they are affected by injury at the base of the brain. Subjective sensations—olfactory hallucinations—often arise in brain diseases, especially in the psychoses, cerebral tumours, etc. A unilateral or bilateral anosmia is by no means an unusual symptom in brain diseases.

[1] Of the recent reviews we may mention those of Collet, " L'Odorat et ses troubles," Paris, Baillière ; Sternberg, " Geschmack und Geruch," Physiol. Unters., Berlin, 1906.

It is observed in basal fractures, hydrocephalus, cerebral tumour, arteriosclerosis, and focal diseases, etc. It may also follow concussion of the brain. There is a senile and a congenital anosmia. The latter is found in idiots and rarely in otherwise normal persons (Zwaardemaker, Placzek, Blasi, Fischer). Abundo reports an inherited defect. I have noted anosmia to appear for a time as an isolated symptom after influenza. Hyperæsthesia of the sense of smell is of no real diagnostic importance.

The *sense of taste* is examined by placing upon the tongue by means of a glass rod or a dropping glass, a solution of some substance which can be tasted. The four varieties of taste—sweet, acid, salt and bitter—should all be carefully examined. These may be tested for by dropping on to the tongue vinegar, sugar, salt and solution of quinine. If they are used at one examination the mouth should be washed or rinsed after each test (quinine should be used last on account of its persistent after-taste). As the regions of the tongue and mucous membrane of the palate which are sensitive to taste are under the control of different nerves, it is usually necessary to test each area of innervation separately : (1) on the anterior two-thirds of the tongue (remembering that the sense of taste is present specially at the edges and the tip) ; (2) on the posterior parts of the tongue and in the region of the palate and pharynx. The anterior segments of the tongue taste acids better than bitter substances, whilst the posterior parts and the soft palate are more sensitive to bitter tastes.

Taste stimuli are said not to be perceived on the uvula, the posterior palatine arches and the tonsils (Kiesow and Hahn).

In order that the tongue should not be drawn back, as it must be in speaking, the patient should be asked whether he has a sweet, acid, salt or bitter taste, and should answer by a nod of the head. It is still better to make use of a table or a sheet of paper upon which the four taste qualities are noted, so that he can indicate the corresponding one with his finger.

It should be remembered that the distinction between "acid, salt and bitter" is not a sharp one to the inexperienced and imperfectly educated, and that these terms are used promiscuously.

A gustometer, which supplies the substances in the form of gas, is recommended by Sternberg (*D. m. W.*, 1905).

The sense of taste may be affected by a thickly furred tongue, by catarrh of the mouth, and by excessive tobacco-chewing. An unilateral or bilateral ageusia may be present in nervous diseases of various kinds. It may also be a sign of senility (ageusia senilis).

Electrical examination of the sense of taste may always be dispensed with.

The *sense of hearing.* As affections of hearing are so very often caused by diseases of the sound-conducting apparatus, one should always begin with an *otoscopic examination.* The functional test is carried out by determining the distance at which the patient, with one ear closed and his face turned away, can perceive a whisper (numbers or words) or the ticking of a watch. As the result varies greatly according to the intensity of the whisper, it is advisable to associate an individual

with normal hearing in the examination. We must remember also that different whispering voices are heard at varying distances (the pitch of the tone playing a part in this), and that the various forms of deafness do not influence the hearing in the same way. If a watch is used it is necessary to ascertain at what distance its tick can be heard by a healthy ear. It should also be remembered that a watch will be heard at a greater distance if it is gradually removed from the ear than if it is brought from a distance towards the ear. It is useful also to ask the patient to indicate the exact moment at which a tuning-fork held in front of the ear ceases to sound, holding one's own ear at the same distance from the tuning-fork. We may as a rule dispense with the use of special apparatus for testing the hearing (Politzer's acumeter, etc.), but examination with Bezold's *continuous* series of tuning-forks has given such valuable results that this method should be generally employed.

It enables us to determine the range of hearing, which normally extends from C_2 (16 vibrations) to G^8 (50,000 vibrations) of the Galton whistle. In disease of the auditory apparatus there is a reduction of the range of hearing through loss of the higher or lower tones, or from gaps in the scale, and there seems to be a relation between the site of the disease and the kind of tone-loss; in disease of the auditory nerve, for instance, the range is reduced specially in the upper tones. These relations are still uncertain.

To test the *bone-conduction* one may place tuning-forks (varying in pitch) or a watch with a loud tick upon various parts of the skull, the external auditory meatus being closed. (In old people the bone conduction for very high tones is often so much diminished that the ticking of the watch cannot be heard through the bones.)

In disease of the sound-conducting apparatus, the tuning-fork can still be heard through the skull, perhaps to an exaggerated degree. On the other hand bone conduction is diminished or absent in diseases of the auditory nerves (the labyrinth, or trunk and centres of the auditory nerve). Various methods have been employed to determine this question. Rinne's method is the chief one. If the sounding tuning-fork is placed on the parietal region or on the mastoid process of an individual with normal hearing, and as soon as it has ceased to be heard there is placed in front of the ear, it is again distinctly heard. This is the case in nerve deafness (which of course must not be absolute). Rinne's test is therefore positive in these cases, and negative in disease of the sound-conducting apparatus (or in rare cases it is indeed positive, but with marked shortening of the duration of perception, which in healthy persons lasts about thirty seconds).

The test may, as Lachmund shows (*M. f. P.*, Ergänz. xx.), have an apparently negative result in nerve deafness, because the feeling of the vibration of the tuning-fork on the mastoid process is mistaken for sound, or because the sound is better conducted through the bone to the other ear, provided there is better hearing in it.

The tuning-fork or the watch will be distinctly heard through the bones of the head, the external auditory meatus being closed, because the occlusion of the meatus "converts the cavity into a resonating chamber." If, in a person with normal hearing, the tuning-fork is placed on the middle of the head or the forehead, the sound is heard as if from a distance or is localised in both ears. When one external meatus is

closed, the sound will be localised in the ear of the same side. In diseases of the ear which affect the sound-conducting apparatus, the tuning-fork will in this examination be perceived by the affected ear (Weber's test) If on the other hand there is nerve deafness of one ear, the sound goes towards the healthy side. According to Schwabach, in bilateral deafness the tuning-fork of medium tone placed upon the bones of the head will be heard longer than normally in affections of the middle ear, and not so long as normal in cerebral deafness. The loss of single tones out of the scale is always to be regarded as a sign of nerve deafness.

Unfortunately there are still great differences of opinion as to the value of all these methods in differential diagnosis, and the distinction between nerve deafness and the other forms is still indefinite. Ostmann (*D. m. W.*, 1904) regards two facts as established: (1) If in unilateral disease of the ear the tone C of the tuning-fork placed on the middle of the vertex is heard on the affected side alone or to an exaggerated degree, and if the duration of the tone is greater than the normal for bone conduction and less for air conduction, then the normal conditions are deranged in favour of bone conduction; if there is further an exclusive reduction of the range of hearing for the low tones, we may diagnose a pure disease of the sound-conducting apparatus. (2) If, on the other hand, in unilateral ear disease the tone C of the tuning-fork is perceived from the middle of the vertex in the healthy ear; if its duration by bone conduction is not in the least prolonged, and is distinctly shortened for air conduction; and if the range of hearing is reduced mainly upwards, these conditions are in favour of a unilateral disease of the labyrinth, therefore of nerve deafness. Lachmund suggests the hypothesis that in disease of the cochlea there is present merely a general diminution of the acuity of hearing, whilst the circumscribed loss of the acoustic field (scotoma of hearing) is characteristic specially of affection of the ramus cochlearis and of central conduction; but this statement is of value rather as aiding the endeavour to establish analogies between the acoustic and the optic nerves than as a practical aid to observation and experience. The author also endeavours to distinguish in the auditory apparatus a phenomenon analogous to the pupil-reflex, which would occur between the acoustic nerve and the tensor tympani (motor trig.). On this question see also Wittmaack (*Med. kl.*, 1905) and Passow (*B. k. W.*, 1905).

With regard to the so-called vestibular symptoms and their examination see section on brain diseases.

By *hyperæsthesia acustica* we mean the condition in which all sound sensations are accompanied with a feeling of discomfort. The hearing of a tone higher or lower is known as paracusis. This becomes diplacusis when the tone is correctly perceived in the sound ear. Paracusis Willisii is the name given to the condition where the patient hears better in the midst of a loud noise.

Sight.—Examination of the organ of vision and the power of sight is so extremely important that it should never be omitted. The optic nerve as part of the central nervous system which reaches the surface and can be seen by direct and indirect ophthalmoscopic examination, affords information about many diseases of the central nervous system and constitutes one of the best guides in diagnosis. He who does not understand how to use the ophthalmoscope is no neurologist.

Testing of the sight is carried out in the well-known method. The condition of the refraction and of the accommodation should be thoroughly considered. A description of the methods of examination for these is out of place here.

Examination of the *eccentric vision* is, however, so important in the diagnosis of nervous diseases, that the most important points must be mentioned.

A simple method of examining the eccentric vision is the following. The patient has one eye closed and his back towards the window ; the examiner keeps one of his hands steady at the height of the eye and about 1½ feet in front of it and moves the other hand slowly from the periphery into the field of vision ; the patient indicates the moment at which he first sees the hand. If the hand is in this way brought into the field of vision from all directions its limits can be determined at least so far that the loss of large areas (a half, a quadrant) can be recognised. A somewhat more precise result is obtained if, instead of the hand, a piece of white paper, about 1 cm. square, fastened to a rod (a penholder, for instance), is brought from the periphery into the field of vision. Care must of course be taken that the eye does not move laterally, and that fixation is as absolute as possible. If this method be used, then not only are the limits of the field of vision determined, but marked defects at any site are also discovered if the white piece of paper is moved through the whole field of vision and the patient indicates where it is not seen or is seen indistinctly. In the same way colours can be tested, by using red, blue, or green coloured paper instead of white.

A somewhat more exact measurement can be made when the patient is placed about 1½ to 2 feet from a black board and, one eye being shut and the other kept fixed on a point or cross (marked with chalk), the piece of paper is moved from the periphery of the board towards the fixed point, and the point where it is first seen is indicated.

A method which is exact and applicable to all affections of the field of vision is *perimetric measurement.* The instrument most in use is a perimeter which represents an arc and which can be rotated round its axis so that it can be stopped at every meridian. I have principally used a perimeter made by Sydow of Berlin—a hemispherical apparatus supplied with meridians. The eye of the patient is put at the level of the fixed point, the other is kept shut with the fore-finger. The meridians of the perimeter carry the figures 10, 20, up to 90. The piece of paper should be moved slowly and the attention of the patient kept so alert that he at once indicates the first glimmer of a visual impression. If the paper is moved in little jerks it will be perceived somewhat sooner. If the results found—each meridian should be examined, preferably in a certain order—were arranged in a scheme which any one can construct for himself, the normal field of vision projected on to the surface would be as represented in Fig. 34 (after Hirschberg).

Upwards the extent of the field of vision for white covers				50-60°
Downwards	,,	,,	,,	60-70°
Outwards	,,	,,	,,	90°
Inwards	,,	,,	,,	55-65°

The field of vision for colours is less extensive than for white. Blue has the largest circumference, then comes red, then green. Examination for these colours is sufficient.

We know that the field of vision extends further outwards than inwards. This is because the most external parts of the retina are less sensitive than the inner parts ; the bridge of the nose also obscures a part of the field of vision.

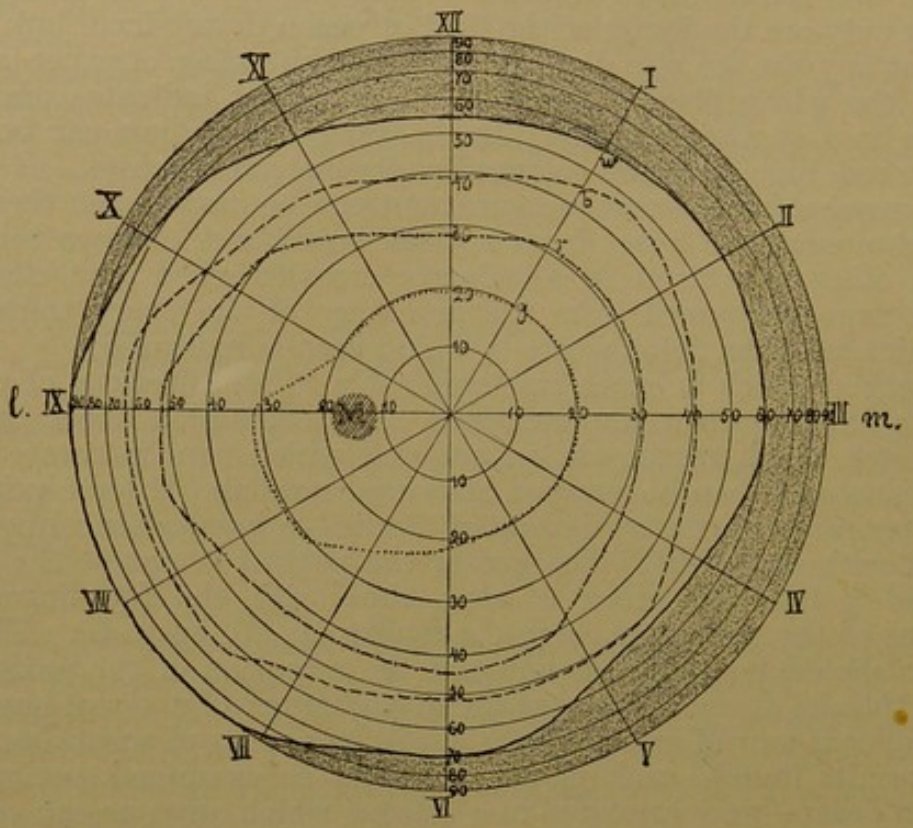
In estimating pathological conditions it should be made a rule to lay no weight on small deviations from the normal, as certain individual

differences, as well as the mode of examination, the lighting, the strength of colour of the object, the condition of refraction, etc., influence the result to a certain extent. In doubtful cases the field of vision of a healthy person should be examined previous to the consideration of the abnormal field.

Amongst the affections of vision special consideration should be given to the following :—

1. Concentric narrowing of the field of vision ;
2. Scotoma ;
3. Hemianopsia.

Concentric narrowing of the field of vision consists, as the name



——— Margins for white.
- - - - - - - - „ „ blue.
— · — · — · — „ „ red
· · · · · · · · · „ „ green.
M = Mariotte's blind spot.
l = lateral ; *m* = median side.

FIG. 34.—Normal field of vision of the 6 ft. eye. (After Hirschberg.)

implies, in an almost equal retraction in all directions. If it is present for white, then the excentric vision for colours is almost always correspondingly limited, so that the concentrically reduced field of vision forms to a certain extent a normal field of vision in miniature. There may, however, also be a limitation of the colour vision, whilst the reduction for white is absent or merely slight. Myopia, myosis and paresis of the accommodation may cause a moderate limitation of the field of vision. Opaque spots on the cornea may also reduce the area of the field of vision.

Examination by means of the dark-perimeter, after Wildbrand's method, is of no real value in practice. It depends on the fact that the sensitiveness of the retina to light is diminished when it is kept in a bright light, whilst in the dark it is greater than normal (adaptation or recovery). If the eye is brought in a dark room to a perimeter, where the fixation point is formed by a spot of bright colour the size of the head of a pin (of some material which shines in the dark after being exposed to diffuse daylight), then the object of examination is not at first seen at the normal limits, but there exists a concentric narrowing which is only lost after a long stay in the dark. The retina shows this power of recovery in the normal as well as in the pathological eye, the normal by a steady proportionate increase towards the periphery in all the meridians, the neurasthenic retina by an extreme slowness of recovery, and the retina injured by organic lesion, by an irregular recovery or by the presence of absolute defects. In simulation the patient contradicts himself or conceals the extent of recovery, etc.

Scotoma is a defect within the area of the field of vision, the loss of sight not lying at the periphery. Scotoma usually affects the fixation-point and its neighbourhood. The sight is not always entirely lost; there may be a relative scotoma, the sight being merely indistinct in the affected area. The scotoma sometimes involves the colour sense only.

Hemianopsia (or hemianopia) is the loss of one-half of the field of

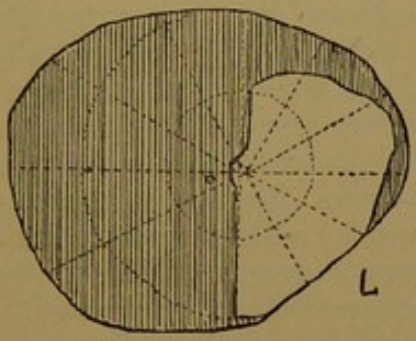


Fig. 35.

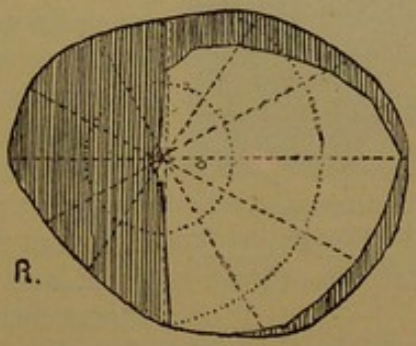


Fig. 36.

The Hemianopic Field of Vision.

Left bilateral homonymous hemianopia. The external outline shows the margin of the normal field of vision for white; the shaded portion corresponds to the area of the visual field in which sight is absent. (After Gowers.)

vision, and almost always affects both eyes. If the sight is lost in the left or right halves of the field of vision on both sides, the condition is known as homonymous bilateral hemianopia (left or right) (Figs. 35 and 36). If the external halves of both fields of vision are affected, if, that is, there is amblyopia of the internal halves of the retina, then we speak of bitemporal hemianopia. This form is much rarer than the homonymous variety. A simple examination is usually sufficient to reveal hemianopia. It is difficult to recognise when there is loss of consciousness, aphasia and mental weakness. With patients who cannot be induced to keep the eye fixed I bring two objects before it at the same time (key and knife, or two colours, red and blue), one into the inner, the other into the outer half of the field of vision; the patient has then to say what he has seen, and in hemianopia he will always indicate only one object or one colour.

Nasal hemianopia is so rare that it has hardly any practical significance. The same is true even in greater degree as regards inferior and superior hemianopia.

Electrical examination of the optic nerve has no real diagnostic interest.

Examination of the Functions of the Other Cranial Nerves

Oculomotor Nerves. — The levator palpebræ superioris muscle elevates the upper lid and keeps the eyes open. Paralysis of this nerve causes the upper eyelid to droop (ptosis). If it is complete the eye is quite shut, or the patient only succeeds, by raising the eyebrow by means of the frontalis (marked transverse lines on the forehead), in slightly lifting the lid (Fig. 37). If the eyebrow is kept fixed with the finger, the ptosis again appears in its completeness. There is also a pseudo-ptosis, which is produced by spasm of the orbicularis palpebrarum; but the spasmodic character can quickly be recognised from the tension and resistance which is offered by the lid on the attempt to raise it passively.

Fig. 37.—Contraction of the frontalis muscle in bilateral ptosis. (Oppenheim.)

Ptosis may be accompanied by appreciable changes of the electrical excitability (see chapter on Oculo-motor Paralysis). The lids contain smooth muscle fibres, innervated by the sympathetic, which dilate the palpebral fissure. Paralysis of these muscles produces a narrowing which would be very difficult to distinguish from an incomplete ptosis, if the other usual signs of paralysis of the sympathetic (contraction of the pupils with conservation of their reaction) were not present. The patient often succeeds, by a special effort of will, in completely raising the drooping lid. A persistent spasm of these unstriped muscle fibres with a resulting dilatation of the palpebral fissure may also sometimes occur.

The Muscles which move the Eyeball

The external rectus muscle moves the eyeball directly outwards; the internal rectus moves it directly inwards; the superior rectus directs it upwards and inwards and at the same time rotates the eyeball slightly, so that if a vertical axis were laid through it its upper end would be turned inwards; the inferior rectus moves the eyeball downwards and slightly inwards. The superior oblique muscle directs the eyeball downwards and outwards and rotates it so that the upper end of its vertical axis turns inwards. The inferior oblique moves the eye upwards and outwards (and rotates the upper end of its vertical axis outwards.)

In lateral deviation of the eyeballs towards the right and left, the external rectus of one eye and the internal rectus of the other contract at the same time. Normally the eyeballs may be turned in the lateral direction so far that the margin of the cornea reaches the corresponding canthus. A small defect, however, is not necessarily pathological. If the lateral movement of both eyeballs in a certain direction is abolished, we speak of *conjugate* or *associated oculo-motor paralysis* (*Blicklähmung*). In such cases the internal rectus, which is not contracted at all or but slightly so in the lateral movement, comes into action in the movement of convergence along with the internal rectus of the other eye. The

reverse condition may occur where the internal rectus contracts in the normal way in lateral movement of the eyeball, whilst it fails to act in the movement of convergence. In fixation, both eyes, or frequently one only, may turn outwards (insufficiency of the internal recti). This symptom appears in errors of refraction (especially myopia) and in certain diseases of the nervous system (exophthalmic goitre, neurasthenia).

We may also speak of upward or downward conjugate paralysis when it is in either of these directions only that the eyeballs cannot be moved.

Paralysis of the ocular muscles leads to the following symptoms :

1. Loss or limitation of a certain movement of the eyeballs ;
2. Diplopia ;
3. Secondary contracture of the antagonists ;
4. Secondary deviation of the unaffected eye ;
5. False projection of the field of vision and abnormal attitude of the head.

Limitation of the power of movement varies according to the degree of paralysis. Even where there is no evident sign of defect in the movements of the eye, there may be a slight paresis which gives rise to diplopia. We must first test carefully whether the eyeballs can be moved in all directions to the normal extent. Weakness of an ocular muscle may sometimes be revealed by the fact that the corresponding position can only be maintained for a moment, and is accompanied by slight quivering of the eyeball. Paralysis of an ocular muscle frequently causes a secondary contracture of its antagonists, so that in paralysis of the external rectus, for instance, the eyeball will be persistently turned inwards from the pull of the internal rectus.

The effort of the patient to direct the eyeball towards the side of the paralysed muscle has sometimes the effect of causing an excessive innervation of the synergic muscle of the other side. In paralysis of the right abducens, for example, the patient strives so intensely to turn the eyeballs towards the right that the internal rectus of the left eye is excessively contracted. Thus there is produced a secondary deviation of the unaffected eye (towards the direction in which the paralysed muscle would develop its power of traction). This becomes distinctly visible only when the affected eye is the fixing one. It occurs in only paralytic strabismus.

Diplopia is the most important sign of oculo-motor paralysis. It is caused by the image of an object being received on the macula lutea of the fixing eye, whilst in the other eye it strikes another part of the retina. In long-standing oculo-motor paralysis diplopia may be absent, the patient fixing with one eye and disregarding the second picture. Very frequently he keeps one eye closed in order to avoid the distressing diplopia, which also gives a sensation of vertigo.

Diplopia is tested for in a simple manner by asking the patient to follow with his eyes the extended finger and to indicate the moment at which a second image appears. If that does not suffice to reveal the diplopia or to give sufficient evidence as to its nature, a coloured (red) glass may be held in front of the sound eye. If the patient then gazes at a flame or at a piece of paper, the diplopia is revealed by his seeing a red-coloured picture along with another of the usual colour. We have then further to ascertain at what part of the field of vision

the diplopia appears, the position of the images, and their separation from each other when the direction of the gaze is altered. The image which appears in the healthy eye is the "true" one, the other the "false." If the false image corresponds with the side of the eye by which it is seen, then we have *homonymous*, in other cases *crossed* diplopia. If on closing the right eye the image on the right side disappears, the diplopia is homonymous. This is more rapidly tested by using a coloured glass. As a rule convergent strabismus is accompanied by homonymous, divergent strabismus by crossed diplopia.

As the diplopia appears first, and often exclusively in the part of the field of vision to which the eyeball would have been turned by the traction of the paralysed muscle, the patient endeavours by keeping his head in a certain position to use only that part of the visual field in which no double image makes its appearance. But as a result of secondary contracture, the diplopia may be present in the whole visual field.

Finally, the oculo-motor paralysis creates a false projection of the field of vision. From the excessive strain which the patient must exert in order to move the eyeball towards the direction in which the paralysis exists, he makes a false estimate of the position of the object in space and reaches beyond it. This false projection disappears when the affected eye is closed.

Paralysis of an ocular muscle may be simulated by contracture of its antagonist (in hysteria, for instance).

Monocular diplopia is very rare. When the patient complains of diplopia—and especially when he sees the object three or four times (polyopia)—this symptom must be tested for by examining each eye with the other closed. It is caused by the structure of the lens and the monochromatic aberration (Helmholtz) to which it gives rise. Diplopia, however, only occurs along with defective accommodation. This may be the result of errors of refraction (myopia). But in the great majority of cases the symptom is of an *hysterical* nature.

The following laws should also be remembered: if the images lie close to each other (without difference of level) and if the diplopia is homonymous, then the paralysed muscle belongs to the eye on the side towards which the object has to be moved in order to increase the distance between the images. If one image lies above the other, then the paralysed eye is the one whose image moves upwards when the patient looks up, and downwards when he looks down.

According to Kunn there is in addition to paralysis of the ocular muscles, a dissociation of the eye movements, *i.e.* a loosening of the normal associative connection when the eyeballs are in the lateral position. This would cause a kind of limitation of mobility and a strabismus, which would be characterised by their inconsistency and irregularity. He has observed this disturbance in hysteria.

Paralysis of Individual Muscles

External Rectus.—Limitation of the power of outward movement. Convergent strabismus, which becomes gradually increased by secondary contracture of the internal rectus. Diplopia in the half of the field of vision corresponding to the affected eye. Homonymous diplopia. The images diverge from each other when the object in front of the affected eye is moved outwards. The head is rotated towards the side of the affected muscle.

Internal Rectus.—Defective movement inwards. Divergent strabismus. Crossed diplopia in the side of the field of vision corresponding to that of the unaffected eye. Head rotated towards this side.

Superior Rectus.—Limitation of the upward movement. In the effort to move the eyeball upwards, the inferior oblique is contracted and produces a simultaneous rotation of the eyeball. Crossed diplopia in the upper half of the field of vision. The upper image, the false one, is inclined and becomes separated from the other when the object is moved upwards. Head is bent backwards and towards the unaffected side.

Inferior Rectus.—Limitation of downward movement. In attempting this movement, the eye is deviated outwards (superior oblique) and is at the same time rotated. Diplopia in the lower half of the field of vision, crossed, the false image lying lower than the true one and diverging downwards from it when the object is moved downwards. The false image is oblique. Head is bent downwards and towards the affected side.

Inferior Oblique. Limitation of upward and outward movement. In looking upwards the eyeball deviates inwards. Homonymous diplopia in the upper half of the field of vision. The false image is raised and inclined outwards.

Superior Oblique.—Limitation of movement is slight. In looking downwards the eye deviates somewhat inwards. Homonymous diplopia in the lower half of the field of vision. Convergent strabismus. The diplopia becomes specially evident in going down stairs, the steps appearing double.

In paralysis of all the muscles which move the eyeball, it is immovable, looks straight forwards, and there is a slight exophthalmos, *i.e.* the eyeball protrudes slightly from the orbit. If all the muscles are paralysed with the exception of the superior oblique and the external rectus, the eye is directed outwards and downwards. In looking downwards rotation takes place, so that the upper end of the vertical meridian is turned inwards. There is diplopia in the whole field of vision.

Exophthalmos, i.e. pathological prominence of the eyeball, may be so marked that the protruding eyeballs cannot be covered by the lids. This symptom is most frequent in Graves' disease. It may occur to a slight degree in exaggeration of the brain pressure, especially in hydrocephalus. Tumours may also develop behind the eyes (also aneurysms, abscesses, etc.) and press the eyeball forwards. In such cases the exophthalmos is almost always unilateral. An intermittent exophthalmos has also been observed, which occurs only on bending, and is due to varicose dilatation of the retrobulbar veins or to some transient condition of swelling in the retrobulbar tissue (Vossius).

Paralysis of *convergence* and *divergence* (Parinaud) has hitherto been rarely observed. In the first case the convergent movement is defective or quite absent. There is either no movement of the eyes when the finger at which patient is gazing is approached, or movement is incomplete, whilst in the associated lateral movement of the eyeballs, the function of the internal muscles is quite normal. There is crossed diplopia, the images remaining at an almost equal distance in all directions. Sometimes the distance is lessened when the object is moved laterally. It may also happen that the double images become blended when at a

distance of about 4 to 5 yards. Accommodation may be normal or diminished, and the convergence reaction of the pupils (see above) may be affected, whilst the light reaction is conserved.

Paralysis of divergence is still more uncommon and difficult to recognise. The patient can bring the eyeballs into the position of convergence for near objects, but cannot bring the axis of vision back into the parallel position or fix them on a point at a distance of 1 to 2 yards. The double images appear in the median plane only at certain distances ; the diplopia is homonymous at a short distance and persists wherever the eyes are directed. It is assumed that a special centre exists, not for convergence only, but also for the movement of divergence, as it represents an active process (Dor).

Paralysis may be combined, affecting both convergence and divergence. Vertigo almost always accompanies this form of paralysis.

We must here mention another symptom which may be caused by paralysis as well as by spasm of the eye muscles ; that is *conjugate deviation* of the eyes, which is frequently associated with a turning of the head in the same direction. It is due either to a tonic contraction of the associated muscles which rotate both eyeballs to one side, or to a paralysis of the antagonists.

Nystagmus is the name given to tremor of the eyeballs, which may occur in the position of repose, but which becomes especially evident during movement. It may occur in the horizontal and vertical directions ; rotatory-nystagmus is less common. (For particulars see the special part.)

The *function of the internal ocular muscles* should also be tested, as its disturbance is sometimes the first sign of a commencing disease.[1]

Of the muscles of the iris, the *sphincter pupillæ* (oculo-motor nerve) is the contracting muscle, the *dilator pupillæ* (sympathetic nerve) is the dilator of the pupils. Contraction of the pupils occurs physiologically under three conditions :—

1. In illumination of the retina. If the shaded eye is suddenly exposed to the light, the pupils distinctly contract. This contraction is also consensual, *i.e.* in illumination of either eye the pupils of both eyes are contracted. The test is made by keeping one eye closed, whilst the other is at first shaded by the hand and then suddenly illuminated by its removal. Ordinary daylight is usually sufficient ; the patient should not, however, be in the half darkness at the back of the room, but should be examined beside the window. If daylight is not sufficient, the examination should be repeated with a strong artificial light, so that in the dark room we can throw the lamplight on to the retina, preferably through a convex lens. The electric pocket-lamp which every doctor should possess, is admirably adapted for this examination.

We cannot here describe the apparatus which have in recent years been recommended for an exact examination (Schadow, Sommer, Hess, Bach, Weiler, Hübner and others), such as the Westien lens and the various pupillometers, or the methods of photography of the pupils (Fuchs, *J. f. P.*, xxiv.).

[1] As sources of literature we would specially mention : O. Marburg, "Die diagnostische Bedeutung der Pupillenreaction." *Wiener Klinik*, 1903, H. 8. Bumke, "Die Pupillenstörungen bei Geistes- und Nervenkrankheiten," Jena, 1904. Wilbrand und Saenger, "Die Neurologie des Auges," 1900 *et seq.*

The eye should at first be so feebly illuminated that the examiner can just see the pupils, and then the lens is brought between the light and the eye. Whilst the patient is gazing into the distance, the light is suddenly turned on to the pupils, and they instantaneously become contracted. With the electric pocket-lamp the lens is quite unnecessary.

The contraction on the entrance of light is a reflex movement. The stimulus which falls upon the retina, is conducted into the optic tract and transmitted to the oculo-motor nucleus, whence it passes through the oculo-motor nerve to the sphincter pupillæ. It follows, therefore, that this reflex may be affected by morbid processes at different sites.

We cannot at present discuss the theories according to which this reflex centre is situated in the ciliary ganglion or in the lowest part of the medulla oblongata or upper cervical cord, nor the view that the corresponding centripetal tract finds its way into the cervical cord. We shall return to these in the special part.

We may also neglect the hypothesis of a special tract in the trigeminus for the dilator reflex in shadow, which Kreuzfuchs has recently put forward.

If the light reflex is absent even in intense illumination, there is either an *absolute rigidity* (immobility) or a *reflex rigidity of the pupils*. This latter condition is often termed the *Argyll Robertson symptom*. The reaction of the pupils to light is *sluggish*, or incomplete, if on intense illumination of the iris they contract but slightly and slowly, or if the contraction is limited to a part of the pupillary area. Much skill and experience are required in judging of these conditions. It should be specially remembered that in pupils which are congenitally narrow, there is very little room for contraction to take place, so that one must be specially careful in such cases before assuming that the reaction is absent or sluggish. According to the investigations of Gudden, Retzius, Monakow, and to the observations of Heddæus and others, the optic nerve appears to contain special fibres for the conduction of the stimuli causing the movements of the pupil, which are not identical with the optic nerve bundles in the strict sense (*i.e.* concerned with vision). These fibres probably undergo partial crossing in the optic chiasma, but their further course is not yet definitely established. It appears that these fibres may still remain capable of function in rare cases of disease of the optic nerve.

In *unilateral reflex immobility of the pupils* the affected pupil does not contract from illumination either of the same or of the other eye, whilst the normal pupil reacts both directly and consensually. In contrast with this there are cases in which the direct light reaction of one eye and the consensual reaction of the other are both absent, a condition which some writers have sought to differentiate from immobility of the pupils by the vague name of "reflex-deafness" (Reflextaubheit) and to make use of for diagnosis (Möbius, Schwarz), but this attempt has hitherto at least led to no practical results.

In unilateral disease of the optic nerve it may happen that in direct illumination the pupil remains immobile or reacts incompletely, whilst in illumination of the retina of the other eye, it reacts consensually and may even be excessively contracted. This is a diagnostic sign of some

value in cases in which retrobulbar disease of the optic nerve cannot be recognised opthalmoscopically (Hirschberg).

Saenger has observed that under certain conditions the rigid pupil may again become capable of reacting after remaining for a long time in the dark. Babinski has also noted this fact (*R. n.*, 1905).

Under prolonged intense illumination a spasm of the sphincter iridis may develop (Heddæus).

The light reflex of the pupil may also be abnormal in activity and in range of movement (Oppenheim, Hübner).

2. There is a contraction of the pupil in *convergence*. The movement of convergence (due to the synergic contraction of the internal recti) is accompanied by a contraction of the pupils. This process is an *associated movement*, *i.e.* one movement is necessarily associated with the other, the voluntary impulse which produces contraction in the internal recti simultaneously reaches the sphincter iridis.

The experimental investigations of Marina and Cofler have not succeeded in explaining the close connection of these processes.

The reaction of convergence is frequently retained when the light reflex is lost. In order to test this point, the patient should be asked during the examination of the light reflex to gaze fixedly into the distance and to avoid any movement of convergence.

3. *Accommodation for Near Objects.*—This condition as a rule coincides with that described under 2, as the accommodation is combined with a movement of convergence. As accommodation may, however, be retained in paralysis of the internal recti, there is an accommodative contraction of the pupils without the movement of convergence. In order to test this, the patient should first look into the distance, and then at the point of his own nose. The accommodation reflex is sometimes so excessively active that it may be difficult to recognize the existing immobility to light. This test is only satisfactory when the gaze has been directed steadily into the distance for a considerable time.

A new theory, according to which the contraction of the pupils in accommodation is caused by the ciliary body expressing its blood into the iris, excess of blood in the latter producing the contraction (Knies), does not appear to us to be founded on sufficient grounds.

A marked sluggishness of the pupils in accommodation and convergence, the contracted pupil dilating again very slowly, is a symptom which has sometimes been noted (Strassburger, Saenger, Nonne, Rothmann, Piltz; see *N. C.*, 1903). The pupils in these cases did not react to light.

We speak of *absolute rigidity of the pupils* when there is no reaction either to the stimulation of light or in the movements of convergence and accommodation.

The views of Levinsohn and Arndt, that the reflex immobility of the pupils resulting from inactivity of the sphincter pupillæ gradually becomes absolute rigidity, and that these phenomena merely differ in degree, are opposed by important experimental facts.

Isolated cases are described (Raggi, Obersteiner, Kahler, Lépine, Morselli, Bechterew, Silex, Frenkel, Piltz,[1] Bojadjeff and others) in which the pupils are apparently dilated under illumination (*paradoxical pupil reaction*). This is probably in most cases merely an illusion, there being either

[1] *N. C.*, 1902 (with literature).

a secondary dilatation which was preceded by a transient and unnoticed contraction, or a pupil insensible to light which is dilated because at the moment of illumination the eyeballs were passing into the position of divergence (adaptation for distance). It has also been ascribed to the stimulation of heat. There are a few cases, however, in which none of these factors appear to have been at work and in which there was a real paradoxical reaction of the pupils (Piltz). A paradoxical or perverted reaction of accommodation is also described (Vysin, Spiller).[1] In any case we cannot be too careful in regard to the assumption of these paradoxical reactions.

We must also notice the fact that strong sensory stimulations cause a dilatation of the pupils. This symptom has been taken to prove that the sensory stimulus is conveyed to the centre for the dilator pupillæ muscle (sympathetic reaction of the pupils). According to the investigations of Schiff, Foa, Braunstein, and Bumke, however, it appears to be a case rather of a reflex inhibition of the oculo-motor centre for the sphincter. Sensory stimulations, acoustic for instance, may have a similar effect according to some writers. This process may be tested by stimulating the skin in the region of the forehead, temples, throat or neck by pricking or by a faradic brush. So far, however, this examination has not proved of real value as regards diagnosis, in spite of some noteworthy results which Hirschl, Stefani, Nordera, Várady and Hübner obtained from their investigations.

Loewy's proposal, which is not new, to make use of the reaction to distinguish between organic and psychogenic pain, will not stand critical investigation.

Pathological conditions such as foreign bodies in the nose, ear, etc., which keep up a constant irritation, may also have an effect on the size of the pupils (H. Frenkel, Moos, Sabrazés).

A. Westphal (ref. *N. C.*, 1903) has drawn attention to a pupil symptom which had been already observed by Graefe, Wundt and Galassi, but which has not been generally known; viz. that in strong contraction of the orbicularis oculi the pupils become contracted. This symptom can be demonstrated in immobile pupils by asking the patient to shut the eyes firmly, whilst by fixing the lids we prevent their being closed. The eyeballs fly upwards and the contraction of the pupils appears—apparently as an associated movement, though Schantz does not agree with this view. Piltz (*N. C.*, 1903) has closely studied this phenomenon, but it has not yet come to be of any diagnostic importance.

The *size of the pupil* varies in different individuals within very wide limits, but there is a degree of contraction and of dilatation which must be regarded as pathological. A contraction of the pupils to the size of the head of a pin (myosis) is almost always pathological, and is caused either by poisons (morphia especially), or by disease of the nervous system. In old age the pupils are frequently narrow and sluggish in their reaction (Möbius, Möli).

We cannot say at all definitely at what degree dilatation of the pupils should be considered pathological.

Schirmer (*D. m. W.*, 1902) desires that the *power of adaptation*, *i.e.* the capacity of the pupils to adapt themselves to various degrees of brightness, should be taken into consideration, and that the maximum adaptation of 4 mm. should be regarded as the physiological size, but this idea has hitherto found little acceptance.

[1] Reference in *N. C.*, 1904.

It cannot in each case be always determined whether the pathological dilatation is due to a condition of irritation in the sympathetic and therefore to a spasm of the dilator pupillæ muscle, or to a paralysis of the sphincter pupillæ (oculo-motor).

In paralysis of the sympathetic, the pupil is persistently narrower than normally, but it contracts to light and convergence as the other eye does, although the dilatation in the shade is incomplete. Cocaine also fails to exert a dilating influence on the pupil.

The dilator contracts also in mental excitement. This fact as well as experimental observations (Schiff, Bechterew, Piltz) and clinical experience shows that there is a cortical centre for this muscle. Haab has found that even the effort to direct the attention to some source of light in the periphery of the field of vision, without moving the eyeball, causes narrowing of the pupil. In some individuals the idea of a dark room or even of pain is sufficient to cause the pupils to dilate, and Piltz has shown that this "ideomotor" or "attention-reflex" of the pupil is a physiological phenomenon, the idea of light producing a contraction, and that of the dark a dilatation of the pupils. Bechterew had previously found in one person the remarkable symptom of ability to dilate the pupils "at will"; a certain impulse directed towards the right eye sufficed to produce this dilatation. Bloch [1] relates a similar observation. We must therefore conclude that the sympathetic nervous system is in exceptional cases and under certain conditions accessible to the influence of the will.

Atropin produces a maximum dilatation of the pupil (mydriasis) from paralysis of the sphincter; it is doubtful whether the dilator has any influence. Duboisin and hyoscyamin have a similar action. The effect of the atropin lasts for several days, sometimes for ten. The influence of homatropin is much more transient. Cocaine also dilates the pupil, but slightly and for a short time only, as it stimulates the endings of the sympathetic pupillary fibres, and when in strong solution also has a paralysing effect upon the endings of the short ciliary nerve. The pupils are wide and immobile in chloroform narcosis. The reaction of the pupil to light may be diminished by acute alcoholic intoxication (Gudden), but this is found mainly in alcoholics and degenerates (Cramer, H. Vogt).[2] Eserin causes a maximum contraction from stimulation of the sphincter and paralysis of the dilator. The pupil becomes narrow and reacts sluggishly to light under the influence of morphia. Rigidity of the pupil has been observed in poisoning with Filix mas (Knies).

In paralysis of the sphincter iridis the pupil is wide (paralytic mydriasis) and rigid. When the sphincter is irritated the pupil becomes narrow (spastic myosis) and its reaction is more or less diminished. In paralysis of the dilator the pupil is moderately narrowed (paralytic myosis) but it still reacts completely to light. If there is spasm of the dilator, the pupil is strongly dilated (spastic mydriasis), but as a rule it still reacts to light. An absolute rigidity of the pupil may be simulated by tonic contraction of the dilator muscles. This is the explanation of the pupil rigidity which appears exceptionally in hysterical attacks. It has also been observed as an accessory symptom in conditions of extreme

[1] *D. m. W.*, 1906.

[2] *B. k. W.*, 1905.

anxiety (Bumke). In paralysis of the sphincter and dilator the pupil is of medium size and is rigid.

Inequality of the pupils (aniscoria) is also a very important symptom. No weight should be laid on very slight differences, as these may appear in health and even as a congenital symptom (H. Frenkel). Schaumann, who confirmed this, regarded it as a sign of neuropathic or psychopathic diathesis. Inequality of pupils is also not uncommon in diseases of the internal organs. In marked degree it is always morbid. It may of course also be caused by differences in the condition of refraction of the two eyes and by differences in the light, as direct reaction exceeds consensual reaction in strength (Bach). When this is not the case and when there is no ocular disease, the symptom points to disease of the nervous system.

The symptom of irregularity in the form of the pupil, in its margin, etc., will be discussed in the special part.[1]

An uncommon phenomenon is that of "*springing pupil*," or "*springing mydriasis*," *i.e.* the dilatation within a short space of time now of the pupil of one eye, now of the other; inequality of the pupils is therefore almost always present, but the mydriasis affects now the right, now the left eye. This may be associated with rigidity of the pupils, and is then always of ominous significance, as it then forms a symptom or a prodroma of paralytic dementia or tabes. Springing pupil may, nevertheless, also occur in neurasthenia (Pelizaeus, Schaumann[2]), may be produced in heart disease by change of position (Geronne[3]), and has even been observed in health (Iblitz, W. Koenig).

A rapid alternation between narrowing and dilatation of the pupils without any external stimulation is not infrequently noted in healthy persons. This extremely rapid oscillation, which is known as *hippus*, occurs specially under pathological conditions, but the symptom is not of much importance in diagnosis, in spite of the data of Damsch, Michel, Pisenti, Siemerling, Vidal and others.

Paralysis of the accommodation muscles is revealed by the inability to adapt the eye to near distance, to recognise small objects, to read small print, whilst the sight for distance is unaffected and near vision is aided by use of a convex glass. If the eye is myopic, the affection of accommodation may be quite latent.

The Facial Muscles,

which are controlled by the facial nerve, seldom show isolated paralysis. There is usually paralysis of all the muscles of one side or of the greater part of them. The corresponding functional symptoms will be described in the chapter on facial paralysis (*see below*).

Sensibility of the Face

The cutaneous area of innervation of the trigeminus may be seen on Fig. 28. The posterior boundary is approximately formed by a line which connects the upper insertions of the ear with each other. The conjunctiva, cornea, nasal mucous membrane, tympanic cavity,

[1] The latest work on this subject is that of Piltz, *N. C.*, 1903.
[2] *Z. f. kl. M.*, Bd. 49.
[3] *Z. f. kl. M.*, Bd. 60.

and mucous membrane of the tongue and mouth are also innervated by the trigeminus.

Among the reflexes of this area the most important are the *conjunctival* and the *corneal*. These are tested as follows. The palpebral fissures are held open, so gently—and without touching the eyelashes—that the movements of the eyelids are not inhibited ; then the conjunctiva and cornea are lightly touched with a small pointless object, such as the head of a pin. In health this stimulation will elicit a contraction of the orbicularis palpebrarum, *i.e.* a closing of the eyelids. The intensity of this reflex varies greatly in different persons and diminishes in old age (Möbius). The conjunctival reflex especially is often feeble and may be voluntarily suppressed ; the corneal, however, is constant, and its absence is always pathological, although it seems to me that even in health the reflex may be occasionally extremely slight.

The *nasal reflex*, which has little diagnostic interest, consists in contraction of the nasal muscles and zygomatici, etc., which follows stimulation of the nasal mucous membrane by a pin.

The Masseter Muscles (Trigeminal Nerve)

The *masseter* and *temporal muscles* approach the lower to the upper jaw and when contracted on both sides they powerfully close the jaw. The *pterygoid muscles* cause lateral movement of the lower jaw. When they are contracted on one side the lower jaw is pushed towards the opposite side.

In unilateral *masseter paralysis*, the patient can only chew on the unaffected side ; the muscular contraction on closing the jaw can be felt and seen on this side only. In opening the mouth the lower jaw deviates towards the paralysed side, and the external pterygoid moves the condyloid process forwards on the healthy side only. The patient cannot move the lower jaw towards the healthy side

In bilateral masseter paralysis the jaws cannot be closed at all ; in paresis they can be feebly closed, but this can be more or less easily prevented by the examiner. Mastication is affected (dysmasesis). One of my patients helped himself by pressing his hand against his chin and so supplementing the masticating movements.

The action of the masseter muscles is sometimes combined with a peculiar associated movement : contraction of the levator palpebræ superioris. Thus it has been occasionally noted in patients suffering from ptosis that in opening the mouth and also in biting, the drooping eyelid is raised (Gunn, Helfreich, Bernhardt,[1] Coburn). G. Flatau observed in a hysterical patient a spasmodic movement of the eyeball inwards and upwards in opening of the mouth, *i.e.* in depressing the lower jaw.

Muscles of the Tongue (Hypoglossal Nerve)

In protrusion of the tongue, the *genioglossus* is specially concerned and in unilateral action, on account of its radiation into the tongue, it draws the latter obliquely towards the opposite side. In paralytic conditions, therefore, the protruded tongue deviates towards the paralysed side.

The tongue is moved backwards by the *styloglossus*.

[1] *N. C.*, 1894 ; see literature here and in Wilbrand-Saenger, " Neurologie des Auges," Bd. I.

In the other movements of the tongue, such as pointing, moving upwards and downwards, curling, etc., the intrinsic muscles, the lingual and transverse lingual, are specially contracted.

In unilateral paralysis, the tongue, so long as it lies on the floor of the mouth, deviates as a rule towards the healthy side. As soon, however, as it is protruded, the tip points towards the paralysed side.

If there is unilateral atrophy (hemiatrophy) the curving of the point towards the affected side is particularly marked; the affected half is diminished, flattened (Fig. 38), wrinkled and flaccid, forms hills and dales, and shows strong fibrillary tremors. This condition can be first recognised by palpation, as the half of the tongue taken between the fingers does not feel elastic and firm, like muscular tissue, but is flaccid.

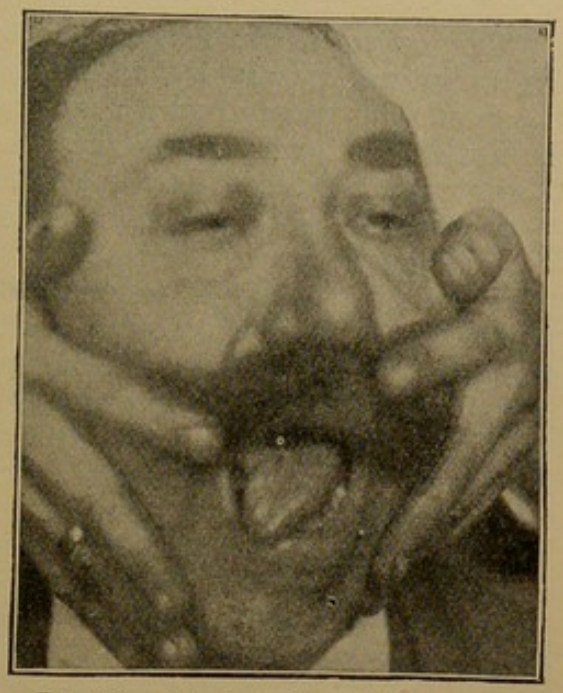


Fig. 38.—Hemiatrophy of the tongue. (Oppenheim.)

In bilateral paralysis, the tongue lies motionless in the floor of the mouth. Bilateral paresis is much more frequent. In this the tongue can only with difficulty be protruded slightly beyond the teeth, and drawn back again, the lateral movements being absent or very slow and incomplete. Bilateral atrophy (Fig. 39) can be inferred from the description of hemiatrophy.

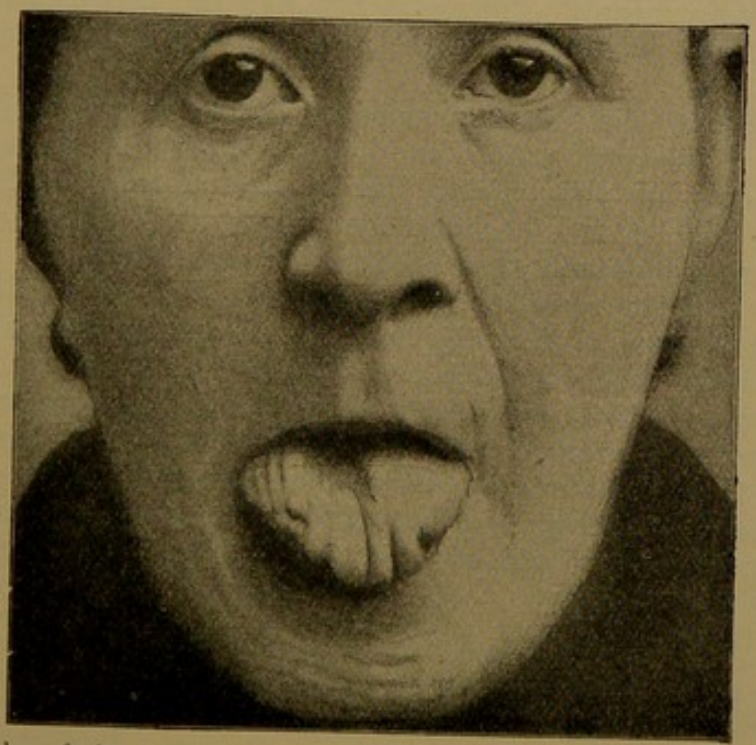


Fig. 39.—Atrophy of the entire tongue in bilateral paralysis of the hypoglossal nerve. (Oppenheim.)

Muscles of Palate and Pharynx. The Act of Swallowing and its Paresis

The *azygos uvulæ* muscle shortens the uvula and bends it backwards. In unilateral paralysis the uvula is curved towards the healthy side. It should be remembered that the uvula is not always exactly straight in health, but very frequently shows a deviation to one or other side. Unilateral paralysis does not affect speech and deglutition; bilateral paralysis does so (to a slight extent). Speech becomes nasal and fluids may return through the nose.

The *levator palati* muscle raises the soft palate. When it is paralysed the soft palate on the corresponding side is lower, the arch which forms the free margin is flatter than on the sound side, and this difference is clearly marked when the patient intones an "a." In bilateral paralysis the soft palate hangs loosely down and is not raised in phonation. The voice is markedly nasal. In drinking, part of the fluid returns through the nose.

The *palatoglossus* and *palatopharyngeus* muscles close the nasal cavity against the pharynx, whilst they approach the tongue to the palate and the posterior palatal arch. The palatopharyngeus muscle draws the soft palate downwards.

The *tensor palati* (innervated by the trigeminus) is also known as the dilator tubæ. Its paralysis causes insufficient opening or closing of the Eustachian tubes, and thus impedes the entrance of air into the tympanic cavity and affects the hearing. Paralysis of the palate was, however, not found by Krause in section of the 3rd branch of the trigeminal. The main nerve of the soft palate is the vago-accessorius. Some writers think the soft palate is innervated exclusively by it.

In order to test the function of the palatal muscles, we first observe the position of the soft palate in quiet breathing. To avoid eliciting reflex movements of the palate, the protruded tongue should not be pulled upon. The patient should be asked to phonate (to say "a" aloud). The soft palate is then distinctly contracted, the curve formed by the uvula and the anterior palatal arch is bolder, but the degree of tension is not the same in every individual, so that the signs of slight bilateral paralysis are difficult to recognise. It may be noted in passing that hypertrophy of the tonsils may produce a paresis of the soft palate.

Speech is always affected and has a *nasal* quality. There is however by no means always a complete parallelism between the speech affection and the degree of visible paralysis. The speech may be distinctly nasal, whilst the movements of the soft palate in phonation do not appear to be materially affected.

H. Schlesinger states that the affection of speech is less obvious in the lying position.

Mann has recently (*Z. f. Ohr.*, Bd. 47) attempted to give an exact characterisation of the paralysis of the various muscles.

The *palatal reflexes* may be retained or abolished in paralysis. These are tested by touching or tickling the uvula with a probe or the handle of a spoon. This is followed by a contraction of all the palatal muscles.

The speech is often markedly nasal although swallowing does not

seem to be affected. If the paralysis is complete, there is almost always regurgitation of fluid through the nose, and this may occur as an isolated symptom.

A certain degree of insensibility of the palate may be congenital; therefore absence of the reflex is not of itself a certain sign of a pathological condition.

In swallowing, the muscles of the lips, tongue, palate and pharynx are involved. The orbicularis oris is active in eating, and when it is paralysed, fluid tends to escape between the lips. The tongue is, in swallowing, pressed against the hard palate, first with the point, then with the back, and thus fluid food is forced back through the pharynx. The tongue is also used in order to remove the food which has been caught in the cheek pouches, or at any point in the cavity of the mouth, and to pass it backwards. If the tongue is paralysed, fluid does not pass backwards but returns towards the mouth. Small morsels of food remain on the tongue and in the buccal cavity.

Solid food is swallowed and passed into the stomach by the muscles of the pharynx and œsophagus. Paralysis of the œsophagus does not prevent drinking. When these muscles are incompletely paralysed, solid food can only be swallowed slowly and with great effort. In unilateral paralysis of the pharyngeal muscles, the corresponding half of the pharynx is dilated, and during the movement of swallowing does not contract, whilst the other half is protruded into it.

" Swallowing the wrong way " is a symptom which often accompanies paralysis of deglutition. The patient coughs in drinking, especially if the fluid contains finely broken-up particles of solid. This is due to the fact that while the fluid is passing down, some of it makes its way into the entrance to the larynx, which is either fully open or imperfectly closed.

The sounds produced in the act of swallowing are of no real diagnostic importance.

The Muscles of the Larynx. Their Functional Disorders

Affections of the voice or of phonation can immediately be recognised from *hoarseness* or *loss of voice* (aphonia). But as a matter of fact the function of the tensors of the vocal cords may be affected without producing any marked change of voice ; indeed the vocal cord of one side may be almost entirely paralysed without giving rise to any very evident disorder of phonation. This shows the necessity for a *laryngoscopic examination*, which is the more requisite from the fact that hoarseness and aphonia (as well as all the changes in the timbre of the voice) may be caused by disease of the mucous membrane and the deeper lying tissue as well as by paralysis of the muscles. It is only by laryngoscopic examination that we can determine which muscles are involved in the paralysis.

This is not the place to discuss the nature and technique of laryngoscopic investigation.

The annexed figures of laryngoscopic pictures show the most important forms of laryngeal paralysis (Figs. 40-44).

The respiratory movement of the vocal cords, the dilatation of the rima glottidis in inspiration is controlled by the crico-arytenoids.

In *unilateral recurrent paralysis* (Fig. 40) the vocal cord of the

paralysed side is in a position between adduction and abduction (cadaveric position) and is not approached to the middle line in phonation. The vocal cord of the healthy side extends beyond the middle line and the arytenoid cartilages are crossed. In inspiration the paralysed vocal cord does not move and the unaffected cord assumes the position of abduction.

In *bilateral recurrent paralysis* (Fig. 41) both vocal cords assume the cadaveric position and maintain it both in phonation and in respiration. There is aphonia and considerable difficulty in respiration (inspiratory dyspnœa).

Paralysis of the internal thyro-arytenoids (internus paralysis) (Fig. 42)

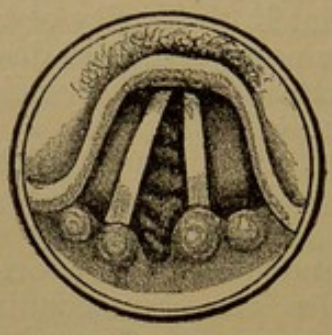


Fig. 40.
Unilateral recurrent paralysis.
Position of inspiration.

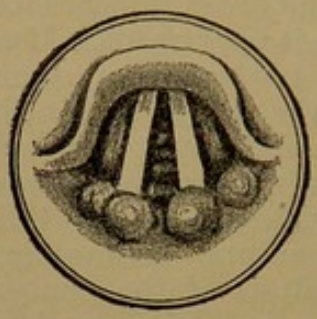


Fig. 41.
Bilateral recurrent paralysis.
Cadaveric position of the vocal cords.

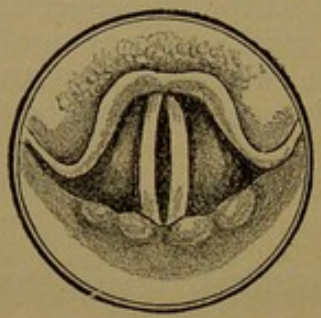


Fig. 42.
Paralysis of the internal thyro-arytenoid muscles.
Attempt at phonation.

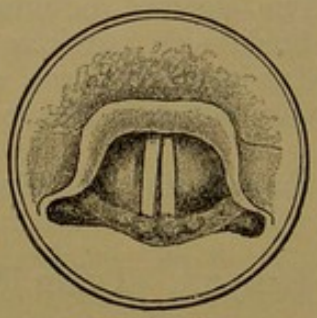


Fig. 43.
Paralysis of both posterior crico-arytenoid muscles.
Position of inspiration.

Fig. 44.
Bilateral paralysis of the internal thyro-arytenoids combined with paresis of the arytenoids.

Figs. 40-44.—(After Strümpell and Eichhorst, taken from Hirt's textbook.)

causes hoarseness. In phonation the glottis is not completely closed, but an oval fissure remains between the vocal cords, which remain flaccid. If the arytenoids only are paralysed, the glottis is closed only in its anterior section during phonation, a small triangular slit remaining posteriorly. Combined paralysis of the arytenoids and the internal thyro-arytenoids is shown in Fig. 44.

Paralysis of the *crico-arytenoidei postici* (posticus paralysis) causes inspiratory dyspnœa. If the paralysis is unilateral or incomplete, inspiration is noisy; an inspiratory stridor is heard and difficulty first arises during efforts which require increased respiration. In inspiration the rima glottidis is not dilated; the vocal cords are in fact brought still closer to each other, sucked together (Fig. 43). (A somewhat similar condition may, however, be observed in the normal larynx, if a forced

inspiration is made; we must therefore request the patient to breathe quietly).

Paralysis of the crico-thyroids makes the voice rough, and impedes the production of high notes; laryngoscopic appearances are not characteristic, but this muscle should be regarded as an accessory muscle of the phonators.

Electrical examination of the vocal muscles is possible if the electrode is introduced into the larynx, but it is very difficult to limit the stimulus to one muscle. This method of examination should be avoided on account of its uncertainty. The *recurrent laryngeal* may, however, be stimulated in the neck, between the larynx and the inner margin of the sterno-mastoid muscle at the level of the crycoid cartilage. Galvanic stimulation is most successful, but the currents employed must be strong. At the moment of closure (C.C.C.) we see a powerful adduction of one or both vocal cords. The stimulation does not always succeed, especially when the patient is fat and has a short neck.

Speech Disturbances

Speech depends upon the combined action of the respiratory, laryngeal, palatal, lingual and lip muscles.

Speech may be mechanically affected by cleft palate, or perforation of the palate, or even by loss of teeth. These mechanical impediments are readily detected. The form of speech affection caused by paralysis of part of the muscles involved in speaking, *affection of articulation or dysarthria*, is of more importance. It is characterised by changes in the sounds produced in speaking; some vowels and consonants are indistinctly pronounced, and speech itself is thereby indistinct and articulation defective. According as the paralysis affects the muscles of the lips, tongue or palate, there appears a disorder of the articulation limited to the formation of the labials, linguals or palatals respectively. If all these muscles are affected and their functions incomplete, speech will be difficult to understand and patient will speak through his nose and as if he had his mouth full. This form and the higher grades of disturbance of articulation are specially, though not exclusively, observed in diseases of the pons and medulla oblongata, and are therefore briefly termed *bulbar speech*. If speech has become an incomprehensible babble, or if it is quite abolished as the result of complete paralysis of the muscles of articulation, the condition is known as *anarthria* (which must not be confused with aphasia).

If the weakness affects the *lip muscles*, the vowel u and the consonants b, p, v and f are incompletely formed. In paralysis or weakness of the tongue muscles the vowels i and e and the consonants d, t, s, l, r; and in paralysis of the palatal muscles g, k, ch (guttural as in loch), ng, are indistinctly pronounced. A nasal quality of speech indicates weakness of the soft palate, b and p sound like m-b, m-p, etc.

Speech may show pathological slowing—*bradylalia*. In the higher degrees of this condition the words are clipped or broken up into syllables which are separated by perceptible intervals—*Scanning speech*. This affection may be recognised by asking the patient to pronounce a word of many syllables, such as Constantinople, etc., as rapidly as he can.

Stuttering is a spasmodic form of speech disorder. Spasmodic

muscular contractions keep the speech apparatus, especially the lips and tongue, fixed in the position requisite for the formation of a sound, especially the commencing consonant, so that the speaker cleaves to this sound, or in trying to get on further repeats it several times, until finally the whole word is forcibly shot out, as t-t-t-t-terror. The spasmodic muscular contractions are easily recognised. There are also not infrequently sympathetic movements and spasmodic contractions in muscles which are not involved in articulation. In singing the stuttering does not usually occur. There is also a mental inhibition of speech, such as the loss of power of speech caused by a sudden feeling of anxiety, which only lasts for a moment. These disorders appear in school children, for instance, who have to stop in the middle of a sentence or cannot answer a question suddenly put to them, although they have no difficulty about the answer itself. (With regard to mutism and other speech disorders see the special part.)

Pararthria exists when the single sounds and syllables cannot be pronounced in proper succession, but are confused with each other and mingled with other sounds which do not belong to the word, as "artillerary" or "artrillery" instead of "artillery," etc.

Aphasia is loss of power to translate ideas into words, although the speech muscles are unaffected, and loss of power to understand what is spoken, although the power of hearing is retained. This is described in detail in the Special Part.

II. SPECIAL PART

DISEASES OF THE SPINAL CORD

The Anatomy, Physiology and General Pathology of the Spinal Cord

The spinal cord is continuous above with the medulla oblongata. There is no line of demarcation and the origin of the first cervical nerve is looked upon as the upper limit; below, it reaches normally as far as the lower border of the first or the upper border of the second lumbar vertebra. Here it ends in the *conus terminalis*, while a bundle of nerve roots, forming the cauda equina, is prolonged downwards to reach the lumbar and the sacral intervertebral foramina (Figs. 45 and 46). Injuries and diseases of the second and lower lumbar vertebræ, therefore, cannot affect the spinal cord.

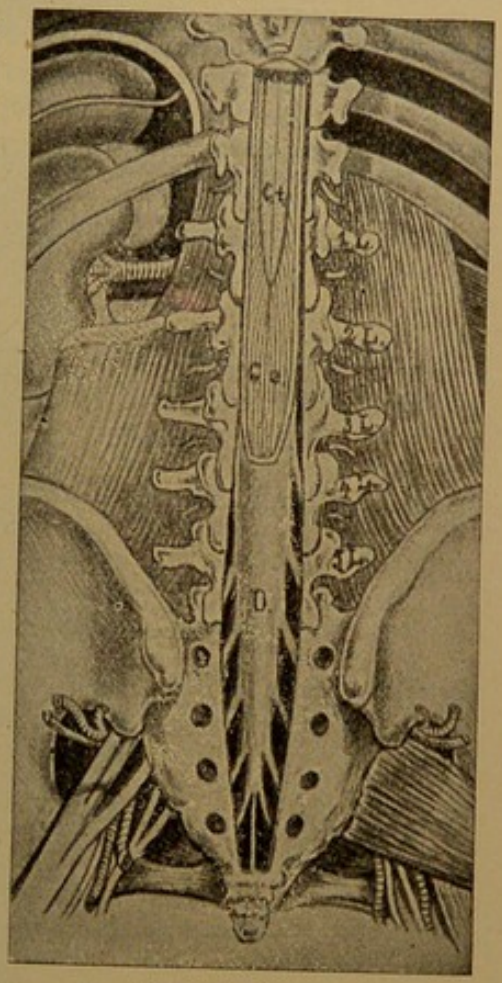


Fig. 45.—(After Henke). Position of the conus terminalis and the cauda equina in the spinal and in the sacral canal.
C. tr.—Conus terminalis.
C. e.—Cauda equina.
1 L.—1st lumbar vertebra.
D.—Dura mater.

The *cauda equina* consists of two halves, separated from one another by a narrow, elongated space containing fluid (cisterna terminalis of *Dönitz*). The *dura mater* is separated from the periosteum lining the vertebral canal by an areolar and adipose tissue containing many vessels, especially venous plexuses. The dura mater is separated also from the spinal cord by a fairly wide space; it is thick and dense and offers considerable resistance to diseases attacking the cord from without. The *arachnoid* is a double membrane, the outer fold of which is intimately connected with the inner surface of the dura mater, the inner one with the outer surface of the pia mater. Numerous very delicate trabeculæ stretch from one fold to the other. According to another view only the fold in relation to the dura mater is to be looked upon as arachnoid; the wide space lying within this, which contains the cerebro-spinal fluid, would then be called the subarachnoid space. This space is directly continuous with the subarachnoid space of the brain and with the lymph channels of the peripheral nerves—whose roots receive a covering from the dura and arachnoid. The cerebro-spinal fluid is clear, alkaline, poor in solid constituents (barely 1 per cent.), has a specific gravity of 1005-1010, and contains about 0·3 ‰ albuminous substances with traces of sugar. According to *Axel Key* and *Retzius* ("Studien über die Anatomie des Nervensystems," Stockholm, 1876), the subarachnoid spaces contain about 50-150 c.cm. of this fluid.

The *pia mater* adheres closely to the spinal cord and cannot be separated from it without loss of substance. From the pia mater numerous small septa pass into the cord and also into the anterior median fissure, where it is a thick, connective-tissue fold carrying blood-vessels—the anterior median septum. The pia contains non-medullated and also medullated nerve fibres; the latter are sometimes met with in the anterior septum arranged in bundles.

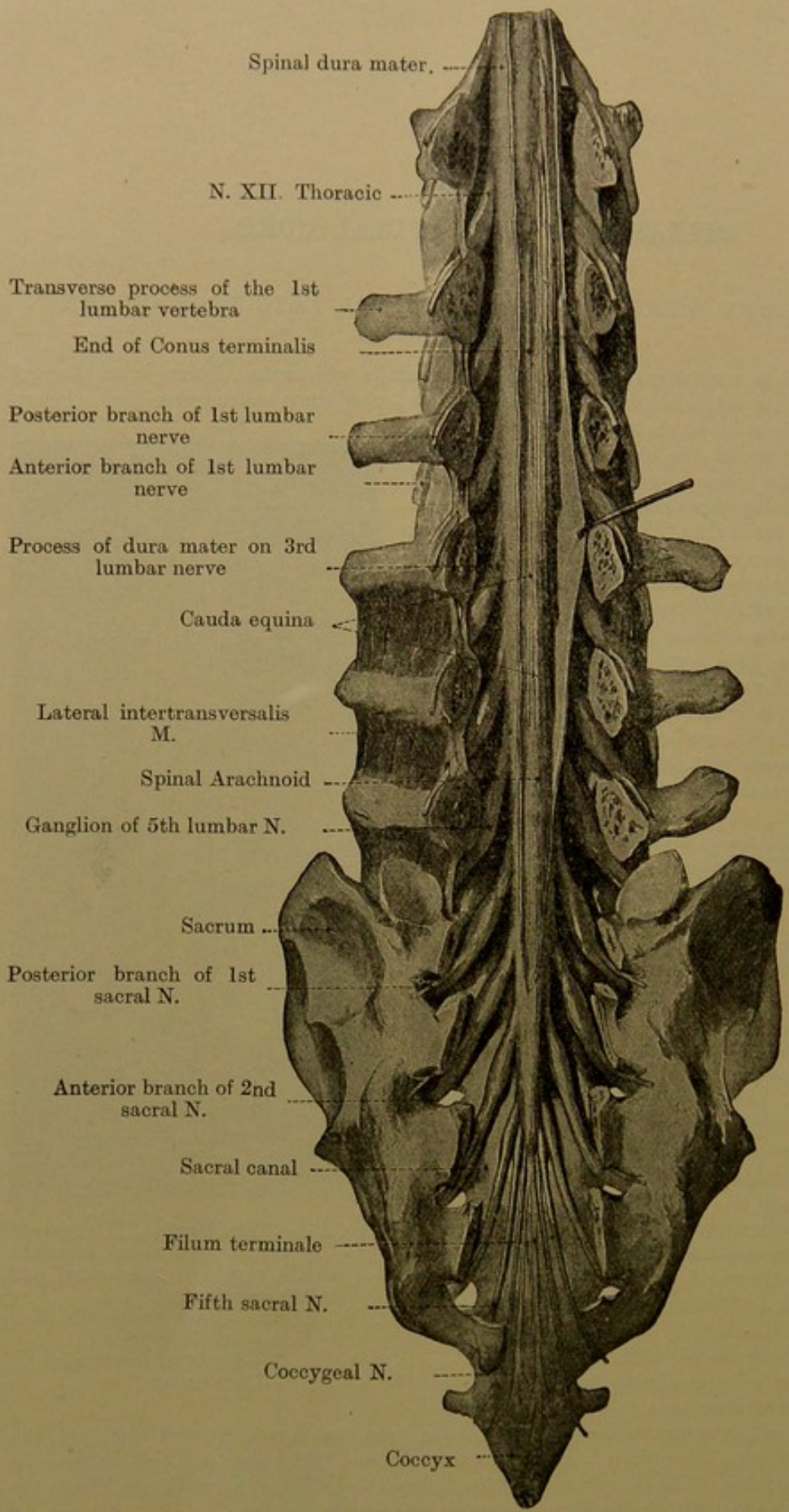


FIG. 46.—Lower end of the spinal cord with the membranes in the spinal canal. From behind. (After Spalteholz.)

The *circumference* of the spinal cord varies at the different levels. It remains almost the same at all parts of the dorsal cord.

The *cervical enlargement* shows a very marked increase in size. This commences at the level of the third and fourth cervical vertebræ, reaches its maximum at the fifth and sixth, and gradually diminishes till it passes into the dorsal portion at the level of the second dorsal vertebra.

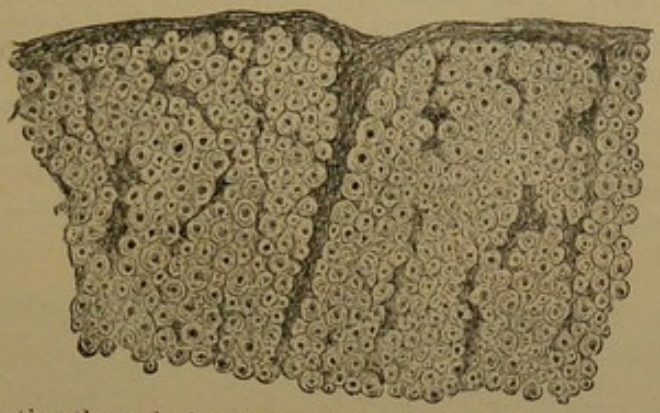


FIG. 47.—Transverse section through the white matter of the cord. From a section stained with carmine. Medium power.

The *lumbar enlargement* is shorter and the increase in circumference is not so great. It begins at the level of the tenth dorsal vertebra, reaches its maximum about the twelfth, then it diminishes to the sacral portion of the cord and ends in the conus terminalis. The cauda equina comprises the roots of the lumbar and sacral cord: the three upper lumbar roots soon branch off and the first can hardly be included in it.

On a transverse section of the spinal cord, taken at any level, the more centrally-lying *grey* matter stands out clearly from the *white* matter

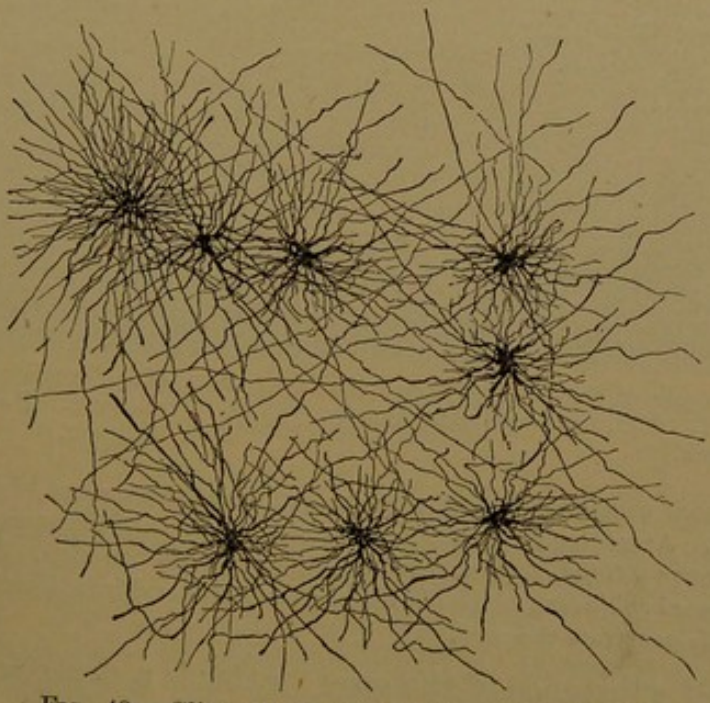


FIG. 48.—Glia cells (Golgi's type). (After Kölliker.)

which surrounds it on all sides. The contrast in the staining depends upon the difference in the histological structure. The white matter consists partly of medullated nerve fibres, which run chiefly in the longitudinal direction of the cord, and partly of the framework of the neuroglia supporting these. When a transverse section of the white matter is

examined under the microscope it presents the appearance of a series of fine, closely applied circles varying in size, each with a dot in the centre. These are the transversely divided medullated nerve fibres with their axis cylinders—the spaces between these are filled by the neuroglia (Fig. 46). The neuroglia cells have a narrow rim of protoplasm around the nucleus and numerous ramifying processes, some of which are very long (Fig. 48).

Kölliker divides the glia cells, according to the length of the processes, into *short-* and *long-*rayed cells. In the white matter of the cord it is chiefly the latter which are found. The neuroglia cells and their processes are disposed around the nerve fibres so that each of these may be said to lie in a tunnel of neuroglia—the vessels are similarly surrounded. The processes of these

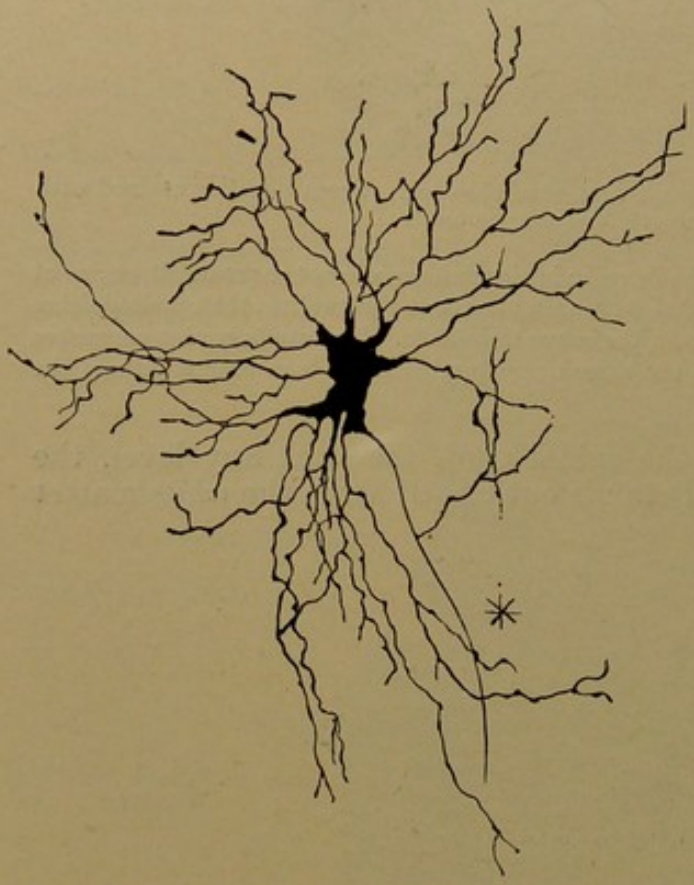


FIG. 49.—Ganglion cells of the anterior horn with Golgi's method. * The axis-cylinder process. (After Lenhossek.)

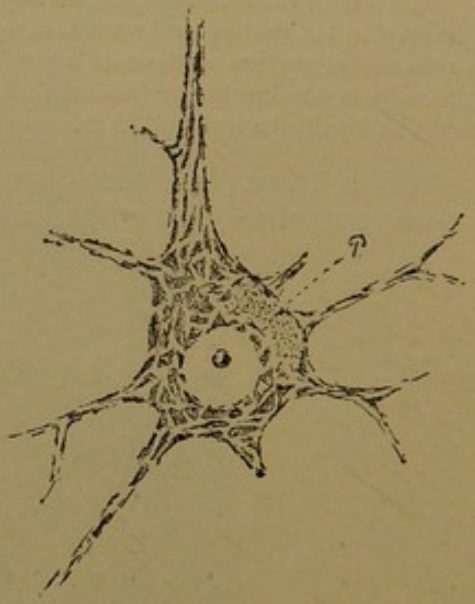


FIG. 50.—Ganglion cells by Nissl's method. P = pigment. (After Edinger.)

cells form a meshwork which traverses the whole cord, but the processes of different cells do not anastomose (*Golgi*). According to *Ranvier* and *Weigert* the fibrils of the neuroglia are independent of the cells, but *Ramon y Cajal, Ziehen, Held* and others dispute this view. A thin layer of neuroglia surrounds the cord subjacent to the pia mater; from this and from the glia forming the septa processes of the cells penetrate the cord.

The ground substance of the grey matter is a dense feltwork of fibres interlacing in all directions. These are derived from the medullated nerves and their offshoots, from the numerous processes of the ganglion cells and their branches, and from the glia tissue which is specially well-developed here. The blood-vessels are more numerous than in the white matter. The nerve cells, which form the specially important constitutent, are scattered throughout the grey matter, partly isolated and partly in groups.

In recent times views regarding the structure of the nerve cells and their relation to the nerve fibres have altered greatly, owing to the advance in staining technique. The observations of *Golgi*, *Ramon y Cajal*, *Kölliker*, *His*, and *Waldeyer*, made especially by means of the *Golgi* methods of silver impregnation, have helped to establish a conception—the so-called *neuron theory*—which has become the prevailing one and has greatly influenced our knowledge of the physiology and pathology of the nervous system. Recent investigations have thrown doubt on this theory, and it must at least be modified; yet I consider myself justified, together with many others, in holding to it in the meantime and making it the basis for any further observations.

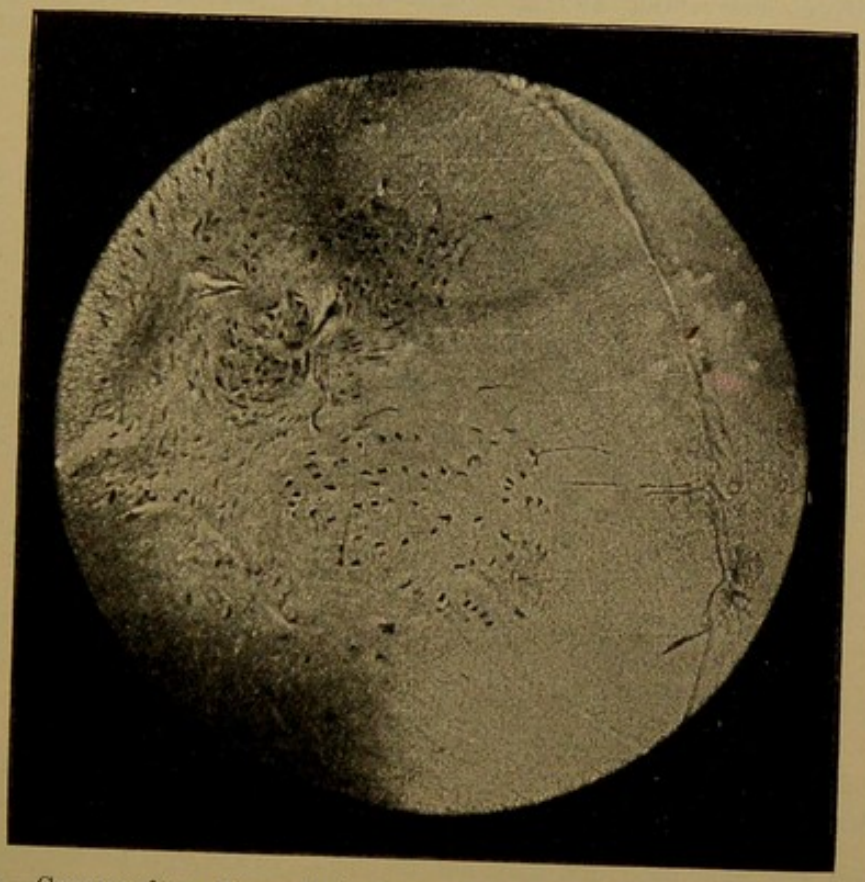


Fig. 51.—Groups of ganglion cells in the anterior horn of the lumbar enlargement. (From a photograph.)

According to this theory the nerve cells have one *nerve-process* (axon, neurite) and numerous protoplasmic offshoots (dendrites) which divide and subdivide, forming a dense network, and end in free arborisations (Fig. 49). They are not, therefore, in direct connection with the processes of other cells or with the offshoots of nerve fibres. The unit consisting of the nerve cell, the nerve-process and its terminal ramifications is designated a *neuron* (Waldeyer). The nerve-process of most cells becomes the axis-cylinder of a medullated nerve fibre. It is distinguished by its uniform calibre from the dendrites which divide antler-like, immediately after they leave the cell; the nerve-process, however, gives off lateral branches (*collaterals*). There are, moroever, nerve cells whose process breaks up within the grey matter (inner cells, reflex cells, *Golgi* cells).

Held had already taught that, in adults, the terminal branches of the nerve fibres end in direct and actual relation to the nerve cells when *Apáthy*, in particular, and then *Bethe*, on the strength of his own special methods of investigation, published observations which tended to undermine the neuron theory. They point out, as the principal result of their investigations, that the fibrils (neurofibrils) are the essential element of nerve tissue and that these traverse the nerve fibres as well as the nerve cells. From the nerve fibres they enter into the nerve cells, pass through their substance and into the processes—no distinction being made between nerve processes and the so-called dendrites. The fibrils do not terminate in the nerve cells but leave by the processes to form part of the meshwork of the grey matter (elementary lattice-work), and in this a free nerve-termination can nowhere be seen. Thus a continuity of the whole nervous system is brought about by the fibrils, and therewith the conception of the neuron has become untenable. In corroboration of this newer view is an experiment carried out by *Bethe* with the antennæ of the common crab, according to which reflex movements are brought about without the intervention of nerve cells. *Nissl*,[1] *Schenk*, *Hill*, *Durante*, *Pflüger*, *Held* and others have also disputed the neuron theory. *Lenhossek*, *Hoche*, *Verworn*, *Schmaus-Sacki*, *Münzer*, *Bardeleben*, *Lugaro*, *Dejerine* and others have doubted the conclusiveness of Bethe's

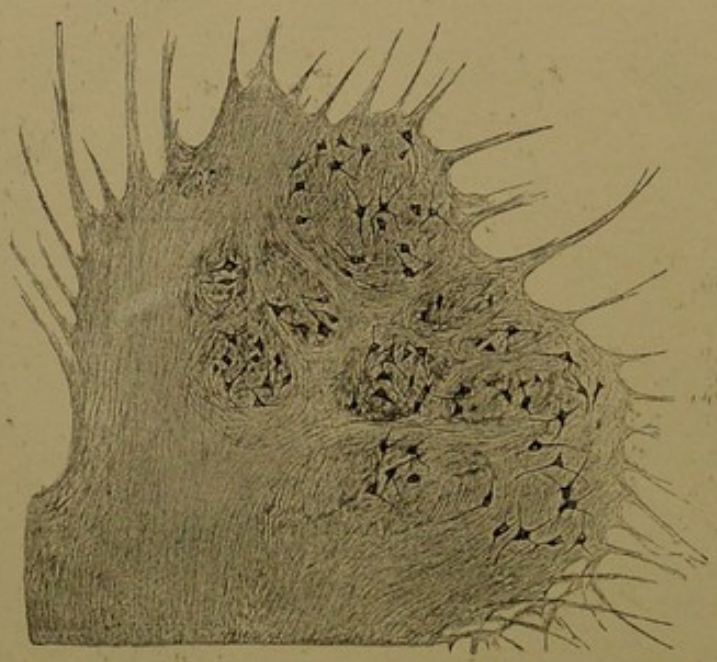


Fig. 52.—Groups of ganglion cells in the grey matter. (From a section stained with carmine.)

experiment and objected to his interpretation of the fibrils. In any case it is impossible from such experiments to generalize in regard to the higher animals and man. As yet we have no sufficient grounds to replace the neuron theory by the *Apáthy-Bethe* theory, although the accuracy of one portion of their observations—the evidence of the fibrillar structure of the ganglion cells and of the axis-cylinders to which the elder *Remak* and *Max Schultze* had before referred—can scarcely be questioned. The conception of the histological unity or independence of the neuron, however, probably requires modification. Compare also *Bielschowsky*,[2] who maintains the fundamental position of the neuron theory for the vertebrates, also *Merzbacher* and *F. Hartmann*.[3]

The grey matter forms the anterior and posterior horns, which are very easily distinguished from one another as the latter reach almost to the peripheral border of the spinal cord. Histologically the essential distinction is that the anterior horns have very numerous and large cells; some are so large that in a stained section they can be recognised by the naked eye.

[1] "Die Neuronlehre und ihre Anhänger." Jena, 1903.
[2] "Die histol. Seite der Neuronlehre." *Journ. f. Psych.*, Bd. V.
[3] "Die Neurofibrillentheorie und ihre Bedeutung," etc. Wien, 1905.

In the enlargements of the cord the cells are specially abundant and beautifully developed. In the anterior horns they are found lying together in well-defined groups (Figs. 51 and 53). It is possible to recognise with *Waldeyer*, a medial and a lateral principal group, each of which can again be divided into a ventral or anterior and a dorsal or posterior group; the lateral posterior group is the most marked and it also can be sub-divided. The anterior roots proceed chiefly from the lateral groups. The posterior horn cells form no distinct groups.

In the lower cervical and in the dorsal cord a *lateral horn* stands out clearly (intermedio-lateral tract) (Fig. 54). In the angle between the lateral horn and the outer border of the posterior horn lies the formatio reticularis.

In the conus medullaris the large nerve cells are found less fre-

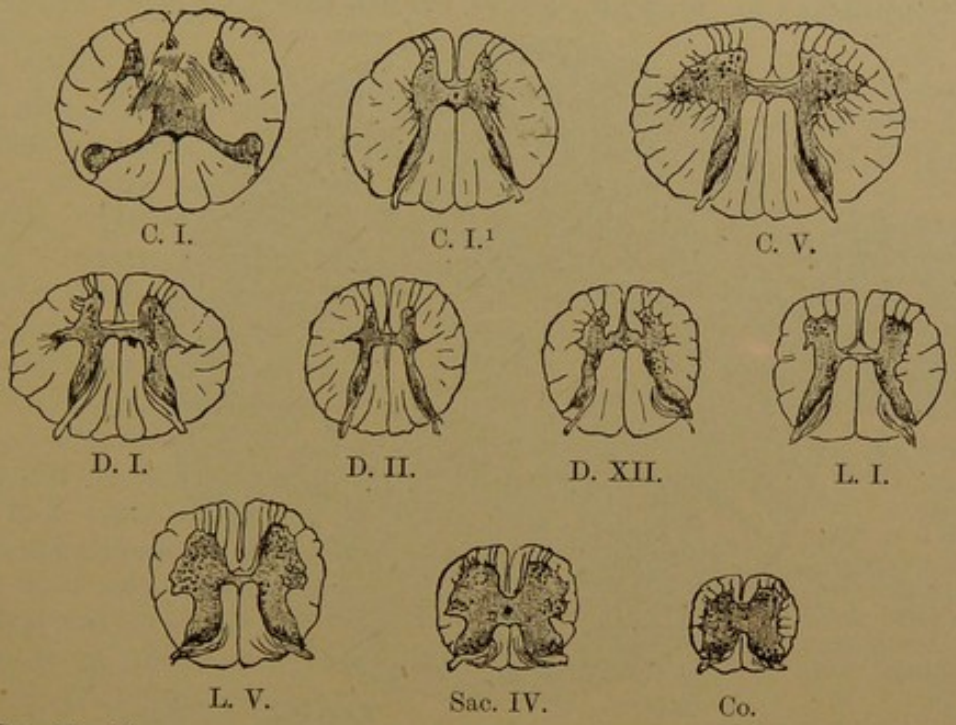


FIG. 53.—Transverse sections at various levels of the cord. (After Quain.)
C = cervical cord. D = dorsal cord. L = lumbar cord. Sac. = sacral cord. Co. = conus terminalis.
The Roman figures mark the root pairs at the level corresponding to the transverse section.

quently in the anterior horn than in the the intermediate area of grey matter which lies between the anterior and posterior horns (*L. R. Müller*).[1]

Onuf[2] gives a very accurate description of the groups of nerve cells in the sacral cord.

The configuration of the grey and white matter and the relation that these bear to each other vary at the different levels. With a little practice the portion of the cord from which the transverse section is taken can be recognised.

The illustration (Fig. 53) gives a picture of the transverse sections at the various levels. The increase of grey matter in the enlargements (C.v., L.v., etc.) and also its relative predominance in the sacral cord and in the conus terminalis are specially to be noticed.

[1] *Z. f. N.*, xiv. [2] *Arch. of Neurol.*, iii.

The spinal cord consists of two symmetrical halves connected by two commissures—the anterior white and the posterior grey (Fig. 54). The latter encloses the central canal which, in adults, is frequently filled up by a cell-accumulation. It is surrounded by gelatinous substance (*Stilling's* subst. gelat. centralis, *Kölliker's* central ependyma fibres). The ependyma cells lining the central canal send out long processes which traverse the spinal cord substance and some of which, in embryonic life at least, reach to the periphery—up to the pia mater. These elements share in the formation of the neuroglial framework.

Anteriorly, the two halves of the spinal cord are separated by a cleft, the anterior longitudinal fissure, into which a thick connective tissue fold of the pia passes. Posteriorly, the two halves are separated

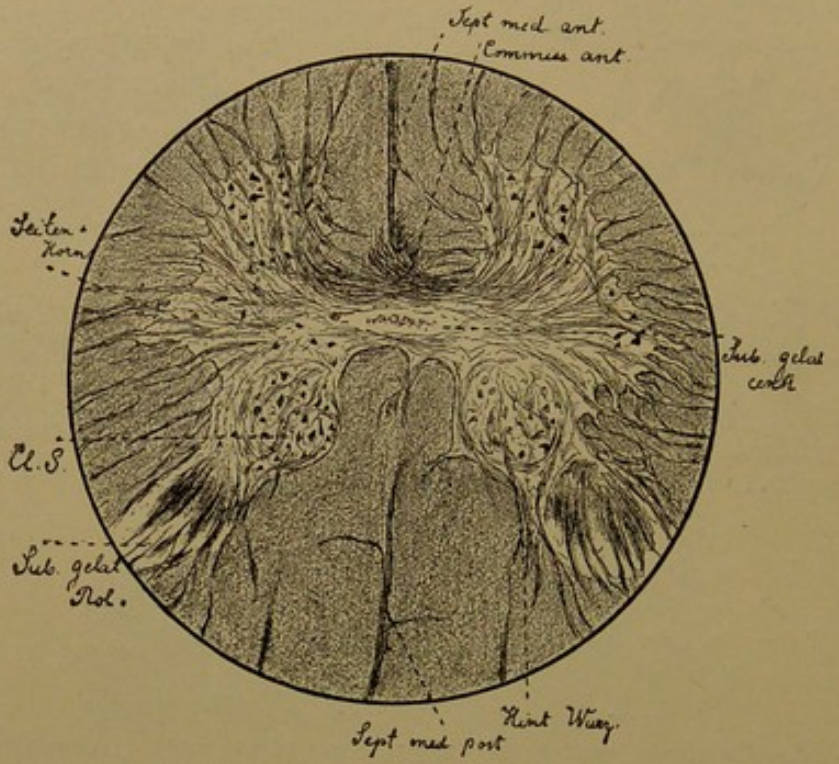


FIG. 54.—Transverse section of cord in the dorsal region. Cl. S. = Clarke's column. Seiten horn = Lateral horn. (From a section stained with Weigert's hæmatoxylin.)

by a septum (posterior or dorsal median septum) which may in part arise out of the processes of the ependyma cells of the central canal. A posterior longitudinal cleft (posterior fissure) can be distinguished only in the cervical cord and in the conus.

The anterior roots, arising from the anterior horns, pass through the white matter in bundles. They spring directly from the large nerve cells whose nerve-processes they form. The posterior roots enter the cord as one compact bundle, partly just where the posterior horn reaches near the periphery, and partly close to this in the white matter. Some of the fibres enter the grey matter of the posterior horn directly, some indirectly, and one portion remains for a time definitely with the white matter.

Within the white matter one distinguishes :—the area of the *posterior columns*—that part of the white matter which is included within the posterior horns ; the area of the *anterior columns*—the portion lying

between the anterior horns and the anterior fissure; lastly, the area of the *lateral columns*, *i.e.* the part situated on the outer side of the grey matter. The anterior and lateral columns are not clearly defined as the boundary is formed by a relatively broad area—the part of the anterior columns traversed by the anterior roots.

Studies in embryology and in experimental pathology, and observations in morbid anatomy have shown us that the white substance and, indeed, the individual columns can be subdivided into a number of tracts according to their development and function. These, it is true, cannot histologically be distinguished one from the other in the fully developed spinal cord, but in fœtal life and in early infancy they stand out clearly, because the various tracts of fibres acquire their myelin sheath at different periods (*Flechsig*). Thus, in the newly born child all the fibres have a myelin sheath except those of the pyramidal tracts (Fig. 55).

The following fibre-systems can be distinguished from one another and are all to be regarded as independent tracts (Fig. 56; *cf.* also Fig. 58).

1. The posterior column consists of (*a*) the column of *Goll* (funiculus gracilis); (*b*) the column of *Burdach* (funiculus cuneatus). The column of *Goll* lies close to the posterior median septum, that of *Burdach* on the outer side of and, in the cervical portion at least, distinctly separated from that of *Goll* by a septum (postero-intermediate septum).[1]

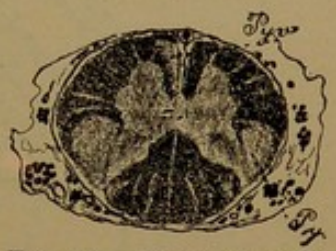


Fig. 55.—Section through a fœtal spinal cord. The crossed pyramidal and left direct pyramidal tracts are still undeveloped, the fibres being still non-medullated. (Weigert's stain.)

2. The lateral column contains (*a*) *the lateral or crossed pyramidal tract.* On transverse section it is triangular in shape. In the cervical and dorsal regions this tract does not extend to the pia, but in the lumbar region it reaches the surface of the cord; it does not extend quite to the grey matter. Its greatest extent is in the cervical part; it then decreases from above downwards, and in the sacral cord it is only just perceptible. (*b*) *The lateral cerebellar tract.* This tract lies on the outer side of the lateral pyramidal tract, between it and the pia and is almost crescentic in shape.

It is first distinctly developed in the lower dorsal region and increases in size from there upwards. With reference to this, however, experimental investigations (*Rothmann* and others) and individual observations in man (*Barbacci*) show that it can begin in the lumbar portion.

3. The *anterior column* contains the *anterior* or *direct pyramidal tract*, situated close to the anterior median fissure and forming there a narrow border. It is found usually only in the upper portions of the spinal cord and as a rule extends as far as the middle dorsal region, but individual variations are frequent. Investigations, especially by means of *Marchi's* method, have shown it to extend occasionally as far as the sacral cord (*Stewart, Marie-Guillain*).

The tract of *Gowers* or the *ascending antero-lateral fasciculus* is less sharply defined. It lies in the antero-lateral column and commences in front of the lateral cerebellar tract, reaches a little further in than this tract, and extends forward on the surface of the cord as far as the

[1] The further structure of the posterior columns need not be entered into here, as they have been described by *Flechsig, Trepinsky, Schaffer* and others, on the basis of their embryological relations.

entrance of the anterior roots or even further. Gowers' tract begins in the lumbar region.

With reference to the significance of these tracts we know that the columns of *Goll* and *Burdach*, the lateral cerebellar tract, and the antero-lateral fasciculus are centripetal tracts. They carry sensory impulses which, in part, reach the cortex as conscious sensations, in part reach lower centres influencing, in particular, the co-ordinating apparatus in the cerebellum, etc.

The pyramidal tracts are the paths of conduction of motor impulses; they connect the motor centres of the brain with the muscles. The lateral pyramidal tract is the crossed motor path and the anterior pyramidal tract the uncrossed path; that is, in the lower portion of the medulla oblongata the motor tract undergoes a partial crossing (Fig. 57)—its larger portion reaching the lateral pyramidal tract of the

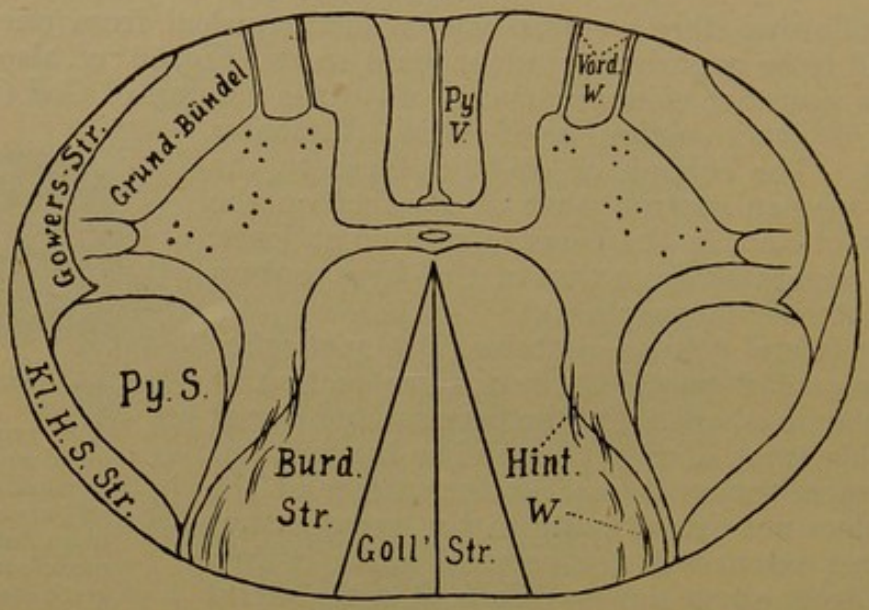


FIG. 56.—Diagram of the tracts of the spinal cord.

Goll' Str. = Goll's column.
Burd. Str. = Burdach's column.
Hint. W. = Posterior root.
Vord. W. = Anterior root.
Py. V. = Direct pyramidal tract.
Py. S. = Crossed pyramidal tract.
Kl. H. S. Str. = Direct cerebellar tract.
Gowers Str. = Gowers' tract.

opposite half of the spinal cord, while the smaller portion remains in the anterior pyramidal tract of the same side. Probably the latter undergoes a crossing in the cord itself, because at all levels its fibres are seen to bend from the longitudinal course to a more or less horizontal one and, by the white commissure, to reach the other side and pass into the anterior horn.

Kölliker, *Stoddart* and others think that all the fibres participate in this crossing, *Ziehen* that only some do, while *Lenhossek* denies any crossing of the fibres of this tract. The view that they are found at all levels of the cord has also been called in question.

Individual variations in the relations of the pyramidal tracts which may be considered as congenital abnormalities (absence of the pyramidal decussation, decussation of the anterior pyramidal tract, aberrant bundles of fibres, displacement of the pyramid, etc.) need not be here discussed. The fact that an unimportant bundle of fibres passes into the lateral pyramidal tract of the same side, and the controversy between *Marie-Gullain* and *Dejerine* (*R. N.*, 1904) which is still continuing, may be also disregarded here.

Lewandowsky ("Untersuchungen über die Leitungsbahnen," etc. Jena, 1904) thinks that the pyramidal fibres do not radiate directly into the anterior horns, but into the intermediate grey matter.

With reference to the portion of the white matter still to be considered not much is definitely known. Yet numerous recent and exhaustive investigations have greatly added to our knowledge. The white fibres around the anterior horn, not included in the tracts named, have been designated as *ground bundles* of the *anterior* and *lateral* columns, or as *antero-lateral ground bundle*. Flechsig distinguishes in the lateral column in the position not occupied by long fibre-systems the *anterior mixed lateral zone*, and the *lateral limiting layer* which adjoins the posterior horn, and in the posterior portion of the latter a special bundle (*median bundle of lateral column*). They do certainly contain many commissural fibres, *i.e.* fibres which unite the different levels of

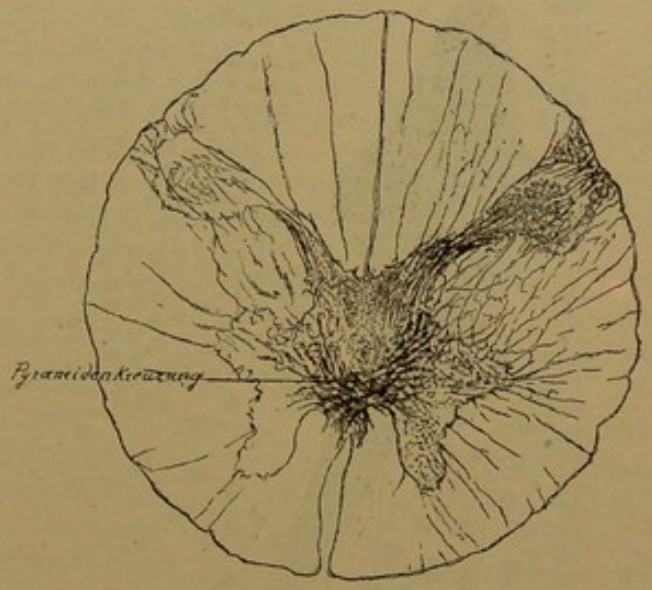


FIG. 57.—Transverse section in the region of the commencing pyramidal decussation. (Pyramidenkreuzung.)

the cord, passing from the grey matter of one segment to a segment higher or lower (intersegmental inner tracts of Ziehen).[1]

Further, Loewenthal and Marie have defined in the antero-lateral column fasciculi of fibres, of which one—the marginal bundle, *sulco-marginal* zone—on the anterior median border of the cord mingles to some extent with the fibres of the anterior pyramidal tract; the other runs in the antero-lateral ground bundle on the inner side of the lateral cerebellar tract and anterior to the lateral pyramidal tract (fasciculus intermedius, *cf.* Fig. 58). Though the function of these fasciculi of fibres has not yet been ascertained recent investigation has shown that in the antero-lateral column, in addition to short intersegmental tracts, there are also others containing fibres of long course. One portion of these conveys impulses from certain definite areas of the mid-brain, and from the nuclei of the medulla oblongata, probably also from the cerebellum (chiefly on the same side) to the spinal cord, while another portion

[1] According to *Flatau* the short ascending and descending fibres run near the grey matter, whereas the long ones tend to pass to the marginal zones of the cord (law of the eccentric position of the long tracts in the spinal cord). Observations of *Henschen* and others coincide with this.

ascending in the cord represents sensory tracts, especially a crossed sensory path of conduction (II. Order, see below).

The assertion of *Marchi* that fasciculi of fibres pass from the cerebellum to the spinal cord has met with much contradiction, but the existence of such cerebello-spinal tracts can no longer be doubted after the observations of *Kölliker, Bechterew, Biedl, Thomas* and others. It is uncertain whether they pass directly from the cerebellum to the spinal cord (*Russell*) or, as is more probable, are interrupted in *Deiters'* nucleus. They run in the antero-lateral columns and to some extent in the fasciculus intermedius and indeed among the fibres of the lateral pyramidal tract (possibly also in the lateral cerebellar tract), and thus enter into connection with the nerve cells of the anterior horn. Also from the medulla oblongata and from *Deiters'* nucleus, from the formatio reticularis and also from the corpora quadrigemina and the red nucleus, centrifugal

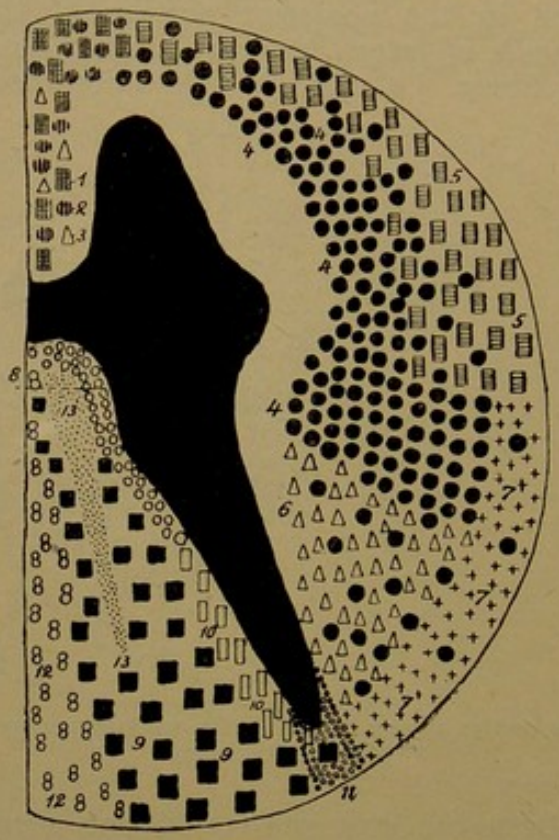


FIG. 58.—Diagram of the tracts in the spinal cord. (After Marie.)

1. and 2. Anterior basis bundle.
3. Direct pyramidal tract.
4. Lateral basis bundle.
5. Gowers' tract.
6. Crossed pyramidal tract.
7. Direct cerebellar tract.
8. Ventral portion of posterior column area.
9. Burdach's tract.
10. Entering posterior root.
11. Lissauer's tract.
12. Goll's tract.
13. Comma tract.

fasciculi of fibres pass to the spinal cord. (Recent observations of *Ferrier, Bechterew, Turner, Russell, Redlich, Monakow, Held, Tschermak, Gebhardt, Kohnstamm, Pawlow, Collier-Buzzard, Rothmann* and others.) These fibres run in the antero-lateral column, partly of the same side and partly of the opposite side. *Monakow* has described fibres descending from the red nucleus to the cord under the name of "aberrant lateral column fasciculus." This has since then been known as *Monakow's* bundle and its course has been exhaustively studied by *Rothmann* (*N. C.*, 1900). *Lewandowsky* also ("Untersuchungen über die Leitungsbahnen," etc., *Neurol. Arb. v. O. Vogt.*, Bd. i.) has devoted much attention to it. *Marie* and *Guillain* (*R. N.*, 1904) refer to parapyramidal fasciculi of fibres in the anterior columns, etc.

It is unnecessary to refer here to some wholly hypothetical fasciculi of fibres described by *Spiller, Barnes, Marie-Guillain, Stewart* and others.

The tracts of fibres in the white matter of the spinal cord may be differentiated thus :—

I. In the POSTERIOR COLUMNS
- (*a*) the column of *Goll.*
- (*b*) the column of *Burdach.*
- (*c*) less extenive and less sharply defined centrifugal tracts, *e.g.* the so-called ventral posterior column field, the dorso-medial sacral bundle, etc.

II. in the LATERAL COLUMNS
- (*a*) the *lateral pyramidal tract.*
- (*b*) the *lateral cerebellar tract.*
- (*c*) the tract of *Gowers.*

III. In the ANTERIOR COLUMNS
- (*a*) the direct pyramidal tract.
- (*b*) compare under IV.

IV. In the region of the anterior and lateral columns there are a number of less well defined tracts not yet completely investigated, viz. :
- (*a*) the anterior marginal bundle of *Loewenthal.*
- (*b*) the fasciculus intermedius of *Loewenthal.*
- (*c*) the lateral limiting layer, and, within this, an antero-medial bundle.
- (*d*) cerebello-spinal or vestibulo-spinal, bulbo-spinal, thalamo-spinal fasciculi, Monakow's bundle, etc.
- (*e*) tracts ascending from the spinal cord to the medulla oblongata, pons, or mid-brain—probably crossed sensory paths of conduction of the second order (spino-thalamic and spino-tectal fasciculi).
- (*f*) commissural tracts, endogenous fibre-systems.

The grey matter is also divided, with reference to its function, into the anterior and posterior horns.

The anterior horns in the first place contain the trophic centres for the muscles of the trunk and extremities. The impulses from the motor conducting paths do not, therefore, pass directly to the anterior roots, but first to the grey matter, and within this to the nerve cells before they are transferred to the anterior roots. Affections of the anterior horns, therefore, cause not only paralysis but also atrophy of the motor nerves and of the muscles, because they contain the trophic centres for the same.

In addition to important facts already referred to, the observations of *Golgi*, *Ramon y Cajal*, *Kölliker*, *His* and others—which though made chiefly on embryos, may yet be applicable to adults—have proved respecting the motor tract and its entrance into the grey matter that the nerve fibres contained in the pyramidal tracts everywhere give off fine lateral branches (collaterals) which pass from the lateral pyramidal tract to the anterior horn of the same side. They are here resolved into a network of delicate fibrils, the free endings of which are directed towards the nerve cells (Fig. 59). These terminal branches to a certain extent embrace the cell without, however, entering into actual connection with it. The transmission of the impulse takes place, therefore, by contact. The objections recently raised to this view have already been considered.

A further function of the grey matter of the anterior horn is the bringing about of reflex movements with which we should probably also class tendon reflexes (*cf.* page 7). We shall refer later to the tracts and cells involved in these processes.

The grey matter, further, contains vasomotor centres for the unstriped muscle of the vessels and also for the viscera. On page (64)

it has been stated that these centres lie either in the lateral horn or in the intermediate area between the anterior and posterior horns. The small cells of the anterior horn have been said by some observers to possess this function. At all events the great majority of, if not all the impulses from the grey matter pass in the anterior roots and by these reach the rami communicantes and the sympathetic. Electrical stimulation of the anterior roots with tetanising currents causes constriction of the arteries (*Pflüger*). According to some observers vaso-dilator fibres run in the posterior roots (*Stricker*, *Bayliss* and others).

Gaskell maintains that in dogs, the nerves from the second dorsal to the second lumbar and the second and third sacral contain the fibres for the muscles of the blood-vessels and intes-

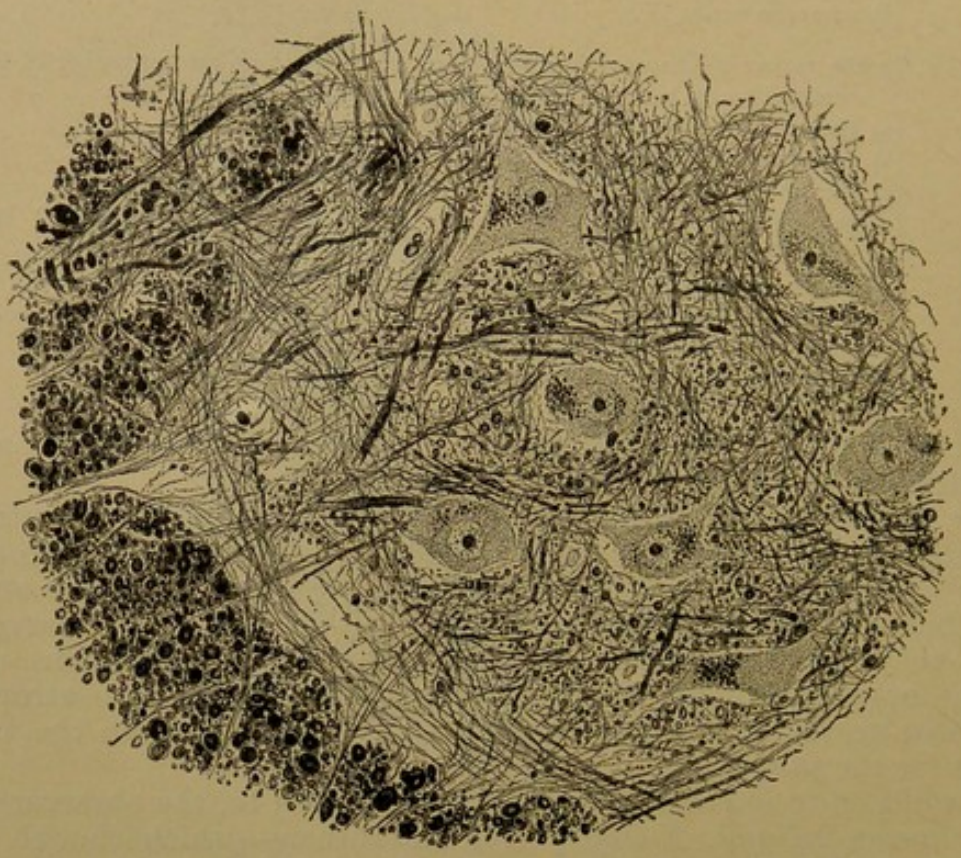


FIG. 59.—Part of the grey matter of the anterior horn with the adjacent parts of the lateral column, represented to show the fine medullated fibres which go from the lateral column into the grey matter and surround the (pigmented) nerve cells. Stained by the Weigert and Pal methods. (After Kölliker.)

tines, and therefore for involuntary movements; and that these nerves are the bundles of fine medullated fibres contained in the anterior roots. These statements also have been contradicted.

The cerebro-spinal elements of the sympathetic are, according to *Kölliker*, partly of a sensory and partly of a motor nature; the sensory convey the few sensations which we have from the viscera. The motor carry to the sympathetic from the cerebro-spinal centre the impulses for the unstriped muscles of the vessels (indeed for the intestines, glands, etc.). Reflex processes may also be carried out through the connection of the spinal cord with the sympathetic.

The cell groups of the intermedio-lateral tract have been looked upon as the area of origin of the sympathetic elements in the spinal cord by *Gaskell*, *Laignel-Lavastine* (*R. N.*, 1904) *Bruce-Pirie* (*R. of N.*, 1907), *Lewandowsky* and others; but *Sano* and others have doubted this.

On the relation of the spinal cord to secretion of sweat, *cf.* p. 68.

Head has attempted to ascertain the exact spinal innervation of the viscera, and claims definite segments of the spinal cord for each of these, *e.g.* the first to third dorsal segment for the heart, the

first to fifth for the lungs, the sixth to the ninth for the stomach, etc. Depressor fibres for the heart are contained in the second to fifth dorsal. *Head* maintains that he has established that the affections of each of the viscera are expressed by pain and hyperæsthesia localised in that posterior root area which corresponds to the relative segment. This had already been assumed by *Ross*. Herpes zoster may also have an analogous distribution. In this way have been identified sensory zones of the skin (especially for sensations of pain and temperature) pertaining to the different posterior roots or spinal cord segments, and also the spinal sensory innervation of the different viscera. In regard to the latter the results are similar to those *Gaskell* and *Edgeworth* wished to establish for the motor innervation of the area controlled by the sympathetic. With reference to this question pathology has, as yet, little to say. *Kausch* suggests that dilatation of the stomach may be brought about by localised disease of the ninth and tenth dorsal segments, and *Head* was able to connect paralysis of the small intestine with an affection of the tenth dorsal segment.

Head's theory has been supported by the investigations of *Haenel*, *Bartenstein*, *Petrén* and *Carlström* (*Z. f. N.*, 27), *Horsley* (*Practitioner*, 1904) and others, in spite of individually differing results, but it has been attacked by many (*Guillain*, *Winkler*, *Moll de Charante* [1]).

The posterior horns form a terminal and transit station for the sensory tracts, especially for the fibres conducting sensations of pain and temperature. The fibres for the reflexes also traverse a considerable part of the posterior horns. Numerous observations tend to prove that the grey matter has a trophic influence on the skin (as well as on the bones and joints), yet the course of these hypothetical trophic fibres is unknown (*cf.* pp. 64 *et seq.*). From the cells of the posterior horns fibres seem to proceed which share in the structure of the posterior columns. Such fibres are specially found in the areas adjoining the posterior commissure (in the so-called ventral field of the posterior columns, Strümpell). According to other observers this area is occupied chiefly by the descending branches of the posterior root fibres.

At the border between the columns of *Goll* and *Burdach* in the dorsal cord is found a small field (the so-called comma of *Schultze*). In the lumbar and sacral cord also individual bundles have been defined in the posterior columns—dorso-medial sacral bundles, *Flechsig's* oval posterior column field, etc.—bundles which contain partly descending endogenous fibres and partly descending fibres of posterior roots. This question has been studied in detail by *Schultze*, *Hoche*, *Wallenberg*, *Redlich*, *Margulies*, *Gombault-Philippe*, *Bikeles*, *Dejerine*, *Marburg*, *Homén*, *Stewart*, *Petrén*, *Schaffer*, *Nageotte*, *Dydynski*, *Orr*, *Collier-Buzzard* (*Br.*, 1904). *L. R. Müller* assumes that the impulses controlling the functions of the bladder and rectum are conveyed in these tracts.

In the dorsal cord, occupying an area at the inner or mesial angle of the base of the posterior horn, lies a well-defined rounded prominence of grey matter, containing numerous roundish cells, and an abundant network of fibres—*Clarke's* column [vesicular column of *Stilling* (Fig. 54)]. Small cell-groups corresponding to these are found also at other levels.

Schacherl ("Obersteiner," VIII.) has made very exhaustive investigations regarding the position and extent of *Clarke's* column.

Regarding the intimate relations of the parts, *i.e.* the *course of the fibres*, in the spinal cord, the following may be stated.

The posterior roots arise wholly, or in great part from the spinal ganglia. The nerve cells contained in these are bi-polar—in the adult they appear uni-polar, because the two processes, immediately after they leave the cell, lie close to one another— the one process passes by the posterior root to the spinal cord, the other as a sensory nerve fibre to the periphery.

[1] "Die hyperalgetischen Zonen von Head," Leyden. The whole question is treated by *Grosser* in *C. f. Gr.*, 1904. *Cf.* also the section : Localisation in the Spinal Cord, in this text-book.

It has recently been suggested that the posterior roots also contain fibres, independent of the spinal ganglion cells, whose trophic centre is in the periphery—in the skin and mucous membrane, or even in the spinal cord—but this is very doubtful. The posterior roots, at their entrance into the spinal cord, consist of a lateral and a more marked median bundle. The lateral fibres lie opposite the tip of the posterior horns in the area of white matter, which here forms the marginal zone (*Lissauer*) or the spinal bridge (*Waldeyer*): the fibres of the median bundle pass first into the column of *Burdach* (*cf.* Fig. 60). Each posterior root fibre, directly after entering the cord, divides into an ascending and a descending branch. The descending soon enters the grey matter,

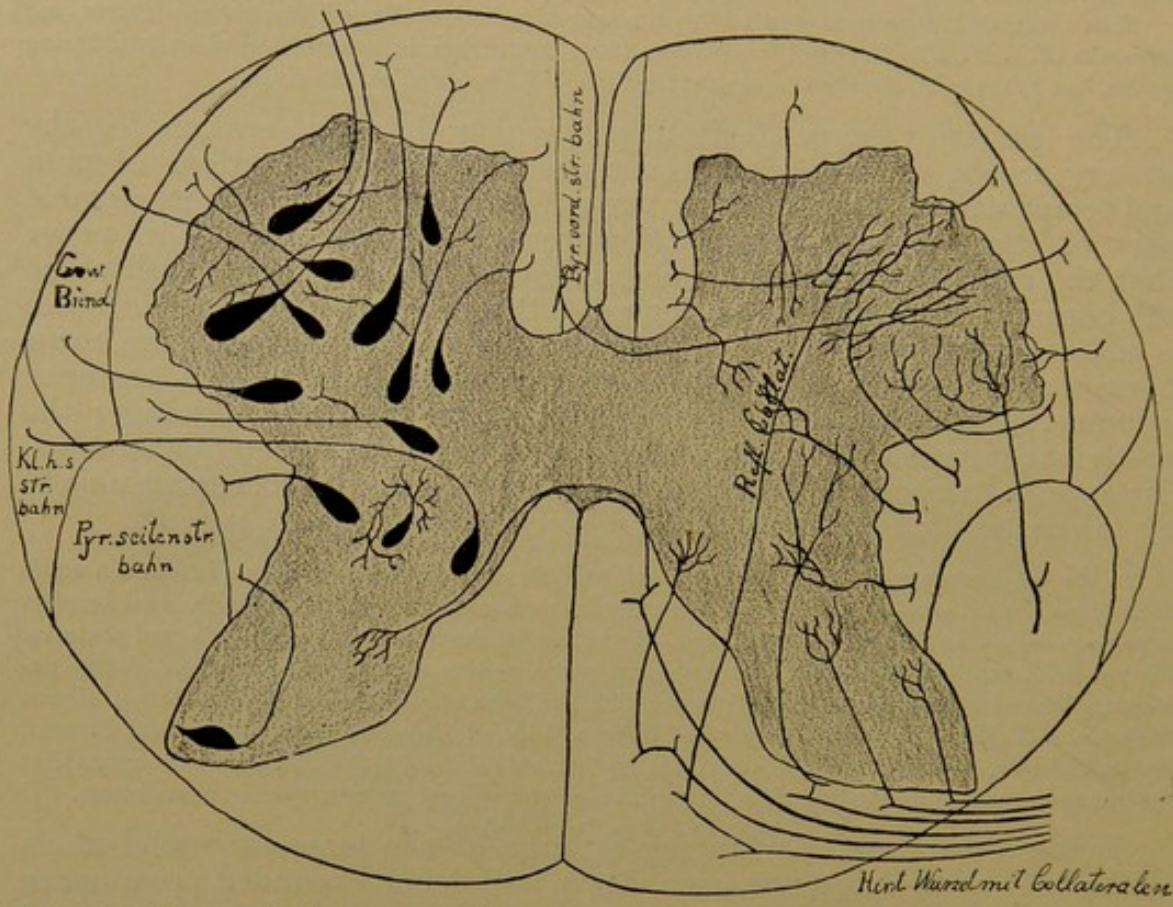


FIG. 60.—Diagram of the structure of the spinal cord. On the left the various cells and the course of their axis-cylinder processes, on the right the course of the posterior root-fibres, etc. Some of those represented in the figure are still hypothetical. (Simplified from Lenhossek.)

Pyr. vord. str. bahn = Direct pyramidal tract.
Pyr. seiten str. bahn. = Crossed pyramidal tract.
Gow. Bünd. = Gowers' tract.
Kl. h. s. str. bahn. = Direct cerebellar tract.
Hint. Wurzel mit collateralen = Posterior roots with collaterals.

and divides into numerous branches. The ascending fibres at first lie in the column of *Burdach* next to the posterior horn, but as each successive root enters the cord, those which have already entered become pushed inwards by the new fibres till they come to lie in the column of *Goll*. The lower in the cord the sensory roots enter, the further inwards they reach, so that in the cervical region the sensory fibres of the sacral roots lie in the medial area of the column of Goll. From the trunk

of the posterior root fibres, as well as from the longitudinal branches, collaterals are everywhere given off which penetrate into the grey matter of the posterior horns. This radiation into the grey matter is specially marked in the middle third of the column of *Burdach* (root entry zone, radiation zone). Thus, one portion of the posterior root fibres passes directly into the grey matter, another portion into the grey matter at higher levels, and a third remains in the posterior columns till, in the medulla oblongata, it passes into the grey matter of the so-called posterior column nuclei (nucleus gracilis and nucleus cuneatus).

In the dorsal cord a large proportion of the posterior root fibres passes to *Clarke's* column.

The collaterals which enter the posterior horns and *Clarke's* column resolve themselves into a reticulum, the threads of which twine round the nerve cells of the posterior horns or of *Clarke's* column and their dendrites, and transfer to them the sensory impulses. It may be that here a direct union takes place or that this transmission is accomplished by means of contact. The substantia gelatinosa *Rolandi* (Fig. 54) contains a large number of such collaterals.

One portion of the sensory fibres passing into the posterior horns reaches the grey matter of the anterior horn, and the fibres ramify around the anterior horn cells. These are probably the fibres which effect the reflexes [*reflex collaterals* (Fig. 61)]. This direct passage of posterior root fibres to the anterior horns is, however, disputed by *Lewandowsky* and others. Whether fibres reach also to the opposite anterior horn by the white commissure is not yet settled.

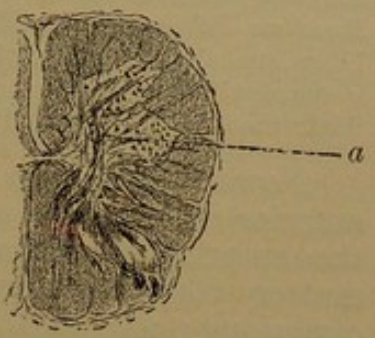


Fig. 61.—Part of transverse section of spinal cord. Pal's method. *a* = fibres passing from the area of the posterior roots into the anterior horn (reflex collaterals ?).

Further, one portion of the sensory tracts or their collaterals seems to pass from the white matter of the posterior columns, by the posterior commissure to the posterior horn of the other side. It is still uncertain whether a portion of the sensory tracts in man crosses in the *anterior commissure*, but this is now assumed by many observers ; some even say that the crossing of the sensory tracts occurs chiefly or altogether in this way. There may be fibres arising from the cells of the posterior horns (perhaps also of other areas of the grey matter) which pass over in the anterior commissure to the antero-lateral column of the other side—thus forming a sensory path of conduction of the second order. *Edinger*, *Bechterew*, *Kölliker*, and *Kohnstamm* have asserted the existence of such fibres, while *Dejerine*, *Mott* and *Russell* deny this.[1]

Some investigators have found in the tract of *Gowers* other spinal fasciculi of fibres—spino-thalamic and spino-tectal—the limits of which are not yet settled.

From *Clarke's* column arises the lateral cerebellar tract, *i.e.* the fibres of this tract are the nerve processes of the nerve cells contained in *Clarke's* column. Whether this is the only origin of the fibres of this

[1] Compare also the rather different statements of *Piltz* (*N. C.*, 1905, and *A. f. P.*, Bd. xli.) and the opposing views based on animal experiments of *Lewandowsky* (Unters. über die Leitungsbahnen, etc., *Neurol. Arbeiten. v. O. Vogt.*, etc., I.).

tract, and whether the tract is the only continuation of *Clarke's* column, is undecided. The fibres are continued upwards to the restiform body and thence to the superior vermiform process of the cerebellum (*Flechsig, Monakow, Bechterew, Bruce*). Probably this tract contains also short fibres (*Sherrington-Laslett*).

The tract of *Gowers* [1] is probably also a sensory path of conduction of the second order, crossed (wholly or mainly). There is no uniformity of view as to its origin. *Bechterew* thinks the fibres arise from cells of the intermediate grey matter lying between the anterior and posterior horns. The crossing must take place in the anterior commissure.

With regard to its central termination also statements do not agree. *Mott, Hoche, Auerbach, Tooth, Russell, Bruce* and others think it probable that in the medulla oblongata it separates from the lateral cerebellar tract and passes to the upper pons, there to bend backwards in order to reach the cerebellum. According to later investigations this refers only to one part of this tract. The other part passes through the corpora quadrigemina—the lateral fillet and internal geniculate body—to the thalamus, and thus enters into connection with the cerebrum. (*Quensel, Henneberg, Rossolimo, Schmaus-Sacki, Thiele-Horsley, Amabilino, Kohnstamm, Marburg, Dydynski, Hunt.*[2]) There are also physiological and pathological grounds for this view. *M. Lewandowsky* is amongst those who assert that this tract passes wholly to the cerebellum, while *Kohnstamm* thinks that the larger part, after first passing to a nucleus of the crusta, goes on to the thalamus.

Now in addition to the motor cells of the anterior horn from which the anterior roots spring, and in addition to the posterior horn cells, to which the entering posterior root fibres convey sensory impulses, the grey matter contains numerous nerve cells which are differentiated as *commissure cells* and *column cells*. The commissure cells lie specially in the central area of the anterior horns, their nerve processes pass through the anterior commissure to the other side and curve round into the anterior column in a longitudinal direction. Individual cells also send their processes to the grey matter of the other side (*Lenhossek*). The *column* cells are scattered throughout the grey matter. Each sends its nerve process into the white matter of the anterior and lateral columns, where it divides into an ascending and a descending branch, or turns upwards and at various levels gives off collaterals to the grey matter, so that by these fibres a far-reaching union is established between the different segments of the cord. The posterior columns also, as stated above, may contain such commissural-fibres. These endogenous fibres of the spinal cord run chiefly in the parts closely adjoining the grey matter. Regarding the course of the fibres concerned with so-called reflex inhibition, which in part descend from the brain and in part arise in the grey matter of the cord itself, we know nothing definite; possibly they coincide with the pyramidal tract (*Kölliker, Strümpell*). Some investigators, however, deny the existence of such tracts.

In the above mentioned cerebello-spinal, bulbo-spinal, tecto-spinal, tracts, etc., it is probable that impulses are carried to the spinal cord from co-ordinating centres, possibly also impulses which influence the muscle tone and originate automatic movements. Thus recent observations have proved that the motor stimuli are conveyed to the grey matter of the cord not exclusively by the pyramidal tracts but that the

[1] *Edinger* names this the ventral cerebello-spinal fasciculus, and the lateral cerebellar tract he names dorsal cerebello-spinal fasciculus.

[2] *R. of N.*, 1904.

stimuli, for unconscious movements at least, may be transmitted by other channels.

We hold that the impulse for voluntary movements of the muscles of the extremities is conducted by the pyramidal tract, but we do not deny that there are additional tracts which, after destruction of the pyramidal tract, might be able—to a certain though only incomplete extent—to take over this function. This view has been specially advocated by *Rothmann* (*B. k. W.*, 1901; *N. C.*, 1902) on the basis of experiments on animals by *Starlinger* and himself. Compare also for the results of animal experiments, *A. Schüller*, *N. C.*, 1905.

Let us now bring together the essential points.

One division of the sensory fibres, after its entry into the cord, passes into the grey matter of the posterior horns, partly directly, and partly after a longer or shorter course in the column of *Burdach*. Its fibres pass, for the most part, into the posterior horn of the same side, but a small number seems to cross over in the posterior commissure. In the cells of the grey matter of the posterior horn a second neuron begins, the axis-cylinders of which probably cross in the anterior commissure to the antero-lateral column of the opposite side, apparently go into the tract of *Gowers* or into special—spino-thalamic, spino-tectal—tracts, and in these pass upwards. Another division of the sensory fibres passes in the columns of *Burdach* and *Goll* to the medulla oblongata. In the dorsal cord a large number of the posterior roots radiating into the grey matter reach the region of Clarke's column; the sensory fibres here and in the posterior horn form a reticulum whose fibres twine around the ganglion cells and their processes. From the cells of *Clarke's* column arise the fibres of the lateral cerebellar tract. This is therefore a sensory path of conduction of the second order, probably conveying to the cerebellum sensory impulses influencing co-ordination.[1] This also holds good for the tract of *Gowers*, which forms a crossed, or mostly crossed, sensory path of conduction of the second order whose function has not yet been settled. In agreement with *Gowers*, some writers claim for his tract the conduction of the sensations of pain and temperature. This could be the case only if the greater part of the tract reached the thalamus, and in this way the cerebrum. Probably there are special fasciculi of fibres (spino-thalamic, etc.) that are concerned with these sensations. With reference to the significance of the different centripetal tracts in the conduction of the various sensory stimuli, compare the chapter on *Brown-Séquard* paralysis. One division of the fibres passing to the grey matter reaches as far as the anterior horn cells, and serves probably to effect reflex movements. The path concerned with reflex movements may also have a longer course, for the sensory stimuli may be conveyed first of all to column cells and from these, by means of their processes or their collaterals, pass to cells at other levels of the spinal cord. (*Cf.* Figs. 62 and 63.)

The motor impulses conveyed in the lateral and direct pyramidal tracts are carried by the collaterals into the grey matter of the anterior horn and transmitted to the motor ganglion cells, whence they pass to the anterior roots. The motor path of conduction consists, therefore, of two neurons: the one (direct motor neuron, or neuron of the first order) extends from the anterior horn cell to the terminal ramifications

[1] *O. Marburg* (*A. f. A.*, 1904, Suppl.) ascribes to the lateral cerebellar tract influences regulating specially the muscles of the pelvic girdle and therefore the equilibrium in standing, walking, etc.

of its nerve process in the muscle, the second (indirect, or neuron of the second order) from the cell in the motor cortex to its terminal branch in the anterior horn. *Waldeyer* designates the central, *archineuron*; and the peripheral, *teleneuron*. The sensory paths of conduction consist of at least two neurons, probably more.

In each spinal-cord segment there are *centres* for definite groups of muscles, and *reflex centres* for the reflex movements occurring in it. Probably the ganglion cell-groups, which come into action in the transference of the motor impulse descending in the pyramidal tract to the anterior roots, are the same as those carrying out reflex movements. There are, in addition, fibres for the inhibition of reflex movement.

The skin reflexes probably take the following course: the impulse

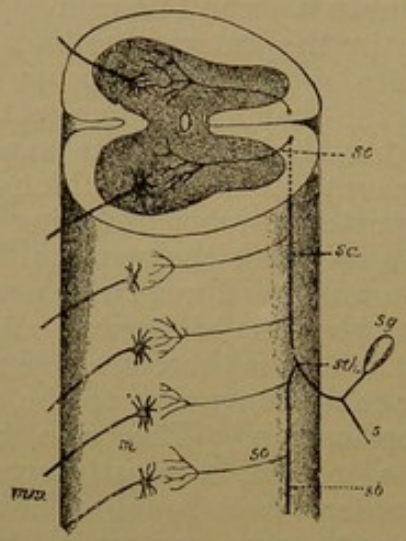


Fig. 62.—(After Kölliker.) Drawing of elements composing the spinal reflexes.

A sensory root-fibre (s) connected with a spinal ganglion cell (sg) gives off from both its branches (sth), the ascending (sa) and the descending (sb) collaterals (sc) which act upon the motor cells (m). mw = motor roots.

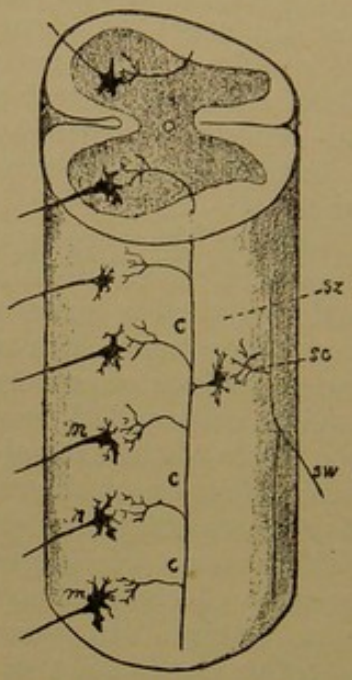


Fig. 63.—(After Kölliker.)

A dividing sensory root-fibre (sw) gives off a collateral (sc) which acts upon a nerve cell (sz), and by means of the collaterals of its bifurcated nerve process (c) excites a number of motor cells (m).

passes by the posterior root to the grey matter, where it reaches the neighbourhood of the corresponding anterior horn cell, either directly by a branch of the posterior root (reflex collateral), or by the interposition of another cell. This cell, by which the sensory impulse is at first transmitted, may possibly be an inner cell (reflex cell, *Golgi* cell), or a so-called column-cell. In this way the impulse is carried by the nerve processes and their collaterals to the various spinal-cord segments (Fig. 63). The stimulus may also by means of the long ascending posterior column fibres be conveyed directly to the motor cells of several spinal-cord segments (Fig. 62).

It is assumed that the reflex stimuli try first of all the shortest route, so that the simple reflexes have their reflex arc in the same spinal-cord segment as that in which the posterior root enters. More complicated reflexes which have, on the other hand, a long reflex arc, distribute them-

selves in several spinal-cord segments, or are transmitted through the medulla oblongata. According to Pflüger's law, which is consistent with recent views regarding the structure of the spinal cord, it is easily understood how the increase of the stimulus causes an almost unrestricted extension of the excitability in the area of origin of the different groups of muscles.

Numerous observers have called in question the prevailing theory that the shortest route is used in bringing about skin reflexes with minimal stimuli. *Rosenthal* and *Mendelssohn* regard the reflex-transfer as taking place in the lower part of the medulla oblongata. *Sherrington, Jendrássik, Munch-Petersen, Gehuchten* and others look for it in the cerebral cortex. We have no sufficient grounds for relinquishing the older conception.

Every lesion of the reflex arc which lessens the power of conduction leads to weakness or loss of the corresponding reflex movements If the excitability of the reflex centres be abnormally increased then the reflexes are increased. In affections situated above the reflex arc, especially when the lesion is an incomplete one, the reflexes are increased. If there is a complete interruption of the conduction in the segment of the spinal cord situated above the reflex arc, the reflex excitability may be lowered or raised.

This fact, aleady ascertained by earliest observations, has been specially noted by *Vulpian* and *Kahler-Pick*. But while it had previously been believed that diffuse affections situated in the supra-lumbar regions of the spinal cord caused a *spastic* paralysis with increased superficial reflexes and tendon jerks, and that only very exceptionally and under special circumstances did this become a flaccid paralysis with loss of reflexes, *Bastian* has pointed out (*Medic.-Chir. Transact.*, London, 1890) that it is the rule that diseases or injuries which lead to a *complete interruption* of conduction in the cord and therefore to complete anæsthesia, even when the lesion is situated above the reflex-arc, cause a loss of all reflexes and tendon jerks falling within the area below the spinal-cord lesion. This is explained by *Bastian* and *Hughlings Jackson* as follows: The tonus of the muscles, upon which the tendon reflexes depend, is maintained by the influence of the cerebellum upon the anterior horn cells, while the cerebrum inhibits this tonus by means of the lateral pyramidal tract. Cerebral lesions, therefore, intensify the tendon jerks and cerebellar lesions may abolish them. If cerebrum and cerebellum be cut off from the spinal cord, as in a complete transverse lesion of the cord, the tendon reflexes are absent. This hypothesis in a modified form was accepted by *Gehuchten*, while *Sternberg* sought an explanation for the loss of reflexes in a condition of irritation in the reflex inhibitory tracts. *Bruns* (*A. f. P.*, xxv.), in a case which fulfilled the conditions laid down by *Bastian*, showed that the tonus of the muscles and the condition of the reflexes were in keeping with the latter's teaching. Since this proof of Bruns, this view has been accepted unreservedly, or at least with few reservations, by many investigators, *e.g. Dejerine, Nonne, Marinesco*, and *Bruns* himself. Animal experiments had given it no confirmation, for high transsection of the cord in animals produced either no loss of tonus or of reflexes, or merely a transient loss in consequence of the shock, while the reflexes were increased subsequently. *Sherrington* alone arrived at other results in apes, but even in his cases the loss of tendon reflexes was not permanent, and *Margulies* pointed out in reference to this that the result of the experiment was influenced by the violence with which the lesion was made. *J. Kron* also reached results which contradicted the Bastian theory. Regarding observations in man, some authors (*Senator, Egger, Hoche, Habel, Marinesco, Nonne, Cestan, Winter, Bittorf*, and especially *Bruns* and *Collier, Br.*, 1904), seemed to agree with the Bastian theory. But on the one hand cases were observed which showed that flaccidity and loss of reflexes may be present even in an incomplete transverse lesion, and also in a simple compression of the cord causing complete interruption of conduction without anatomical injury. On the other hand many cases have been observed in which, in spite of apparent total destruction of the cord in one of the cervico-dorsal segments, the reflexes and tendon jerks remained (*Schultze, Fürbringer, Senator, Gerhardt, Brauer*, and others). *Bruns* does not allow that any of these cases was free from objection, and considers the *Bastian* theory, at least with reference to the condition of the tendon jerks, as well founded and incontrovertible. *Kausch* has since published one case showing unmistakably that in total

transverse lesion of the cord in the dorsal region the tendon jerks may be not only maintained but even increased up to a short time before death. *Jolly, Lapinsky* (*A. f. P.*, Bd. xlii.), and *Henneberg* (*Charité-Annalen*, xxi.) report a similar case, and thereby the Bastian theory falls to the ground.

If now, in spite of this, it is found, as is shown for instance by the work of *F. Rose* ("Du tonus et des réflexes dans les sections et compressions," Paris, 1905), that after a severe destructive transverse lesion of the cervical or dorsal cord, the resulting paralysis is often flaccid and associated with loss of reflexes, this may be explained on the following grounds: (1) In injuries, which are frequently the cause, *shock* plays a great rôle. This may be prolonged for a period varying according to the intensity of the injury. I have recently seen a series of cases, in which the immediate consequence of the enucleation of a tumour compressing the cord in the cervical or dorsal region was the transition of the spastic paralysis to a flaccied one, with complete loss of reflexes and tendon jerks—a condition which under favourable circumstances tends to recover in a few weeks. (2) The material consequences of the injury are often not limited to the portion of the spinal cord directly affected, but reach far beyond this. (3) Complete section of the cord causes altered vital conditions for the portion situated below the level of the lesion, the blood and lymph circulation being especially disturbed, and in this way the function of the lumbar cord is more or less gravely perverted (*Strümpell, Brissaud, Brauer, Lapinsky, Raymond-Cestan*, and others).[1] In particular an accumulation of cerebro-spinal fluid, resulting in an increased pressure, could affect the posterior roots of the lumbar portion even to the arresting of the conduction. (4) As under these circumstances it may happen that degenerative changes may occur in those portions of the cord and its roots which serve as the reflex arcs, this may depend upon the fact that the nature of the affections which lead to the compression of the cord causes degeneration *throughout the whole of the spinal cord*, especially in the anterior horn cells and the roots of the lumbar portion, and these changes explain the loss of the reflexes. The causal conditions are very frequently malignant neoplasma and tuberculosis. Thus *Siemerling*, and I also in 1886, pointed out with reference to this that in dorsal tubercular spondylitis the spastic paralysis of the legs was transformed into a flaccid one in this way, and that the peripheral nerves became degenerated in consequence of the general condition. *Brissaud* has confirmed this; *Bálint* also has found a similar condition, and *A. Westphal* has shown that under such conditions the motor cells of the anterior horn throughout the whole cord may undergo degenerative changes which materially impair their function. Finally *Lapinsky* (*Z. f. N.*, xxx., and *A. f. P.*, Bd. xlii.) has stated that a strong sensory irritation, such as is occasioned, *e.g.*, by compression of the posterior roots, may have a depressor influence on the reflex movements. For an explanation of *Bastian's* observations, *Monakow* gives his hypothesis of Diaschisis.

Regarding the tendon reflexes all that has been stated with reference to the superficial reflexes in general holds good, yet in disease the condition of the two reflexes by no means runs a parallel course.

With reference to the path of the so-called reflex-inhibiting fibres we know nothing definite—probably it coincides with the motor path. Thus the voluntary innervation of the muscles concerned may tend to inhibit the reflex. It was mentioned above that it is assumed by many observers that the pyramidal tract has a depressing influence on the muscular tone, whereas in the cerebello-spinal tracts stimulating impulses are conveyed to the anterior horn-cells which increase the tone of the muscles; but this must be regarded as wholly hypothetical.

From ascertained facts with reference to the tendon reflexes we deduce the following statements: the *knee jerks are absent*—

1. In a lesion of the corresponding centripetal limb of the reflex arc, and therefore, in neuritis of the crural nerve, in affections of the posterior roots and posterior columns in the area of the spinal cord involved.

[1] Amongst physiologists *H. Munk* has specially appreciated this fact. According to *Munk* the lumbar cord, through its severance from the rest of the central nervous system, undergoes progressive internal changes, "Isolation changes." The author tries in this to find an explanation for the increased reflexes and declines the theory of reflex inhibiting tracts. In this connection we may recall the experiments of *Porter, Rothmann* and others.

2. In an affection of the grey matter at the level of the reflex arc.

3. In an affection of the centrifugal limb and, therefore, of the anterior roots pertaining thereto, and of the motor fibres of the crural nerve.

4. In an affection of the spinal cord above the reflex arc, which is associated with a complete loss of continuity, but only under the conditions already discussed.

5. In deep coma.

6. Sometimes in cerebral affections accompanied by increased pressure of the cerebro-spinal fluid, *e.g.* in tumours of the cerebellum and other cerebral areas—such a condition, however, not being quite explained (see chapter on Cerebral Tumours).

Isolated observations (*Bloch, Oppenheim, Weimersheim*[1]) have shown that the knee jerks may be absent from birth onwards—the *Westphal* sign belonging to the congenital stigmata of degeneration. This is, however, so exceedingly rare that in practice it need not be taken into consideration.

In pyrexial states and in the climax of certain acute infectious diseases, *e.g.* croupous pneumonia, the knee jerks may be absent (*Marinian, Sternberg, Pfaundler*), (*M. m. W.*, 1902).

Increase of the knee jerk may arise—

1. From an irritative condition of the centripetal limb of the reflex arc. Thus slight lesions of the posterior roots, which occasion no interruption of conduction, may be associated with an increase of the knee jerk.

2. From irritative conditions in the reflex centre itself (strychninism, tetanus).

3. From disease of the lateral pyramidal tracts, with certain restrictions.

4. In diffuse affections of the spinal cord above the level of the reflex arc—at least those which do not cause a complete interruption in the conduction.

5. In functional neuroses which are accompanied by a general increase of reflex excitability.

We have still to consider the *centres* which are contained in definite vertical sections of the grey matter.

The lowest cervical and highest dorsal regions—indeed the area from which the first dorsal root arises—contain the *cilio-spinal centre*, a centre for the dilator pupillæ muscle and the unstriped muscle of the eyelid whose contraction widens the palpebral fissure. The stimulation of this centre leads to dilatation of the pupil and palpebral fissure of the same side. The path of conduction leaves the spinal cord in the *anterior root of the first dorsal nerve*, and through this, by means of the ramus communicans, reaches the sympathetic. Possibly the oculo-pupillary centre extends even to the eighth cervical segment, so that some of the fibres may emerge from the cord by the eighth cervical root. This is doubtful, and cannot, in any case, be admitted for the second and third dorsal roots in man, as I have produced a maximum dilatation of the pupil by electrical stimulation of the first dorsal root, but, from the second, I have been unable to obtain this.[2]

Seguin saw myosis set in after section of the lower roots of the plexus.

[1] "Uber den angeborenen Mangel der Patellarreflexe." Würzburg, 1906. Ref. *B. k. W.*, 1906.
[2] *B. k. W.*, 1896.

The old view that the cilio-spinal centre extends from the sixth cervical nerve to the third dorsal nerve is incorrect.

Kocher (*Mitt. aus d. Grenzgeb.*, i.) states—as *Schiff* had previously done—that the oculo-pupillary fibres arise in the medulla oblongata, traverse the whole cervical cord, and ultimately leave it by the first dorsal root. Other investigators, also, have recently advocated the view that the centre for the dilator pupillæ is in the medulla oblongata, and that the fibres descend in the cervical cord and leave the spinal cord at the level of the first dorsal nerve, or they refer to an oculo-pupillary path from the cerebrum, descending by pons and medulla oblongata to the spinal cord. The so-called cilio-spinal centre may be only a rendezvous, in which the fibres conveying the reflex collect before they emerge from the cord (?). In our opinion the view of the cilio-spinal centre rests upon surer grounds and is the correct one. *Jacobsohn* has described small cell groups in the lateral horn as carrying out this function.

In the lowest level of the cord, in the sacral cord or in the conus terminalis lie the centres for the *bladder*, the *rectum*, and the *sexual apparatus*. These centres regulate the evacuation of the bladder and of the rectum and the ejection of the seminal fluid. Some observations point to the fact that the centre for the sexual apparatus is not in the immediate neighbourhood of the ano-vesical centre but is above it, and that the centre for erection is above that for ejaculation.[1] *Pansini*[2] gives more detailed statements regarding the position of these centres. *Schlesinger's* observations seem to point to a bilateral position of these centres.

The laws which govern these functions, and the path which connects these centres to the brain are not fully understood. We are justified in assuming that the grey matter of the lowest part of the spinal cord contains a centre for the sphincter and the detrusor of the bladder (see below). From these centres the motor path of conduction passes by the anterior roots of the third and fourth sacral nerves into the common pudendal nerve, or into the middle hæmorrhoidal nerve (and sympathetic), and, thence, to the bladder muscles. From the mucous membrane of the bladder, sensory fibres pass to the spinal cord by the roots of the second to fourth sacral nerves, and remain in relation to the centres. From them also sensory stimuli are conveyed to the brain.

Rehfisch distinguishes two groups of bladder nerves. The upper leaves the lumbar cord and pass as the rami-communicantes to the lumbar division of the sympathetic, and as mesenteric nerves to the inferior mesenteric ganglion: from this they pass as hypogastric nerves to the hypogastric plexus and, thence, to the bladder muscles. The lower group leaves the spinal cord in the three upper sacral nerves, unite in the nervi erigentes, and appear in the hypogastric plexus, from which they pass to the bladder muscles. (See Fig. 64, Plate i.)

According to recent observations (*Zeissl*, *Rehfisch*, *Frankl-Hochwart*)[3] chronic retention of urine is to be traced to the persistent contraction of the internal sphincter of the bladder, while the striated outer muscle comes into play in voluntary inhibition and interference with the flow of urine.

The act of micturition may be analysed thus. When the bladder is full, the sensory nerves become stimulated and the impulse is conveyed by the sensory branches of the nervi erigentes and hypogastric to the

[1] The nervi erigentes arise from the first to the third sacral nerves; the fibres for the ischio-cavernosus, transversus perinei, and bulbo-cavernosus from the third and fourth. The sympathetic also shares in the vaso-motor innervation of the penis, and this is the essential element in erection. *L. R. Müller* thinks that the centrifugal fibres going to the centre for erection in the sympathetic leave the cord in the upper lumbar region, while the centripetal fibres reach the lowest level of the cord in the common pudendal nerve.

[2] *Rif. med.*, 1903.

[3] "Die nervösen Erkrankungen der Harnröhre und Blase." "Handbuch d. Urologie." Wien, 1904. Also Frankl-Hochwart and Zuckerkandl in Nothnagel's "Handbuch," xi.

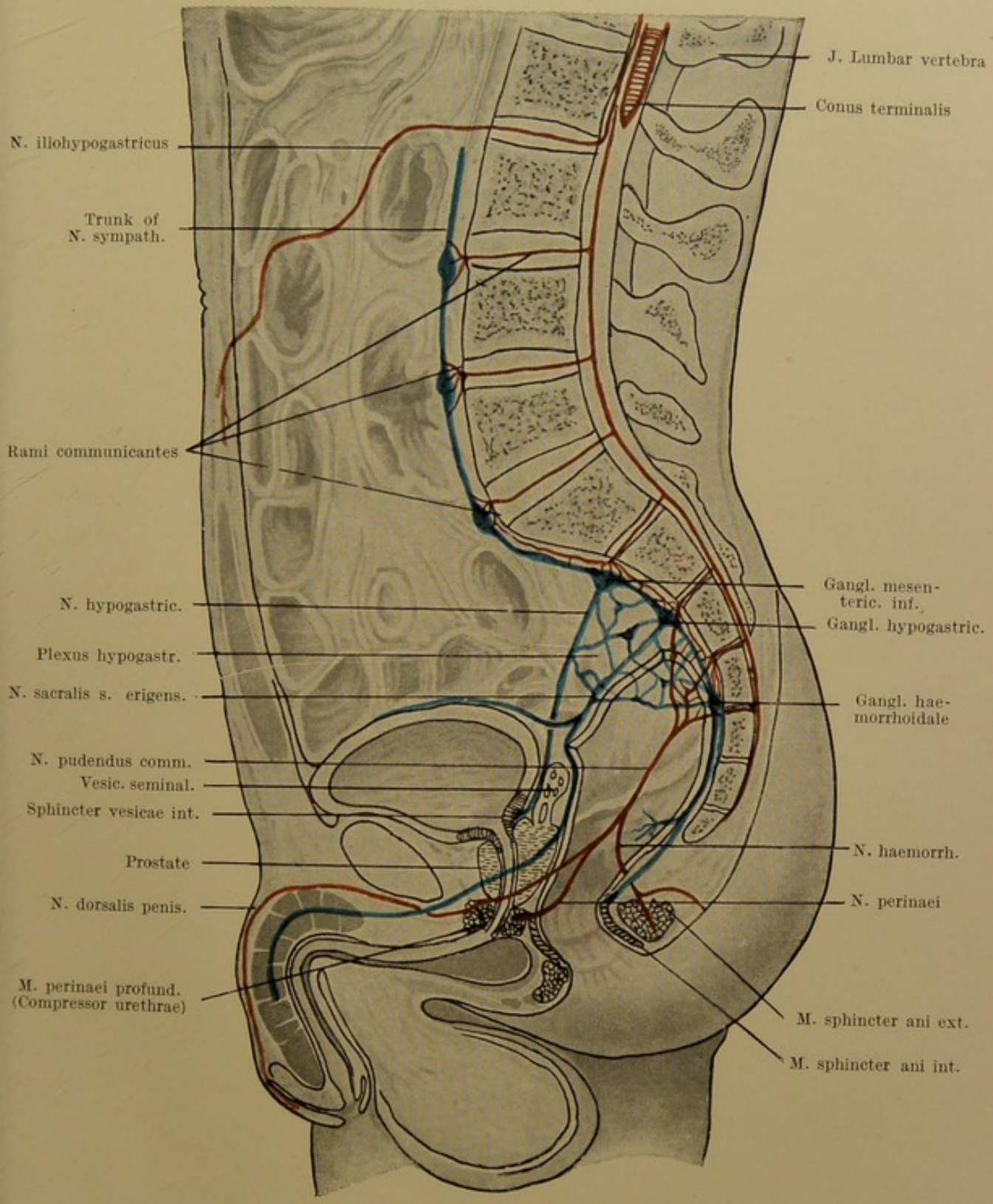


Fig. 64. (After L. R. Müller.)

Otto Schulze & Co., Edinburgh.

bladder-centre in the spinal cord. This centre, by means of the centrifugal nerves, effects the motor innervation of the bladder wall. The resulting contractions produce the stimulus, which passes by the sensory tracts to the brain, and thus gives rise to the expulsive effort of voluntary micturition (*Guyon, Dubois-Genouville*). It is assumed that between the detrusor and sphincter centre there exists an antagonism of such a nature that a stimulation of the former causes an inhibition of the latter and thereby a relaxation of the sphincter. Yet investigations by *Zeissl* and *Hauc* have shown that the relaxation of the sphincter is not a consequence of the detrusor action, but is in direct dependence on the nervous system. The impulses suspending the tone of the sphincter are carried in the nervi erigentes. The will can at any time regulate the action of the sphincter and cause it to contract or relax and can also inhibit its reflex contraction. In any case the will does not influence the detrusor directly ; but whether the hypothetical antagonism (which *A. Rehfisch* and *Müller* among others have disputed) shows itself in such a way that the voluntary inhibition of the sphincter causes a stimulation of the detrusor cannot definitely be decided. But the will has certainly the power to stimulate the abdominal muscles to contract and thus to aid the evacuation of the bladder. We do not know definitely in which columns of the spinal cord the impulses are carried—probably the antero-lateral column.[1]

From the above it may be understood that derangements of the bladder function may be brought about from any level of the spinal cord. If the interruption to conduction be above the bladder centres, and therefore above the sacral cord, and if it be complete, voluntary micturition is suspended, as the will no longer controls this function. When the bladder is full the discharge of urine takes place reflexly, and the patient is not able to retain the urine (*intermittent incontinence of urine*). If the centres are themselves destroyed then the sphincter is permanently relaxed, the detrusor is inactive, and constant dribbling of urine is present. The elasticity of the bladder-outlet may, however, effect the closure of the bladder to such an extent that there is no dribbling until there is a considerable accumulation of urine in the bladder. Other factors also, *e.g.* the altering pressure of the viscera, can under certain circumstances influence the discharge from the bladder, just as by pressure upon the abdomen, in sphincter paralysis, a portion of the bladder contents may be expelled (*Heddæus, Wagner, Frankl-Hochwart-Zuckerkandl*). Therefore in the diagnosis of bladder paralysis those factors, which act wholly mechanically, must always be borne in mind. According to *Kocher, Head*, and others, under these conditions strangury may be present. Moreover, some observations seem to prove that in a *complete transverse*

[1] *L. R. Müller* (*Z. f. N.*, xiv. and xix.) has studied very carefully the course of the fibres in the conus and states that the conditions here differ fundamentally from those elsewhere in the cord. The groups of large cells are found not in the anterior horn, but in the intermediate area between anterior and posterior horns. Further, fibres are seen to curve forwards from the posterior column into the grey matter and, finally, from here the root fibres enter, through the postero-lateral column, directly into posterior roots. Upon these anatomical observations he bases his view that there are in the posterior columns centrifugal tracts which bear a relation to the functions of the bladder, rectum, and sexual apparatus, and which reach the corresponding centres in the intermediate zone of grey matter. The nerve processes arising from these cells pass through the lateral column into posterior roots. Recently he has suggested that the tracts in question may be contained in the fasciculi of the triangular field, etc. Without more conclusive grounds, however, this view cannot be accepted. *Ziehen* has also opposed it. With reference to statements by other authors (*Budge, Mosso, Stewart*, etc.) compare *Frankl-Hochwart.*

lesion of the cord at any level the *reflex discharge of the bladder and of the rectum may be lost* (see p. 115). Yet this is improbable. Paralysis of the detrusor causes retention of urine (*ischuria*). The bladder becomes at first greatly distended till the urine ultimately dribbles away mechanically (*ischuria paradoxa*). Retention of urine may also be occasioned by spasm of the sphincter or by the inability to relax the sphincter voluntarily. Retention of urine, owing to spasm of the sphincter, is a very usual symptom in the first period after any interruption to conduction in the cord above the vesico-spinal centre. Retention of urine and incontinence may also result from an affection limited to the sensory tracts, which are concerned with micturition. The question concerning the relations between the derangements of the function of the bladder and the site of the spinal-cord lesion requires further elucidation.

The evacuation of the rectum takes place through a similar mechanism.[1] If the sphincter-centre itself be destroyed then incontinence of fæces exists. Yet hardened fæces can, through the elasticity of the anal aperture, be retained for a long time. If the path of conduction in the spinal cord, connecting this centre with the brain, be obstructed, the will loses its influence upon the sphincter ani externus, while its contraction may still take place reflexly. The reflex-contraction may even be increased. By introducing the finger into the anus the reflex contraction may be felt, and thereby proof is sometimes given that the affection must have its site above the centre.

I have seen one case in which, in an affection immediately above the conus, slight stroking of the skin in the gluteal, perineal, or even plantar region caused active contraction of the sphincter ani and tonic contractions in the glutei.

When the interruption of conduction is complete, this reflex may be wanting. The will has no influence on the intestinal musculature governing defæcation, yet, by controlling the tension of the abdominal muscles and regulating abdominal pressure, it may assist the expulsion of the fæces.

These views regarding the sacral centres for the bladder, rectum, and genital apparatus have recently been very energetically attacked. Experimental observations by *Goltz* and *Ewald*, *Langley*, *Arloing*, *Fuld*, and others had already pointed out the fact that the sphincter ani externus has a distinct, distinctive position, and *Frankl-Hochwart* and *Fröhlich*[2] have stated that this muscle resembles the non-striped muscles in its reaction and function. They, therefore, transferred the centre for its tonus to the sympathetic ganglia, but ascribed a regulating influence on this centre to the grey matter of the spinal cord in the conus. L. R. Müller has gone further than this. According to him *the centres for the evacuation of the bladder and rectum, and the erection of the penis are not in the spinal cord at all but are in the sympathetic ganglia of the pelvis.* He supports this view by experimental evidence, and also by the known fact that

[1] *Müller* makes the following statements regarding the innervation. As far as the sphincter ani internus the rectum is innervated only by the sympathetic. The greater portion of the fibres arise from the hæmorrhoidal plexus and the smaller portion from the inferior mesenteric plexus. From these plexuses rami communicantes pass centripetally to the spinal cord. Spinal medullated nerves appear only in the sphincter ani externus and the outer anal skin, *i.e.* in the inferior hæmorrhoidal nerve—a branch of the common pudendal nerve. *Bálint* and *Benedict* have come to similar conclusions on the ground of their own observations (*Z. f. N.*, xxx.).

[2] *N. C.*, 1902.

the discharge of urine and fæces is only incited by the will, but represents really an involuntary process, and, lastly, by the complete similarity, according to his observations, of the functional disturbances in transverse lesions affecting different levels of the spinal cord, including the conus medullaris. The earliest sign is always ischuria paradoxa, which, as a rule, is followed by incontinence, with periodic, automatic discharge of almost equal quantities of urine. Müller then resumes : "The view up till now held that there exist in the lowest portion of the cord centres for the discharge of urine and fæces is incorrect, we have there only the nerve cells for the external sphincter. It is not the centres for defæcation but only the centre for the anal reflex that is localised in the conus." *Fürnrohr* and *Pick* agree with *Müller*, and *Bálint-Benedict* confirm many of his statements.

The act of erection seems to persist even when the conus is destroyed. This is in keeping with *Müller's* assumption—which has recently received further experimental support (*Z. f. N.*, xxx.)—that the centre is to be found in the ganglia of the sympathetic (hypogastric plexus). But these observations, as also those of *Bálint-Benedict*, do not include the orgasm and the ejaculation, as these are connected with the spinal centres for the ischio- and bulbo-cavernosus muscles. These authors describe a case of normal labour in a woman whose last sacral segments were destroyed — the confinement was naturally painless. The activity of labour pains, therefore, cannot be related to the spinal cord, as was also shown by the experiments of *Goltz-Ewald* and *Rein*.

We do not consider ourselves justified in accepting Müller's conception in its entirety, and believe that, in addition to the sympathetic centres, there are spinal centres in the sacral cord in man, so that for lesions and affections of this part of the spinal cord the above statements hold good. Yet it must be admitted that even in man, after destruction of the conus centre through a lesion in its grey matter, the sympathetic apparatus is able to carry on the functions of evacuation of urine and fæces, though not in such a perfect manner. *Gehuchten, Berger, Sahli, Frankl-Hochwart,*[1] *Bálint-Benedict, Zimmer* and *Minkowski*[2] express themselves likewise.

Secondary Degeneration

The motor conduction-path, which is represented by the lateral and anterior pyramidal tract, has its trophic centre in the cerebral cortex, in the nerve cells of the motor area. The fibres which run in these tracts are the direct offshoots, *i.e.* the nerve-processes of these nerve cells. The cell of the motor cortical area, along with the nerve fibre descending in the pyramidal tract, forms a nerve unit. If the nerve-processes become separated from the cell-body, they can no longer exist and must atrophy.

Therefore every morbid process, which destroys the motor tract at any point, causes a degeneration of the part that no longer remains in connection with the trophic centre—therefore a *descending degeneration* (Türck). If, therefore, the motor tract in the cerebrum, the pons, or medulla oblongata be destroyed, we find an atrophy of the corresponding anterior and opposite lateral pyramidal tract in the spinal cord (Fig. 65; *cf.* also Fig. 66).

If the interruption to conduction occurs in the spinal cord—as in cases where injuries and diseases cause complete circumscribed trans-

[1] *L.c.* and *Obersteiner*, Festschrift, 1907. [2] *Z. f. N.*, xxxiii.

verse destruction of the cord, *e.g.* transverse myelitis, compression of the cord, etc.—we find both pyramidal tracts degenerated in the spinal cord below the point of lesion. The anterior pyramidal tract naturally participates in the secondary degeneration only if the lesion concerns the higher segments of the cord, as this tract normally reaches only as far as the middle or lower dorsal region. It has been stated above, however, that, by means of the *Marchi* method, the degeneration may be followed still lower. Fig. 69 represents a degeneration limited to the lateral pyramidal tract.

While, then, the motor tracts degenerate in a descending direction,

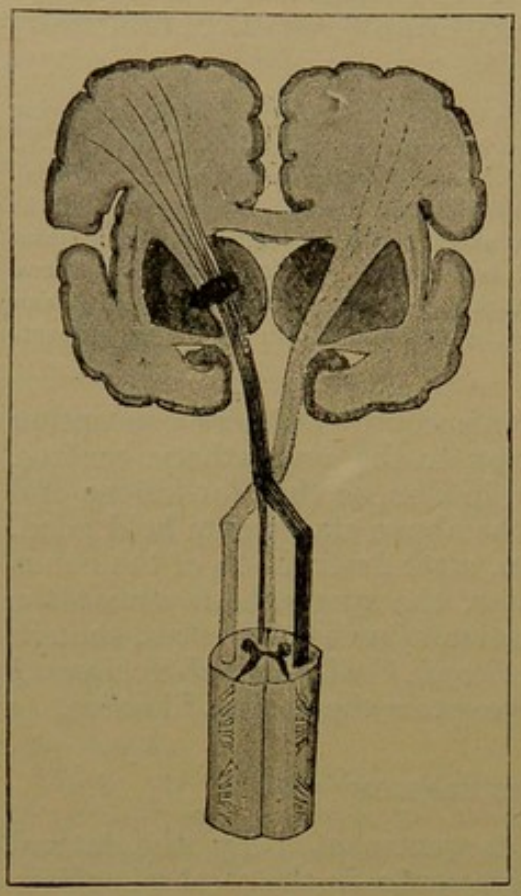


Fig. 65. — Descending degeneration in focal disease of the left internal capsule. (After Edinger.)

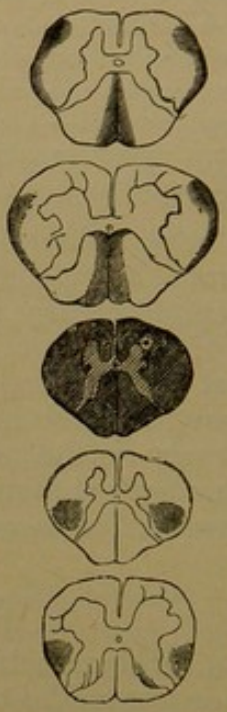


Fig. 66.—Secondary ascending and descending degeneration in transverse lesion of the upper thoracic cord. (After Strümpell.)

the secondary degeneration of the sensory tracts is an ascending one, because these arise from cells contained in the ganglia of the posterior roots in *Clarke's* column, and in other parts of the grey matter, and from these cells fibres run upwards in a centripetal direction.

Trans-section of the spinal cord at any level or any corresponding affection leads to the following changes in the part of the cord above the lesion : immediately adjoining the lesion the whole area of the *posterior columns* is degenerated, and also the *lateral cerebellar tract*, and the *fasciculus antero-lateralis*. The degeneration of the columns of Burdach, however, soon disappears, because they are, to a certain extent, reformed by the posterior roots entering at every level. In the cervical cord and in the medulla oblongata one finds, therefore, only degeneration of the

columns of Goll, the *lateral cerebellar tracts*, and *the tracts of Gowers* (*cf.* Fig. 66 and Fig. 67).

The degeneration in the fibres in the above mentioned fasciculi in the antero-lateral columns is not distinct and uniform, and is frequently recognisable for only a short distance. The determination of the degeneration is rendered specially difficult because fibres which degenerate in opposite directions lie side by side in the same tract. This holds good, *e.g.*, for the lateral limiting layer of the grey matter, for the fasciculus intermedius, etc. Yet the bundles defined by *Loewenthal* and *Marie* seem to degenerate chiefly in a descending direction, corresponding to the course of the already mentioned cerebello-spinal and bulbo-spinal fibres. This is equally true for the bundle described by *Held* and *Monakow* (*Rothmann* and others).

It may be remarked incidentally that there is also a descending degeneration in the posterior

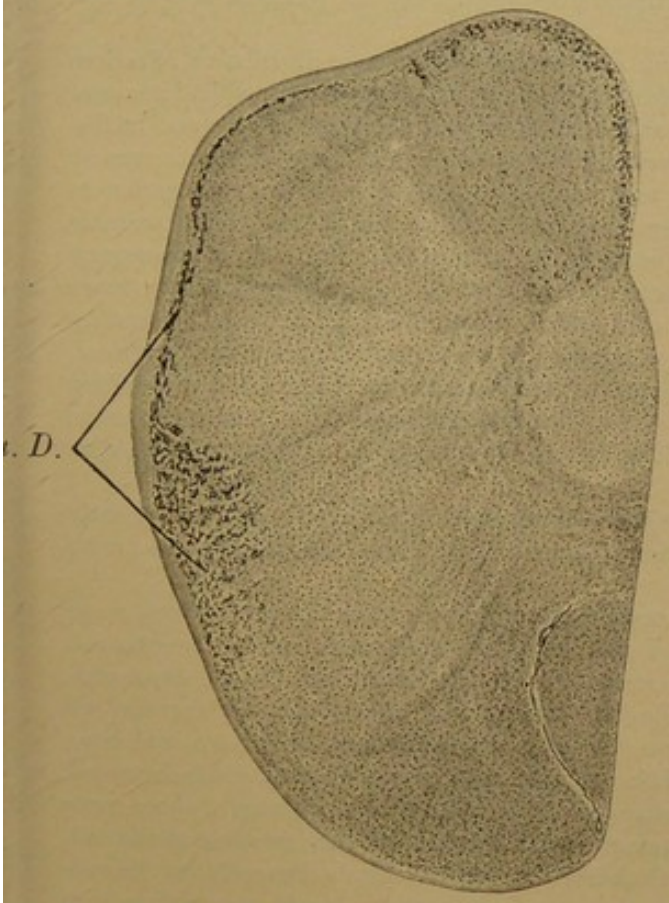


FIG. 67.—Ascending degeneration in the lateral cerebellar tract and in Gowers's tract in the medulla oblongata, by Marchi's method. a.D. =ascending degeneration.

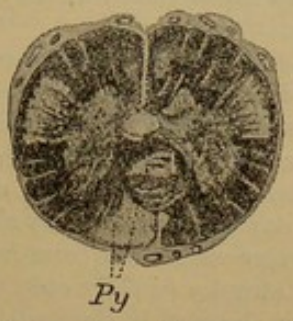


FIG. 68.—Descending degeneration of left pyramidal tract in a brain disease. (Pal's method.)

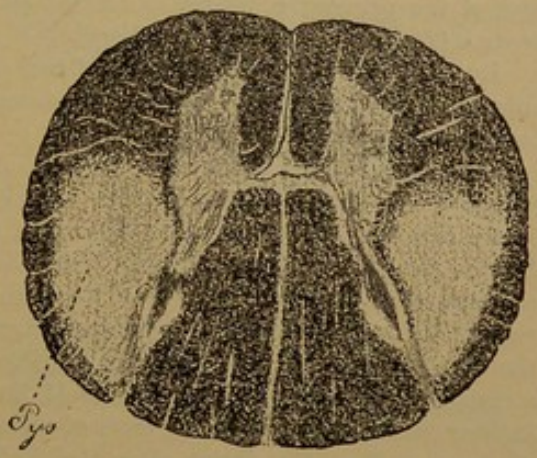


FIG. 69.—Degeneration of lateral pyramidal tract. (From a section stained by Weigert's method.)

columns. This is perceptible for only a short distance in two small comma-like areas (*Schultze*). These represent the descending branches of the sensory roots, spoken of on page 109, which likewise degenerate. Possibly the same holds good for the oval field which *Flechsig* describes as situated at the posterior septum of the lumbar cord, and also for the triangular field, found in the lower levels of the spinal cord, situated in the dorso-medial section of the posterior columns (Obersteiner's dorso-medial sacral bundle, Gombault-Philippe's median triangle). The question whether these three bundles, along with the ventral field of the posterior columns, all form one single connected tract, as also the other question, whether this consists chiefly of endogenous posterior column fibres or of descending branches of the posterior roots, has lately occupied the attention of many writers (*Loewenthal, Redlich, Hoche, Marinesco, Dejerine, Wallenberg, Russell, Campbell, Thomas, Schaffer, Zappert, Giese, Margulies, Fickler, Bikeles, Homén, Petrén, Marburg, Goldstein*,[1] *Matuszewski*, etc). Most of these authors think that both kinds of fibres are present.

[1] *M. f. P.*, xiv., also for the corresponding literature.

In the lateral cerebellar tract also and in the tract of *Gowers* there is an inconsiderable number of fibres which seem to degenerate in a descending direction—possibly they are descending branches of the nerve processes which come from the cells of *Clarke's* column.

An ascending degeneration is observed also after division or disease of the posterior roots. This has been proved by the investigations of *Schiefferdecker*, *Kahler*, *Singer and Münzer*, *Sottas*, *Tooth*, *Schaffer* and others, which show that the posterior columns form the direct continuation of the posterior roots.

This teaching in regard to secondary degeneration accords well with the neuron theory. If, however, the soundness of the *Apáthy-Bethe* fibril theory were acknowledged then the explanation of secondary degeneration would be wanting. Bethe's statements on this point cannot be discussed here.

The so-called *retrograde degeneration* is a form of atrophy which, after division or affection of the nerves as also after an interruption to conduction in the tracts of the cerebro-spinal nervous system, can develop in that part of the neuron still connected with the original cells and in the cells themselves. *Gudden*, *Monakow*, *Forel*, *Durante*, *Klippel*, *Gehuchten*, and others have turned their attention specially to the occurrence of these secondary changes. The degeneration is one which develops usually very slowly and irregularly, and is more marked in young animals and in diseases which date from infancy, or at least far back from early life. To this category belong the atrophy (usually only slight) of a nerve nucleus after disease of its peripheral nerve; the atrophy observed in the roots of the spinal cord and in the cord itself, after a long standing amputation (*Vulpian*, *Dejerine*, *Marinesco*, etc., as opposed to the negative findings of *Obersteiner*, *Orzechowski*); the rarely observed ascending degeneration in motor tracts and the descending one in sensory tracts of the cord; and finally, the still-unexplained atrophy of the optic nerve and of the primary optic nerve centres after long-existing lesions of the corresponding higher centres (*Monakow*, *Moeli*, and others).

By the use of the methods of *Nissl* and *Marchi* the circle of these discoveries has been greatly enlarged, and it seems almost as if the foundation, upon which the *Wallerian* theory rests, must be shaken.

It has been proved that after division of a peripheral nerve certain changes very soon occur (even after twenty-four hours) in the central end and also in the nucleus to which it is attached. These changes are recognisable only with the staining methods mentioned (*Nissl*, *Bregmann*, *Darkschewitsch*, *Marinesco*, *Flatau*, *Gehuchten*, *Lugaro*, and others). Soon after division the cell of origin, or in the case of section of a spinal nerve its cells of origin in the anterior horn, undergo a change. *Nissl's* staining shows disintegration of the granules in the nerve cells and displacement of the nucleus (Fig. 70 ,B. ; *cf.* with A.). These changes can be rapidly compensated if there come a restoration in the peripheral part. If, however, the regeneration fail, then in some of the cells further changes develop in the form of atrophy. According to *Gehuchten's* observations the cells of the spinal ganglia perish after the division of the sensory nerve. *Marinesco* distinguishes in this process a stage of reaction (réaction à distance) which is marked by the appearance of chromatolysis and displacement of nucleus, and a stage of regeneration in which there results a transitory swelling of the cells and other changes. *Marinesco* has since again discussed this question (*R. n.*, 1905).

These appearances have been variously interpreted. First, it appeared as if it were a retrograde degeneration, which was in direct opposition to the Wallerian theory. More recently the view has been brought forward that, after division of the nerve, its cells of origin atrophy, because the stimulus necessary for their life—the sensory impulse connected with the movements of the corresponding members, the central voluntary impulse, etc.—is no longer carried to them (*Marinesco-Goldscheider*, p. 66), and the functional activity of the ganglion cells is perverted (*Lenhossék*). The affection of the central end is, according to this conception, the result of the affection of the cell. This is all hypothetical.

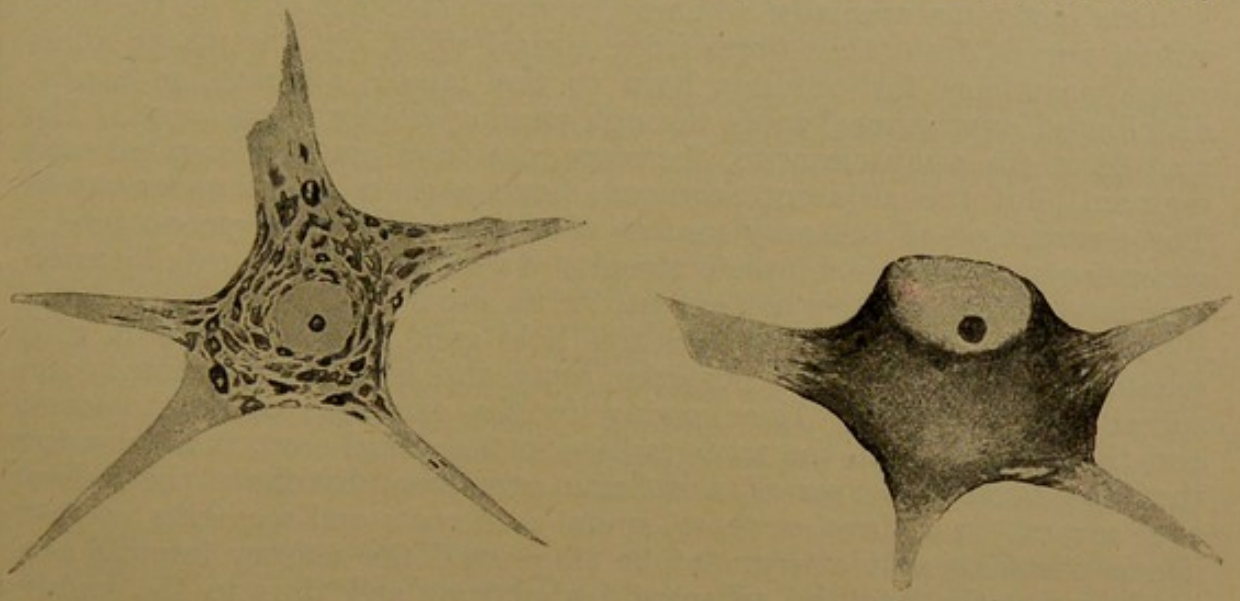
In agreement with these observations were found, after division and other lesions of sensory nerves, corresponding alterations in the cells of the spinal ganglia (*Lugaro*, *Cassirer*, *G. Koester*,[1] *Kleist* and others). It was seen that under certain conditions this *réaction à distance* extended

[1] "Zur Physiologie der Spinalganglien und der trophischen Nerven sowie zur Pathogenese der Tabes dorsalis." Leipzig, 1904.

even to the posterior roots and their continuation, *i.e.* to the posterior columns (*Redlich, Darkschewitsch*). It was attempted to explain these observations by the *Marinesco-Goldscheider* view, but the positive and negative results are both opposed to this (*L. R. Müller, N. C.*, 1904).

The *Nissl* method seems to be an unusually good means of demonstrating the changes occurring in the ganglion cells, and shows that a lesion of any part of the neurone involves the whole neurone. Yet it is necessary in the meantime to use great caution in the interpretation of all these results, and above all we must not regard the changes described as degenerative, since they represent fine separate processes and the Nissl's bodies, which are chiefly affected, form no vital constituent of the cell. Many years ago (even in the second edition of this text-book) I referred to the *Nissl* method in this same way, and also urged caution in the interpretation of the cord changes found only with the *Marchi* staining. More recently *Ziehen, Philippe, Marcus, Meyer, Heilbronner, Obersteiner, Spielmeyer* (*C. f. N.*, 1903) and others have had similar misgivings, and my standpoint in the question of the so-called retrograde degeneration has also been accepted by *Raimann, Schmaus-Sacki, Sträussler*, and others. On the other hand *Ziehen* rightly points out that the existence of retrograde degeneration renders the determination of secondary degeneration (*Wallerian*) and of the course of fibres more difficult.

In addition it may here be emphasised that the *Nissl* method has been employed in the study



A. B.

FIG. 70.—(After Marinesco and Raymond.)
A. Normal nerve cell by Nissl's method.
B. Affection of the cell after section of the peripheral nerve (chromatolysis and displacement of nucleus).

of affections of the nerve-cells produced experimentally—by poisoning, anæmia, rise of temperature, etc.—and that the investigations of *Nissl, Schaffer, Sarbo, Goldscheider, Flatau, Juliusburger, Marinesco, Dejerine, Babes, A. Fraenkel*, and others have led to important results.

Marinesco thinks that primary diseases of the nerve-cells, such as are occasioned by poisoning, are to be distinguished from the secondary affections above described. The latter affect chiefly the chromatophile substance, the former the achromatic cell substance.

LOCALISATION IN THE SPINAL CORD

assumes an accurate knowledge of the motor functions controlled by the individual segments of the cord—*i.e.* the area of origin of the various root-pairs, and of the area of innervation of the skin corresponding to the posterior roots. Our knowledge regarding these is, however, still very imperfect.

The following statements are based upon the investigations of *Ross, Thorburn, Starr, Sherrington, Mills, Head*, and others, also those of *Bruns*,

Kocher, *Chipault* and *Démoulin*, *Bolk*, and *Wichmann*.[1] On most points, however, I have relied upon my own knowledge of the subject. The known facts have been ascertained partly by experiments on animals (division of the various roots and stimulation of the same),[2] partly by anatomical investigations, and chiefly by the symptoms of irritation and of paralysis present in man in the injuries and diseases of the spinal cord or its different segments and roots. In man experimental observations could only rarely be carried out, such as *my own*[3] by electrical stimulation of the first dorsal and, later, of the eighth dorsal, and those of *Chipault* and *Seguin* by means of the division of various roots. *Head*, as already mentioned, has recognised in the distribution of herpes zoster a further guide to the investigation of these relations, and his views are founded on the basis of abundant pathological material.[4] They have as yet found no general acceptance, and have been specially contested by *Winkler* and *Charante*.

One fact must specially be borne in mind, that not only do the individual nerves draw their fibres from several roots, but the motor fibres which pass from the anterior horn to the muscle are also, as a rule, distributed over several roots through passing chiefly to one root. The root fibres for a muscle arise, therefore, not from one single spinal cord segment, but the adjacent segments have a share in its innervation. The destruction of the grey matter of one spinal cord segment would, accordingly, not cause complete atrophy of a muscle, but the neighbouring segments suffice, to a certain extent, for its supply.

To a still greater extent does this hold good, and we assert it with greater confidence, for the posterior roots and their distribution in the spinal cord segments. Here, owing to the existence of anastomoses, and the way in which the roots radiate in the spinal cord, it is provided that the sensory nerves of a definite skin area distribute themselves over several (two, three, or more) spinal cord roots and segments, so that if one posterior root or segment be eliminated the sensory impulse can reach indirectly into the adjacent higher root and thus to the higher segment of the cord. Consequently the injury of one pair of posterior roots does not necessarily occasion sensory disturbance nor, in an affection of the cord at a certain level, is the sensation in the area of innervation of the root originating at this level necessarily lost.

Regarding the topographical relation of the sensory innervation of the skin to the spinal cord or to the posterior roots, from the investigations and statements of writers[5] we may infer the following :—

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[1] *R. Wichmann*, "Die Rückenmarksnerven und ihre Segmentbezüge," Berlin, 1900, contians an almost complete summary of the literature. For the references to the sensory innervation, see article by *Grosser* in *C. f. Gr.*, 1904. Among more recent papers that by *Harris* (*Journ. of Anat. and Physiol.*, 1904) deserves special mention.

[2] Investigations of this nature have already been made by *Türck* in 1856, and very exhaustively also by *Sherrington*. Recently also, observations have been made by *Marinesco*, *Dejerine*, *Sano*, *Buck*, *Gehuchten*, *Bruce*, *Knape*, *Deneef*, *Parhon-Goldstein*, *Rosenberg*, *Lapinsky*, *Bikeles-Franke*, and others. These investigations have been partly experimental and partly observations on the relations of muscles and nerves to definite segments and nuclear groups of the cord, after amputations. The results obtained are still very uncertain and conflicting.

[3] *B. k. W.*, 1896, and *Z. f. N.*, xxiv.

[4] *Head* and *Campbell*, *Br.*, xxiii.

[5] *Sherrington*, *Head*, *Bolk*, *Coenen* ("Over de periphere uitbreiding van de achterwortels van het ruggemerg.," Dissert. Amsterdam), *Winkler* (*M. f. P.*, xiii.), *Winkler* and *Rijnberk* (IV., *Proceedings of the Royal Acad. of Sciences, Amsterdam*, etc.), *Blaschko* (*A. f. m. A.*, Bd. xxx.).

The extent and limits of the skin areas (dermatome, dermatomere, rhizomere, etc.) innervated by the posterior roots is explained by the originally metameric structure of the vertebrate animal body. The area of skin supplied by a root is an uniform continuous field, divided into two zones by the division of the spinal nerves into anterior and posterior branches. The posterior, the smaller and more variable of these, may for the present remain unconsidered. The dermatome does not quite coincide with the myotome but is usually towards its caudal side (*Sherrington*), so that, *e.g.*, in a lesion of an anterior and posterior pair of roots, the limit of paralysis lies higher than that of the anæsthesia.

Originally the dermatome forms a band perpendicular to the vertebral column and reaches from the posterior to the anterior axial line of the body; yet this law obtains only in the trunk, while at the extremities new secondary axial lines, an anterior and a posterior, can be defined for the arrangement of the dermatome. Individual variations, especially in the extremities, are frequently found (*Sherrington's* prefixed and postfixed types, etc.). The dermatomes in part overlap each other so that an area of skin is innervated not only by the one posterior root directly related to it, but also by the adjoining two, three, or more roots. The overlapping naturally occurs most at the margins. The division of a single posterior root does not therefore necessarily occasion any marked loss of sensibility, for tactile sensation at least—according to the clinical observations of *Charcot* (*Arch. de Neurol.*, xxii.) and *Prince* (*Br.*, xxiv.), and according to the experimental results of *Sherrington* (*Horsley* [*Practitioner*, 1904] seems in regard to this question to advocate another view); for the sensations of pain and temperature, whose dermatomes are apparently smaller and overlap less, there is a local diminution. Regarding these questions much has still to be explained. This is particularly so with reference both to the relation of the dermatome to *Head's* zones of hyperalgesia and of herpes, and to the share of the sympathetic fibres in the metameric innervation (*cf. Gross., C. f. Gr.*, 1904).

Another view, with which *Grasset* agrees, is given by *Brissaud*, who states that the sensory disturbances, brought about by a lesion in the individual spinal cord segments, extend in zones ("zones ségmentaires") which do not coincide with the dermatomes of the posterior roots, but refer to segments of the trunk and extremities. This view, however, finds no sufficient basis in clincal observations. In lesions of the posterior horns, though an anæsthesia is frequently found which extends to whole limbs or segments of limbs, and lesions of posterior roots produce a sensory disturbance which is distributed in longitudinal striæ over the extremity, this distinction is explained by the fact that in the first case it is not one single segment but a number of adjacent ones that are affected, and that the different outline is due to the confluence of anæsthetic zones.

With *Dejerine* ("Sémiologie du Système nerveux," and *Journal de Physiol.*, 1903) I must also contest the views of *Sano* and *Gehuchten*. The former of these observers ascribes to every muscle a distinct nucleus in the grey matter of the cord, while the latter assumes a segmental arrangement, *i.e.* that definite ganglion groups in the anterior horn represent individual segments of limbs. It is much more probable that synergesic muscles, *i.e.* muscles associated in a definite movement, *e.g.* flexion of the forearm, have a common nucleus and are innervated by the same segment. *Marinesco* (*N. C.*, 1902) at one time agreed with this view, but later (*M. f. P.*, xii.) has taken an intermediate standpoint by trying to define muscle nuclei—thus arriving at results similar to *Gehuchten*, *Buck*, *Bruce*, and others. *Sano*, also, has reached a like conclusion, for he ascribes to each muscle a nucleus, to each group of muscles a nuclear group, and to each segment of a limb a nuclear zone. *Remak*, in particular, has found in lesions of the anterior horns types for localisation, and to this view the Paris school has lately returned. *Remak* draws attention to the correctness of the conception that claims circumscribed sections of the grey matter as nuclei for functionally co-operative muscles. *Lapinsky* (*Z. f. N.*, xxvi) has further modified this view, and *Lazarus* (*Z. f. k. M.*, Bd. lvii.) agrees with him. *Bikeles* and *Franke*, in opposition to the authors named, accept only morphological principles for the conditions of innervation. They believe that a motor cell group, corresponding to the area of the anterior branch, innervates a muscular longitudinal stria of the extremity or of the myotome, and that the lateral cells of the anterior horn innervate the dorsally placed muscles of the extremities (Radialis, Peroneus, etc.). *Lewandowsky* agrees with this view.

The spinal nerves immediately after the union of the two roots divide into a ventral and a dorsal branch, the latter of which (except in the two upper cervical nerves) is the smaller. The posterior branches supply

the dorsal muscles as well as the skin of the back and nape of the neck, but take no share in the innervation of the extremities. This is effected through the ventral branches.

From the first, second, and third cervical segments arise the motor fibres for the deep cervical and neck muscles. The *levator anguli scapulæ*, the *trapezius*, and the *sterno-mastoid* also receive root fibres from these segments.

The nucleus of the spinal accessory (Fig. 71) commences in a cell group in the anterior horn of the upper cervical cord, and reaches downwards almost to the sixth cervical segment. For its roots, however, it is chiefly the three upper segments that are concerned.

According to *Marinesco* the nucleus for the sterno-mastoid must lie nearer the middle line than that for the trapezius ; *Parhon* places it in the central group of the two first cervical segments.

The motor supply for the *platysma* is, according to *Kocher*, derived from the third cervical segment, while *Frohse* derives it wholly from the facial.

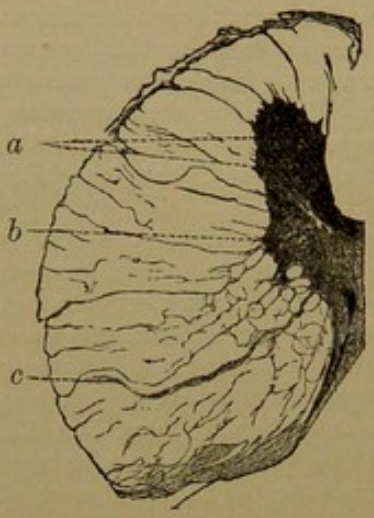


Fig. 71.—Part of transverse section of upper cervical cord. (After Grabower.)

a = spinal accessory nucleus.
b = lateral groups of nuclei.
c = root fibre of accessory nerve.

The phrenic nerve arises from the fourth cervical segment, but possibly its area of origin lies also in the third segment.[1] From this segment also probably proceed root fibres for the *rhomboidei*, the *supra-* and *infra-spinatus*, the *serratus magnus* (?), and the *scaleni*, but these are also connected with lower segments (according to Wichmann even to the eighth).

The fifth and sixth cervical segments distribute motor fibres, in their anterior roots, to the *deltoid*, *biceps*, *brachialis anticus*, and *supinator longus*.

The fifth root has a special relation to these muscles, and it also seems to have a relation to the *supra-* and *infra-spinatus*, to the *scaleni*, *rhomboidei*, *serratus magnus*, and *pectoralis major* (possibly only the clavicular portion of this muscle, while the costal portion is innervated by a lower segment). I have seen one case of plexus-paralysis which involved the seventh and eighth cervical and first dorsal, in which only the costal portion of the pectoralis major was affected.

The nuclear centres for the *extensors of the hand* and the *extensor communis digitorum* are to a certain extent in the sixth, but more especially in the seventh, cervical segment. The sixth segment has first of all a share in the innervation of the muscles ascribed to the fifth. Then it sends fibres to the pronators and to the triceps. From my own observations I would ascribe to the triceps a lower origin, as I have frequently found it affected in common with the muscles supplied by the seventh, and especially the eighth cervical segment, and in man contraction of the triceps can be brought about by electrical stimulation of these roots.

Wichmann claims this segment also for the flexor carpi radialis and pollicis, but this does not agree with my observations.

[1] According to *Luschka* the diaphragm has some relation to the five lowest cervical nerves, as *Wichmann* also mentions, but this is so slight as to be of no importance.

From the seventh cervical segment arise the root fibres for *the long extensors of the wrist and fingers*. Further, to it are ascribed centres for the innervation of the *latissimus dorsi*, *the teres major*, *triceps*, and possibly also for the *flexors of the wrist*. Yet there is no doubt that the nuclei of origin of the extensors of the wrist lie above those of the flexors, and those of the supinators apparently above those of the pronators.

But the extensor carpi ulnaris would seem to have a lower origin : thus I have seen two cases in which—in lesions of the eighth cervical and first dorsal—only the extensor carpi ulnaris was affected in the extension of the hand.

The eighth cervical segment is probably the main source of innervation for the *flexors of the fingers*, likewise for the *small muscles of the hand*, possibly also (my own observations make this probable) for the *extensor pollicis*, *longus* and *brevis*, and for the *flexor carpi ulnaris*.

The first dorsal segment has also a share in the innervation of the *small muscles of the hand* (especially the muscles of the balls of the thumb and little finger), and it is the main area of origin of the *oculo-pupillary fibres of the sympathetic* (*cf.* p. 117). According to *Dastre* and *Morat*, vaso-dilator fibres for the face pass out, in the eighth cervical and in the two first dorsal roots.

Regarding the relation of the individual cervical roots and spinal cord segments to the innervation of the skin, the following facts may be deduced from the observations and investigations before us (*cf.* Figs. 72 and 73). From the different well-known *schemata* (*Head*, *Starr*, *Kocher*, *Edinger*, *Seiffer*, *Blaschko*, and others), I have reproduced that of *Seiffer*, because, being based on the careful comparison of all the available data, it alone gives in a simple and comprehensive way, facts which are definitely established, or in the highest degree probable. One must not lose sight of the fact that here individual factors play a great part and may occasion many deviations.

The second, third, and fourth posterior cervical roots supply sensory fibres to the skin over the head, neck, cervical, and upper thoracic regions (to the second intercostal space, and as far back as the spinous process of the fifth cervical vertebra). This region is defined by the auriculo-parietal line, from that supplied by the trigeminal nerve. Each dermatome forms a narrow or broad band passing from behind forwards.

The skin of the upper extremities is supplied by the ventral branches of the spinal nerves from the fifth cervical to the first dorsal root (C_5-D_1). The fifth innervates the region of the shoulder over the deltoid muscle below the joint. The eighth cervical and first dorsal roots innervate the skin on the inner surface of the upper- and fore-arm and the ulnar side of the hand and finger. Some observers deny that its branches supply the upper arm. *Fröhlich* and *Grosser* claim a more extensive area for the eighth cervical. The remaining regions, *i.e.* a lateral one in the fore-arm and the radial and median areas of the hand, are supplied by the sixth and seventh cervical.

Wichmann states that the ventral branch of the sixth cervical supplies the skin over the external condyle and head of the humerus, by the external brachial branch of the musculo-cutaneous nerve ; further, by the lateral cutaneous nerve (antibrachii) it supplies the anterior surface of the radial side of the forearm ; by the superior and inferior posterior cutaneous nerves the dorsal surface of the upper arm and the elbow, and also the radial side of the forearm ; and, lastly, by the superficial radial the ball of the thumb, the thumb, and the index finger.

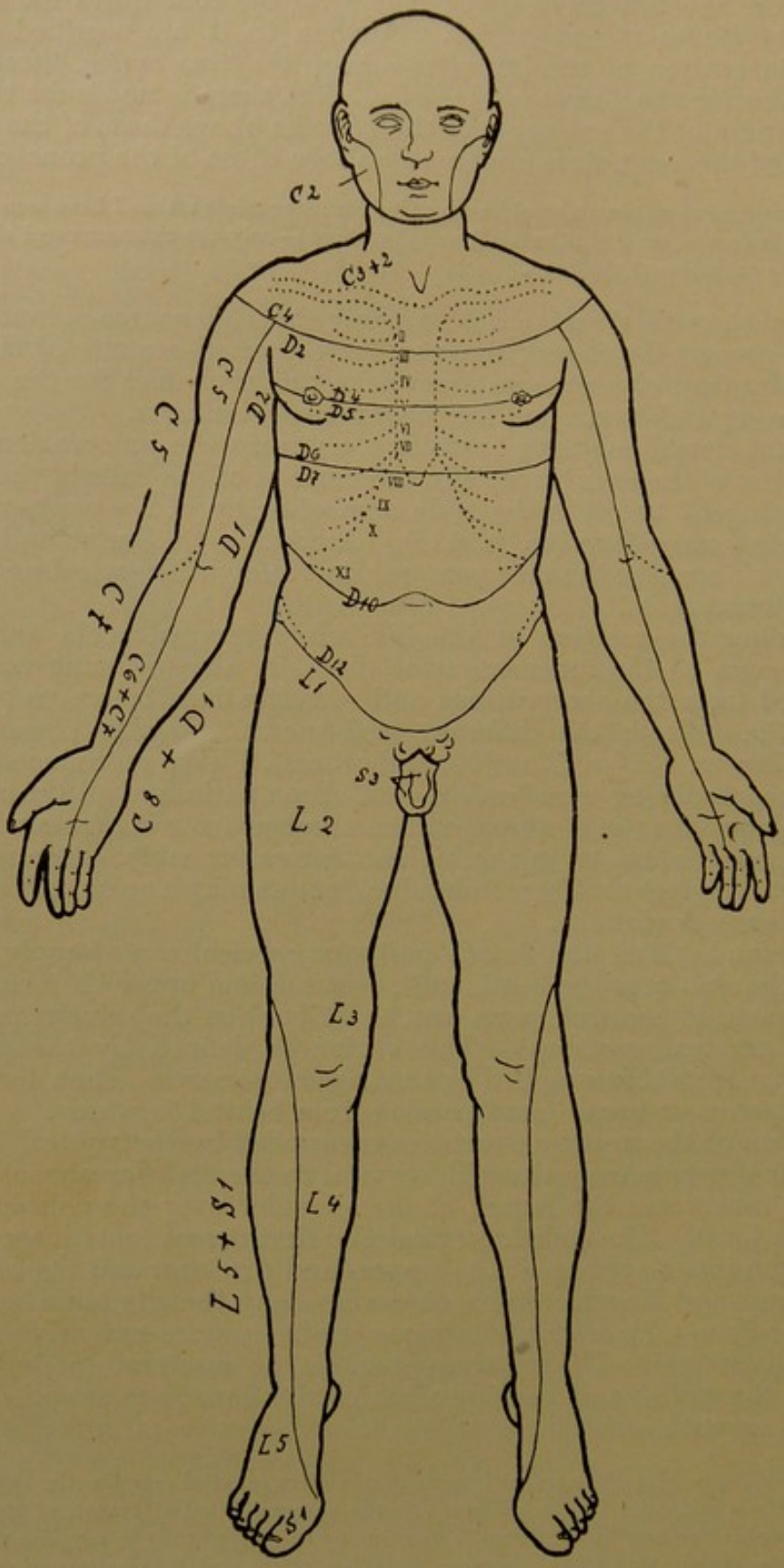


FIG. 72.—Diagram of spinal sensibility. (After Seiffer.)

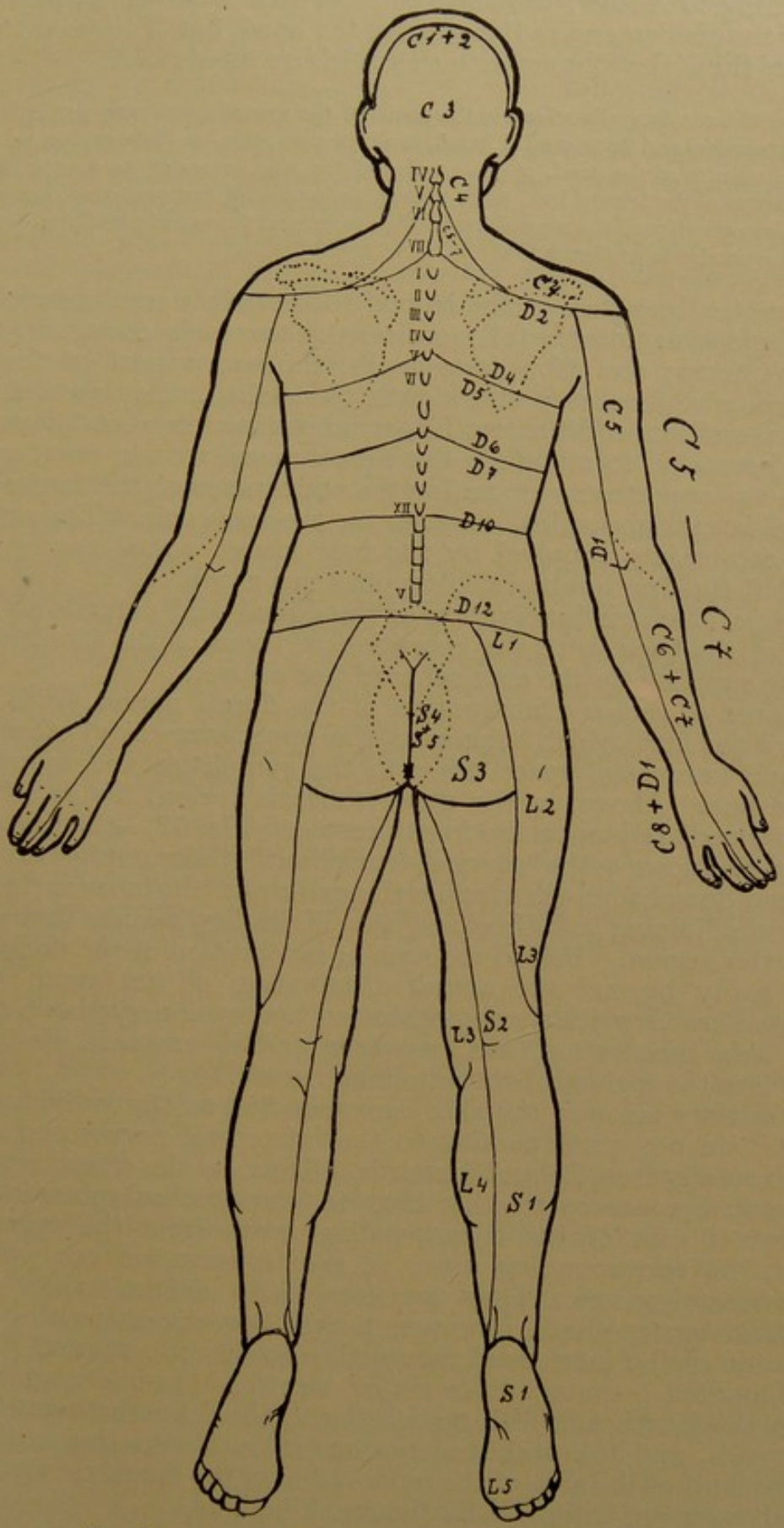


FIG. 73.—Diagram of spinal sensibility. (After Seiffer.)

According to *Kocher*, the sixth cervical root alone takes part in the supply of the median and radial areas of the hand, but *Wichmann*, *Grosser*, and others think that the seventh shares in this innervation.

In a case of hæmatomyelia affecting the nuclei of the seventh, but more particularly those of the eighth cervical and first dorsal, I found, in addition to oculo-pupillary symptoms, paralysis of the small muscles of the hand, of one portion of the long flexors of the fingers, and of the triceps, with loss of the triceps jerk, while that of the tendon of the supinator longus was preserved. I have since found the same thing in other cases, and the experimental results of *Bikeles* and *Franke* seem also to confirm it.

The motor branches of the second to seventh dorsal nerves supply the corresponding intercostal muscles, the levatores costarum, the triangularis sterni; the fifth to seventh, the upper part of the rectus and obliquus abdominis; the eighth to twelfth, the muscles of the abdominal wall, in addition to the corresponding intercostal muscles. I have had occasion to stimulate the eighth dorsal root in man, and have thus had the opportunity of noting the appearance of the contraction in the three lateral abdominal muscles on the same side. The abdominal muscles are mainly supplied by the four lowest dorsal nerves. *Stertz* claims that the first lumbar also supplies this group.

The area of motor innervation of the dorsal cord extends also to the muscles of the back, with the exception of those of the shoulder-girdle, which are supplied by the cervical cord (see above).

The area of sensory innervation of the dorsal nerves (with the exception of the first, which belongs to the upper extremity) extends from the second intercostal space down to the symphysis and round to the gluteal region. Above, in the upper thoracic region, the second dorsal borders immediately upon the fourth cervical (Fig. 72)—the line dividing these zones is known as the "cervical-trunk limit" (Halsrumpfgrenze). From the second dorsal a branch supplies the axilla and the inner surface of the upper arm adjoining it. *Head* and also *Wallenberg* assume that the third supplies this region. Below, the area of innervation of the dorsal nerves reaches really beyond the actual distribution of the main branches. *Sherrington* has very clearly demonstrated that each nerve has a principal area of skin innervation, but, upwards and downwards, its influence extends into the regions of the adjoining nerves.

The sensory areas of the skin corresponding to the individual dorsal segments, are not quite parallel to the intercostal nerves and ribs, but run horizontally from behind forwards. Thus, on the dorsal surface the upper limit of a sensory area lies three to four vertebral spines lower than the point of exit of the corresponding nerve from the spinal canal. Further, the corresponding areas of skin innervation are wider than the intercostal spaces. They are irregularly defined, show different levels (*Eichhorst*), which, however, nearly correspond to the areas of distribution of the intercostal nerves themselves (*Grosser* and *Fröhlich*).[1] As well-marked boundary lines *Seiffer* mentions the intermamillary line between the fourth and fifth dorsal, the xyphoid line between the sixth and seventh, and the umbilical line on a level with the tenth dorsal, while the limit of the area of the innervation of the dorsal nerves, towards the lumbar region, is formed by the trunk-leg line.

[1] *Morphol. Jahrbücher*, xxx. and *Z. f. N.*, xxiii.; see also *Grosser*, "Die Metamerie der Haut," *C. f. Gr.*, 1904.

The dermatome of the dorsal nerves, therefore, embraces the whole trunk.

Regarding the sphere of innervation of the *lumbar* and *sacral roots*, our knowledge is much less complete. The following facts may be deduced from experimental and clinical observations.

The first lumbar segment has possibly a share in the innervation of the *abdominal* muscles, it also supplies the *ileo-psoas* muscle and takes part in the innervation of the *cremaster*.

The second and third contain trophic centres for the *cremaster*, the *flexors*, and *adductors* of the thigh, possibly also for the *sartorius*, which some investigators connect with the first lumbar segment.

The third and fourth supply the *extensors* and *adductors* of the thigh, the *abductors* (?), the *extensor cruris quadriceps*, which is mainly innervated by the fourth lumbar, possibly also the *tibialis anticus*. Wichmann says that the nuclei for the gastrocnemii and extensor longus digitorum reach into the fourth lumbar segment—a statement with which, so far as it refers to the gastrocnemii, I do not agree.

Stertz also places the nucleus of the tibialis anticus muscle in the fourth lumbar segment.

The *fifth lumbar and first sacral segments* supply the *flexors of the knee*, the *glutei* (probably also the pyriformis, the obturator internus, and gemelli), and the *long extensors of the foot and toe*—which are chiefly supplied by the first sacral. *Bruns* thinks that the peronei have their centres below the nuclei for the extensors of the foot and toe. Some writers say that the glutei are supplied by the lower sacral segments, and this recent observations of my own tend to confirm. *Gierlich* places the flexors of the knee still lower.

Parhon-Goldstein (*N. C.*, 1905) make the following statements : the nucleus for the quadriceps lies in the external group in the third and fourth lumbar segments, for the sartorius in the antero-external group of the third lumbar, for the adductor magnus in the central group of the fourth lumbar, for the semi-membranosus in the central group of the fifth lumbar, for the tibialis anticus at the same level. The nucleus for the peronei lies in the postero-external group of the first sacral segment.

From the *first and second sacral segments* arise the root fibres for *the muscles of the calf* (including flexors of the toes), and the *small muscles of the foot*. Some observers (*L. Müller*, *Minor*, *Leyden-Goldscheider*) think that the centre for the peroneus reaches lower than that for the tibialis posticus.

From my own observations I would contest this statement, for I have seen a case of spinal-cord compression at the level of the twelfth dorsal vertebra, in which ankle-clonus co-existed with a degenerative paralysis of the peroneal group. *Minor* has himself recently (*Z. f. N.*, xxxi.) adopted this view.

From the third and fourth come the fibres for the *muscles of the perinæum, the bladder, and the rectum* (*cf.*, however, the statements on p. 120). The bulbo-cavernosus and ischio-cavernosus also are supplied by this area.

The fifth sacral and the coccygeal nerves innervate the *levator ani*, yet Wichmann associates this muscle with the third sacral.

As regards the relations of the *posterior roots* of this region to *the innervation of the skin* of the lower limbs, Fig. 72 and Fig. 73 show the present position of this question. The limits are not sharply defined, and

the areas overlap in part, as each skin area is innervated by several roots. Individual variations are also frequently found.

It may again be specially emphasised that the third and fourth sacral roots contain the sensory fibres for *the region of the perinæum*, for the *anus* and its *neighbourhood*, for *the mucous membrane of the bladder*, as well as for a narrow strip at *the posterior and inner surface of the thigh. Sherrington* names this portion of the skin which lies over the nates, perineum, and inner surface of the thigh, and which is supplied together with the genitals, the genital flap or sexual skin.

The coccygeal (and fifth sacral) send sensory fibres to the region of the coccyx.

For details of this arrangement, *cf. Grosser.*

According to *Kocher*, *Schlesinger*, and others the motor centres for the bladder and anus are contained in the lower part of the conus medullaris, just where the fourth sacral nerve arises, while the corresponding sensory areas are situated somewhat higher. The reflex for erection passes, according to these authors, probably in the second sacral segment, while the centre for ejaculation lies lower (*cf.*, however, the statements of Müller, p. 121). In affections of the conus, in spite of anæsthesia of the skin of the scrotum, the testis itself is sensitive; the cremasteric reflex is also retained, and the sexual desire and power of erection may be preserved although the power of ejaculation of semen is lost.

Finally, concerning the relation of roots and segments to the reflex functions, in addition to all that has been said previously, the following statements are of importance for localisation in the cord: the abdominal reflex is effected by the eighth to the twelfth dorsal roots, the cremasteric reflex by the first and second lumbar roots.[1] I must, however, state that by abdominal reflex I mean only those reflex movements of the muscles which can be originated from the skin of the abdomen. Further, the supra-umbilical abdominal (epigastric) reflex can be differentiated from the infra-umbilical, and, for the former, I claim the eighth and ninth, and, for the latter, the tenth and eleventh dorsal roots. The reflex-arc for the knee jerk passes through the second, third, and fourth lumbar segments and corresponding roots. According to some writers it is chiefly the fourth.

For the Achilles tendon jerk, the fifth lumbar and especially the first sacral root are concerned—according to my own oberservations the first and second sacral; for the plantar reflex, the first and second sacral roots come into play. The so-called anal reflex—the contraction of the sphincter ani when the finger is introduced into the anus (frequently the irritation of the skin in the neighbourhood of the anus will cause this contraction)—is caused by the lowest part of the spinal cord and its roots (fifth sacral and coccygeal). I have found the reflex greatly increased in an affection of the epiconus. These statements may be qualified by further observation.

Kaiser, Collins, Hammond, Onuf, and *Marinesco* have sought, by anatomical investigations, to define the position and extent of the nuclei of the various spinal nerves.

Experimental observations and those after amputations have been specially numerous and exhaustive, and, chiefly by means of *Nissl's* method, have supplied many valuable contributions

[1] *Kocher* describes a special testicle reflex which is carried out by the lower dorsal cord.

to the study of this subject. As the results are very diverse and their interpretation by no means settled, I limit myself to the most important. I refer to those of *Sano, Gehuchten, Buck, Crocq, Marinesco, E. Flatau, Parhon-Goldstein, Bruce, Bikeles-Franke, Blumenau-Nielsen,* and others.

Valuable facts regarding the spinal localisation of muscles have also been ascertained by *Monakow, Dejerine, Stewart-Turner, Onuf, Rosenberg, Brissaud-Bauer,* and others.

Many authors—especially *Marinesco, Sano, Parhon-Goldstein,* have studied on transverse sections the position and arrangement of the cell-groups interpreted as nuclei, and have obtained remarkable results.

The atrophic paralysis of a muscle group therefore, if it be of spinal origin, enables a definite conclusion to be arrived at regarding the site of the lesion. Great caution is necessary in the interpretation of sensory disturbances in the diagnosis of the level of the disease. It must be remembered that total anæsthesia in an area occurs only if the neighbouring roots or segments be also affected. Anæsthesia of the skin, supplied *e.g.* by the sixth dorsal segment, would be brought about only when the fifth and probably also the fourth are affected.

Sherrington has shown, as already mentioned, that division of the posterior root in the monkey caused a diminution of the sensations of pain and temperature, but, on the other hand, occasioned no disturbance of tactile sensibility. From this he inferred that the overlapping of the segmental fields for the sense of touch is greater than for the other sensations. With this view *Muskens* agrees.

In man lesions of single roots have only very rarely been observed—as those by *Charcot, Prince, Wallenberg, E. Bramwell, F. Buzzard,* and *Respinger.*

As hyperæsthesia frequently develops in root areas only partially affected, it can be taken as the upper limit of the disease and thus be of great value in the diagnosis of the level of the lesion.

The condition of the reflexes also forms a guide as to the localisation. Thus, to take only one example in a case of tumour of the spinal cord, which I diagnosed correctly to be at the level of the eighth dorsal nerve, the absence of the abdominal reflex on this side was the first objective sign of the disease. But it must be remembered that affections of the spinal cord which cause a complete interruption in the conduction may, even although their site is far above the reflex centre, bring about loss of the reflexes, especially the deep reflexes (*Bastian, Bruns,* and others); *cf.* pp. 115 *et seq.* Nor must it be forgotten that even in health some of the reflexes are inconstant.

Regarding this question, the relation of the spinal-cord segments to the vertebræ is of great importance, and also that of the area of origin of the roots to their place of exit from the vertebral canal.

The origin in the spinal cord, if we except the highest part of the cervical cord, lies higher than the point of convergence from the vertebral canal, and the distance increases from above downwards. Individual variations regarding this are, however, very marked (*Reid, Starr*). As a rule the seventh cervical vertebra corresponds to the first dorsal segment, and the eighth cervical root arises opposite to the lower border of the body of the sixth cervical vertebra. In the dorsal cord the roots arise from the cord one, one and a half, to three vertebral bodies higher than they emerge from the vertebral canal. The difference is accentuated from above downwards. Thus the place of origin of the sixth dorsal root corresponds to the intervertebral disc between the fourth and fifth vertebral body; the origin of the tenth corresponds to the eighth vertebra; opposite to the eleventh dorsal vertebra the first lumbar nerve arises; between the eleventh and twelfth the second lumbar nerve;

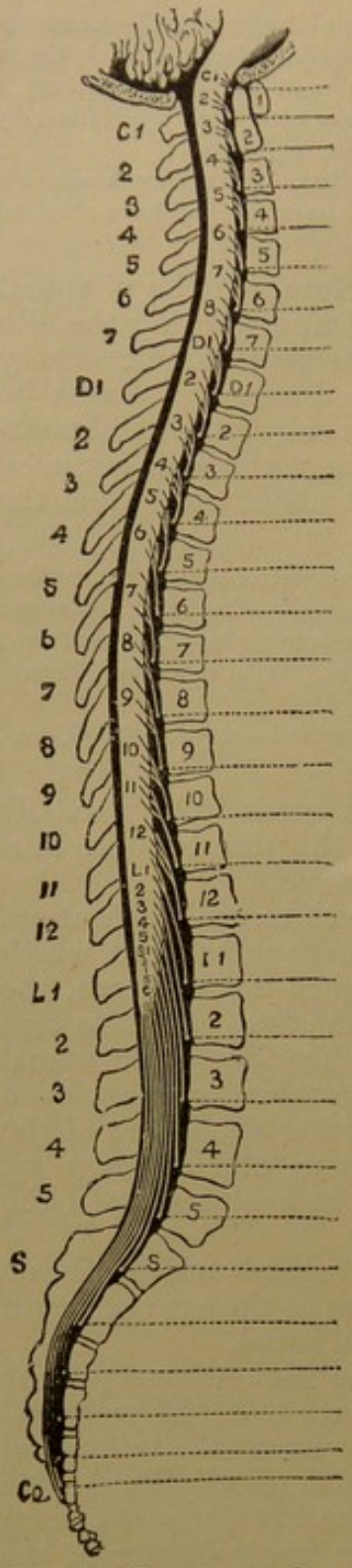


Fig. 74.—Drawing to show the relation of the spinous processes to the vertebral bodies, and the sites of origin of the nerve roots. (After Gowers.)

opposite the twelfth, the third and fourth lumbar nerves; and between this and the first lumbar vertebra the fifth lumbar and first sacral nerve; the other sacral nerves opposite to the first lumbar vertebra, or possibly the intervertebral disc between the first and second lumbar vertebræ (Fig. 74). The entire lumbar and sacral cord, therefore, corresponds to the portion of the vertebral canal from the eleventh dorsal vertebra to the first lumbar inclusive.

In descending from their origin to their emergence from the vertebral canal—a more or less considerable length of the cord—the roots touch the segments from which a greater or lesser number of the roots below arise. This is specially the case with the lumbar roots, which in their course come in contact with almost the whole sacral cord.

Further difficulties as to the diagnosis of the level of the lesion in the cord arise from the circumstance that in deciding the vertebral level we have to judge by the spinous processes; and the relation of the intervertebral foramen—the point from where the roots emerge—to the spinous process is by no means the same at every level and is altogether very variable. In the cervical region where the processes pass directly backwards, the intervertebral foramen is found in the middle between the spinous processes of the adjoining vertebræ; in the dorsal region, where the processes incline downwards, the corresponding intervertebral foramen lies about one spinous process higher, *e.g.* that lying between the ninth and tenth dorsal vertebræ almost corresponds with the tip of the spinous process of the eighth dorsal vertebra (according to *Chipault* it lies even higher). In the lumbar vertebræ the spinous process is again on a level with the body of the vertebra.

Uni-lateral Lesions of the Spinal Cord—Brown-Séquard's Paralysis

The experiments of *Brown-Séquard* first made known the signs which are presented in an unilateral lesion of the spinal cord, *i.e.* a lesion in which the conduction in one-half of the transverse section of the cord is interrupted. They are as follows:—

Paralysis of the corresponding side and anæsthesia on the opposite side. This anæsthesia is, however, not complete, the sensitiveness of the deep parts (bathyanæsthesia) being preserved. The perception of position in particular is as a rule diminished on the side of the lesion, or may be lost. There is, in addition, a certain hyperæsthesia, especially for painful stimuli, and usually a local rise of temperature from 0·5 to 1·0 C. Derangements of the bladder and rectal functions may be present, but are not constant.

These appearances may be thus interpreted: the paralysis on the same side is easily explained by the fact that the motor path, at least the main part concerned—the lateral pyramidal tract—is a direct tract in the spinal cord. The condition of the sensibility, on the other hand, must lead one to assume a complete crossing of the sensory tract immediately after its entrance into the spinal cord—the fibres concerned with the conduction of muscular sense being alone excepted.

The experimental results of *Brown-Séquard* were only partly confirmed by the investigations of *Ferrier*, *Turner*,[1] and others. But *Mott* in particular, *Horsley*, *Schäfer* and others obtained results in direct opposition to the teaching of *Brown-Séquard*, and the explanation of the symptoms called forth such marked opposition that ultimately it was withdrawn by the author himself.

But clinical observations[2] are essentially in agreement with the *Brown-Séquard* teaching, and more recent views regarding the course of the fibres in the spinal cord confirm on the whole the explanation of the facts which he had previously given.

The unilateral lesion usually affects the dorsal cord. We then find: (*a*) *on the corresponding side*: 1. Paralysis of the leg, associated as a rule with increased tendon jerks, but at the commencement—and, as I have observed, even for weeks—these may be diminished or lost. 2. Hyperæsthesia of the skin for painful or possibly for all stimuli. This is not always distinctly marked, and may be rapidly restored or may persist for a long time. 3. Loss of the sense of position (bathyanæsthesia). This is certainly not a very constant phenomenon, but, according to the *Brown-Séquard* teaching, it is diminished on the affected side. 4. With the return of movement in the leg, ataxia may set in (*Bottazzi*, *Herhold*, *Kocher*, *Oppenheim*). (*b*) *On the opposite side* to the lesion: anæsthesia for all sensations, with the exception of the sense of position or far more frequently only analgesia and thermanæsthesia (*Gowers*, *Oppenheim*, *Mann*, *Kocher*, *Laehr*, *Brissaud*, *Petrén*).

On the side corresponding to the lesion, within the area of supply of the roots directly affected, semi-girdle pains and hypæsthesia or anæsthesia; while on the opposite side the anæsthetic zone is limited above by a hyperæsthetic zone; this may extend, however, to the paralysed side, and may occupy a narrow area above the anæsthetic zone. *Brissaud*, in Fig. 75, endeavours to give an explanation of these phenomena; it refers, however, only to those sensory tracts which cross soon after their entrance into the spinal cord.

If the lesion be in the cervical cord, then spinal hemiplegia results, *i.e.* arm and leg are paralysed on the same side as the lesion. The paralysis is usually associated with contracture, which may be either slight or very marked. On the opposite side anæsthesia is present over the leg and trunk, and extends upwards as far as the area of distribution

[1] *Turner* agreed with *Brown-Séquard's* observations for the lower extremities, but, for the upper, asserted that only the tracts for the sensation of pain cross. According to *Bechterew*, the crossing of the tract for the sensation of touch is imperfect, but more perfect for the lower extremity than for the upper. He agrees with *Turner*.

[2] We must refer specially to the communications of *Brown-Séquard* himself, then to those of *Köbner*, *Enderlen*, *Raymond*, *Brissaud*, *Laehr*, *Mann*, *Kocher*, *Schlesinger*, my own (*A. f. A.*, 1899), as also to those of *Jolly*, *Henneberg*, *Dejerine*, *Petrén*, *Wagner-Stolper*, *Peugnier-Philippe*, etc., *Cf.* also the summary of the literature by *G. Flatau*, "Die Stichverletzungen des Rückenmarks," *C. f. Gr.*, 1905, also *Krauss* (*Journ. of Nerv. and Ment. Dis.*, 1906), and the observations of *Head* and *Thompson* (*Br.*, 1906), practically confirming the old *Brown-Séquard* teaching.

of the roots which join the cord at the level of the lesion, inasmuch as these have a partial crossing first at a higher level of the cord. On the other hand, oculo-pupillary signs may appear on the side of the lesion.

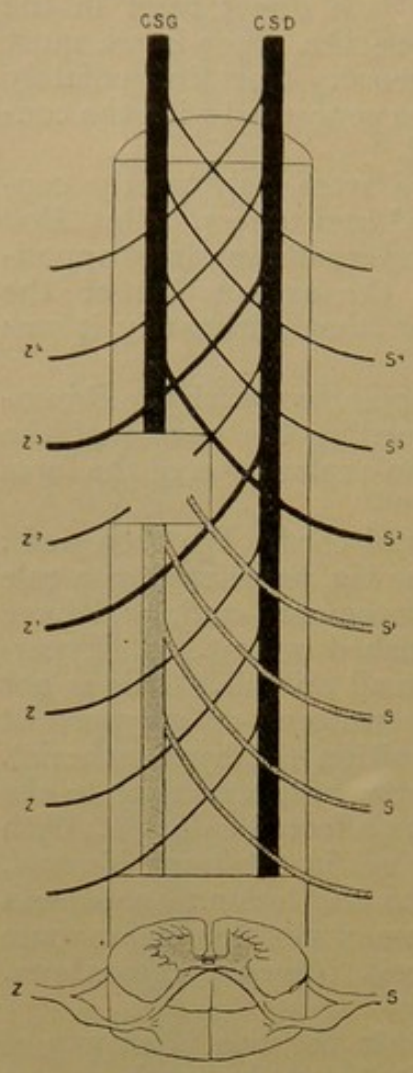


FIG. 75.—(After Brissaud.)

Diagram to explain the condition of the sensibility in Brown-Séquard paralysis. CSG and CSD, sensory nerve tracts of right and left sides. S-S⁴, posterior roots of left side. Z-Z⁴, posterior roots of right side. The incision between Z² and Z³ represents the unilateral lesion. The anæsthesia affects the roots of the left side entering the cord below the focus (S, S, S,), the root of the right side opening into the focus itself, Z², whilst there is hyperæsthesia in the area of Z¹ and Z³ of the right, and of S² of the left side.

If the unilateral lesion be low in the lumbar cord or in the sacral cord, then the paralysis and the sensory disturbances both affect the leg on the same side, as at this level, naturally, only a few sensory roots have crossed over to the other side.

Wernicke and *Mann* (*Z. f. N.*, x.) have shown that there is a type of unilateral lesion of the lumbar cord, where the affection is so high that the sensory tracts have already crossed to the opposite side. They found anæsthesia of the paralysed, atrophic leg, and also on the opposite side anæsthesia in the region of the scrotum, perinæum, penis. I have recently had a similar case.

If the disease causing the unilateral paralysis occur in youth, before the end of the period of growth, the paralysed limbs remain short and the muscles are retarded in development. This arrest of development was very marked in one of my cases where the paralysis had almost entirely disappeared.

The condition of sensibility in the *Brown-Séquard* unilateral lesion can be now more satisfactorily explained by the more accurate knowledge of the course of the fibres in the spinal cord. It is very probable that the path in the spinal cord that conveys the sense of position takes an uncrossed course in the posterior columns, possibly in part by *Clarke's* columns to the lateral cerebellar tract. The tracts for the conduction of pain and temperature sensations pass, chiefly or altogether, through the grey matter. It must not, however, be assumed that they continue upwards in this, as there are no long paths of conduction in the grey matter. They remain in it for only a short distance (Böttiger says for four segments). According to the prevailing view the majority or all of them reach the anterolateral column of the opposite side[1]; it is still undecided whether this crossing is effected chiefly in the anterior or posterior commissure. More recent observations, as above (p. 111), support the former view. While, therefore, the grey matter forms chiefly

[1] *Woroschiloff*, *Holzinger*, and *Bechterew* found that division of the lateral column produced analgesia. It has been stated that in the lateral limiting layer of the grey matter, therefore in the deeper part of the lateral column, there are tracts for the sense of touch, which, arising directly from the posterior roots, pass up uncrossed. This is, however, very uncertain.

a transit station for the sensory fibres joining the segment in question, the antero-lateral column of the dorsal cord contains all the conducting tracts for the sensations of pain and temperature of the leg of the opposite side. Among recent writers on this subject *Ziehen* especially advocates another view. From experimental and clinical observations *Ziehen* draws the conclusion that the paths of conduction for the sensations of touch, pain, and temperature in man (monkey and dog) are both *crossed* and *uncrossed*. *Ziehen*, in coming to this conclusion, is doubtless guided largely by the results of experiments.

Both *Lewandowsky* and *Rothmann* are sceptical of or opposed to the old *Brown-Séquard* teaching, yet *Rothmann* (*B. k. W.*, 1906) at one time held views very closely resembling it in regard to man.

This is also the opinion of *Head* and *Thompson* (*Br.*, 1906). According to their very exhaustive investigations the tracts for the different forms of sensation, on their entry into the spinal cord, undergo a re-arrangement (opposed to that in the peripheral nerves) by which a complete (or almost complete) crossing takes place for the tracts for pain- and temperature- sensation, as well as for that for deep pressure. This is completed within a few segments by a separate course for the tracts for cold and heat. The fibres conveying the sense of touch reach also to the other side, but very gradually, so that the conduction for a long distance is on both sides. The conducting tract for the perception of movements is, throughout the whole cord, an uncrossed one.

Kalischer and *Lewandowsky* have, by means of the former's "training" method, showed in the dog the crossing of the tract for the sense of temperature.

As the condition of tactile sensation in *Brown-Séquard's* paralysis is the least easy to understand, so the question of the conduction of the tactile stimuli still remains unsolved. Concerning this many teach that it is conveyed upwards in the posterior columns—*Schiff*, in particular, upholds this view—but this seems inconsistent with the fact that tactile sensibility is often retained in unilateral lesions. Further, it has been supposed that fibres may pass directly from the posterior roots to the limiting layer of the grey matter (?), and further by a system of short tracts with frequent intercalations in the grey matter, etc. *Mann's* view undoubtedly deserves most consideration. He supposes that the tactile impulses pass up any open centripetal path, and that so long as fibres are present for the transmission of centripetal stimuli, these can be followed by the tactile sensations, even although the posterior columns mainly subserve this function. *Petrén* also assumes an uncrossed and a crossed tract for the transmission of this sensation (also *Borchert*, *Rothmann*, and others). A case of *Jolly's* shows the importance of the latter. Some writers (*Langendorf*, *Münzer-Weiner*, *Ziehen*) hold that the sensations for touch follow the same paths as those for pain and temperature, and with this the experimental results of *Borchert* and *Borowikow* agree. Other investigators also, altogether deny that there are separate tracts for the conduction of the different forms of sensation.

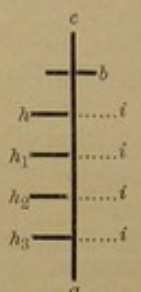
Rothmann, on the grounds of his experiments, concludes that no order of sensation is associated with any definite tract.

Strümpell holds that the fibres not only for the muscular sense, but also for the sense of pressure, and probably also for the sense of touch, run uncrossed in the posterior columns.

Head and *Thompson* think that in spinal affections (as opposed to peripheral) the sense of pressure behaves in the same way as the sense of touch, whereas the power of discriminating the two points of a compass remains parallel to the muscular sense; disturbance of this sense, therefore, just as with bathyanæsthesia, corresponds to the side of the paralysis.

For the hyperæsthesia of the paralysed leg no satisfactory explanation has been given, in spite of the experiments of *Woroschiloff*, *Raymond*, *Gowers* and others.

I [1] have suggested the following hypothesis: in the long ascending tracts special impulses are carried upwards, which pass to sub-cortical (cerebellar, bulbar, pontine) centres (co-ordination centres, etc.) without awakening consciousness, whereas the stimuli which produce conscious sensation go chiefly or solely through the lateral branches on the crossed side. Each stimulus employed in the sense-test is divided, therefore, after it reaches the cord by the roots (h-h_3) into two components, the one of which passes upwards in a c (which represents the long direct ascending tract) and influences the sub-cortical centres, while the other (i), after passing over to the opposite side and the secondary ascending tract, incites conscious sensation. By an interruption in conduction at b the transmission of the impulse upwards is prevented, and thus the stimuli producing conscious sensation, carried from h to i, become intensified.



Münzer and *Wiener* have independently arrived at the same conception, and this also finds support in an observation by *Henneberg*.

Sensory signs of irritation play, in other respects, no essential part in this "symptom complex": yet pain may be caused by the primary disease, especially if it be a new growth. *Spasmodynia cruciata* (tonic spasm in paralysed muscles with simultaneous pain in the limbs on the opposite side of the body), described by me, occurs very rarely. Allocheiria (*Brown-Séquard*, *Jolly*) has occasionally been described, and, according to Jolly, is explained by preformed connections of the sensory tracts on both sides, which are, however, used only under special circumstances. *Rothmann* assumes that in man there is, on the one side, a smaller path of conduction for pain- and temperature-sensations, on the other side a larger tract, and that only after the loss of the latter does the former come into use. *Schittenhelm* and also *A. Schmidt* (*Z. f. N.*, xxvi.) describe marked disturbance of the power of localisation on the opposite side.

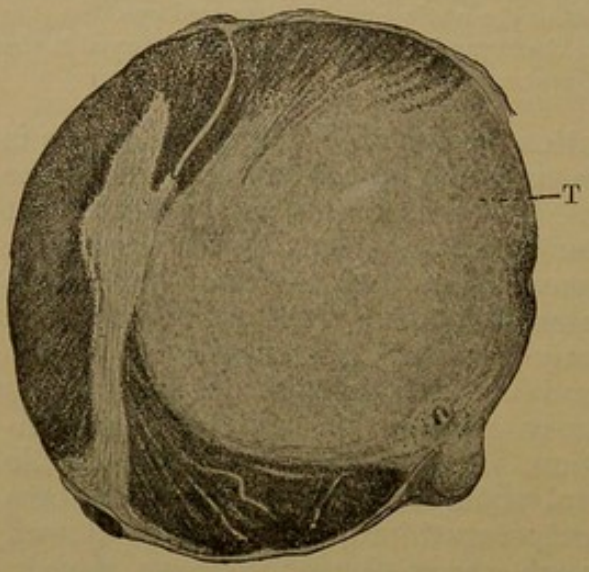


Fig. 76.—Transverse section of spinal cord from a case in which a tumour of the right side of the cord (T) had produced incompletely developed symptoms of unilateral lesion. (From a section of Oppenheim's prepared by Henneberg.)

Pathology teaches that it is chiefly injuries, and especially punctured wounds and gunshot wounds, that bring about the signs of a unilateral lesion in the cord. Hæmorrhage may also lead to an interruption to conduction on one side. In one case I saw *Brown-Séquard* paralysis come on after a quick movement while bowling, and disappear only gradually. Recently, surgical operations—division of roots for neuralgia (*Monod-Chipault*, *Prince*)—also have been found to be the cause of the development of this group of symptoms. Tumours, also, may produce it, and specially frequently, spinal syphilis (*Oppenheim*, *Lamy*, *Brissaud*, and others). I have shown that it also occurs in multiple sclerosis, but rarely in myelitis. It is natural that the complete picture is rarely seen, more frequently it is incompletely and indistinctly developed, so that paralysis prevails in one leg, anæsthesia in the other.

[1] *A. f. A. u. P.*, 1899. Supplementband.

Further, it is not unusual to find this group of symptoms preceding the development of a more extensive lesion (paraplegia or paraparesis), or it may follow such a lesion as is seen in non-traumatic cases, and in a specially characteristic way in tumours.

Lastly, it must be further pointed out that the prognosis of unilateral lesion, when of traumatic origin, is relatively favourable, since a partial —more rarely a complete—retrogression usually takes place. This holds good also, to a certain extent, for the forms produced by syphilis and hæmatomyelia. *Brown-Séquard* paralysis may also be a temporary manifestation during the course of multiple sclerosis.

A bilateral *Brown-Séquard* paralysis has been described by *Hanot* and *Meunier*, also by *Jolly*; I myself have also observed a temporary alternating of the same, the first attack proceeding from the left, the second from the right half of the cord (alternating *Brown-Séquard* paralysis), while, in a case of multiple sclerosis I have seen, in addition to a localised hemiplegia affecting the lower extremities, another in the upper extremities, but on the opposite side.

The results of treatment in the syphilitic and traumatic forms of Brown-Séquard paralysis have been specially favourable. Thus numerous cases have recently been noted in which, after the removal of foreign bodies and shot, localised by means of the Röntgen rays, the paralysis has been cured. Even when compression of the cord by an extra-medullary tumour, is the cause of this group of symptoms, operation is the effective treatment (*cf.* the section tumours of the spinal cord). I have observed a large number of such cases.

The Vessels of the Spinal Cord

The arteries supplying the cord arise from the spinal arteries, from the intercostal, lumbar, and sacral arteries. The spinal arise from the vertebral artery as two anterior, which blend into

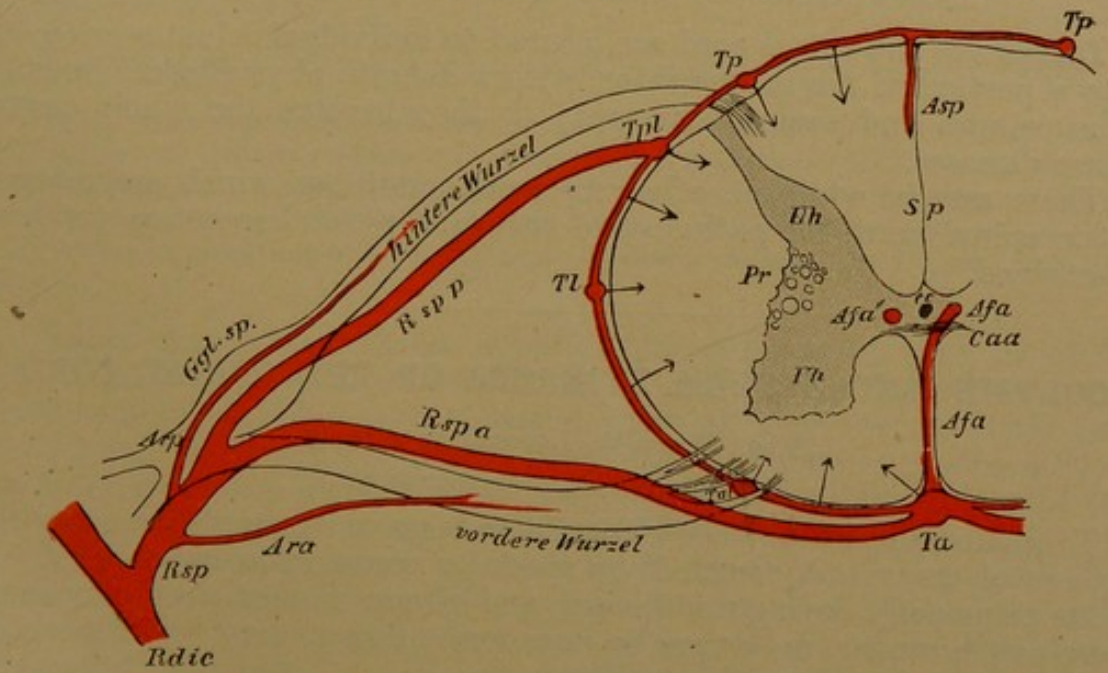


Fig. 77.—Diagram of the arterial vessels of a spinal-cord segment. (After Ziehen.) Afa, artery of the anterior fissure. Ara, artery of anterior root. Arp, artery of the posterior root. Asp, artery of post. med. septum. Rdic, dorsal branch of intercostal artery. Rspa, ant. spinal branch. Ta, anterior artery. Tal, antero-lateral artery. Tp, posterior artery.

one, and two posterior. These spinal arteries run on the anterior and posterior surfaces of the cord, and everywhere give off small branches which they send into this organ. Even at the level of the middle cervical cord they form an arterial system. The branches arising from the intercostals, the lumbar, and sacral arteries pass along the spinal nerve roots, and, dividing into ascending and descending branches, enter into communication with the vessels arising from the

spinal arteries. Thus arise the so-called arterial chains—the arterial system—one anterior and two lateral or posterior (tractus arteriosus posterolateralis and posterior), of which the anterior is the largest. The branches penetrate partly from the bottom of the anterior fissure into the central part of the spinal cord—these supply the grey matter except one large area of the posterior horns—partly from the periphery into the white matter and the posterior horns. The anterior spinal arterial system gives off at all levels branches which pass into the anterior fissure of the cord as " arteriæ sulci " or " fissuræ anterioris," and reach the anterior commissure. Here these arteries divide (*Adamkiewicz*) or curve without division (*Kadyi* [1]) to right or left as *arteria sulco-commissuralis*, and pass to the anterior horn where, by dividing into ascending and descending branches, they supply the greater portion of the anterior horn and also a small part of the adjacent white matter. A branch reaches as far as *Clarke's* column. Lateral branches, also, pass off from the anterior system to form an anastomotic chain in the neighbourhood of the anterior roots—the tractus arteriosus anterolateralis (*cf.* Fig. 77).

In addition to the central arteries, vessels pass radially from the periphery into the spinal cord substance—the *vasocorona*, which is formed by the anterolateral, the posterolateral, and posterior systems. This sends branches into the white matter, an " arteria cornu posterioris," to the posterior horn, etc. Although the white and the grey matter are not supplied by two independent vessel-systems, yet there are areas, *e.g.* the inner portion of the grey matter, which are supplied exclusively by central arteries, and other areas, such as the peripheral area of the white matter, which are supplied solely by the vasocorona.

Both the arteriæ sulci, and all the small branches of the vasocorona which penetrate the spinal-cord substance, are terminal arteries (*Adamkiewicz, Hoche*), while on the surface of the cord the vessels anastomose freely.

The veins of the spinal cord have an arrangement similar to that of the arteries, but on the surface of the cord they are much larger than the arteries. We may refrain from a detailed description.

Classification of Diseases of the Spinal Cord

Diseases of the spinal cord are limited to individual columns or tracts, or to a portion of the grey matter, with a definite physiological function —the column and system diseases; or they involve the whole cord—diffuse diseases.

There are, in addition, other diseased conditions which are referred to the spinal cord, the pathological nature of which has not as yet been ascertained.

COLUMN AND SYSTEM DISEASES OF THE SPINAL CORD

Tabes Dorsalis, Locomotor Ataxia

(German: *Rückenmarksschwindsucht*)

Tabes dorsalis, owing to its frequency, takes the first place among the diseases of the spinal cord. It is specially common in large cities. It occurs principally during middle age, and oftener in men than in women. There can, however, no longer be any doubt that it may begin in childhood (Tabes infantilis and juvenilis).

Cases of this kind have been described by B. Remak, Kellog, Gombault-Mallet, Bloch, Raymond, Dydynski, Halban, Rad, Idelsohn, Brasch, Kaufmann, Linzer, Collins, Hirtz-Lemaire, Fischler, and I myself have seen about a dozen, some of which have been described by O. Maas and Hagelstamm (*Z. f. N.*, xxvi.).

For the pathological changes an important article by Koester, who has collected the cases by the above-named writers, may be consulted (*M. f. P.*, xviii.).

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[1] " Über die Blutgefässe des menschlichen Rückenmarks." Lemberg, 1889. Recently this question has been discussed by *Sterzi* (" Die Blutgefässe des Rückenmarkes." Wiesbaden, 1904).

It is only exceptionally that the disease is acquired at an advanced age.

Causes.—Notwithstanding the objections until quite recently raised against this view, the only fact which I regard as established is that there is a relationship between tabes and syphilis. This has been ascertained statistically (Fournier, Erb [1]).

It is very exceptional that individuals who have not been infected with syphilis become tabetic. But we are by no means justified in regarding syphilis as the *conditio sine qua non* of tabes as Brissaud, Babinski and others do. Tabes is, further, not a syphilitic disease in the pathologico-anatomical sense; the pathological changes have nothing in common with the well-known lesions of visceral syphilis (although Erb does not admit this distinction). It is nevertheless conceivable that as a result of the syphilitic cachexia, poisons (chemical substances) are produced in the organism which bring about a simple degeneration in certain portions of the nervous system.

The observations made in the last few years by cyto-diagnosis, *e.g.* by the discovery of lymphocytosis (see below), are specially adapted to support the doctrine of the syphilitic origin of tabes.

The discovery by Schaudinn and Hoffmann of the spirochæta pallida, which is regarded as the infective agent in syphilis, has also given an impulse to corresponding investigations in tabes, hitherto with negative results (Marinesco, *Semaine méd.*, 1906).

On the other hand the evidence obtained by the use of Wassermann's sero-diagnostic method appears to place the doctrine of the syphilitic origin of tabes on a sure foundation (S. Plaut,[2] Citron,[3] and others), since it has been shown that the serum of tabetics almost invariably contains the antibodies of syphilis.

It is possible that the disease may be produced by other poisons.

Tuczek, whose observations have been confirmed by Jahrmärker (*A. f. P.*, xxxv.), has shown that ergotin can produce a disease with symptoms and pathological lesions similar to those of tabes, although non-progressive in character. The experimental researches of Mosse and Rothmann, as well as those of Spielmeyer (*M. m. W.*, 1906) also deserve to be mentioned here. Some cases appear to indicate that there may be a relationship between chronic lead-poisoning and tabes.

Tabes usually follows the syphilitic infection at an interval of from five to fifteen years, seldom less or more. The symptoms of the syphilis have usually not been severe, indeed, often all that can be made out is, that there has been a venereal ulcer, but the history is not sufficient to make its nature clear. Hitzig has suggested that the poison of tabes may also be conveyed along with the soft chancre.

I know of several cases in which tabes developed during adult life in men who had no knowledge of any syphilitic infection, but whose fathers had suffered from syphilis (or even from tabes). Erb, Fournier, S. Kalischer, Babinski, and Nonne describe similar cases. Infantile and juvenile tabes may also be often attributed to hereditary syphilis, yet in a number of our cases I have failed to find any grounds for this explanation. The occurrence of tabes in married couples (conjugal tabes),

[1] Among the newest works on this subject that of Fischler, "Über die syphilogenen Erkrankungen des zentralen Nervensystems," etc. (*Z. f. N.*, xxviii.), deserves mention. See further, Erb (*Z. f. N.*, xxxiii.).

[2] *M. m. W.*, 1907.

[3] *D. m. W.*, 1907. For details and technique, see Plaut, *M. f. P.*, xxii., also Fornet, *M. m. W.*, 1908.

which has now been established in a large number of cases, can be best explained by the assumption of a syphilitic origin.

Those suffering from tabes seldom present any signs of constitutional syphilis.[1] In female patients the information furnished by the history of the case is usually negative.

Other causes of tabes are not known. It is beyond doubt, however, that the predisposition to this disease is increased by hereditary taint, the importance of which was long ago emphasised by Charcot, and has been recently pointed out by Bittorf. Exposure to cold, bodily over-fatigue,[2] injuries, sexual excesses, alcoholism, are to be looked upon as contributory causes.

With regard to a traumatic origin, I have been able to demonstrate in the majority of cases in which an injury was alleged to be the cause that the tabetic symptoms had already existed before the injury, and that they had been preceded by syphilitic infection. Without doubt, however, injuries may materially hasten the progress of the disease, and may so determine its course that the injured part of the body may become affected in a special degree by the tabetic symptoms.

I have repeatedly stated that this is my experience. I will mention only the following out of many examples: A workman, in the initial stage of tabes, whose arms had been hitherto quite normal, suffered from a severe crush of the left hand, and within a few months thereafter there developed a high degree of anæsthesia and ataxia of the left arm. In a man suffering from cervical tabes (see below), whose limbs had been hitherto free, there developed after a fall on the back an anæsthesia, ataxia, and hypotonia of the legs with disappearance of the tendon-reflexes.

Hitzig, "Über traumatische Tabes," Berlin, 1894, Klemperer, and others have thoroughly investigated this question.

Leyden and others consider that trauma is one of the direct causes of the disease.

Symptomatology.—The symptoms and signs of tabes are extremely numerous, yet there are a certain number which recur in almost every case, and which may be regarded as the essential and initial signs of the disease. In the early stages they may constitute its only manifestation. In the later stages they always afford the surest ground for the diagnosis; but I should like to state at this stage that no sign of tabes is so constant that its absence should necessarily invalidate the diagnosis. Although the grouping of the symptoms differs greatly in the various cases, and in one case a sign of disease may appear in full development, at the very beginning, which in another case appears only at a later stage, we are nevertheless justified in considering the initial stage by itself, and then in describing the disease as it may appear when fully established.

The *initial stage* is specially indicated by the following symptoms: 1. *Westphal's sign*, that is the *loss of the knee jerk*; 2. *Reflex immobility of the pupil* (*Argyll Robertson's sign*); 3. *Lancinating pains*; 4. *Analgesia of the lower extremities and diminution of tactile sensibility of the trunk.*

Westphal's sign may appear years before the other symptoms, and is one of the most frequent signs of the initial stage. It is sometimes present on one side only, whilst the knee jerk can still be distinctly or somewhat

[1] See, however, the cases collected by Adrian in *Z. f. k. M.*, lv.

[2] Edinger has produced experimentally degeneration of the posterior columns of animals, by over-fatigue. With regard to his exhaustion-theory, see *D. m. W.*, 1904, and Bing (*Z. f. N.*, xxvi.).

feebly elicited on the other leg. It should, however, be noted that there are also not a few cases in which this sign is absent, although the nature of the disease has been rendered certain by many other signs.

It had been noted even by early authorities (Erb, Leimbach, and others) that the *Achilles tendon reflex* might also disappear in tabes. The full significance of this fact, however, was not recognised until examination by Babinski's method (p. 8) showed that the Achilles jerk might be regarded as being practically constant in healthy persons. We may now agree with Babinski, Goldflam, Sarbó, Kollarits, and others in regarding absence of this reflex as one of the early symptoms of tabes, admitting that it is frequently absent while the knee jerk is still conserved. We should, however, guard against putting too great a value upon this sign in the diagnosis of tabes, as there are many other affections (sciatica, for instance, and those mentioned on p. 11) in which this reflex may be abolished.

Immobility of the pupils[1] competes for the first place with Westphal's sign, in so far that it may precede the fully developed disease for a still longer time, and is often the first definite premonitory symptom. Indeed, I have seen cases in which, for ten or fifteen years, it constituted the only objective sign of tabes. On the other hand it is not of constant occurrence, and is often absent at the commencement or during the later course of the disease. Sometimes it is distinctly present in one eye only. Complete loss of the reflex is sometimes preceded by a weakening, or by a slowing of the reaction.[2] The pupils are often not perfectly circular and may be irregular in outline. In isolated cases the reflex immobility of the pupils has been found to be intermittent (Eichhorst, Treupel, Mantoux), *i.e.* it may be present only for a time, and then disappear. As a rule the contraction of the pupil during convergence takes place in the normal way, in spite of the loss of the light reflex, but there are exceptions to this.

The *lightning pains* would also be considered an equally important sign, were it not that the symptom is a subjective one, and that in judging of it we usually have to depend practically entirely upon the statements of the patient. The pains are characterised by *their severity*, by the *suddenness of their onset*, and by the *lightning-like rapidity of each separate pain*. The length of the intervals between the attacks of pain may vary from days or weeks to months. The attacks themselves usually last only for some hours, but sometimes they are of so short a duration that the patient disregards them, and only recalls after special questioning that he has now and again suffered from "twinges," "rheumatism," etc. They may, however, persist for days and weeks. They have their seat in various places, most frequently in the lower extremities. They may appear also in the arms, the trunk, the area of the fifth nerve, in the testicle, bladder, anus, etc. Sometimes the pain is like a short wrench, sometimes like a "flash of lightning," which darts through the whole limb or a large part of it. The direction varies. The patient usually thinks he feels the pain deeply "in the firm flesh," or in the bones. But there is another form of the pain in which it is limited to a circumscribed point on the skin, boring into it, as it were, and usually accompanied by a marked hyperæsthesia of the skin, so that even the pressure of the bed-clothes is painful.

[1] Consult the literature in Bumke, "Die Pupillenstörungen bei Geistes- und Nervenkrankheiten." Jena, 1904.

[2] Kutner thinks he has observed an abnormal fatigability of the light reaction of the pupils in tabes.

In this condition a firm grasp is better borne than a light touch. In the later stages the lightning pains may be accompanied by others of a slow, dull, boring character. If the paroxysms are severe, they exhaust the patient and exert an unfavourable influence on his general condition. In isolated cases the attacks of pain are accompanied by pyrexia.[1]

Pal (*M. m. W.*, 1903, and "Gefässkrisen," ref. *N. C.*, 1906) found an increase of the blood pressure. Subcutaneous hæmorrhages and local œdema may also occur along with the lightning pains (Strauss, Oppenheim). It is unusual that symptoms of motor irritation amounting to clonic spasms should be present in these attacks or in combination with the paræsthesiæ We have seen (*A. f. P.*, xviii., Beob. iv.) and Foerster also has described such cases.

Dejerine observed in one case a transient paraparesis (from exhaustion) which developed at the height of such an attack.

Girdle-pains frequently occur. There is sometimes a painful sensation in the anus, "as if a wedge were being driven in."

The lightning pains occur in almost every case. There are some in which they do so only at long intervals, and in which they are very transient, but it is only in exceptional cases that they are entirely absent. They may, on the other hand, precede the onset of the other symptoms by ten to thirty years.

Analgesia is another sign of the disease, which may be discovered in the majority of cases in their earliest stages. It is usually found in the lower extremities. Although the sensibility in general is still unaffected, this peculiar symptom may occur, so that the prick of a pin produces little or no feeling of pain. If we pinch up a fold of skin and pass the needle through it, the patient feels that it has been touched or pressed, that a needle has been inserted, but the sensation is not painful. One of my patients had accidentally fastened himself to the bedcover by a needle through a fold of skin on the thigh, and he only noticed it by chance some hours later. Analgesia is, however, a less constant symptom than the others already mentioned. On the other hand, recent observations (Hitzig and Laehr,[2] confirmed by Partick, Marinesco, Burr, Grebner, Muskens, and by cases of my own) have shown that, even in the initial stage of the disease, there is very frequently a zone on the trunk, particularly at the level of the mammæ or from the third to the sixth dorsal nerves, in which tactile sensibility is diminished or abolished. At first, this anæsthesia has a quite circumscribed, patchy distribution, which later becomes girdle-form (radicular). Analgesia is very frequently found in this area, but the diminution of tactile sensibility usually precedes it.

Frey's investigations show that the threshold of stimulation of sensation is particularly high at and around the nipple, so that this fact should be taken into consideration in doubtful cases. The pathological character of the hypæsthesia of tabes can usually be definitely recognised from its degree and from the fact that its extent corresponds with the distribution of one or more nerve roots (radicular distribution), and also from the fact that the zone usually lies below the nipple, or extends above and below this area.

A diminution of the sense of vibration (see p. 50) not infrequently occurs even in the early stages of the disease, especially on the leg.

Swaying when the eyes are shut (Romberg's sign) may also be included among the symptoms of the initial stage, although it does not usually

[1] I have observed this, and so has Goldflam (*N. C.*, 1902). Attacks of pyrexia during the course of tabes have also been noted (J. Hoffmann), and have been explained as "crises fébriles" (Pel, *B. k. W.*, 1899).

[2] *A. f. P.*, xxvii.

appear so early as those already mentioned. If the patient stands first with his feet together and the eyes open, a swaying movement may be seen, which becomes much more marked when the eyes are closed. A very slight swaying movement occasionally occurs even in healthy persons, especially in those of an anxious temperament, but it usually disappears when the attention is diverted. The symptom becomes much more marked if the patient is asked to bend down and then to raise himself up again, his eyes being closed. The test, which I modify in this way, may be of diagnostic value in doubtful cases.

Along with these symptoms, others may appear and become so prominent, even at the commencement of the disease, that the patient's attention is directed to his illness mainly by them. I shall first mention and then analyse them. *Difficulty in passing urine, lessening of sexual power*, in many cases *oculo-motor paralysis*, in not a few *gradual failure of sight* may be the earliest symptoms. *Gastric disturbances, spasms of coughing, paralysis of the vocal cords, affections of the bones and joints*, etc., etc.—all these symptoms may become evident in the early stages, and may be the outstanding feature of the disease. Although commencing tabes may in this way simulate ocular, abdominal, laryngeal, articular, or bladder disease, yet in almost every case the symptoms have so definite a character that they either indicate, or at least suggest the real nature of the disease. This can then be confirmed by the presence of some or all of its early symptoms.

The disease attains its full development with the evolution of the *ataxia*. The motor disturbance in tabes—in so far as it concerns the extremities at least—is not a motor weakness, but an *inco-ordination*. Whilst individual movements of the legs and arms can be carried out in their full extent and with normal power, a severe lesion exists on account of which the movement is executed, not in the shortest way, but with repeated diversions and with an irregular and excessive expenditure of motor energy.

The main points as to the nature of the ataxia have been discussed on p. 27. The ataxia of tabes is almost always associated with affections of conscious sensation, that is of sensibility in the ordinary sense, but it depends mainly on the absence of centripetal impulses that are not concerned in conscious sensation. The oculo-motor paralysis and other motor paralyses in tabes are sometimes ascribed to the absence of such centripetal impulses as Feilchenfeld and Mirallié-Desclaus have recently tried to show, but this theory does not seem to us to be defensible. Compare also Lapinsky's interesting investigations (*A. f. P.*, 42).

This inco-ordination in the majority of cases affects the muscles of the lower extremities first, and gives the patient a feeling of uncertainty in walking (especially in the dark), and in going down stairs. It can frequently be observed by others before the patient is aware that there is anything amiss. It is of special importance to note that the ataxia becomes exaggerated in tabes when the control of the eyes is excluded. The tabetic superintends each movement with his eyes, and thus materially limits the degree of inco-ordination. If we desire to appreciate this in its full significance the same movements should be executed with the eyes closed.

The ataxia of the lower limbs produces a most *characteristic gait*, which is almost pathognomonic of tabes. In its highest development it is distinguished by the following features : the patient raises his advancing leg too far by over-flexion and external rotation in the hip-joint, then throws

it heavily down, with an excessive extension of the knee and ankle, so that he brings the foot to the ground with a stamp of the heel, and then puts abnormal pressure upon the knee of the other leg. He usually walks rapidly with uneven steps, fixes his eyes steadily on the ground, and is inclined to fall as soon as he looks away. It sometimes happens that his knees suddenly give way and he falls down.

The ataxia may be apparent when the patient is lying on his back, even although it has not yet become visible in his gait. The contrary may also be the case. Slighter degrees of the ataxic gait can be recognised, especially by the stamp with which the heel is brought to the ground. The movement of turning is uncertain, and the patient easily tumbles. The way in which he sits down and rises up is also characteristically affected by the ataxia, as has been well shown in O. Foerster's [1] masterly investigations.

The ataxia often appears first in the upper extremities. It is mainly the fine, complicated movements of the hands, such as writing, fastening the clothes, etc., which are carried out with difficulty. In such cases other symptoms—pains, paræsthesiæ, and diminution of the sensibility usually affect the upper extremities, either beginning in or remaining limited to them (cervical tabes).

A peculiar symptom can be often seen along with the ataxia, especially when it affects the arms: the patient cannot keep them quite steady, even when they are supported. One or more fingers are slowly extended, then flexed, or abducted and adducted, or the whole hand is raised; in severe cases the arm itself may be raised from the bed and put into the vertical position, the patient having no feeling of the movement. I have described this symptom as "*spontaneous movements*"; [2] but they cannot be sharply distinguished from static ataxia. They have merely a superficial resemblance to athetosis, and should not be confounded with it.

Abnormal associated movements may also occur in parts of the body other than those which are being moved voluntarily (Oppenheim,[3] Stintzing [4]).

The ataxia may in very rare cases involve the muscles of the face and tongue, and may then affect the speech in a peculiar way, owing to excessive movement of the muscles of the lips, tongue, and jaws. The muscles of deglutition may be involved in exceptional cases.

Ataxia is by no means one of the early symptoms of tabes. By the time it is developed, a number of other symptoms can always be made out. It is accompanied by atony of the muscles, and during the later stages of the disease there is also a flaccidity of the ligamentous and capsular apparatus, so that passive movement is extremely easy.[5] Compare Fig. 1, p. 11. Hypotonia may be apparent even in the early stage of the disease.

[1] "Die Physiologie und Pathologie der Koordination." Jena, 1902.

[2] Compare Oppenheim and Siemerling, "Beiträge zur Path. der Tabes dors.," etc., *A. f. P.*, xviii.

[3] *B. k. W.*, 1884.

[4] *C. f. N.*, 1886.

[5] This symptom has long been recognised, and indeed Westphal ascribed the disappearance of the tendon-phenomena directly to it. Westphal allowed no session to pass without demonstrating to his students to what an excessive degree passive movements might be carried out in advanced tabes. It is true that this symptom first attracted general attention after Frenkel's work, but one is not justified all the same in associating an already long known symptom with the name of this writer. Orschansky (*N. C.*, 1906) draws attention to the fact that the heel of the leg which lies extended can be elevated to a greater or less height from the bed, and he regards this over-extension of the knee as an important evidence of the hypotonia of tabes.

The symptoms now to be discussed may develop at any stage of the illness. We shall first take those which appear with great regularity during the advanced stages, and then the less frequent symptoms.

Of the disturbances of sensation, pain has already been considered. Paræsthesiæ of various kinds are almost constantly complained of: a feeling of tingling, numbness, formication, etc., at the extremities, in the region of the thorax and abdomen, and especially a feeling as if the soles of the feet were covered with india-rubber, felt, skins, etc., as if patient were sitting on an india-rubber cushion, as if the trunk were girt in with a belt, a strap, or an iron band, etc. A *painful sensation of cold* is very frequently complained of, especially in the legs. A paræsthesia of cold in the form of a girdle also occurs.

Whilst the paræsthesiæ are not associated with the area of any particular nerve in the lower extremities, in the upper they mainly and usually first affect the areas of the skin supplied by the *ulnar nerve*, or by the nerves arising from the eighth cervical and the first dorsal root, especially on the fourth and fifth fingers.

Amongst the objective affections of sensibility, the hypæsthesia on the trunk and the analgesia in the legs, already mentioned, appear to be the earliest symptoms. A *persistent hyperæsthesia*, in which even the slightest touch will produce a distressing sensation of pain, especially on the trunk, is much less common.

I have already drawn attention to this symptom in the second edition, and have frequently observed it since then. The patients thus affected are much distressed by the fact that the contact of the skin with the shirt or undervest upon the thorax is extremely painful. (Dejerine also mentions this in his *Semiologie*.) Others (Riche-Gothard) have found hyperæsthesia especially with regard to the temperature sense, but the sensitiveness to thermal stimuli in the thorax is often very marked, even in healthy individuals, so that careful control examinations are required in determining this condition.

The temperature sense is frequently conserved, even to the latest stages, and tactile sensation may also remain unaffected for a considerable time.

The diminution of sensibility soon affects a diffuse distribution, being found over the whole lower limb or on both, though it is more marked in places. The anæsthesia or hyperæsthesia is more often found in circumscribed parts of the skin, such as on the external surface of the thigh, the inner border of the foot, the sole of the foot, etc. There may even be small spots in an otherwise sensitive area which show a dulness of sensation, so that of two pin-pricks applied close to each other, one will be felt as painful, the other as painless. On the whole, however, recent investigations (Laehr, Dejerine, and others) show that the areas of the anæsthesia correspond to the cutaneous distribution of nerve roots (*cf.* p. 126).

It appears from Marinesco, Frenkel-Foerster, and especially from Grebner, that in the lower limbs it corresponds as a rule to the area of the first sacral and fifth lumbar nerves.

Pain conduction is often retarded, a painful stimulation being felt only after an interval of two to five seconds. The double-sensation described by Naunyn and Remak [1] is closely related to this symptom. When this is present the prick of a needle at first produces a sensation of contact, and then, after an appreciable interval, a sensation of pain. In a few cases, I have noted that a single prick of a needle provoked a sensation as if the

[1] *A. f. P.*, vii. See also B. Stern, *A. f. P.*, xvii.

skin had been affected simultaneously or after a short interval by pricks at two neighbouring points.

Another phenomenon deserves mention, viz., that at areas on the skin at which the prick of a needle is not perceived as painful, a scratch with the needle will occasionally cause severe and prolonged pain.

This has been established by earlier observations (my own and others), and has been experimentally verified recently by Egger, and attributed to a summation of stimulations.

The *sensibility of the deep parts* is usually involved at a comparatively late stage, and there is practically always some diminution, where ataxia has developed, although this stands in no definite relation to the ataxia. In advanced cases of tabes the patient as a rule has no idea of the position of his limbs, and does not know whether they are in bed or out of it. The sense of pain of the deep parts may be diminished even in the initial stages of the disease. I found this condition in some cases of tabetic joint disease, in which the cutaneous sensibility was still quite normal, and movements of the affected joint were absolutely painless. Surgical operations on tabetic joints may frequently be carried out without anæsthesia (Bier). The "sensation of vibration," or "pallæsthesia," is often diminished at an early stage (Egger, Seiffer-Rydel, Oppenheim), but no definite rule can be established with regard to this. The analgesia also may extend, especially in the later stages, to the inner or deep-lying organs, so that pressure on the trachea, the abdomen, the testicle, the toes, muscles, etc., often excites no pain (Pitres, Dejerine, Sicard, Abadie, Racine, Bechterew). The nerve trunks are often insensitive to pressure (Biernacki).

The *excitability of the cutaneous reflexes* undergoes no material change in tabes. It is worthy of note, however, that the cutaneous reflexes, such as the plantar and abdominal, are frequently exaggerated at the commencement, and may be very active, even when there is anæsthesia and complete loss of the tendon reflexes. I have found that the abdominal reflex was usually exaggerated in the early stages. The plantar reflex always conforms to the normal type of plantar flexion.

The *function of the bladder* is interfered with in almost every case, and it may be at any stage. The first sign of this is usually a difficulty in emptying the bladder. The patient is obliged to use stronger and more prolonged pressure before the urine passes. At other times he finds with a certain satisfaction that he can retain his urine for a very long time, the desire to micturate being felt only once or twice in the day. This also indicates a pathological condition. Indeed, it seems to me usually to precede the difficulty in passing urine. Severe bladder troubles, such as complete incontinence or retention, usually appear only in the later stages, and they may, in spite of the progressive character of the disease, again disappear.

Incontinence of the bowels is very rare. Constipation is usual, and a distressing tenesmus may be present in rare cases.

Impotence is sometimes an early symptom, but sexual power may be retained for a considerable time. *Satyriasis* may also appear at the commencement of the disease. In one of my cases *priapism*, lasting for weeks and not accompanied by voluptuous sensation, making the passing of urine very painful, was the first trouble. It was followed by absolute impotence. In another case there were nocturnal attacks of priapism

with incontenence of urine. Persistent priapism is also described by Pitres and Raichline. Clitoridean crises in tabetic women are sometimes present in the form of occasional paroxysms of voluptuous sensation with vulvo-vaginal secretion (Pitres, Köster).

Let us now consider the *symptoms produced by affections of the cranial nerves*. The most frequent of these is *oculo-motor paralysis*.[1] Sometimes the abducens, sometimes one or more branches of the oculo-motor nerve are paralysed. Thus ptosis is very frequent, although it does not always depend upon paralysis, but may be caused by atony of the levator and may then be transient and inconstant. In such cases the eyelid droops very low at times, but may be completely raised by voluntary effort. Paralysis of accommodation may occur, and may even be one of the earliest signs. The oculo-motor paralysis and the subsequent diplopia are usually temporary, and may disappear and return. A persistent oculo-motor paralysis may, indeed, appear quite at the commencement, but it is more usually observed during the later stages of the disease. The paralysis is never an associated one, but is irregular and involves various muscles of one or both eyes. A complete *ophthalmoplegia* may even occur, but *isolated, complete ophthalmoplegia interna* is an exceptional occurrence in tabes (Uhtoff). The pupils are frequently abnormally narrowed, even in the early stage. Unless the condition is very marked, it should not be regarded as being due to disease. Mydriasis is rarer, inequalities of the pupils more frequent, and the symptom of "springing" pupils appears in isolated cases (see p. 85).

In many cases of tabes the optic nerve is involved. The mode of this involvement is typical. It consists almost constantly of a *bilateral, simple progressive atrophy of the optic nerve*, which usually leads to complete blindness. Although one eye is as a rule affected more severely and sooner than the other, yet the atrophy is hardly ever permanently confined to one side. The ophthalmoscopic picture is characteristic.

The affection of the sight is usually an irregular, concentric, or sector-shaped reduction of the field of vision, which at an early stage extends also to colours (red and green). On the other hand, central scotoma is very exceptional and should be ascribed to a complication, or to some special cause (alcoholism, lead-poisoning, etc.). At least in the cases under my own observation I have been usually, though not always, able to demonstrate this.

During recent years Marie and Léri [2] especially have been occupied with the study of tabetic optic atrophy.

The atrophy of the optic nerves commences as a rule at an early period. It may for a long time be the only sign of tabes. It is only in exceptional cases that it develops during the late stages of the disease.

The *fifth nerve* is often involved. Not only is it the site of pain and paræsthesiæ (tingling, feeling of numbness or as if the face was covered with a mask or the teeth pressed against each other), but often also of anæsthesia or hypæsthesia, particularly for painful stimuli.

The analgesia may be limited to the mucous membrane of the mouth and jaw. In exceptional cases the anæsthesia affects the cornea. Occasionally also there are trophic disturbances, amongst which the *spontaneous falling out of the teeth* (Demange, Oppenheim,[3] and others) is the most

[1] An exhaustive discussion and the literature on the question are to be found in Uhtoff, "Die Augenveränderungen bei den Erkrank. d. Nervensyst." Graefe-Saemisch Handbuch, 2. Aufl., Bd. xi., Kap. 22, 1904.

[2] *Thèse de Paris*, 1904; *R. n.*, 1904 and 1905, etc.

[3] *A. f. P.*, xx.

common. The patient notices that the teeth become loose, and within the space of a few days or weeks he can, without pain or bleeding, extract a great number with his fingers. The teeth themselves may be quite normal. Sometimes small sequestra from the jaws also drop off. The jaws atrophy after the teeth have fallen out. In a case described by Sabrazès-Fouquet,[1] an attempt to extract the teeth led to fracture of both jaws. Trophic lesions of other kinds (neuroparalytic keratitis, ulcerations of the mucous membrane of the mouth, formation of abscesses in the face, herpes, facial hemiatrophy) have been rarely observed by ourselves and others. Diminution of taste on the corresponding anterior half of the tongue is rare. One of my patients complained that he had the sensation of taste only for the first moment of eating, and then was obliged to eat his food without tasting it. Another had the sensation of taste only during the act of swallowing. Disagreeable sensations of taste and smell are mentioned by Erben and Umber. Constant dropping of tears, which sometimes occurs in tabetics, is an affection of the fifth nerve which should be mentioned.

There have also been described (Pel, Haskovec) by the name of "eye-crises," attacks of severe pain in the eyes, with dropping of tears, violent contractions of the orbicularis and hyperæsthesia of the eye and its neighbourhood. We must, however, wait for further experience before we include these attacks amongst the symptoms of tabes. I have frequently found a hyperæsthesia of the retina, which made examination of the light reflex exceedingly difficult, as every attempt caused the eyes to fill with tears.

It is only in very exceptional cases that symptoms appear which point to involvement of the *motor fifth nerve* : viz., *weakness*, and atrophy of the muscles of mastication (Schultze,[2] Marie-Léri [3]). Affections of hearing are in a few cases ascribed to a lesion of the acoustic nerve, but the clinical and pathological proofs of this are still scanty. According to Pierre Bonnier,[4] many of the symptoms of tabes are due to involvement of the nerves of the labyrinth, which gives rise not only to auditory affections but also to vertigo and disturbances of equilibration. Affection of taste and smell is specially mentioned by Klippel [5] and Jullian,[6] but this is rare in my experience.

A short time ago I was consulted by a chemist, in whom anosmia and partial loss of taste constituted the first signs of tabes. He stated that he found this specially troublesome as he was very dependent upon his olfactory nerves in his occupation. This loss may be explained on Edinger's exhaustion-theory.

A number of symptoms which are due to lesions of the vago-accessory nerve are worthy of note. To these belong the habitual acceleration of the pulse rate, which is found in many cases of tabes, often at an early period. The patient is himself sometimes conscious of it. Other uncommon conditions are described as cardiac crises, in which there occurs severe pain in the region of the heart—radiating from here towards the shoulders, especially towards the left—accompanied by a feeling of oppression and an acceleration and irregularity of the heart's action during the attack. The condition has a resemblance to angina pectoris. Tachypnœa may also be included among the symptoms. A marked bradypnœa was noted in one case (Egger) and was ascribed to bilateral paralysis of the vagus. Pal [7] speaks of vascular crises.

Gastric crises (Topinard, Delamarre) or spasmodic attacks of vomiting

[1] *Nouv. Icon.*, 1900. [2] *A. f. P.*, xxi. [3] *R. n.*, 1905.
[4] *Nouv. Icon.*, 1899. [5] *Arch. de Neurol.*, 1897. [6] *Thèse de Paris*, 1900.
[7] *M. m. W.*, 1903, and "Gefässkrisen," Leipzig, 1905.

are much more common than the symptoms described above. They usually have a typical course. The attack comes on suddenly; the patient feels a severe pain in the abdominal region or a painful sensation of constriction; he begins to retch and to vomit violently, at first only the food which he has taken, and later bilious matter. Although all the nourishment which he has absorbed has been rejected, the vomiting as well as the distressing pains still persist. There is great exhaustion, the face is pale and wasted, the urine scanty and concentrated; the pulse may be rapid and irregular, the tongue is usually in a normal condition. After the attack has lasted for hours, days, or even weeks, it suddenly ceases, and the patient can at once resume his ordinary food. Gradual transitions do, however, also occur. The intervals between the attacks vary greatly in duration. There are also incomplete forms (attacks of pain without vomiting, attacks of vomiting without pain, etc.).

I have often, especially in the last few years, observed forms of gastric crises which differ materially from those described as typical. It often happens that the attack develops gradually, and especially that it ceases gradually or that the crisis, apparently ended, re-commences. In several cases a disturbance of the intestinal functions is given as the cause of the relapse or of the attack. For instance, an individual suffering from constipation has used an aperient or an enema, and with the stimulation of the action of the bowels a fresh attack of vomiting has occurred. It is not unusual, however, that diarrhœa may accompany the attack of vomiting from the beginning.

The attacks may also be ushered in by hallucinations of taste and smell (Umber). Roux (*Thèse de Paris*, 1900) mentions a form of gastric crisis in which digestive derangement played an essential part, and errors of diet brought on the various attacks.

In one of my cases the attacks always commenced with a pain in the nape of the neck, in another with pain in the "region of the bladder."

Hæmatemesis occurs in exceptional cases during the gastric crises (Vulpian, Charcot, Oppenheim, Noorden, Eckert,[1] Rubin, Neumann).

It is also of interest that in a few cases an anæsthesia (Egger, Heitz, and Lortat-Jacob) or a hyperæsthesia of the trunk (Foerster) could be found only during the attack. It has also been observed that immobility of the pupils was absolute only during the crises, or was only present then (Mantoux).

Simple anorexia, or a complete loss of appetite, may be a symptom of tabes. In one case which I treated there was also *glycosuria*, and there also developed symptoms of diabetes mellitus, which were, however, only transient. Similar observations are reported by Reumont, E. Meyer, and others.

Laryngeal crises, spasmodic attacks of coughing (Féréol) are much less frequent than gastric crises. There is a sudden feeling of suffocation, with long-drawn, inspiratory stridor lasting from a few seconds to some minutes, in which the breathlessness and cyanosis is considerable. These attacks occur without any recognisable cause, or are brought on by choking. In one of our [2] cases spasms of sneezing were associated with the attacks of coughing. French authors (Klippel, Sullivan, Jullian) have recently described such spasms of sneezing as "nasal crises." Laryngoscopic examination usually shows nothing abnormal. *Paralysis of the laryngeal muscles* can, however, hardly be termed an unusual symptom of tabes.[3] The most frequent form is *paralysis of the crico-*

[1] "Inaug. Diss.," Berlin, 1887.

[2] *B. k. W.*, 1885.

[3] Observations by Schnitzler, Rosenthal, Krishaber-Charcot, Oppenheim, Semon, Landgraf, Fraenkel, Schüller, Green, Faure, and especially in the collected statistics and monographs of Krause, Semon, Burger, Sendziak, and Dorendorf. See the earlier literature in my work, "Über Vagus-Erkrankung im Verlauf der Tabes dors.," *B. k. W.*, 1885, and "Neue Beitr. zur Path. d. Tab.," *A. f. P.*, xx.; further, more recent work in Sendziak, "Klin. Vorträge aus d. Geb. d. Otol.," etc., iv., 1901.

arytænoidei postici, the abductors of the vocal cords. If it is not complete, it does not cause any marked subjective troubles, but dyspnœa is usually complained of, and even in quiet breathing a more or less marked stridor may be perceived. The adductors may also be affected. Complete paralysis of one vocal cord has even been observed. I have found that in cases of this kind the electrical excitability of the recurrent laryngeal was abolished, and that on the neck between the larynx and the sterno-mastoid a spot could sometimes be discovered where slight pressure caused pain, and stronger pressure sometimes produced an attack of coughing. In isolated cases paralysis of the palate is observed, and more rarely symptoms of paralysis of the muscles supplied by the spinal accessory (Martius,[1] Avdakoff, Seiffer, Ilberg[2]). I have recently seen a case in which the tabes was completely masked by a unilateral total paralysis of the vagus and accessory. Unilateral atrophy of the tongue (Ballet, Westphal, Koch[3]) is an uncommon symptom. (See Fig. 78.) Unilateral paralysis of the phrenic nerve is mentioned only in isolated cases (Gerhardt,[4] Levy-Dorn).

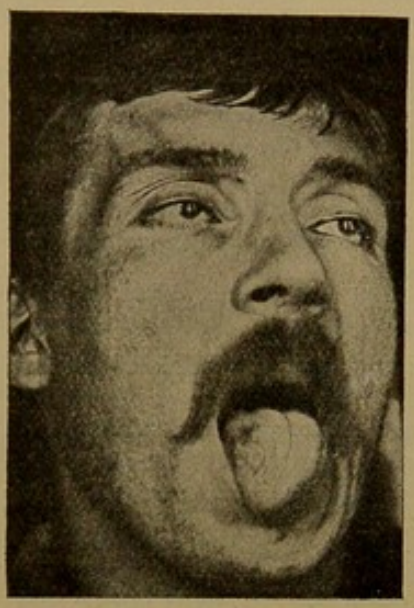

Fig. 78.—Hemiatrophy of the tongue in tabes. (Oppenheim.)

In a few cases (Oppenheim, Eisenlohr, Howard, Jacobsohn, Bloch, Cassirer-Schiff,[5] Spiller) the symptoms described were so marked in the larynx, the muscles of the palate and throat, that one might have spoken of the "bulbar-paralytic form of tabes." Salivation also was present, whilst in one of my cases swallowing was difficult because of the absence of the secretion of saliva during mastication.

I have described a rare form of crisis under the name of *pharyngeal crises*.[6] These are attacks in which violent movements of deglutition accompanied by a gurgling, gulping noise, follow each other in rapid succession. The attack lasts from several minutes to half an hour. There may be twenty-four swallowing movements in the minute. The paroxysms are usually produced by a deep pressure at the side of the upper part of the larynx. Courmont[7] has subsequently described spasmodic symptoms of other kinds in the pharynx, and Bechterew also speaks of a periodic hiccough. A kind of globus also appears in tabes. Stembo mentions attacks of hiccough.

Attacks of colic with diarrhœa, which may appear even in the beginning of the disease, are described as intestinal crises, and bladder attacks with severe pain in the region of the bladder and kidneys, eventually accompanied with dysuria, as renal crises. Liver crises are also mentioned (A. Jolly, Bernhardt).

O. Foerster (*M. f. P.*, ix.) also speaks of crises of taste and smell, and of crises in the extremities, by which he signifies lightning pains with tonic and clonic muscular spasms. According to him conditions of irritation in the sensory tracts and the accompanying reflex symptoms determine the nature of the crises, and they consequently show three elements: (1) symptoms of sensory irritation appearing spasmodically; (2) spasmodic motor acts (vomiting, coughing, etc.); (3) severe hyperæsthesia of the affected organ.

[1] *B. k. W.*, 1878. [2] "Charité-Annalen," 1893. [3] *Revue de Méd.*, 1888. [4] *B. k. W.*, 1893. [5] *Obersteiner*, 1896. [6] *A. f. P.*, xx. [7] *Revue de Méd.*, 1894.

Trophic Disturbances.—The muscles of the trunk and extremities retain in tabes their normal size and their normal electrical excitability. It occasionally happens that at the beginning or during the course of the disease a paralysis of a peripheral nerve (popliteal, musculo-spiral, etc.) occurs, the onset of which is favoured by abrupt movements of the ataxic patient as well as by his insensitiveness to pressure and to blows, though it may not be entirely explained by these. Cases of this kind are described by Joffroy, Strümpell, Bernhardt, Fischer, Remak, and others. I have seen a considerable number. In one of our patients the peroneal paralysis was the direct result of arthropathy of the knee-joint, in which the nerve was compressed by a piece of bone and was raised up from the underlying structures (Finkelnburg).

Muscular atrophy may also develop in tabes. This is not confined to the distribution of a peripheral nerve, and is distinguished from that above described by its gradual onset. It affects whole groups of muscles, the small hand or foot muscles, for example.

The question of muscular atrophy in tabes has recently been exhaustively discussed by Dejerine and by Lapinsky (*A. f. P.*, Bd. xl. and xlii.).

Localised atrophy of the muscles of the back and abdomen was observed by Dejerine-Leonhardt (*R. n.*, 1905). Gowers showed me recently in his wards a case of unilateral paralysis of the abdominal muscles in a tabetic.

Cases which are characterised by the comparatively rare symptom of muscular atrophy, sometimes show other peculiarities, either etiological or symptomatological. Two of my patients had been the subjects of lead-poisoning; in two other cases the localised muscular atrophy was accompanied by a pronounced general marasmus (Fig. 79). I think I am justified in concluding, mainly from my own experience, that general emaciation may be one of the symptoms of tabes, and that there is a rare form of this disease (the marantic) which is characterised by the early onset of severe general emaciation.

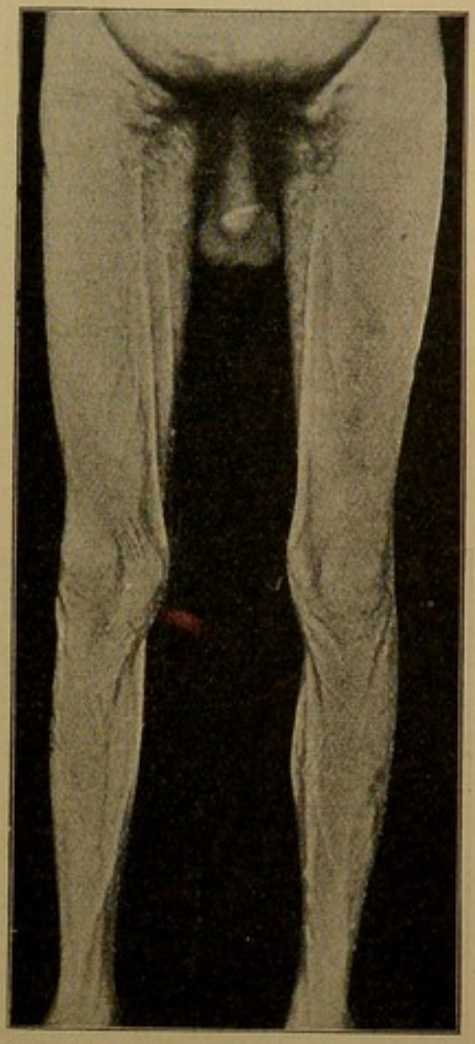

FIG. 79.—Case of tabes dorsalis in which there was marked general emaciation (atrophic tabes). (Oppenheim.)

Trophic disturbances in the joints and bones are by no means unusual. *Tabetic arthropathies* (Charcot [1]) usually appear in the early stages. The knee-joint (of one or both sides) is most frequently affected. The affection generally begins suddenly. The joint swells, and the swelling usually extends beyond the joint, so that an œdema of the whole leg may accompany the exudation in the knee-joint. The accumulation of fluid may be very considerable.[2] There is neither pain, redness, nor fever.

[1] With regard to the literature and characteristics, see the review by S. Adler, "Über tabische Knochen- und Gelenkerkrankungen," *C. f. Gr.*, 1903.

[2] We have also occasionally found and described extravasation of blood in the joints (*A. f. P.*,

Destruction of the ends of the bones follows very rapidly, and on account of this and of the simultaneous atony of the capsule and ligaments, deformities and dislocations occur (Figs. 80 to 86). At the same time there is a new formation of bone, a diffuse and quite irregular protrusion

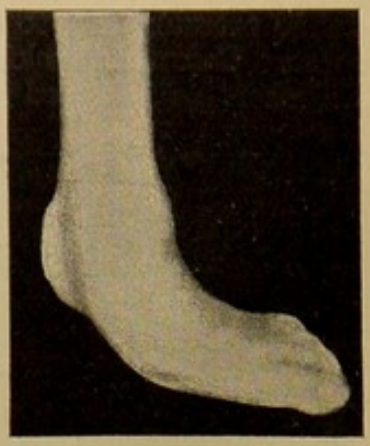

Fig. 80.—Tabetic foot. (Oppenheim.)

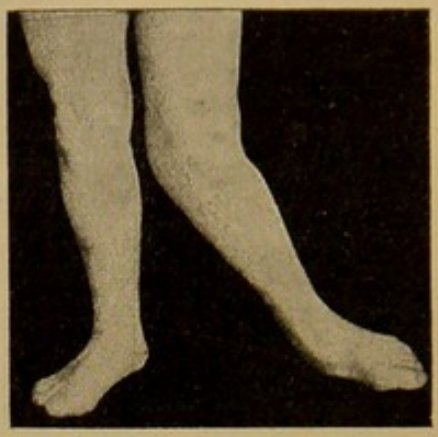

Fig. 81.—Genu inversum in tabetic arthropathy. (Oppenheim.)

of the ends of the joints, with formation of bony excrescences and free bodies. These processes create a condition of the joint which is very closely related to arthritis deformans, but is distinguished from it by the mode of development and the absolute painlessness which is the rule. The peri-articular new formation of bone is also particularly characteristic. The hip-joint may also be affected. I have seen in a patient who was confined to bed, a spontaneous dislocation of the hip-joint, of which he himself was quite unaware. There was in this case an advanced destruction of the hip-joint (Fig. 86). The shoulder (Fig. 84) and elbow-joints, the spinal column (Krönig, Abadie, Spiller, Frank), and the joints of the foot are less frequently affected. In the foot the arthropathy may give rise to a peculiar deformity which is known as the tabetic foot (Charcot, Féré), (Figs. 80 and 81). The patient usually leans on the tarsal articulation, and thus causes a protrusion at the inner border in the region of the cuneiform, whilst the anterior part of the foot turns outwards.

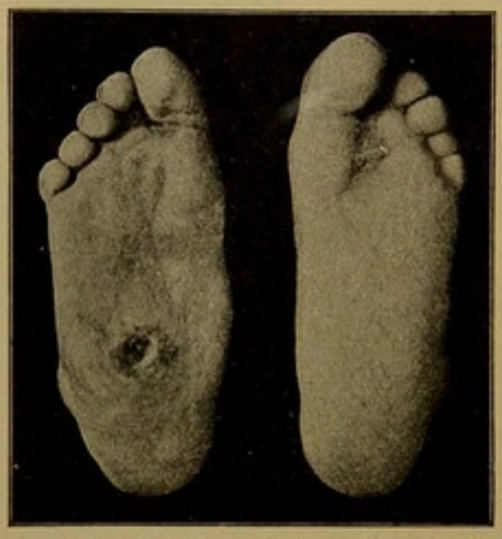

Fig. 82.—Perforating disease in the sole of the right foot, and a cicatrised ulcer of this kind between the 1st and 2nd toes of the left. (Oppenheim.)

An arthropathy of the temporo-maxillary joint is very rare (Infeld).

There is also a form of disease of the joints in tabes, which is distinguished from those above as being benign, giving rise only to an effusion which is rapidly re-absorbed.

xviii.). French writers (Brissaud, Charcot-Dufour) have later bestowed great attention on the occurrence of this symptom, the "hémarthrose tabétique." The secondary appearance of tuberculosis in a tabetic joint has occasionally been noted.

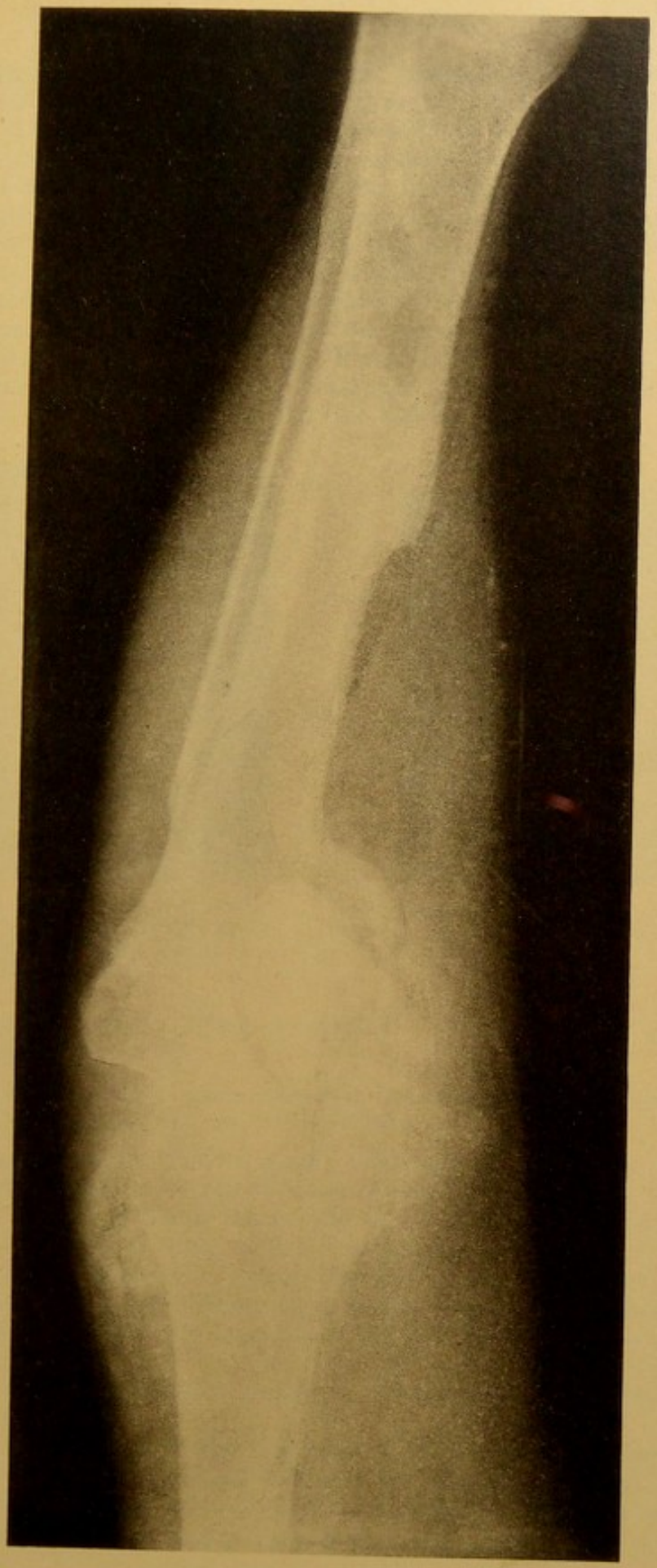

Fig. 87
Spontaneous Fracture and Arthropathy in Tabes
(Author's case).

Otto Schulze & Co., Edinburgh.

Sometimes the bones are abnormally brittle, so that fractures occur spontaneously, without any external violence. I have frequently seen spontaneous fractures of the thigh in cases which were not in an advanced stage. In one of these, it was not until eighteen years after the occurrence of the fracture that the initial symptoms of the tabes were recognisable. Touche reports one case in which spontaneous fracture of both femora took place at the commencement of the disease. Fracture of the ischium has been noted by Siebold, and of the patella by Cohn.

Arthropathy is occasionally associated with fracture (Rotter), as has been confirmed, especially in the last few years, by means of radiography (Oppenheim and others) (see Fig. 87, Plate II.). Fine changes in the bone tissue, which have been variously interpreted, have also been reported (Nalbandoff, Kienböck, Donath, Leyden-Grunmach, Cohn).

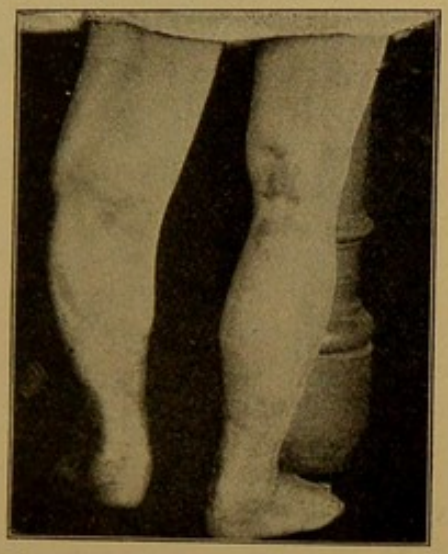

FIG. 83.—Genu eversum in tabetic arthropathy of the left knee-joint. Swelling round it. (Oppenheim.)

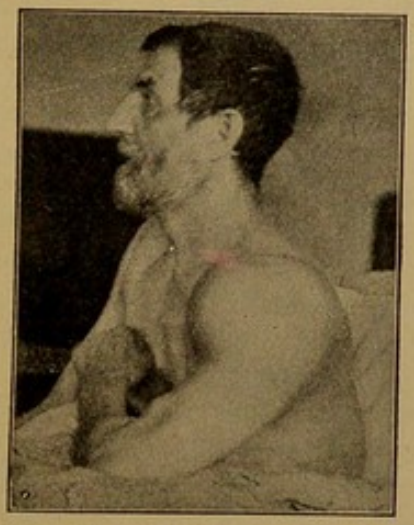

FIG. 84.—Tabetic arthropathy of the left shoulder-joint. (Oppenheim.)

Spontaneous rupture of the tendons—of the Achilles or quadriceps tendon—very rarely occurs.

I have treated a gentleman in whom spontaneous rupture of the quadriceps during a movement on level ground was apparently the first sign of tabes.

Amongst the trophic disturbances I have still to mention—

1. Perforating ulcer—a round ulcer which develops in the sole of the foot (rarely on the dorsum), especially in the region of the balls of the toes, but also at other sites, which has a great tendency to penetrate even as far as the bones and joints, and which offers great resistance to treatment.[1] There is also, however, a benign form. Perforating disease affecting the buccal mucous membrane has been mentioned occasionally in French literature.

2. Spontaneous gangrene (Joffroy, Pitres) has been observed in isolated cases.

Various disturbances of the nutrition of the skin (*e.g.* vitiligo) and of the nails, as well as spontaneous falling of the latter, have been described.

Amongst the rare symptoms there are also hemihyperidrosis, diffuse

[1] This is well described and reviewed in the "*Sammelreferat*" of Adrien, *C. f. Gr.*, 1904.

ecchymosis, which may follow the attacks of pain (Strauss), but which I have also seen without these, and herpes, which in three of our cases formed the first sign of the disease. These, as well as other exanthemata, may appear as accessory symptoms of the gastric crises (Rauschke), and the lightning pains (Seiffer).

Finally there is still another symptom which has been brought into connection with tabes (Debove), although the mode of its relation has hitherto not been made clear. This is the *apoplectiform attacks* which occasionally occur in the commencement or the course of the disease, and

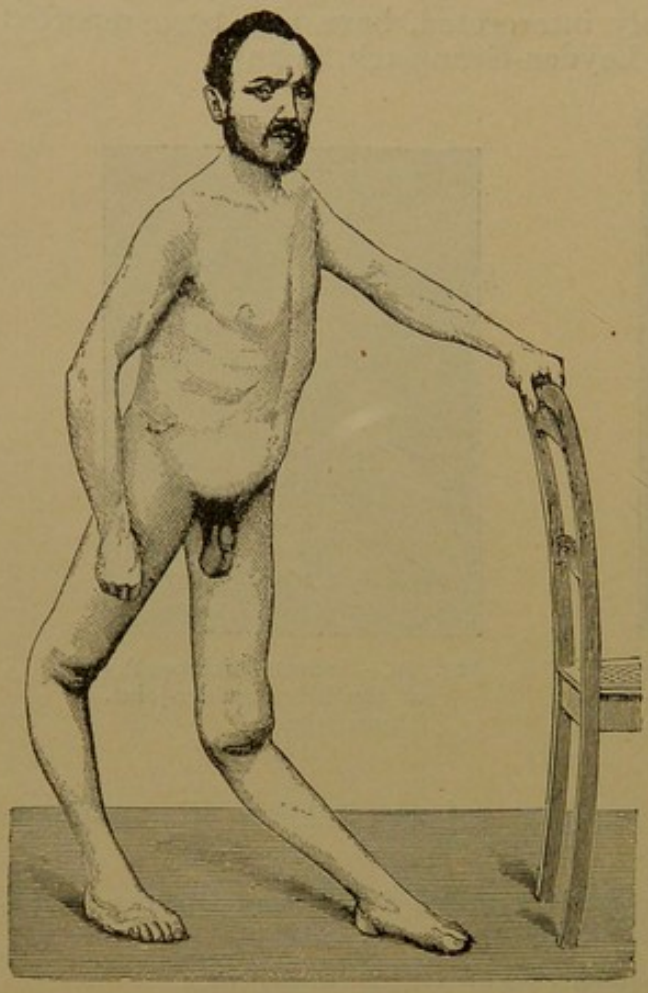

FIG. 85.—Deformity due to tabetic arthropathy of the knee-joints. (After Westphal.)

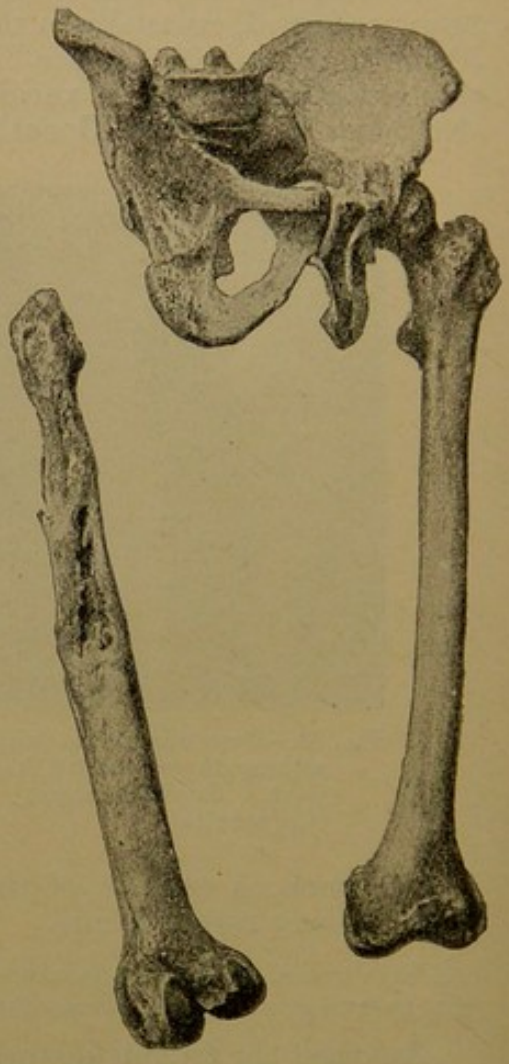

FIG. 86.—Arthropathy of the hip-joint. (Oppenheim and Sonnenburg.)

may leave a hemiplegia, which is usually recovered from. Paraplegic conditions may also appear in the course of tabes and again disappear. Most probably, as I agree with Minor (*Z. f. k. M.*, 1891) and others in thinking, focal diseases usually lie at the root of these disturbances, which are not directly related to the tabes but are dependent on syphilitic processes. Cayla, Lapinsky, and A. Schüller (*W. m. W.*, 1906) have recently discussed the question. The last named describes the condition as *tabetic attacks*. As a rule there are paralytic attacks, usually of paraplegic distribution, associated with hypotonia and absence of reflexes, which come on during the early stage of the tabes and, subsequently, usually disappear entirely or partially. I have observed this condition in one case and have frequently obtained a history of it. Lapinsky (*A. f. P.*, Bd. xlii.) regards it as analogous to the experimental paralysis which follows section of the posterior roots. These attacks may also appear under the form of a very severe temporary ataxia (Schüller, my own observations) or exceptionally under that of Landry's paralysis. The con-

dition must not, of course, be confused with that of ataxic paraplegia, which is caused by a combined affection of the posterior and lateral columns (compare the corresponding chapter).

Insufficiency of the aortic valves, to the coincidence of which with tabes Berger and Rosenbach first drew attention, seems to be merely a complication of tabes, although it may be ascribed also to the trophic disturbances. Aortic aneurysms are also by no means rare in tabetics. Babinski has pointed out their combination with immobility of pupil. The frequency of aortic diseases in tabes and their diagnostic significance has recently been referred to by Strümpell (*D. m. W.*, 1907).

Floating kidney has been repeatedly found in tabes. It is possible that this condition is caused by the relaxation of its ligaments due to the fundamental disease.

Remarks on the Diagnosis

In advanced cases of tabes the diagnosis presents no special difficulty. There is practically only one disease with which any real confusion is likely to arise, viz., *multiple neuritis*. This disease, however, almost always has an acute or subacute development, reaches its height in the space of a few weeks or at most of a few months, and then terminates either fatally, or, in the great majority of cases, in a convalescence which is often very protracted. Multiple neuritis has in common with tabes the pains, sensory disturbances, ataxia, and Westphal's and Romberg's signs, but in it as a rule the bladder disturbances, the girdle sensation, and the immobility of the pupils [1] are absent. The areas of the anæsthesia correspond in polyneuritis to distribution of the peripheral nerves, whilst in tabes it follows that of the nerve roots. Advanced, bilateral atrophy of the optic nerve does not occur in polyneuritis, but there may be an optic neuritis with termination in partial atrophy and a disturbance of vision in the form of central scotoma (specially in alcoholism). On the other hand, in multiple neuritis we find symptoms which are absent in tabes, viz., pain on pressure of the peripheral nerves and muscles, and degenerative paralysis of the peripheral nerves. (The latter may appear as a complication of tabes, but is then confined to one nerve, to the posterior interosseous or peroneal, for instance.) Multiple neuritis is also frequently accompanied by mental disturbances, of a kind which do not appear in tabes. Finally, consideration of the etiology reveals important differences, showing the cause of the tabes to be syphilis and that of the multiple neuritis to be an intoxication (alcohol, lead, arsenic, etc.), or an infection (acute infective diseases, tuberculosis, etc, but very rarely syphilis).

I must, however, remark in qualification that recent experience has convinced me that chronic alcoholism must be included among the factors which help to produce tabes. Nonne (*N. C.*, 1907) in particular has recently shown that combined systemic diseases of the cord may develop as the result of alcoholism.

A form of disease which depends essentially on a multiple neuritis, must be specially mentioned on account of its great resemblance to tabes and its completely different prognosis. This is *diphtheritic paralysis*. In the generalised form, which is by no means uncommon, we find ataxia, Westphal's sign, sensory disturbances in the extremities, Romberg's sign, oculo-motor paralysis, etc. The history, however, almost always shows the difference. We learn that some weeks or months previously there has been a febrile affection of the throat, causing, first, difficulty in

[1] Isolated loss of the pupil reflex (reflex immobility of the pupils), occasionally, though very rarely, appears in chronic alcoholism, whilst absolute immobility of the pupils is apparently less unusual (see Bunke, *loc. cit.*).

swallowing, nasal speech, and later, paralysis of the pharynx and larynx, etc., and that the symptoms in the extremities followed this localised paralysis.

Compare also the discussion of "acute ataxia" in the chapter on myelitis.

Tabes dorsalis has many points of contact with *diabetes mellitus*.

Glycosuria may be either a symptom of tabes or a complication of it; diabetes may also produce a great number of the symptoms which we have included in the description of tabes: the attacks of pain, Westphal's sign (Bouchard), sensory disturbances, oculo-motor paralysis, spontaneous loss of teeth, impotence, attacks of vomiting, etc. The origin of the majority of these symptoms is to be sought in a neuritis (which has also been proved by anatomical investigation in a number of cases by Lapinsky), so that the condition is usually one of a diabetic pseudo-tabes. It is doubtful, however, whether the pupil immobility and the bladder paralysis can develop from a neuritis. The question has recently been studied by Pitres and by Williamson. A degeneration of the posterior columns has, however, been found in isolated cases of diabetes (Williamson, Souques, Marinesco). And finally there can be no doubt that the two diseases may combine, and may exist side by side. In such cases it may be exceedingly difficult to determine whether we are dealing with a diabetes with tabetic symptoms or with a real tabes associated with diabetes.

Addison's disease may also have a few symptoms, such as Westphal's sign, in common with tabes. A case of this kind was sent to me with the diagnosis of tabes. As soon as the pigmentation becomes distinctly visible, however, such an error is no longer possible. In this disease also a degenerative process in the posterior columns has been found (Bonardi).

On the whole, therefore, it is only the early stage of the disease which presents difficulties of diagnosis, because the grouping of the symptoms may be extremely diverse. I shall now mention a number of groups of symptom in which tabes may make its appearance.

Lightning pains, Westphal's sign, reflex immobility of pupils.

Weakness of bladder, Westphal's sign, girdle sensation.

Immobility of pupils and anæsthesia of trunk.

Immobility of pupils and absence of Achilles reflex.

Atrophy of optic nerve, Westphal's sign or girdle sensation with corresponding hypæsthesia, analgesia.

Atrophy of optic nerve, lightning pains, impotence.

Attacks of vomiting, Westphal's sign, or absence of Achilles jerk or pupil immobility.

Gastric crises and anæsthesia of trunk, etc.

Joint affections, analgesia, Westphal's sign or immobility of pupils.

Paralysis of the vocal cords (with or without fits of coughing), Westphal's sign, immobility of pupils.

Spontaneous falling out of the teeth with sensory disturbances in the area of the trigeminus, Westphal's sign, bladder-disturbances, etc.

Oculo-motor paralysis, girdle sensation, analgesia, etc.

Atrophy of optic nerve and anæsthesia of trunk, etc.

Every other combination of symptoms is conceivable and is observed; further discussion is unnecessary.[1]

It is only where there is but a single symptom present, or where there are several which might also be precursors of paralytic dementia, that there is any difficulty. Immobility of the pupils, for instance, as the one

[1] The question of atypical and abortive forms ("formes frustes") of tabes, to which attention was drawn in the earlier editions of this text-book, has been in recent years repeatedly handled by Erb, G. Flatau, Determann, Pennero, Raymond, Schüller, Hudovernig, and others).

recognisable symptom, is not sufficient to justify the diagnosis of tabes, although isolated cases are cited (Cassirer-Strauss, Dufour) in which it was the only sign of a tabetic degeneration of the posterior columns. It is therefore, without doubt, a symptom which must arouse suspicion that the case is one of commencing tabes. This is still more true of simple progressive atrophy of the optic nerve.

Fragstein saw a case in which neuralgia of the fifth nerve, associated later with anæsthesia in the area of its distribution, was for a considerable time the only sign of tabes.

The establishment of a previous syphilis, although not absolute proof of the existence of the disease, is yet a valuable confirmation of the diagnosis. Although we have frequently to dispense with it, yet I cannot sufficiently impress the value in such cases of an examination of the husband or wife (and even of the children) and the establishment of a diagnosis "ex uxore." We may not often find the signs of syphilis in the other partner of the marriage, yet an immobility of pupils or Westphal's sign may reveal the conjugal tabes and also the presence of syphilis.

During the last few years French writers especially (Sicard, Widal, Rivaut, Raymond, Babinski, Nageotte, Brissaud, Marie-Crouzon, Milan, and others) have made the important observation that the cerebro-spinal fluid obtained by lumbar puncture may materially help the establishment of the diagnosis, as a more or less marked lymphocytosis is an almost constant sign, even at the very commencement. Although this occurs more or less in all syphilitic and metasyphilitic diseases of the nervous system, it is nevertheless of great significance in the differential diagnosis of tabes, particularly in the initial stages. Schönborn, Frenkel, Dinkler, J. Fraenkel, Niedner-Mamlock, Apelt, Meyer, and many others have confirmed this observation, and Erb[1] has specially laid stress upon its importance. But, however valuable it may be, I do not believe that the examination will be adopted in general practice, as lumbar puncture is too serious a proceeding to be used as an ordinary method of neurological diagnosis.

Lymphocytosis is also found in cases in which reflex immobility of the pupils is the only morbid sign, and it has therefore been inferred that this symptom is a definite proof of syphilis (Babinski, Cestan, Dupuy-Dutemps). But this seems to me to be going too far.

For the carrying out of this examination one employs a hollow needle of irido-platinum of 1 mm. maximum diameter with a platinum wire to clear it if blocked. The needle is introduced into the space between the laminæ of the third or fourth lumbar vertebræ, the patient being in a sitting position (compare chapter on lumbar puncture). The fluid, of which 4 to 6 c.cm. only are drawn off, is received in a sterilised glass tube and is centrifuged for about twenty minutes. The contents are then poured off, and from the remains of the fluid which still adhere to the bottom of the glass, a drop is drawn into a pipette, then placed on the slide, fixed and stained. For the staining, methylene blue or Ehrlich's triacid or hæmatoxylin-eosin is used. Whilst in normal cerebro-spinal fluid, with a magnifying power of 400 to 450, only 3 to 4 lymphocytes will be found in the microscopical field; in tabes there will be 30 to 40 and even more.

Nonne and Apelt (*A. f. P.*, Bd. xliii.) lay great weight from their own experience on the diagnostic value of the increased amount of albumin in the fluid.

The facts revealed by Wassermann's serodiagnostic method are of great interest and apparently also of value in the diagnosis of tabes.

The atrophy of the optic nerves, the immobility of pupils and Westphal's sign, are not infrequently forerunners of paralytic dementia. The most important points for the differential diagnosis are the mental

[1] *Z. f. N.*, xxxiii.

disturbances, the speech affections, and the paralytic attacks. We must beware, however, of suspecting paralytic dementia from every mental alteration which occurs in the course of tabes.

Strümpell speaks of a disease allied to tabes, which depends on chronic nicotine poisoning, but he gives no opinion as to the nature of differential signs of this affection.

(With reference to the distinction of tabes from syphilitic pseudo-tabes, see the chapter on syphilis of the spinal cord.)

Pathological Anatomy

The most important of the pathological changes in tabes dorsalis is the grey *degeneration of the posterior columns*. This is almost always visible to the naked eye. The posterior columns appear grey in contrast

Fig. 88.—Transverse section of a spinal cord in the early stage of tabes dorsalis. (Weigert's method.)

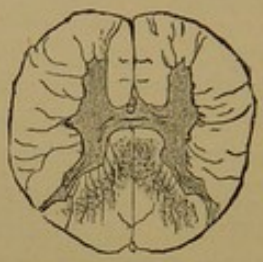

Fig. 89.—Localisation of the posterior column degeneration in the early stage of tabes. The affected parts are shaded dark. (After Westphal.)

with the rest of the white matter, and they are usually diminished in size and sunken inwards. It is only in cases which come early under post-mortem examination that there is any doubt about the naked eye appearances.

Microscopical examination shows that the process commences in

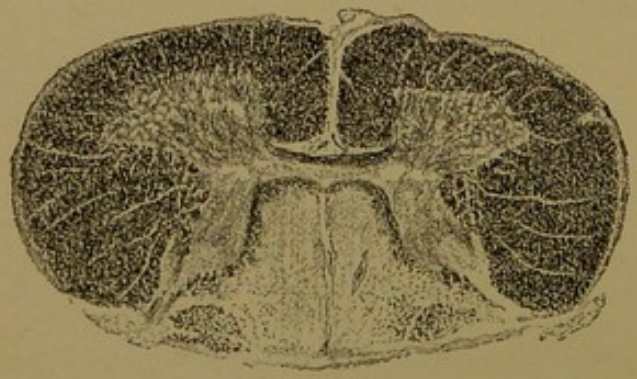

Fig. 90.—Transverse section through the cervical enlargement of the spinal cord in advanced tabes dorsalis. (Weigert's method.)

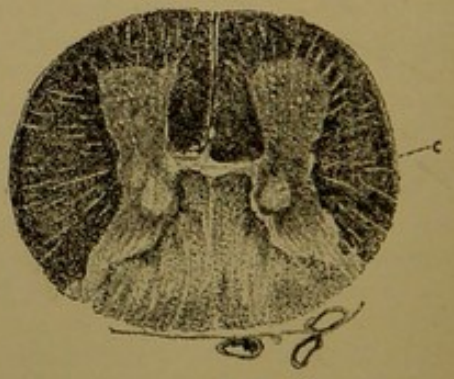

Fig. 91.—Degeneration of the posterior and Clarke's columns in tabes dorsalis. c = Clarke's column. (Weigert's method.)

Burdach's column, usually in the upper lumbar region. At first two symmetrical fields are affected (Figs. 88 and 89), in the region of the root entry-zone, *i.e.* in that segment of Burdach's tract which the posterior roots enter or which they constitute. This atrophy of Burdach's tract

corresponds in higher segments to a degeneration of Goll's column, which is a direct result of the former. In advanced stages, in the lumbar and thoracic regions the whole of the posterior columns are degenerated (with the exception of a small area adjoining the posterior commissure). In the cervical region the affection is at first confined to Goll's column, but, later, portions of the external field of the posterior columns also become affected. The degeneration then usually shows the distribution represented in Fig. 90.

In so-called cervical tabes, Burdach's tract of the cervical cord is the first to be affected.

With regard to the nature of the process, there is a universal degeneration of the nerve fibres, whilst the glia is conserved or even proliferated.

The disease of the spinal cord is not however limited to the white matter. The atrophy of the fibres also affects the grey matter: (1) Clarke's column, which, it appears, is constantly involved (Fig. 91), and (2) the posterior horns, especially Lissauer's marginal zone.

In typical and pure cases the anterior grey matter always remains intact, but in a few cases (Pierret, Charcot, and recently Raymond-Philippe) there appears as a rare symptom or as a complication of the disease, a muscular atrophy which is attributed to disease of the anterior horn cells. Lapinsky has put forward certain signs by which the tabetic muscular atrophy of spinal origin may be distinguished from the peripheral-neuritic form. Whether the fine cell changes revealed by Nissl's method (Schaffer and others) are of real practical significance is very doubtful. *Slight* changes in the lateral columns have been found in a few cases which clinically showed the picture of a pure tabes.

Atrophy of the *posterior roots* is a constant feature. Indeed, in the opinion of some writers this atrophy forms the starting-point of the process, and the changes in the cord are a simple result of it (Leyden, Redlich, etc.). From this point of view, therefore, it is of special interest that we[1] have been able to trace the disease of the posterior roots as far as their trophic centre, into the *spinal ganglia* (Figs. 92A and B), where we as well as Thomas and Hauser,[2] found mainly a destruction of the medullated fibres, but in a few cases (our own, and specially those of Wollenberg, Stroebe, and Babes) an atrophy of the ganglion cells could also be distinguished, whilst Marinesco, Schaffer, and others could not discover essential changes in them, and Marburg regarded them as secondary and insignificant.

The disease not infrequently extends to the medulla oblongata and causes atrophy of the spinal root of the trigeminus, and frequently also of the solitary bundle (Figs. 93A and B), as well as of the posterior vagus nucleus, whilst we never find involvement of the nucleus ambiguus. It is only in exceptional cases that other parts of the medulla oblongata, such as the sensory trigeminal nucleus (Oppenheim), of the motor nucleus, of the trigeminus (Raymond-Artaud), the nucleus of the auditory and the hypoglossal nucleus (Raymond-Artaud, Koch-Marie) participate in the degeneration. In a few cases the atrophy affects the nuclei of the oculo-motor nerves (compare Fig. 94B).

On the other hand, some of the cranial nerves become affected independently, the optic nerve most frequently. The grey degeneration of the optic nerves is an ordinary occurrence. The pathological-histological nature of this affection of the optic nerve has been specially studied in recent years by Marie and Léri. The oculo-motor nerves may

[1] *A. f. P.*, xviii.

[2] See their recent work in *Nouv. Icon.*, 1904.

also be involved in the atrophy, although their nuclei seem to be unaltered. I found this also with regard to the vagus and the recurrent laryngeal, and once with regard to the glosso-pharyngeal. According to Cahn,

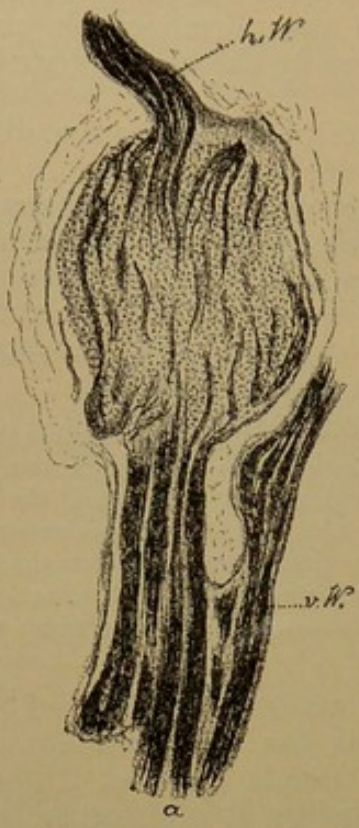

Fig. 92A.—(Compare with B.) Longitudinal horizontal section through a spinal ganglion with posterior (h. W.) and anterior (v. W.) roots and emerging nerves (a) (From a section stained with Weigert's hæmatoxylin.)

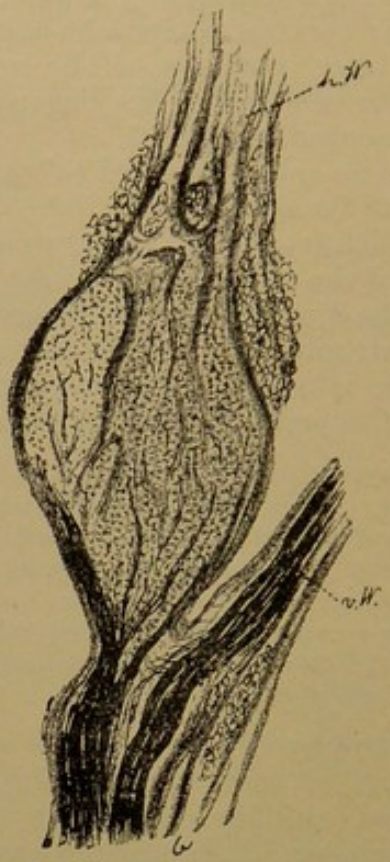

Fig. 92B.—(Compare with A.) Atrophy of posterior root and spinal ganglion in tabes dorsalis. (Weigert's method.)

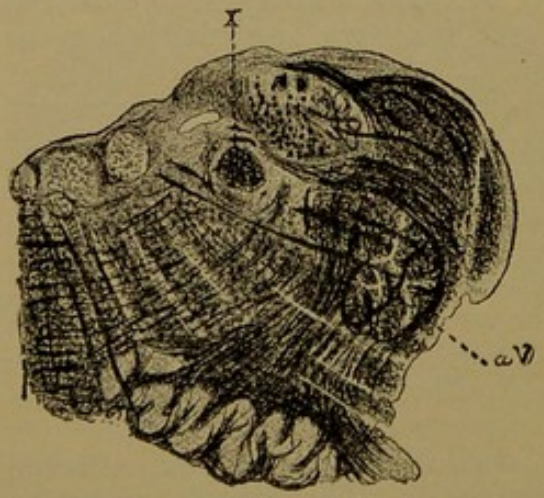

Fig. 93A. — Part of transverse section through medulla oblongata at the level of the xii. and x. cranial nerves. X = solitary bundle. aV = spinal root of the trigeminus. Normal. (Weigert's method.)

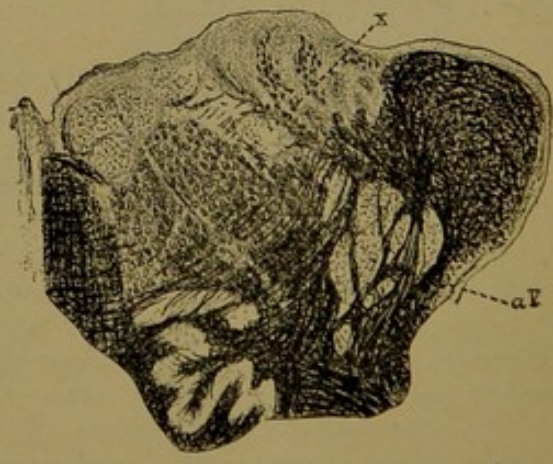

Fig. 93B. — Part of transverse section through medulla oblongata at level of xii. and x. cranial nerves. Atrophy of the solitary bundle x. and of the spinal root of the trigeminus aV in tabes. (Weigert's method.)

tabetic laryngeal paralysis depends, in most cases at least, upon a degeneration of the laryngeal nerves. Heitz noted changes in the nerves

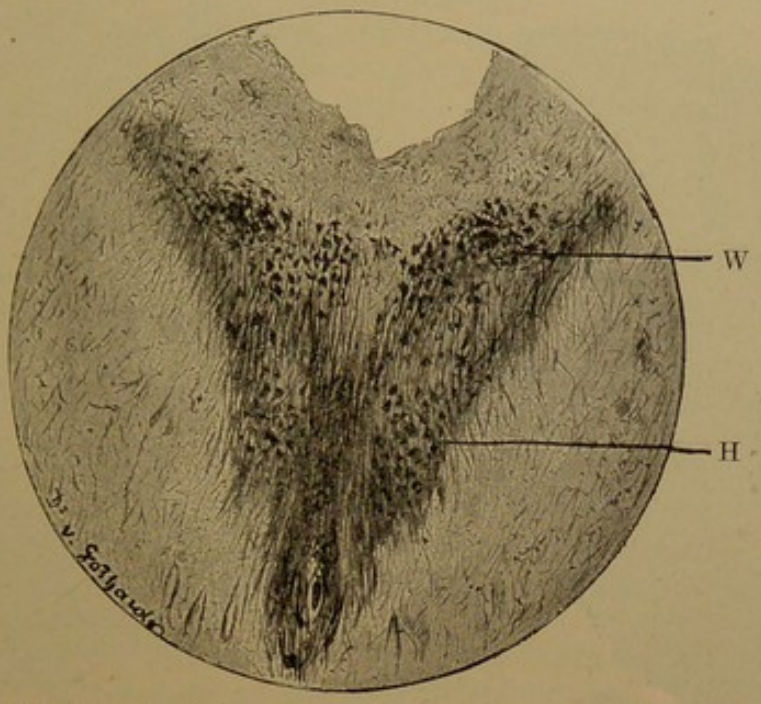

Fig. 94A.—Part of frontal section through anterior corpora quadrigemina from a normal brain. W = Westphal-Edinger nuclear group. H = Main oculo-motor nucleus.

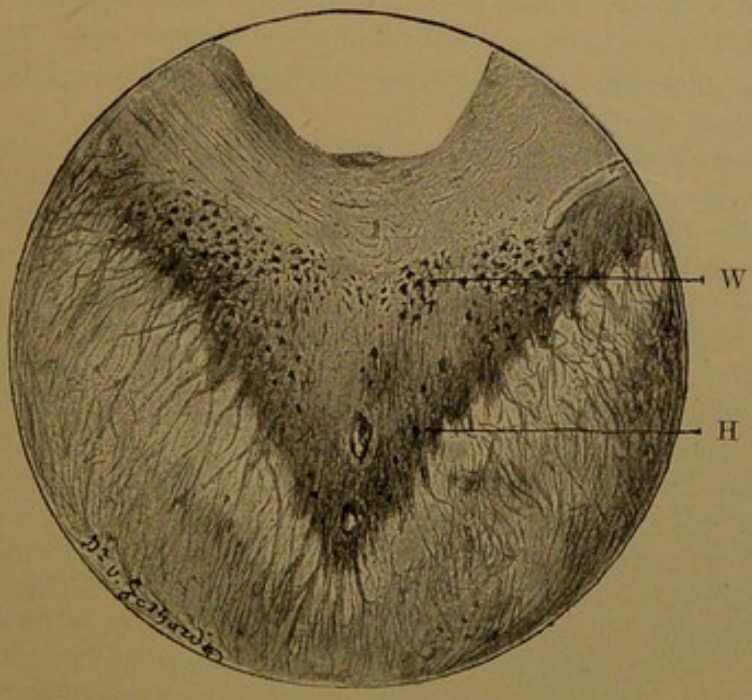

Fig. 94B.—(Compare with 94A.) Part of a frontal section through the anterior corpora quadrigemina in tabes dorsalis, with oculo-motor paralysis. W = Westphal-Edinger nuclear group. H = Main oculo-motor nucleus, which shows a somewhat severe degree of atrophy.

and ganglia of the heart. Hemiatrophy of the tongue may also have a peripheral origin (Obersteiner, Cassirer, and Schiff). The peripheral or radicular genesis of paralysis of the spinal accessory is upheld by Seiffer and others. Grey degeneration of the auditory or its terminal ramifications is very rare (Strümpell, Oppenheim-Siemerling, Brühl).

Fig. 95B shows a degeneration of the *Gasserian ganglion* in connection with the ganglion cells and fibres, as I found in one case. It is worthy of

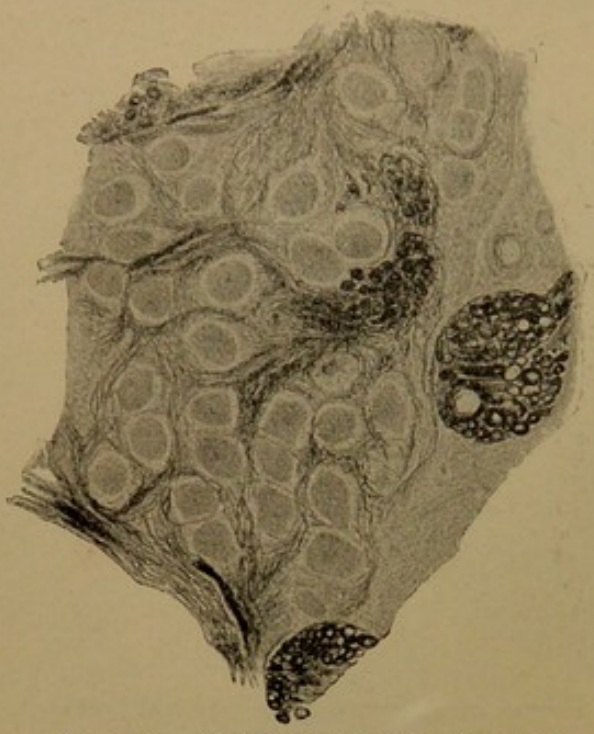

FIG. 95A.—Section through normal Gasserian ganglion. Stained with osmic acid.

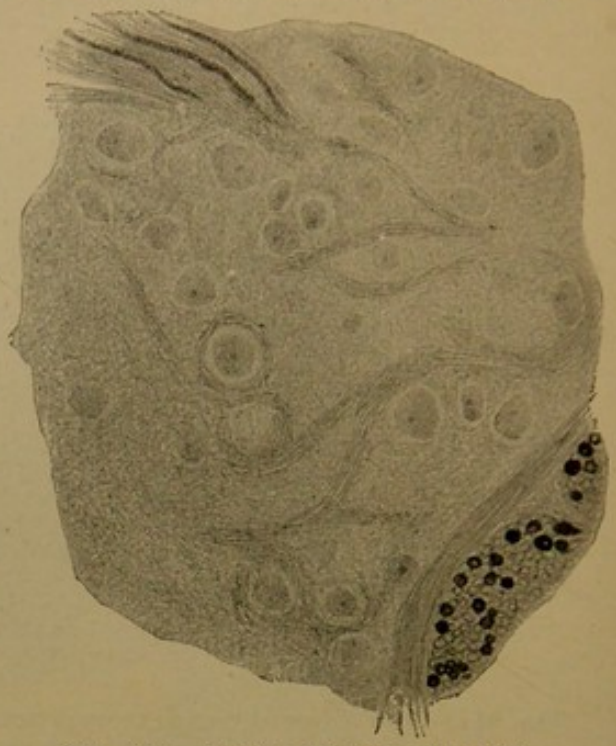

FIG. 95B.—Section through atrophied Gasserian ganglion in tabes dorsalis.

notice on this account, as the Gasserian ganglion forms the origin of the spinal trigeminal root.

Finally, we must mention the *atrophy of the sensory cutaneous nerves*, which was observed long ago by Westphal, and has since been specially investigated by Dejerine, Siemerling, and myself, and has been shown by Gumpertz to exist in a particle of skin taken from a living case. It forms, apparently, a regular occurrence in tabes. The cutaneous sensory nerves of the lower extremities are mainly affected (Fig. 96). We do not know what part these affections play in the symptomatology, nor can we decide whether the changes are primary or accessory, and in what relation they stand to the disease of the spinal cord or posterior root ganglia. That the neuritis of the peripheral nerves—the motor also are occasionally affected in tabes—may, in itself, produce a group of symptoms closely resembling those of tabes dorsalis (peripheral neurotabes) has been specially demonstrated by Dejerine's investigations.

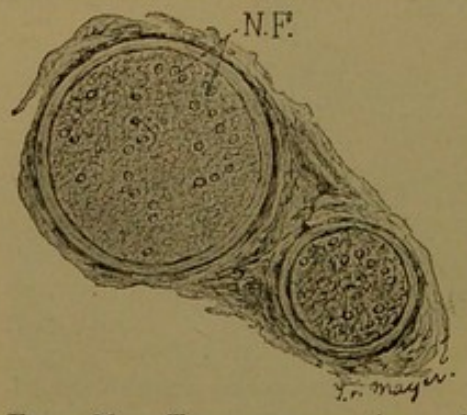

FIG. 96. — Transverse section of atrophied cranial nerve. N.F. = normal fibres.

An involvement of the sympathetic—atrophy of some of the fine medullated fibres—has been recently found, especially by Roux,[1] and has been regarded as the origin of the affections of visceral sensibility

[1] Soc. de Biol., 1899 ;] *Thèse de Paris*, 1900.

Whilst the great majority of the symptoms of the disease may readily be accounted for by the pathological lesions, some have as yet no recognised anatomical basis. Chief amongst these is the reflex immobility of the pupils. According to the opinions and investigations of Rieger, G. Wolff, and Schmaus-Sacki, its origin is to be sought not in the region of the oculo-motor nucleus but in the cervical cord. This seems also to be supported by an observation of Dreyfus's, and Reichardt has specially defended this view, which, however, Bumke [1] justly opposes. The conditions present in several cases are not in accordance with this view. Further, the changes found by Marina in the ciliary ganglion deserve full consideration.

The site of the origin of tabes dorsalis has been sought at various points. Of the prevailing opinions two must specially be discussed here. The first was originated by Marie, modified by myself [2] on the ground of my own investigations, and may be thus formulated : The generating poison which produces tabes, acts upon the spinal ganglia and their homologues (Gasserian and jugular ganglia, etc.), and injures them without at first altering their structure. This lesion is capable of bringing about an atrophy of the sensory fibres which arise from them in the spinal cord, in the medulla oblongata, and in the peripheral nerves, especially at their distal ends. This atrophy gradually extends towards the cells of the posterior root ganglion, and eventually destroys the fibres that arise from them. Marie himself has abandoned this view of the pathogenesis ; he now assumes that the disease depends on a syphilitic affection of the lymphatic system of the posterior columns and of the corresponding meninges.[3]

The second view agrees with that just discussed in so far that it also assumes an *exogenous* origin of tabes, and assigns it, as Leyden had previously done, to the posterior roots. Obersteiner and Redlich [4] have shown that the posterior roots undergo a constriction as they pass through the pia mater, so that every affection of the meninges which causes a thickening and contraction injures the roots at this point and causes them to atrophy. They would therefore attribute the whole morbid process to a posterior meningitis. Nageotte as well as Thomas and Hauser have expressed similar views on the ground of their investigations.

This view has much to commend it, but several objections may be raised against it, especially this (brought forward by myself, Schwarz, and Dambacher), that such a meningitis is not always present and that it does not explain the affection of the cranial nerves, etc. Schaffer has also expressed himself as unfavourable to this view, although he still adheres to the radicular origin of tabes. During recent years the Paris school has again upheld the view that meningitis (syphilitic) is one of the constant and fundamental changes in tabes, claiming special support for this view from the results of cytodiagnosis already mentioned.

A similar view has been expressed as regards the genesis of the optic atrophy (Schlagenhaufer), whilst another writer (Moxter) regards it as being of peripheral origin.

[1] *Klin. Mon. f. Aug.*, 1907. [2] "Zur path. Anat. d. Tabes dors.," *B. k. W.*, 1894.

[3] Compare Marie et Guillain, *R. n.*, 1903. Orr's investigation into the lymph tracts of the spinal cord may well serve as a confirmation of this view.

[4] Redlich, "Die pathol. Anat. der tabischen Hinterstrangerkrankung."

Finally, the researches of the last few years (compare p. 124), according to which lesions of the *peripheral nerves* may lead to an affection of the posterior root ganglia and even of the posterior roots and columns, have once again brought into prominence the possibility of a peripheral origin of tabes dorsalis.

None of these theories can claim to be generally accepted. Many writers regard the disease simply as an affection of certain nerve tracts, which are characterised by their developmental history and their function as associated systems, thus committing themselves in no way as regards its site of origin.

Jendrássik's theory, that changes in the cerebral cortex, especially atrophy of the fibres, constitute an essential element in the disease, has obtained little confirmation or support from recent observations (Philippe). Schaffer, however, corroborated Jendrássik's views, and Strümpell has explained their findings as the result of a degeneration of the terminal processes of the central sensory neurones.

Changes in the cerebellum were found by Jellinek, Weigert, Spielmeyer, and Homburger, but they are of no essential value in the morbid picture. Atrophy of the ganglion cells in the posterior central convolution has been noted by Campbell.

Course and Progress

Tabes is a disease with an absolutely chronic course. It extends on an average over a period of one to two decades, but may last for twenty-five to thirty years and longer. Cases in which the disease ends fatally after a duration of a few years are not common. When the ataxia and bladder troubles develop early and get rapidly worse, a shorter course is to be expected than in the cases in which the disease has lingered long at an early stage. I have occasionally seen cases in which the ataxia appeared remarkably early, with an almost acute onset, and progressed very rapidly. There is also, however, an acute ataxia at the commencement of tabes which tends to incomplete recovery (see above). Even where optic atrophy is one of the initial symptoms, a long duration of the disease is usually to be expected, and under these conditions the other symptoms, the ataxia especially, tend to develop very late. This must depend essentially on the localisation of the lesion. The circumstance, however, that the patient is on account of his blindness preserved from much harm and is compelled to a certain extent to carry out a continuous treatment by exercises (see below) may contribute to the late and incomplete development of the other symptoms. This, however, does not by any means apply to every case of this kind.

In the final stage, which we describe not altogether fittingly as the paraplegic or paralytic stage, the patient is usually permanently confined to bed. The general emaciation, which may be one of the early symptoms, usually reaches a marked degree. The power of moving the legs is at this period greatly limited by the general weakness, the effects of the prolonged inactivity, and by the secondary changes (equinus position of the foot caused by the unchanging position and the pressure of the bedclothes). A real paraplegia is not frequent. In this condition the patient succumbs to marasmus, to cystitis and pyelonephritis, to infection from bedsores (rare), or with special frequency to some intercurrent disease. It has recently been shown, particularly by Marie,[1] that the duration of life is in general not materially shortened by the disease.

[1] *R. n.*, 1904. Compare, on the other hand, Goldflam, *N. C.*, 1905.

The *prognosis* in tabes is on the whole unfavourable. Recovery is but seldom reported, and is extremely rare when the disease has entered upon the ataxic stage. On the other hand it not infrequently happens that the *disease may remain permanently at an early, and often quite tolerable stage of development.* Thus we have reports of several cases which may be regarded as cured in so far that the subjective troubles completely disappeared, whilst the pathological changes in the posterior columns were only recognised on examination after death, which had resulted from a complicating disease.

It is to be remembered also that the prognosis of each individual case is materially influenced by the character of its symptoms. If the lightning pains are very severe and the attacks are separated by but short intervals, the life of the patient is extremely distressing and his power of working is greatly reduced, whatever his occupation may be. This is true also with regard to the gastric crises, which, when they are severe, prolonged, and frequent, give rise to intolerable misery and aggravate the general condition to an extreme degree. In contrast to these are the other cases of tabes in which the symptoms have, one might almost say, merely a diagnostic value, the patient's pleasure in his life and work being practically unaffected by them. I treated a gentleman, for instance, who had for thirty years suffered from mild lightning pains, and who can still, notwithstanding the existing tabes, superintend his business as a merchant quite well. Another in whom the lightning pains first occurred in 1870, was in 1898 still firm upon his legs and capable of full enjoyment of life. A third, who had acquired syphilis forty years previously, and in whom dilatation of the pupils appeared twenty years ago, presented in 1898 no symptoms except the immobility of pupils, Westphal's sign, and weakness of the bladder. A female patient whom I treated in the Charité for tabes, shows to-day the same condition, etc.[1]

These facts were already communicated in the first and second editions. Since then, I have collected a large number of similar experiences, amongst others a case of juvenile tabes in which the disease has been at a standstill for seventeen years. There is another in which the arrest has been controlled by myself for eighteen years. Some time ago, a gentleman of seventy years of age consulted me. The diagnosis of tabes had been made in his case forty years previously by Griesinger and Hitzig, on account of the initial symptoms, and the disease had not progressed beyond this stage nor had he lost his capabilities of work and enjoyment. In the same year, I saw an officer with the initial symptoms of tabes, which had been recognised as present shortly after the Franco-German war. Brissaud, Marie, Raymond, Faure, and others have recently expressed the view, from their own experience, that tabes has very frequently now a benign course and has lost its malignant character.

The question has been exhaustively studied from the material of my polyclinic by my pupil Malaisé ("Die Prognose der Tabes dorsalis," *M. f. P.*, xviii., Ergänzungsheft, Berlin, 1906, S. Karger). Belugon-Faure also give valuable data. See also Oppenheim (*Z. f. N.*, xxxiv.).

[1] The development in the following case is also interesting: A young lady of twenty-six consulted me on account of hemicrania. Since 1880, she had suffered from reflex immobility of the pupil of the right eye, but showed no other symptom. In 1892, twelve years later, lightning pains appeared, and in 1896-98 oculo-motor paralysis, Westphal's sign, girdle sensations, etc. Now as a married woman she admits that as a young girl she was led to masturbation by a maid-servant, who had touched her naked body directly with her genitals. Whether an infection had thus been communicated cannot be determined.

Further, there are symptoms which may improve in spite of the advance of the disease itself. This specially applies to the oculo-motor paralysis. I have myself observed that a complete ophthalmoplegia, which appeared at the commencement of a tabes, almost completely disappeared again. The bladder disturbances are frequently only of transient duration. It is very remarkable that even the attacks of vomiting—and the laryngeal crises also according to Charcot—may gradually cease in the further course of the disease.

I have again seen a female patient whom I had treated fifteen years before for gastric attacks of so severe a nature that she had become addicted to morphia. The attacks have now ceased for five years, and the morphia has been withdrawn from her. She shows now only the initial symptoms of tabes and an arthropathy which was also observed previously.

It has been found in rare instances (Hammond, Spitzka, Goldflam, Pick, A. Westphal) that the knee jerks, after being absent for a long time, can again be elicited. As a rule the tabes was complicated by a hemiplegia, with the onset of which this change coincided. It is less frequent without hemiplegia (Berger, Donath, Dufour). The anatomical conditions of this process have been specially studied by Pick.

I myself have seen inverse cases in which a hemiplegia masked the tabes. As soon as the patient came into my room I recognised, or suspected from the gait and attitude—from the trailing of the leg, which was not spastic but caused by the hypotonia—a combination with tabes, which was then established by a thorough examination. In two cases of this kind the patients had for a considerable time suffered from an unrecognised tabes, to which the hemiplegia was superadded. The paralysis was entirely flaccid, but the Babinski sign and Oppenheim's sign indicated the lesion of the pyramidal tracts. It is interesting to observe in how many ways the symptomatology of tabes and of hemiplegia is modified and influenced when these diseases co-exist.

Spontaneous improvements have been observed even as regards the inco-ordination.

The prognosis is made graver by the fact that paralytic dementia is not infrequently superadded to the tabes.

Neurasthenia and hysteria form a common complication. In a few cases paralysis agitans accompanied the tabes, and a combination with exophthalmic goitre has occasionally been noted. Tabes may also be combined with true syphilitic disease of the central nervous system, as has been proved by numerous observations.

Treatment

The endeavours to cure or to improve the primary disease, and to remove or modify the subjective troubles, have led to the employment of numerous methods of treatment. The results of treatment are on the whole disappointing, yet it is possible sometimes to procure a real improvement and very frequently to alleviate the distressing symptoms by the measures adopted.

Let us first consider the methods recommended :—

1. *Drugs.*—Nitrate of silver (Argent. nitrat.), in doses of 1 centigr. (Argt. nitr. 0·3, Bol. alb. q. s. ut f. pil. Mitte xxx. Sig. three times a day one pill.) This prescription is continued for a few months, and may be again resumed after an interval, but on account of the possibility of argyria, the total amount given should not exceed 10 grm. As a substitute for it protargol (to 0.1) as well as sodium nitrate, the latter by means of subcutaneous solution (1-2 per cent.), have been recently recommended (Darkschewitsch). Further, ergot (0·3 pro dosi), iodide

of potassium or iodipin in the usual doses, sodium-gold chloride (0·003-0·02 pro dosi), keratin (Zypin), mercury (hydrargyrum), as inunctions or subcutaneous injections (see below), may be tried.

To alleviate the pain :

Sodæ salicylat	1·0—3·0 pro dosi	All these drugs must be administered only under medical control.
Antiferbrin	0·25—0·5 ,,	
Antipyrin	0·5—1·0 ,,	
Phenacetin	0·5—1·0 ,,	
Methylenblue	0·1	
Analgen	1·0—2·0 ,,	
Salipyrin	0·5—1·0 ,,	
Lactophenin		
Pyramidon	0·2—0·3 ,,	
Aspirin	1·0—2·0 ,,	
Morphia	0·01—0·02 ,,	
Codeine	0·01—0·05 ,,	

For the attacks of vomiting, morphia and cerium oxalate (see below).

2. *Electricity.*—The galvanic current is used by placing one electrode of fifty to seventy sq. cm. at the neck, and another of corresponding size at the lumbar region, the current being about five to eight milliampères in strength. The electrodes may be kept stationary, or the upper one may be moved by degrees downwards over the whole spinal column. According to a method recommended by Erb, the cathode (of medium size) is placed over the superior sympathetic ganglion of one side, whilst the large anode is at first held steadily on the opposite side of the spinal column immediately beside the spinous process of the lower cervical and upper thoracic vertebræ, and is then gradually moved downwards. This treatment may be continued for months, daily or three times a week, each application lasting for about five minutes. The constant (stabile) galvanic treatment of the nerve trunks in the lower limbs is also recommended.

The Faradic Brush.—A large electrode is placed on the region of the thorax or neck, whilst the other (the wire brush) is moved over the skin of the trunk and the lower extremities, or over the anæsthetic region of the skin. The current should be so strong that it begins to be felt as painful, but without producing muscular contractions. Each application should last about ten minutes.

Attempts to treat other symptoms, such as the gastric crises, by electricity (direct galvanisation of the vagus nerve, galvanic treatment of the medulla oblongata, the solar plexus, etc.), have proved unsuccessful. The magneto-galvanic current has also been used for the lightning pains.

3. *Hydrotherapeutics and Baths.*—The mild carbonic acid warm springs and brine baths of Oeynhausen have been specially recommended, and also the baths at Nauheim, Wildbad, Landeck, Cudowa, etc. Simple lukewarm baths and cold frictions may be prescribed.

4. *Mechanical Treatment.*—Surgical stretching of the nerves has been almost completely abandoned. Benedikt alone advocates it, and Chipault describes certain indications for it (see below).

Suspension, massage, and gymnastics are used, as well as bloodless nerve stretching.

Bloodless nerve stretching is employed by Bonuzzi and Benedikt in the following way:—the patient lies on his back, his legs are fastened with a towel at the ankle-joints and are pulled so far above the head, which lies still on the bed, that the spinal column is strongly flexed and the knee can be brought as far as the forehead or even beyond it. Care must be taken in doing this, and the brittleness of the bones, etc., must be kept in mind. Blondel's method is milder. He brings the flexed knee of the patient close to his chin, and keeps it there for about five minutes by a bandage going from the neck to the popliteal space. Another mode of "extending the spinal cord by elongating the spinal column" is recommended by Gilles de la Tourette and Chipault. The patient is seated on a table, his legs are kept in a position of extended abduction, whilst the trunk is strongly flexed forwards. He is kept in this position for eight to twelve minutes.

None of these methods has attracted so much notice as the mode of muscular exercises recommended by Frenkel, which endeavours to combat the ataxia, and by methodical exercises to restore the co-ordination of the movements (re-education of the muscles, appropriate work, Leyden's compensatory exercises). The essential principle of these methods is to pass from simple to complicated movements, which have for their aim not the strengthening of the muscles, but their orderly co-operation. Various apparatus is recommended by Frenkel and others, which need not here be described. Particular importance is laid on exercise in standing and walking. Goldscheider [1] has collected and described these methods very clearly. The work of O. Foerster is very specially suited to introduce one to the principles of this treatment by its exact analysis of the nature of the ataxia. There is no doubt that special institutions will soon be opened for this method of treatment (this has actually taken place). Any physician, however, can carry out this treatment.

The results of such methods are often considerable, and I myself have seen their benefit, but I think it right not to encourage any extravagant expectations with regard to them.

Apparatus for the support of the spinal column has also been recommended. Hessing's in particular is very popular. In severe hypotonia of the legs such apparatus may be suitable and may facilitate walking.

Method of Treatment.—If the patient comes under treatment at an early stage, do not paint a gloomy picture to him, but represent to him that the disease which is present may, if his mode of life is not regulated, extend to the spinal cord. He must protect himself from chill and from *excessive physical strain*. I have met cases who in the first stage of tabes, when as yet there was no trace of ataxia, allowed themselves to be taken for a tour in the mountains and returned with fully developed ataxia. I do, however, know one tabetic of sixty-seven, who carried out an expedition of this kind (riding for eight hours on mountain paths) with impunity. A half to one hour's walk on the level ground should be allowed. Hill climbing is interdicted. Cycling may be allowed, under great care and restriction. If ataxia has already appeared, a tricycle should be preferred.

Proceeding from his exhaustion-theory, Edinger (*D. m. W.*, 1905) has recently given a prominent place in the treatment of tabes to the avoidance of all strain on the system.

The patient must specially avoid getting wet, and he should not be subjected to a prolonged application of the ice-bag.

Sexual excess must be unconditionally interdicted.

As pregnancy may have an accelerating and aggravating influence on the disease, it may be necessary to bring about a premature confinement (Heitz).

[1] "Anleitung zur Übungsbehandlung der Ataxie." 2 Aufl., Leipzig, 1904.

The patient should accustom himself to pass urine regularly, and at least four times in the course of twenty-four hours, even although he may feel no need to do so. Mild aperients may be given for the constipation. The diet must from the outset be strengthening, in order to counteract the anæmia and emaciation. I saw an extremely rapid progress of the disease in two vegetarians.

Alcohol must be used sparingly, and smoking should be limited as much as possible.

In every case *galvanic treatment of the spinal cord* should be tried. When it is carried out by an expert it is never harmful, frequently causes an essential alleviation of the subjective troubles, and sometimes an improvement which can be objectively recognised. The treatment should extend over several months, and may be repeated after a corresponding interval.

The results of faradisation (by means of the wire-brush) are overestimated by Rumpf. This treatment may be tried as an experiment in cases in which the sensory disturbances are very prominent. I would advise, however, that currents of moderate strength should be used, and that the course of treatment should not last more than one to two months, if the result has not been evident before then. There are tabetics who cannot bear the faradic brush. For incontinence of urine I can from some remarkable results of my own safely recommend *internal faradisation* of the sphincter vesicæ. Stintzing speaks highly of the external application of the electrodes over the symphysis and perineum, a galvanic current of ten to twenty MA. being employed, or of galvanofaradisation.

In cases which have not advanced too far a course of baths at Oeynhausen, Nauheim, or Wildbad, may be prescribed (or even the cooler baths of Gastein). Brine baths have a specially soothing and pain-calming effect. The carbonic chalybeate baths of Cudova, Schwalbach, Franzonbad, etc., are also recommended. The use of hot [1] and cold, of steam or of sea-baths is to be strictly forbidden, although I have heard of a few tabetics who got benefit from the use of cold sea baths. *A careful course of cold-water baths* may be recommended at any stage.

Suspension treatment (Motschutkowsky) is not suitable for advanced cases. It is also counter-indicated in arterio-sclerosis and bulbar symptoms, as well as in threatening paralytic dementia. It must naturally be used only with great care, as not only peripheral paralysis, syncope, etc., may appear, but also, as in one case described by Fischer, softening of the cervical cord. Sprimon's apparatus, however, by means of which the extension is gradually exerted on the patient while sitting, makes the method almost without danger. Sayre's apparatus may be used for suspension in a sitting posture. Jacob and Konindjy recommend lying on an inclined plane. Under the suspension treatment there may be improvement in the pains, the ataxia, the impotence, and even in the optic disturbances. I can also record some such results. On the whole, however, there is not much to be expected from the method, about which there has been very little said during the last few years. I have seen in a few cases the good effect of the application of a supporting corset to the spinal column, especially for the girdle pains.

It is most difficult to answer the question: Should anti-syphilitic treatment be employed? I myself have had mostly failures from its

[1] The hot-water treatment (packs, baths), prescribed by a well-known physician for all nervous diseases and troubles, including tabes, has had a particularly injurious effect in some of the cases under my care.

use, so that, as a rule, I refuse to adopt it, unless indeed special conditions exist, which will be discussed, and particularly if similar treatment has already been employed. Erb's experience is quite opposed to this; he warmly advocates the mercury treatment of tabes. In many cases he has seen improvement in the sensory disturbances, the pains, the oculo-motor paralysis, and the ataxia, but he has found it of no effect upon the crises. One should resolve without delay on specific treatment in the cases in which syphilis has certainly been present and the treatment insufficient, as well as in those where other signs of syphilis exist, or where the morbid picture is an atypical one, and the possibility of a confusion with a real syphilitic disease of the cord cannot be excluded, as I have already described. Amongst Erb's cases there was one in which four thousand grammes of ung. hydrarg. had been used in the course of six years, without any ill effect. Erb specially recommends repeated short courses of treatment, using in the intervals baths, electricity, tonics (especially strychnine). On the other hand Leredde, Lemoine, Coester, and others advocate very energetic courses of treatment, as does Duhot, who has several times procured "recovery" in this way.

I regard treatment with mercury as dangerous in optic atrophy, and many ophthalmologists (Wecker, Silex, and others) have expressed similar views. No objection can be taken, however, to the experimental use of preparations of iodide in these cases (iodide of potassium, iodipin internally or subcutaneously, inunctions with iothin).

I have endeavoured to procure an extract of cells of the posterior root ganglion and to treat tabetics with it, but I had soon to give up the attempt on account of the great difficulty of obtaining the material.

As regards the *treatment of the various symptoms*, we find that the lightning pains are most frequently amenable to medicinal measures. We have first to discover whether wet packs, firm bandaging, rubbing in of chloroform, dry cupping, massage, the faradic brush, or Franklinisation, have a soothing influence. Some patients find cold beneficial. Munter recommends as the most effective form of bath a one per cent. brine bath of 27 to 28° R. In a few cases vibration-massage has done me service in alleviating the lightning pains and the girdle sensation. These methods do not usually have the desired effect, and we have to resort to drugs. The following frequently prove useful: salicylic acid, antipyrin, antifebrin, phenacetin, anæsthesin, and very specially pyramidon [1] and aspirin, two drugs which I find particularly valuable as antineuralgics, and which usually make it possible to dispense with morphia. This should be regarded as the last resort, but indeed it is only heroic natures who can overcome the most severe paroxysms without subcutaneous injections of morphia.

French writers in particular recommend "rachicocainisation," *i.e.* the injection of a weak solution of cocaine into the subarachnoid space of the spinal cord, for the lightning pains, crises, hyperæsthesia, etc.; but on account of the dangers connected with it, and the unpleasant after-effects this method should be adopted only in exceptional cases. To begin with the smallest possible doses should be used—about 0·002-0·003 of cocaine or tropococaine. It need not be said that the most careful

[1] I saw a tabetic who had taken pyramidon in daily doses of 1 to 3 g. for three years, without any real ill effects. But tolerance such as this is an exception.

antisepsis is necessary. Less energetic, though still sufficiently so, is the method advised by Sicard and Cathelin, of injection into the epidural space of the sacral canal (compare chapter on treatment of neuralgia). Negro has produced an alleviation of the lightning pains by santonin, in doses of 0·015, as Bricage [1] has done with larger doses.

Raymond and Zimmern extol the pain-soothing effect of radium. I have employed it in many different kinds of neuralgia, and also in lightning pains with varying results, and am still doubtful whether there is a specific influence at work as well as a suggestive one.

The attacks of vomiting defy all medicinal treatment, yet we may resort to the ordinary drugs for alleviating the nausea. In the early stages morphia greatly reduces the severity of the attacks. Cerium oxalate in doses of 0·05 to 0·1 has been recently recommended (Ostankow).

In some cases I found strychnine to have a good effect, and Basch, who tried the drug on my recommendation, has also found it of use in a few cases (dose 0·001-0·003 subcutaneously). But it very often fails to do any good. The drugs (opium, codeine, dionin) may be used in the form of suppositories. Basch has also in Boas's clinique found some effect from antipyrin (0·25 every hour). He further advises that a few drops of a solution of cocaine or a drop of tincture of iodide in a teaspoonful of water should be tried for the vomiting. Lemoine recommends methylene blue in large doses. In one of my cases a winter sojourn in Egypt stopped the crises for a long time. Another, who had already become subject to the morphia habit, entirely lost the crises (see above).

Injections of cocaine into the epidural and subarachnoid spaces of the spinal canal have also been used for the crises. I have only once felt justified in advising this method. I consider the method advocated by Debove, of simple lumbar puncture with withdrawal of about 30 c.cm. of fluid, still less worthy of imitation.

With regard to the methods just recently advocated by some writers, such as Lhermitte-Lévy (*R. n.*, 1907), Pope (*Brit. Med. Journ.*, 1907), of injections of fibrolysin and of water, alcohol, and stovaine into the subarachnoid space of the spinal cord (Brissaud), we must wait for the results of further observations.

Vallas-Cotte (*Lyon méd.*, 1906) advises stretching of the solar plexus. Alexander reports the favourable influence of subcutaneous injections into the intercostal spaces according to Schleich or Lange.

The main thing is to feed up the patient in the interval between the gastric attacks—a point upon which Leyden lays special stress—in order that he should be in a condition to make good the loss of strength during the crises. The carrying out of this plan has proved of great value in some of my cases. Charcot advocates the use of the button cautery, which he recommends even for the primary disease. Painting with cocaine may be tried for the laryngeal attacks. In a few cases tracheotomy has proved necessary, but such a measure is very seldom indicated. One of my patients who had for years been distressed by these laryngeal attacks discovered eventually that he could cut the attack short by making swallowing movements instead of coughing to clear his throat.

The ataxia is most effectively treated by Frenkel's method.

The joint affections may require mechanical orthopedic treatment (supporting apparatus, particularly Hessing's splint apparatus), less frequently surgical interference—puncture, arthrectomy (Czerny, Wolff, Ullmann). Operations involving loss of blood are dreaded by most surgeons.

For perforating ulcer Chipault recommends stretching of the posterior tibial nerve, especially of its plantar branches; he, Duplay, Sick, and others

[1] *Thèse de Lyon*, 1906.

having used this with success. Crocq recommends faradisation of the posterior tibial nerve. Kindler advises hot-water irrigation.

With regard to the *prevention* of tabes one thing only is to be said, namely, that thorough treatment of syphilis would from all our experience appear to be the most effectual prophylactic measure. It is however by no means an absolute protection.

Spastic Spinal Paralysis (Lateral Sclerosis)

This is clinically a well-defined disease. Our knowledge of its pathological and anatomical nature is still, however, insufficient, though it has been widened and consolidated in recent years. Erb [1] and Charcot, to whom we owe the first clinical description, regard it as having an independent nosological existence. Strümpell [2] in particular has given us a wider view. Although the disease in its pure form is most frequently found in childhood, it is advisable, for reasons that will be discussed later, to consider first the type which appears in later life.

In evolution and course the disease is as a rule eminently chronic. The lower extremities are almost always the first to be affected. The early symptoms are slight. The patient becomes fatigued after a long walk, feels a kind of *strain* and *stiffness* in the legs, "as if the tendons were too short." This difficulty is particularly noticeable in complicated, forcible, and rapid movements of the legs, as in dancing, skating, hill-climbing, and as a rule it affects one leg more than the other, or may for a time be limited to one leg. The stiffness increases very slowly but steadily, until the affection of the gait becomes apparent to others.

If we examine the patient at this stage, the earliest discoverable symptom is an *exaggeration of the tendon reflexes*. At the same time or soon afterwards a slight *rigidity* becomes apparent in forced passive movements, and there may be slight motor weakness. As Strümpell especially has repeatedly pointed out, the latter may be entirely absent for a considerable time, even although the muscular stiffness itself has begun to limit the freedom of active movements. These symptoms gradually increase until they constitute *marked spastic paresis*, as has been described on page 6 *et seq.* Ankle and even patellar clonus can usually now be elicited. Sometimes the contraction provoked by percussion of the patellar tendon may extend to the thigh muscles (particularly the internal rotators and the adductors) of the other leg. In most cases Babinski's sign and the dorsal leg phenomenon appear at an early stage; the former is rarely absent. The cutaneous reflexes are usually exaggerated, the corresponding muscular contractions being more marked and often increased in extent, like the reflexogenous zones. As a rule there is no antagonism between the cutaneous and tendon reflexes, as some French writers describe. The muscles retain their normal size and electrical excitability. The power of walking is retained until the later stages, but the pace grows steadily slower, the feet are raised from the ground with ever-increasing difficulty, and eventually the patient drags his feet with short steps along the ground. The rigidity may be so great that it can hardly be overcome in the examination, and what has been called a "muscular ankylosis of the joints" (Strümpell) is produced. The muscular tension usually affects the extensors of the thigh and leg

[1] *V. A.*, Bd. lxx. and *Z. f. N.*, xxiii. [2] *A. f. P.*, xvii.; *Z. f. N.*, iv. and v.; *Z. f. N.*, xxvii.

in a higher degree than the flexors, so that the extremities are in a position of extension. It is only in the last stage that a flexor contracture sometimes takes the place of the extensor contracture. Active movements are sometimes accompanied by spastic tremor, and the muscular spasm may for a time be so exaggerated that the extremity remains in a condition of tonic contraction and cannot be released from it.

If the patient tries to raise himself from the dorsal position there is usually an extreme degree of flexion of the hip, and consequently the limb is raised to a varying extent from the bed, This symptom, which I described (*Charité-Annalen*, 1889), was later used by Babinski (*Gaz. des hôp.*, 1900) as a criterion in differential diagnosis. The tendency to associated movement in the tibialis anticus in voluntary flexion of the hip-joint, especially if against resistance (Strümpell's tibialis phenomenon) is one of the common symptoms of spastic paresis. If the patient is requested to raise the leg towards the trunk by flexing the hip- and knee-joints, a visible and palpable contraction of the tibialis anticus takes place which cannot be suppressed, and which becomes still more marked when the movement is carried out against the resistance of a hand placed on the thigh. E. Müller (*Z. f. N.*, xxix.) sees in this an original physiological associated movement (in the new-born) which is gradually suppressed, and only returns under pathological conditions. The phenomenon is, however, inconstant, and it is by no means entirely confined to spastic conditions (Oppenheim, Flörsheim). Other muscles, especially the extensor hallucis longus, may be involved in this associated movement.

Pain is absent or slight, and is merely the result of the muscular tension. The *sensibility*, and the *functions of the bladder and bowels*, are unaffected in typical cases, and therefore the *spastic paralysis is the only symptom of the disease*. Later it often extends to the upper extremities; the arm movements become slow and difficult, and there is muscular rigidity and exaggeration of the tendon reflexes. If one leg has from the beginning been more affected than the other, it is the arm on the corresponding side which is first affected. It seems probable from some observations (Strümpell, E. Müller, Kinichi Naka) that this spastic condition may in typical cases come to involve the muscles of articulation and deglutition, and thus represent a pure spastic form of bulbar paralysis; but this abortive form has undoubtedly a great tendency to change into amyotrophic lateral sclerosis (see below). Strümpell has described attacks of laryngeal spasm, and has attributed them to a spastic condition of the laryngeal and respiratory muscles. He describes also bulbar laughing and weeping.

Cases by Spiller and Mills [1] show that there is probably also a *unilateral* type of this disease. I myself have seen a small number of cases which might be so regarded.

The duration of the disease is, one may say, almost without limits. Erb knew cases which had existed ten to twenty years without the appearance of any further symptom. The disease may even cease to progress.

In the great majority of cases, which at first appear to be spastic spinal paralysis, symptoms arise later which show that behind this picture there lurks another disease of the nervous system, chiefly disseminated sclerosis, chronic myelitis, combined-system disease, compression of the spinal cord, less frequently amyotrophic lateral sclerosis or a brain disease (hydrocephalus, etc.). This fact deserves the greatest consideration. We should continually bear it in mind and endeavour to *unmask the so-called spastic spinal paralysis*. Any sign that does not strictly accord with the clinical features of spastic paresis is an indication of some other

[1] University of Pennsylvania, 1906.

underlying disease, and disseminated sclerosis in particular tends in the initial stage, which often extends over several years, to assume the character of spastic spinal paralysis. On the other hand, Strümpell maintains that the presence of slight disturbances of sensibility—especially in the late stages of the disease—is not sufficient to upset the diagnosis. He has observed disturbances in the function of the bladder which he regards as spastic, and includes among the rare symptoms of the disease. It is nevertheless always advisable to be exceedingly reserved, even in cases of the pure clinical forms, as to the nature of the pathological lesion.

Pathological Anatomy

Before any cases of this disease were examined post-mortem, the suggestion was made that a primary degeneration of the lateral columns, and especially of the pyramidal tract, formed their basis (Fig. 97). For a long time there was no positive proof of the correctness of this supposition. In pure cases of this kind the patient lived a long time, and his symptoms were not so severe as to keep him under prolonged treatment in hospital. The cases upon which an autopsy was made were for the most part not clinically pure, and thus did not constitute a pure affection of the lateral columns, but were due to extensive lesions in which the pyramidal tracts were involved along with other areas. These lesions include sclerotic foci, combined column degenerations, chronic myelitis, or combined affections of the anterior horns and lateral columns (amyotrophic lateral sclerosis), tumours, etc. There are, however, a few cases (those of Minkowski, Strümpell, Donaggio, and especially those of Dejerine and Sottas [1]) which have more or less completely justified the conjecture, and proved that there is a *primary isolated lateral sclerosis or disease of the central nervous system in which the anatomical changes are limited for a considerable time or even permanently to the region of the lateral columns, and in which the pyramidal tract is involved in its whole extent.* These were isolated cases of combined disease of the lateral columns and Goll's tract (the latter but slightly involved) which had presented the clinical picture of spastic spinal paralysis in almost entire purity.

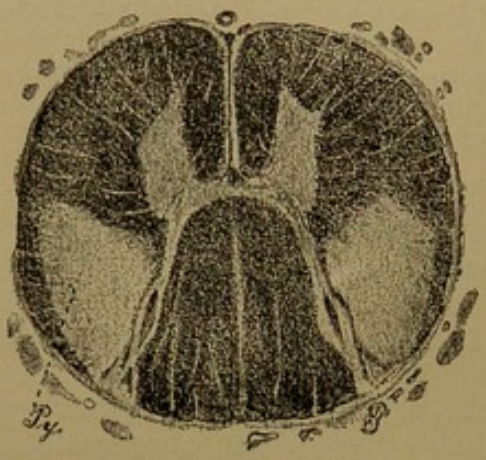

Fig. 97.—Degeneration of lateral pyramidal tracts. (Weigert's method.)

It is known that it also appears in paralytic dementia, but the clinical symptoms are seldom fully developed.

In a case of carcinomatous cachexia, E. A Meyer (*Z. f. N.*, xvi.) observed the symptoms

[1] The case described by Friedmann is of little value on account of the obliterating endarteritis and brain foci; the grey matter was not intact. There are also many objections to the case described by Ida Democh; Erb includes as relevant the cases described by Morgan-Dreschfeld and Bischoff. One or two of Spiller's cases should also be included. Mott and Tredgold speak also of a primary degeneration of the pyramidal tract, but their description applies chiefly to amyotrophic lateral sclerosis. Strümpell has recently given us further clinical and anatomical contributions (*Z. f. N.*, xxvii.).

of spastic paralysis, and found the basis to be a sclerosis of the lateral columns, with special involvement of the pyramidal column.

Rothmann (*D. m. W.*, 1903), on the basis of his experiments, has expressed himself as opposed to the prevailing view, which attributes the spastic symptoms to degeneration of the pyramidal tract. He ascribes the exaggeration of the tendon reflexes alone to the lateral column affection, tracing the spasms and paresis to other, still unexplained factors. We do not, however, regard his views as convincing.

Strümpell has made us acquainted with a *hereditary, family form of spastic spinal paralysis*. The disease, which specially affects the males in a family and may be transmitted through generations, usually commences between the ages of thirty and forty with a pure spastic disturbance of movement of the legs, and only as a rule after many years becomes real spastic paresis and paraplegia. The hypertonia has to the end, however, the predominance over the paresis. The muscles of the arms, lips, and tongue are affected more rarely and much later. The disease may extend over a period of thirty to forty years. In the later stages slight sensory disturbances especially involving the temperature sense may appear, and there may be also slight weakness of the bladder. This constitutes an essential difference between this disease and pure spastic spinal paralysis, and it finds its anatomical expression in the circumstance that in the cases hitherto examined there has been as a rule, in addition to the degeneration of the pyramidal tracts, a slight degeneration of other systems, especially of the cerebellar tract, Gowers' tract, and also in the anterior pyramidal tract. Strictly speaking, therefore, we are not dealing with a lateral sclerosis, but with a combined system disease (see following chapter). The pyramidal degeneration is, however, so predominant, and the affection of the other fibre-systems so slight and inconstant, that we may, with Strümpell, regard this disease as of the nature of a "primary sclerosis of the lateral columns." It is in these cases of the hereditary-family category that the spastic bulbar symptoms described above have sometimes been observed. This applies also to a type, at least related to this condition, which Ballet and Rose[1] have described. It should be noted on the other hand that the hereditary-familial character is not always present, and further, that by no means every form of spastic paralysis of a hereditary and family nature shows an anatomical basis of the type described by Strümpell, the symptoms indicating that a great part of the disease is due to affection of the brain or of the cerebro-spinal nervous system (see section on Little's disease). So far as the anatomical basis goes, the cases described by Bischoff are most like those of Strümpell.

Differential Diagnosis.—As we have already seen, every clinical case of spastic spinal paralysis requires very careful consideration in this respect. It may form the residue of a previous myelitis or compression-myelitis; this can usually be ascertained from the history. I have had the opportunity, for instance, of ascertaining in the case of a man of fifty-nine, who showed the clinical features of spastic spinal paralysis, that he had suffered at the age of fourteen from an acute transverse myelitis, which left behind only the spastic symptoms. Compression of the spinal cord, caused by tumour or affection of the meninges and spine, is as a rule revealed by other symptoms; there may be deformity, sensitiveness of the spinal column to pressure, girdle pain, sensory disturbances, or bladder weakness, etc., but spastic paraparesis may be the first sign of

[1] *Nouv. Icon.*, xviii.

the compression. In order to differentiate it from disseminated sclerosis, we must refer to the symptomatology of this disease. The characteristic cerebral symptoms should be specially considered. All doubt is removed if ophthalmoscopic examination reveals any change in the optic nerve. If degenerative atrophy is present (first in the small hand muscles) there is no danger of confusion between amyotrophic and pure lateral sclerosis.

In the course of *hysteria* a paralysis of the legs, combined with contracture, may appear, but it almost always has an acute onset and follows some mental excitement or an attack of spasm. A spastic condition of the muscles, showing the characteristics described on p. 6 *et seq*, never so far as my experience goes, occurs in hysteria, and this is a point which receives too little consideration. In hysteria there is frequently an exaggeration of the tendon reflexes, which may even become a clonus, and which cannot always be distinguished at once from real clonus,[1] although the characteristic rigidity of the muscles provoked by sudden movements is absent. Or there may be a contracture, characterised as hysterical by its absolute rigidity, its defiance even of the most gentle attempts to overcome it, and by its dependence upon mental processes. A spastic gait in its typical form does not occur in hysteria, so that the expert, after seeing the patient walk, finds no difficulty in determining whether he is suffering from an hysterical paresis or a spastic spinal paralysis. Even before Babinski had described his sign there were seldom great difficulties in arriving at this differentiation; but it must be admitted that in this symptom, as in the almost equally important one described by myself, we possess very valuable guides to the diagnosis. Where there is the typical sluggish dorsi-flexion of the great toe, there is always a true spastic condition and an organic disease. Naturally the other signs are as a rule distinctly present also. I do not lay by any means so much weight upon a negative result of the examination, as I have in some cases found the sign to be absent although spastic paralysis was certainly present. There are also numerous cases in which the results of the test are so indefinite that they can be of no aid in diagnosis. I must admit also, as I have said before, that the phenomenon described by myself may also be absent in spastic conditions. The slowly progressive development characteristic of spastic paralysis is also unknown in hysteria. Finally, hysterical paraparesis and contracture are almost always combined with other signs which reveal the causal disease, although their presence by no means proves that hysteria is the *only* disease present.

Sommer maintains that he has found a fundamental difference in the duration of the latent period of the tendon reflexes in organic and functional nervous diseases. It remains to be seen in how far this statement will prove to be of value.

Although weakness and exaggeration of the tendon reflexes are frequently found in *neurasthenia*, the symptoms of muscular rigidity, the difficulty of passive movement, Babiniski's sign and my own, are always lacking. In my experience, also, spastic spinal paralysis is much more

[1] See also page 8. Hysterical ankle clonus is usually spurious, rapidly exhausted, inconstant, and disappears when the attention is diverted. I must, however, maintain, in opposition to Babinski's view, that a phenomenon corresponding to real ankle clonus may develop in a neuropathic subject, and I am doubtful, also, whether exact physical examination by means of graphic methods can always lead to a definite decision.

frequently mistaken for a hysterical or neurasthenic condition than *vice versâ*.

Age and Causes.—Spastic spinal paralysis of the adult usually appears between the ages of twenty and forty. The causes are but partly known. A history of *syphilis* is not uncommon, and an injury that has occurred many years before has been blamed in some cases. Some of the hereditary forms also (see next chapter) may first develop during adult life. In the case of a lady of forty-eight, for example, in whom the disease had appeared within the last few years, I discovered that her twin-brother had congenital club-foot. The symptoms may develop in the *puerperium*, after *acute infectious illnesses*, and following *lead-poisoning*.

Whether the cases quoted by Bechtold as originating in lead-poisoning and running a favourable course belong to this group, I am unable to determine.

Poisoning with various kinds of lathyrus, especially with *L. sativus* and *cicera*, from which a kind of bread is made in India and Algeria, may, according to the observations of Cantani, Bouchard, Proust, Chabline, Mingazzini, Goltzinger and others, produce a nervous disease which is mainly, though not exclusively, manifested by the symptoms characteristic of spastic paraparesis. Nothing is as yet definitely known as to the anatomical basis of the symptoms, but the idea has been expressed by Trachtenberg, Tuczek, and others that there is an affection of the lateral columns. Mirto has induced in animals a combined column disease of the pyramidal tract and Goll's column by lathyrus-poisoning, whilst Goltzinger and others have had negative results.

Other toxic forms of spastic spinal paralysis have been described (Muchin). The anatomical basis is, however, uncertain. I doubt very much whether a simple exaggeration of the tendon reflexes, which is often to be found in arthritis (even in the gonorrhœal form) can develop into spastic spinal paralysis.

"*Senile spastic paraparesis*," which occurs in arterio-sclerosis, will be discussed in a special chapter (see section on senile paralysis).

Heuck describes a form of spastic paralysis, commencing acutely with pain in the back and recovering in the course of a few weeks; but nothing is definitely known as to the nature and cause of this affection, which is not mentioned by other writers.

Congenital or Early acquired Spastic Paralysis

(Congenital Spastic Stiffness of the Limbs, Little's Disease)

Although this disease is so closely related to paralyses of spinal origin, there can be doubt that in the majority of cases its cause is to be sought in a brain affection which involves the pyramidal tracts in the spinal cord, and either produces in them a condition of sclerosis or atrophy, or causes them to remain incompletely developed. If we remember that the pyramidal tracts form a direct continuation of those fibre systems of the brain which, originating in the motor centres, pass into the internal capsule and thence through the cerebral peduncle, the pons, and the medulla oblongata, we can understand that a morbid process which interrupts this tract at any point whatever will produce practically the same symptoms. Moreover, a destruction of the motor centres in the brain causes a descending degeneration of these nerve tracts, and may thus give rise to the same symptoms. The only standpoint for a differentiation is in the fact that a brain disease is often not limited to this (motor) region, but generally extends beyond it.

Let us first consider the *clinical* aspect. Even in cases where the morbid condition must be regarded as congenital, symptoms pointing to involvement of the muscular system are not always recognised directly after birth. If the lesion is a very marked one, it will certainly be early manifested by the difficulty of separating the thighs, which are closely pressed together, and which at once resume the position of adduction. The condition is frequently first recognised when the child begins to attempt to walk, or shortly thereafter.[1] He learns to walk late and his gait is evidently pathological. The lower limbs are held stiffly, the thighs rotated inwards, and so strongly adducted that the knees touch each other and in walking rub against each other. This indeed may be so very marked that in walking the thighs may cross each other. Whilst the hip- and knee-joints are generally but slightly flexed, the pes equinus position is usually very pronounced, so that in walking the heels are raised in the

FIG. 98.—Mode of standing in simple spastic paralysis of childhood.

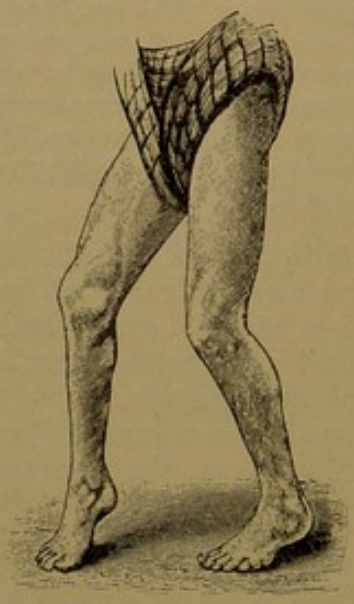

FIG. 99.—Mode of walking in simple spastic paralysis of childhood.

(After Seeligmüller.)

air, and the little patient can only push himself along on the toes or the balls of the toes (Figs. 98 and 99).

The limb is moved as a whole, the pelvis raised and lowered, or pushed very much forwards on the side of the advancing leg.

Even in lying down and standing up the legs are in many cases of this kind moved as a whole without any flexion of the joints (Oppenheim).

If the patient is examined in the dorsal position the well-known phenomena of rigidity and motor weakness are observed, but it should be noted that these are not necessarily equally marked; the former may be very severe, whilst the motor power is little affected or even quite normal. Many writers go so far as to include under Little's disease only those cases in which the motor disturbances are due to muscular rigidity

[1] If we include here the cases of hereditary familial stiffness of the limbs, which at least are very closely related to the congenital form, we have to remember that the disease may occur even in adult age and may have a very chronic, progressive course.

and not to weakness. This distinction cannot, however, be sharply adhered to.

The knee jerk is always exaggerated; ankle-clonus is often absent. The patella is usually drawn somewhat upwards, and the patellar ligament appears to be lengthened (Schulthess, Joachimsthal). This is specially evident when the knee is flexed to an acute angle. Babinski's sign is usually present, but it should be remembered that in very young children dorsal flexion of the toes represents the physiological reflex movement. Oppenheim's sign, as well as the Bechterew-Mendel sign, is not constantly present. In the sitting position the rigidity and the spasm of the extensors are sometimes shown by the fact that the knees cannot be fully flexed, the legs remaining poised in the air. Sitting may in consequence become quite impossible.

Sensory changes, bladder disturbances, etc., are not present, or appear only in rare, atypical cases (Good, Auché-Campanol). The symptoms affect the motor sphere exclusively.

The arms are involved in many cases, but in others are completely spared. If they are affected their attitude is usually a peculiar one. The upper arm is strongly adducted, the fore-arm flexed, sometimes also pronated, the wrist flexed or extended, the fingers flexed in every joint or in the interphalangeal joints only. This may vary in a few cases, but the attitude is always a forced one, at once revealing to the expert the condition of contracture. Passive movements are difficult, the tendon reflexes are exaggerated, the movements slow, awkward, and feeble. The weakness of the arms, however, is not so great as that of the lower extremities. In one case I found the shoulder blades so fixed that they could hardly be moved on the thorax (fixed shoulders).

Deformities of the spinal column (kyphosis, scoliosis) sometimes occur in these cases. Undescended testicles and other anomalies of development may be found; in one case I saw webbing of the toes.

In pure cases of spastic paresis of the lower, or of all four extremities, the symptoms must correspond completely with those of spastic spinal paralysis. There must be no indication of *non-development* or *incomplete development* or *degeneration of the spinal pyramidal tracts*.

In such cases other symptoms, however, are generally present, which at once reveal the *cerebral* origin of the disease (see chapter on infantile cerebral paralysis). Among these are :—

1. *Strabismus.*—There is much difference of opinion as to its causation. Frequently it is merely a concomitant squint due to some error of refraction. In other cases the cause is a spastic condition of some of the eye-muscles. Sometimes there is paralysis of the ocular muscles.

2. *Disturbances of Speech.*—The rigidity sometimes involves the muscles of articulation, and causes difficulty and indistinctness in speech : or the rigidity may be completely of the bulbar type and may in exceptional cases be combined with difficulty in swallowing. There is one class of case in which, besides the spastic symptoms, there is only strabismus, or a combination of strabismus and speech affection. In others several additional symptoms are present.

3. *Mental Disturbances.*—Whilst in many cases the intelligence is unaffected, there is in others weakness of the intellect which may amount to idiocy.

4. *Epilepsy.*—This may appear shortly after birth or during later life.

5. *Choreo-athetotic Symptoms.*—These may affect all four extremities. As a rule they predominate in the upper extremities, and not infrequently appear in the muscles of the face and tongue, forming the symptoms which are earliest recognised.

These cases of spastic-athetotic paraplegia are easily misunderstood, and are frequently confused with chorea.

The family form of spastic rigidity of the limbs may correspond with the pure type of spastic spinal paralysis, but it frequently also bears the stamp of cerebral or cerebro-spinal disease, or becomes atypical on account of unusual symptoms, and then enters into close relation to other affections of a hereditary family character (observations of Pelizæus, Jendrássik, Kollarits, Thomson, Ballet-Rose, Modena).

Etiology and Pathological Anatomy.—The most important cause is premature birth, the significance of which Little had recognised. A *difficult* and *protracted labour*, delayed descent of the head, and the use of forceps may be causes of the disease. Blood relationship of the parents has been blamed. The disease also appears in twins. Inflammatory processes or inhibitions of development in the motor areas occurring during fœtal life are frequent causes. In some cases (Friedmann, De Amicis, Rolly, Dejerine, and others) the disease is attributed to hereditary syphilis. I have also seen cases of this kind in which the picture, however, usually differed from that of Little's disease, changes in the pupils or other complications being present in addition.

Compression of the skull during labour may cause meningeal hæmorrhages, which are limited to the region of the motor zones or even to the leg centres (MacNutt, Haushalter, and others). It is thought that injuries, even when they do not cause hæmorrhage or a gross lesion of the brain, usually affect the pyramidal tracts, and produce in them a condition of degeneration or an inhibition of development, and that this finds its expression in "congenital spastic rigidity of the limbs." In other cases it is encephalitic processes which produce the atrophy, induration of some of the cortical convolutions, or even formation of cavities (porencephaly). If these affect symmetrical areas of the motor zones, we have the clinical result of congenital spastic rigidity of the lower limbs, or of all four extremities.

Inflammatory processes and other affections may involve the motor area of the brain, even in childhood. They are usually limited to one side, and then produce infantile spastic hemiplegia (see corresponding chapter). In exceptional cases they affect both hemispheres, and there then develops a bi-lateral, spastic hemiplegia, usually combined with choreic or athetotic tremors, and so forming a group of symptoms which is again closely related to spastic rigidity of the limbs, except that it is acquired in childhood and is not congenital.

In some cases we cannot say definitely what is the pathological change underlying the disease. The less pure the clinical picture is, and the more prominent the signs of a brain disease, the more probable is it that they are due to an extensive cortical process of encephalitic nature or to an inhibition of development.

The question as to whether there is also a congenital spastic *spinal* paralysis, *i.e.* a congenital spastic paralysis of purely spinal origin, in which the inhibition of development is limited to the motor tracts in the

spinal cord, has received different answers. Pathological anatomy was until recently silent. In spite of this, however, Gehuchten, Souques, and above all Erb, have maintained that there is a spinal form of this disease, and Dejerine has succeeded in a typical case (and recently in a second) in demonstrating that the disease may be due to a spinal lesion, viz., to a focal disease in the cervical cord occurring during fœtal life, with secondary degeneration of the pyramidal tracts.

To this case others have recently been added (Spiller). But it must be admitted—and this has lately been emphasised by Haushalter-Collin (*R. n.*, 1906), that only a small group of cases belong to the spinal form.

Apart from these, the cases which have been examined post-mortem showed the above described cerebral changes, whilst the pyramidal tracts of the spinal cord presented a simple atrophy of the fibres (observations of Binswanger, Ganghofner, Philippe-Cestan, etc.) oftener than a sclerosis or degeneration.

The results of histological investigation in a case by Berghinz (*Riv. di Clin. psych.*, 1903) were quite negative.

The hereditary, family character seems not to exclude a spinal lesion (Strümpell, Erb, Bischoff, Newmark [1]), although, even in the majority of cases in which the spastic paralysis of childhood and youth appeared in several members of the family, the lesion was a cerebral or cerebro-spinal one (observations of Sachs, Schultze, Newmark, Bernhardt, Ganghofner, Pelizæus,[2] Freud, Jendrássik, Lorrain, Sutherland, Krafft-Ebing,[3] Pesker, Rolly, Kühn, Spiller, Raymond, Ormerod, Bischoff, Cestan-Guillain, etc.).

With regard to the differential diagnosis, compare the following chapter and that on diplegia and disseminated sclerosis. The condition described by Nageotte-Wilbouchewitsch as "raideur juvenile" (*Rev. de Méd.*, 1905) is as yet so undefined that it would be out of place to discuss it from the point of differential diagnosis.

Prognosis.—With regard to the congenital or early acquired form of this disease, the prognosis varies with the severity of the symptoms and with the degree in which they conform to the type of spastic spinal paralysis. If there is spastic paresis of the legs without any complication, this may improve, to a certain degree, in later life. The power of free movement in the arms and the normal intelligence enable the patients to follow many callings, and sometimes to attain old age. There are cases of this kind which are so slight that the disease does not for a considerable time become evident to the eye of the layman. The more marked the paralysis and contracture, the more severe is the disease. Involvement of the upper extremities, with athetosis, with mental disturbances, and with epilepsy, are particularly unfavourable.

In the family form of spastic rigidity of the limbs, the tendency to progression is not always marked; the course may be stationary, or even to a certain degree retrogressive (Spiller, Newmark). In the cases where the Little's disease is due to inherited syphilis, the prognosis is somewhat more favourable, as the observations of Gallois-Springer [4] and others show. I myself have found that in typical cases specific treatment

[1] *Z. f. N.*, xxvii. (see literature here).
[2] *A. f. P.*, xvi.
[3] *W. kl. W.*, 1892.
[4] *R. n.*, 1903.

has no effect, but that it gives excellent results in *cerebro-spinal* syphilis of childhood with the symptoms of spastic paralysis.

Treatment.—Early and forced attempts at walking should be forbidden. Even in adult age spastic paresis requires rest and care for the muscles. Treatment depends mainly on the cause and the nature of the disease.

The spasms as a rule diminish in warm baths, but the lessening of the rigidity is merely temporary. The following measures may be useful: massage, gentle rubbing and stroking of the muscles, slow passive movements (also in warm baths) and regulated gymnastics which do not put too great a strain upon the patient, and which counteract the contractures. In the case of congenital rigidity of the limbs, wonderful results may be obtained by such means. It may, however, be necessary to practise tenotomy on muscles which are in a state of contracture. This treatment, specially advocated by Rupprecht, is particularly successful when the contracture constitutes the main hindrance to movement, whilst the paralysis is less pronounced. If there is a high degree of paralysis, surgical treatment is quite out of place. Tenotomy of the adductor tendons, of the fascia lata, the Achilles tendons, etc., and finally lengthening or shortening of the tendons must be followed by orthopædic treatment, which will gradually relax the extremities from their position of contracture. A special splint, which specially counteracts contracture in the flexors of the knee and the pes equinus position, has been recommended by Heusner, and another by Hoffa. Recently Lorenz and Hoffa (as well as Vincent, Lebrun, Gibney, Redard, and others) have strongly advocated surgical-orthopædic treatment of congenital rigidity of the limbs, with tenotomy, myotomy, myorhexis, etc., and forced straightening or over-correction of the position of contraction, and they report excellent results of these measures.[1]

In the most severe cases Lorenz does not even shrink from section of the obturator nerves. I have had the opportunity of examining a case in which section of the anterior ramus of the obturator nerve has almost completely overcome contracture of the flexors and adductors. I have also seen a girl of twenty, who had in her seventh year been successfully treated by Volkmann by means of surgical-orthopædic measures. In another case in which the trouble had developed after smallpox in childhood, this surgical-orthopædic treatment, carried out by J. Wolff on my advice, was of real benefit, although the patient was already in middle life.

Transplantation has also been used in cases of this kind with good results (Vulpius, Hoffa, Reichard, Tubby, Perthes, and others; compare the therapeutic section in the chapter on acute anterior poliomyelitis, and in that on infantile cerebral paralysis). This specially applies to strengthening of the abductors of the thigh at the expense of the adductors, of the extensors of the foot at the cost of the plantar-flexors, etc. Hoffa,[2] however, in his latest work on this question, expresses himself as being sceptical with regard to the value of tendon transplantation in spastic paralysis.

Drugs have practically no influence on spastic conditions; but we may prescribe by way of a trial preparations of bromide, belladonna, hyoscin (not for children), and iodide of potassium. Solanin and veronal have also been recommended.

[1] See Hoffa's recent work in the *D. m. W.*, 1906. [2] *A. f. kl. Chir.*, Bd. lxxxi.

Direct injection of narcotics (morphia, and particularly cocaine) into the subarachnoid space of the spinal cord seems, from the observations of Jaboulay and others, to be an effective method of transforming the spastic condition for a time into a flaccid one; but the unpleasant collateral effects and the dangers of the method are too great to make it worthy of recommendation.

This also applies to stovain, which Goldscheider [1] uses in the same way, and which also has a merely transient effect.

The attempts of Brissaud-Sicard-Tanon (*R. n.*, 1906) to combat the spastic contracture by injection of alcohol into the sheaths of the peripheral nerve-trunks should not be imitated.

Combined Disease of the Posterior and Lateral Columns of the Spinal Cord

Pathology.—In typical cases of tabes dorsalis the affection of the white matter is limited to the posterior columns. In rare cases, such as one which I examined, the lateral cerebellar tract is involved, and we then find in Clarke's column not atrophy of the fibres only, but also destruction of the ganglion cells. Kattwinkel [2] and others have found the same condition. It does not give rise to any recognisable symptom.

It often happens, however, that various columns of the spinal cord, *e.g.* the posterior and lateral columns, are simultaneously affected by disease (Kahler and Pick,[3] Westphal,[4] Strümpell [5]). The process is sometimes a systemic one, *i.e.* the various systems contained in the posterior and lateral columns are affected—Goll's and Burdach's columns, the lateral pyramidal and the lateral cerebellar tracts, or Goll's column, the pyramidal tract, and the cerebellar tract, whilst the anterior pyramidal (Fig. 102) and Gowers' tract are but rarely involved. As a rule, however, a systematic distribution of the degenerative process is not apparent (Figs. 100, 101, 102). As the degeneration is always confined to the two columns of the spinal cord, and affects these in their whole extent, or over a great part of their course, we are justified in thinking that in some of these cases, at least, the disease appears to be diffuse, merely because the site, the course, and extent of the different systems are liable, as is well known, to individual variations.

With regard to the conception of these affections there are still great differences of opinion among investigators. Some (Leyden and his school) would include them under myelitis. A dorsal myelitis with secondary degeneration of atypical extension may simulate the anatomical picture of combined system disease. Erb remains sceptical as to the existence of combined system disease. I myself have described a case (*B. k. W.*, 1896) in which vascular processes in the brain and spinal cord had caused diffuse changes with secondary ascending and descending degeneration, which by their coincidence produced an apparently combined system disease. Henneberg (*A. f. P.*, xxxii.) relates similar conditions in arterio-sclerosis and nephritis. Ballet and Minor (*Arch. de Neurol.*, 1884) had already shown that vascular disease in the region of the posterior and lateral columns of the spinal cord may be followed by a diffuse myelitis limited to these columns.

Dejerine, Marie, Jacob, Grasset, Mayer, Wagner, Teichmüller, Rothman (*Z. f. N.*, vii.), Bruns, Werner, Kattwinkel, Burr and M'Carthy, Marinesco, Taylor-Watermann, Richmond-Williamson, Ormerod, and others have been occupied with this question. Russell, Batten and Collier (*Br.* 1900), whose observations have lately been confirmed by Henneberg (*A. f. P.*, Bd. xl.) and Nonne, have made specially valuable contributions from both clinical and anatomical points of view.

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[1] *Therap. d. Geg.*, 1905.
[2] *A. f. kl. M.*, Bd. lxxv.
[3] *A. f. P.*, viii.
[4] *A. f. P.*, viii. and ix.
[5] *A. f. P.*, xi., xvii.; *Z. f. N.*, xiv.

Changes in the grey matter have been found in a few cases in Clarke's column by Strümpell, Westphal, Oppenheim, Mayer, Hochhaus, Pal ; [1] and in the anterior cornua by Sioli, Münzer, Thomsen, Oppenheim, Luce and Pal—but it is by no means proved that they form the starting-point of the disease. Henneberg (*M. f. P.*, xvi.) describes degeneration both in the anterior horns and Clarke's column, and in the nuclei of the medulla oblongata.

A combination of systemic processes and diffuse changes may also occur.

In observations of Westphal, Dana, and Bruns, and especially in several cases described by Russell, Batten and Collier, the process has assumed a diffuse character at the level of the dorsal cord ; at the other levels, however, it cannot be explained by the assumption of a secondary degeneration, but must be regarded as a combined column disease. Kattwinkel is inclined to attribute the affection to a disease of the vascular-lymphatic system, and to deny it a systemic

FIG. 100.—Combined disease of posterior and lateral columns. (Weigert's method.)

FIG. 101.—Combined disease of posterior and lateral columns. Irregular distribution of the degeneration. Degenerated parts shaded.

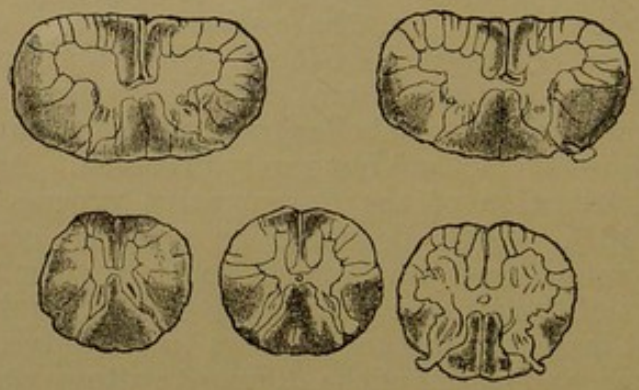

FIG. 102.—Transverse sections at various levels of the cord, in combined disease of the pyramidal, cerebellar, and Goll's tracts of Burdach's column and the anterior pyramidal. Degenerated parts are shaded.

character. This is the view which is also held by his teacher Marie, as well as by Guillain and Crouzon. Henneberg has advanced the view of *funicular myelitis*, *i.e.* a myelitis which in its distribution follows the columns without being attached to the systems. Nonne has agreed with this, but he holds nevertheless to the existence of system diseases.

From the foregoing and my own experiences I come to the conclusion that the picture of combined system disease may indeed be simulated by diffuse processes of degeneration and inflammation, but that *there can be no doubt that this morbid type does exist. For the nosological conception, it is not, however, important whether we are dealing with a systemic process ; it is sufficient to know that there are combined column diseases which are well characterised clinically.*

Etiology.—In some of these cases there may be a *congenital disposition*, which causes a certain weakness and a lessened power of resistance of some of the fibre systems or neurones to injuries. This is undoubtedly so as regards the hereditary and family form already described, which indeed more frequently corresponds to a combined system disease than

[1] "Über amyotr.-paret. Formen der komb. Erkrankungen," etc. Wien, 1898.

to an isolated pyramidal degeneration (observations of Strümpell, Newmark, and others). Syphilis does not play so important a part here as in tabes, but it has been found in a number of cases by myself,[1] Hoppe, Nonne, Batten and Collier, etc. Erb's syphilitic spinal paralysis (see corresponding chapter) has been attributed by Nonne [2] to a combined affection of the posterior and lateral columns. Concussion of the spinal cord is included among the causes. The disease most frequently develops in cases enfeebled by severe *anæmia* and *cachexia*. Lichtheim, Minnich and Nonne, confirmed by Noorden, Boedeker-Juliusburger, Putnam, Moxter, Goebel, Marburg, Taylor, Homén, Collins, Clarke, and others, have seen symptoms of tabes and of combined posterior and lateral column affections in pernicious or severe anæmia, and have found a corresponding spinal-cord disease, which, however, they do not regard as systematic. From the careful observations of these writers and especially from Nonne's [3] abundant experience—I have had an opportunity of seeing his convincing preparations—it appears that there are usually small foci, apparently arising from the vessels, which later become confluent and thus simulate a column disease; this focus-like character is, however, not always marked. The posterior columns are mainly affected, but the lateral columns and the rest of the white matter may also be involved (Fig. 103). The grey matter is also sometimes affected. Whether this is a condition consequent on severe (fatal pernicious, or secondary) anæmia, or whether both affections arise from the effect of a poison, we do not know. The anæmia frequently develops during the course of the disease (Bastianelli, Collier); Nonne, however, does not include these cases in this category, but places them in a special class, and Rheinboldt [4] agrees with him on the ground of a case which he investigated. Some writers, Clarke for instance, think that the anæmia has not always a pernicious character, and this agrees with my own experience and with a recent observation of Nonne's.[5]

In leucæmia [6] also (Schultze, [7] Nonne, Geitlin), septicæmia, ulcerative endocarditis (Nonne), malaria (Oppenheim, Henneberg), carcinomatosis (Oppenheim,[8] Lubarsch, Homén), diabetes (Williamson, Geitlin), tuberculosis (Ransohoff, Süsswein, R. Sand), alcoholism (Homén, and particularly Nonne), lead poisoning (?) (Collins), and as a result of senile degenerations similar changes may occur, and apparently in Addison's disease also.

In the majority of cases of combined column disease which I have observed, the anæmia and the loss of strength were very remarkable, but it seemed to me that this disturbance of nutrition does not produce the disease, but that both are the result of the influence of the same cause, either toxic or organismal. In one of my cases there was a cachexia due to tumour, in another an anæmia due to lactation. In three others the disease developed in individuals who had suffered from malaria in their youth; in the sixth or seventh decades of life they became affected by a cachexia (senile marasmus) for which no definite cause could be

[1] "Zur Kenntnis der syph. Erkrank. des zent. Nerv." Berlin, 1890.
[2] *A. f. P.*, xxix. *u.a.a.O.*
[3] *Z. f. N.*, vi., xiv., etc.
[4] *A. f. P.*, xxxv.
[5] *Mitt. aus. d. Hamburg. Staats.*, 1907.
[6] It should be mentioned here that in leucæmia in the nervous system there have been found hæmorrhages (A. Fraenkel and others), focal degenerations (Bloch, Hirschfeld, Spitz), and leucæmic infiltrations of the grey matter, and that Eichhorst was able to trace a compression of the spinal cord to a lymphoma in the epidural space.
[7] *N. C.*, 1884.
[8] *B. k. W.*, 1891.

discovered, and this was accompanied by the onset of the spinal disease.

Symptomatology.[1]—The clinical picture may be drawn by starting from the symptoms of isolated posterior column disease and of isolated lateral column disease, and by then blending the symptoms which they both present. Here we are at once met by a difficulty. Disease of the posterior columns is characterised by atony of the muscles, Westphal's sign, and ataxia, that of the lateral columns by increase of the muscular tonus, exaggeration of the tendon reflexes, and motor weakness. A combined affection of these columns therefore gives rise to symptoms which partly preclude each other. Westphal's investigations have, however, removed the indefiniteness. He showed that the symptoms depend upon the intensity and the extent of the process in the two columns. If the lateral pyramidal columns are chiefly affected and if the posterior column degeneration does not extend so far down as the lumbar region, the muscular tonus is increased, there is rigidity of the muscles, exaggeration of the tendon reflexes, and motor weakness. Affection of the posterior columns is then revealed by other tabetic symptoms, especially by the

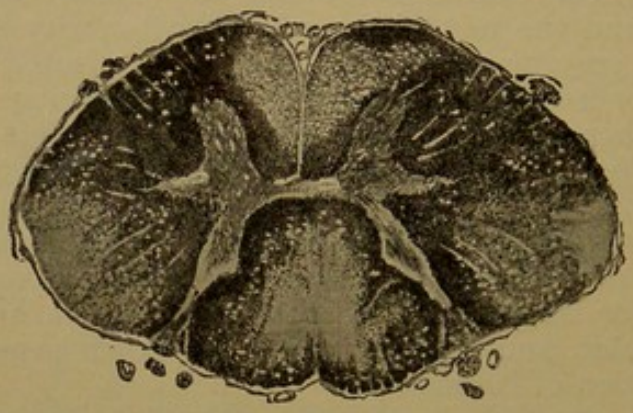

Fig. 103.—From a section prepared by Boedeker in Oppenheim's collection. (Pal's carmine method.) Degeneration of the tracts of the cord in pernicious-anæmia.

ataxia. *Spastic-ataxic paraplegia*, or paraparesis, which leads to a typical disturbance of gait, is then the most pronounced symptom. To this are added other tabetic symptoms—bladder troubles, lightning pains, sensory disturbances—but these may be absent or very slightly marked. Of the cerebral symptoms of tabes, general experience shows that rigidity of pupils is rare and optic atrophy still rarer; in cases which I observed, however, the former was by no means an unusual symptom.

If, from the first, degeneration of the posterior columns preponderates and extends into the lumbar region, then we have a clinical picture of tabes complete even to the inclusion of Westphal's sign, and it is only the motor weakness (which may become actual paralysis) that indicates that the pyramidal tracts are involved. Recent cases (Babinski, Oppenheim, Collier, Marie-Crouzon) have shown that even under these conditions Babinski's toe sign is present as a rule and points to the involvement of

[1] To the symptomatology of this condition special contributions have been made in recent years by Marie and Crouzon, Crouzon (" Des scléroses combinées de la moelle," Paris, 1904, and *Nouv. Icon.*, 1904), and E. Müller (*Z. f. N.*, xxix.). I may, however, mention that the descriptions given even in earlier editions of this text-book contain the essential facts which these writers have recently brought together, and which they have merely completed in a few points.

the pyramidal tract. In several cases of this kind I found the dorsal leg phenomenon to be present.

It not infrequently happens that the signs of involvement of the lateral columns become prominent in the beginning of the disease, and that only in the later stages—when the degeneration of the posterior columns extends down into the lumbar region—the knee jerks, which until then have been exaggerated, disappear, and the muscular rigidity gives place to atony.

This atony may be the first sign that the posterior columns are involved, whilst the other symptoms are absent or but slightly (my own observations). It is rare that as, in a case of Wagner's, the hypotonic stage precedes the spastic.

There are therefore two groups of symptoms which justify us in the diagnosis of combined disease of the posterior and lateral columns :

1. The symptoms of "spastic spinal paralysis," if accompanied by ataxia, lightning pains, bladder weakness, rigidity of pupils, and other tabetic symptoms. There is a form (Strümpell) in which the spastic paralysis entirely dominates the picture, and in which only slight sensory disturbances point to involvement of the other systems (see preceding chapter).
2. The symptoms of tabes, when from the very beginning the ataxia is combined with motor weakness (and with the inversion of the plantar and leg reflexes, which is characteristic of pyramidal lesions), or is even preceded by it, or where the paraplegia gradually develops in the course of the tabes.

It is specially characteristic if the second condition has developed out of the first one. In several cases of this kind, in which Westphal and I made the diagnosis, it was confirmed by autopsy.

Recently I saw two cases belonging to the first category, in which bladder troubles alone had existed for several years before the spastic paraparesis appeared. In addition to the spastic group of symptoms there was only a paralysis of the sphincters, the sensibility being intact. I could only explain the morbid picture by a combined degeneration of the posterior and lateral columns, under the hypothetical view that in this case it was just the vesico-anal posterior column tracts (in the comma area, in the triangular field ?) which was affected on account of a congenital disposition. The presence of certain anomalies of development in one of the cases supported this view.

The disease cannot always be recognised during life. If the degeneration of the posterior columns is but slightly developed, it may remain entirely latent, and the disease may give the impression of a spastic spinal paralysis. Still more commonly the clinical picture agrees so completely with that of myelitis, that no definite distinction can be made between them. If the lightning pains and ataxia form a prominent symptom in a morbid picture which otherwise corresponds to that of myelitis, combined column disease may be suspected. If there is in addition immobility of the pupils, the diagnosis may be regarded as confirmed (my own observations). Bulbar symptoms are described by Mayer,[1] by Henneberg, and E. Müller (see also Strümpell's cases in the preceding chapter).

Our knowledge of this disease has been enriched and deepened in recent years by observations by Dana (*Journ. of Nerv. Dis.*, 1899), but more especially by very thorough investigations by Russell, Batten and Collier (*Br.*, 1900). These writers speak of the *subacute ataxic paraplegia*

[1] "Über die komb. syst. Erkr. der Rückenmarksstränge." Wien, 1894.

or of the "subacute combined degeneration of the columns of the spinal cord," as in the cases they examined the disease developed in the course of a few months, and its whole duration extended to a period of one-half to one and a half years. They distinguish three stages. In the first a slight spastic paraparesis and ataxia appears, along with paræsthesiæ. In the second the paresis increases into spastic paraplegia and is associated with anæsthesia in the legs and on the trunk. In the third the spastic becomes a flaccid paraplegia, with abolition of the tendon reflexes, absolute anæsthesia, and paralysis of the sphincters. General emaciation develops along with atrophy and marked diminution of the excitability of the muscles, and also with œdema. Girdle pain and herpes occasionally appeared, and an irregular rise in the temperature was frequent. Paralysis of the eye muscles occurred in a few cases, but never immobility of the pupils. Towards the end of life stupor and confusion were present, with severe anæmia, which had been in existence from the first or had developed during the disease. Along with a more diffuse, circumscribed disease of the middle dorsal region there was a combined affection of the posterior and lateral columns in the other segments of the spinal cord.

Although the morbid picture reminds us in most of its features of the description given above, it yet presents so many peculiarities that it undoubtedly represents a special type of the disease which they had the opportunity of observing and studying closely. This form takes a middle position between that described above and the spinal affection in pernicious anæmia. Henneberg has reported similar observations, and Nonne (ref. *N. C.*, 1907) has shown that a kindred process with corresponding clinical symptoms occurs in alcoholics.

With regard to the symptomatology of the form which arises in pernicious anæmia and cachexia, it should first be noted that these changes have been found even in cases in which there had been no symptom of any kind pointing to disease of the spinal cord. The posterior column symptoms are the most prominent, and these may be combined with the symptoms caused by the process in the lateral columns. As a rule, there are paræsthesiæ, pains, and objective sensory disturbances in all four extremities. The general motor weakness and the paresis of the limbs are also specially marked, and are frequently combined with ataxia, whilst spastic symptoms are but seldom observed. In opposition to tabes, the upper extremities are very soon affected, frequently at the same time as the lower limbs. Tabetic pupil symptoms are absent according to Nonne and others. Affection of the optic nerve is mentioned by Putnam and Taylor. This disease is specially distinguished from tabes, however, by the rapid course—within a few months or a year, rarely for several years—and by the occasional considerable remissions (Nonne, Bowman). The symptomatology justifies us in discussing the condition along with the continued system diseases, whilst from an anatomo-pathological point of view it might with great or even greater propriety have been classified with myelitis.

In *pellagra* symptoms of *ataxic paraplegia* may appear, and changes are found in the spinal cord which correspond to a combined degeneration of the posterior and lateral columns (sometimes in the anterior horns also) (Tuczek,[1] Marie, Zlatarovic, Babes,[2] Duse, and others).

The morbid picture is very complicated, as the skin, the digestive apparatus, and the nervous system all participate in the clinical symptoms. Amongst the nervous disturbances, the mental and spinal predominate. The symptomatology and pathological anatomy have recently been discussed by Neusser, Sturli, and Tuczek. I myself had an opportunity of seeing cases of this kind during a stay in Czernowitz.

It should further be pointed out that this disease of the spinal cord is not uncommon in *paralytic dementia*. Symptoms are present during

[1] "Klin. und anat. Stud. über Pellagra." Berlin, 1892.
[2] "Die Pellagra, Nothnagels Handbuch," xxiv.

life, but are not usually fully developed, as the brain disease proves fatal comparatively early. Marie ascribes the process in these cases to a primary degeneration of the grey matter (atrophy of the cells of the cord), but this is extremely doubtful.

I have had (*N. C.*, 1883) an opportunity of examining a case of combined disease of the posterior and lateral columns in childhood, which was distinguished from Friedreich's disease, described in the following chapter, by the presence of immobility of pupils, optic atrophy, etc. Besides the affection of the spinal cord there was also found an atrophy of the Westphal-Edinger nucleus and of the cerebral trigeminal root. Luce published a case (*Z. f. N.*, xii.) of combined systemic disease in childhood, in which the anatomical changes corresponded, according to his interpretation, to those of amyotrophic lateral sclerosis combined with those of tabes. However, the forms which essentially involve the anterior grey matter, to which Pal [1] has again drawn attention, will be considered in another place. A case of hereditary combined column disease described by Zahn is difficult to classify.

With regard to the *differential diagnosis* we have specially to consider disseminated sclerosis and spinal or cerebro-spinal syphilis. The cerebral symptoms of disseminated sclerosis are, however, quite different: the partial optic atrophy, the speech disturbances, etc., and later the myosis and immobility of pupils; and, if affection of the optic nerve is present, it is the characteristic pure atrophy. In combined sclerosis, nystagmus has been observed in isolated cases, and tremor has also been noted, but it does not strictly correspond to the intention tremor.

E. Müller, who has recently made an exhaustive study of the differential diagnosis of these two diseases, has come to conclusions similar to those set forth in my earlier editions. He states that in doubtful cases the normal condition of the abdominal reflex, the slow progressive course free from exacerbations and remissions, and also the existence of very marked, persistent, and widely distributed disturbances of the sensibility, are to be regarded as evidence against disseminated sclerosis.

As regards differentiation of the disease from spinal syphilis, the chapter on that subject should be consulted.

Prognosis and Course.—The prognosis is grave, but not hopeless, for an affection developing, as the result or in the course of anæmia, may again disappear, as I believe I have observed in two cases. Nonne [2] has also reported cases of this kind. The course is chronic or subacute; the disease may run its course within a period of a few months and, if we except Strümpell's form, seldom lasts longer than one to two years. It has the tendency to spread from below upwards, but may commence simultaneously in all four extremities.

Treatment is in the first place directed to the exciting cause. Where toxic effects are present the injurious substances should be removed (by withdrawal of alcohol, etc.), and excretion of the products still circulating in the blood should be stimulated by diaphoresis, diuresis, etc.

The anæmia and cachexia should be treated in the usual manner. In two cases, for instance, I have seen improvement follow the use of preparations of iron and arsenic accompanied by a very full diet. E. Grawitz, starting from the hypothesis of the enterogenous origin of pernicious anæmia, attaches the greatest importance to the dietetic measures (vegetable food, nutrient enemata and to washing out the intestine.

[1] "Über amyotrophisch-paret. Formen der komb. Erkrank," etc. Wien, 1898.
[2] "Mitt. aus d. Hamb. Staatskrank." vii. 1907.

The experiences of the result of application of the Röntgen rays in leucæmia, may in time prove to be of much value in the treatment of this disease also.

The principles for symptomatic treatment may be taken from the chapters on Tabes, Myelitis, etc.

Hereditary Ataxia (Friedreich's Disease)

This is a very uncommon condition. It belongs to the family diseases, and affects as a rule a number of members of the same family, several of the brothers and sisters. Bouchaud saw it in twins. It is only in exceptional cases that a single member is affected. Direct heredity is unusual, but that lies practically in the nature of the disease. Epilepsy and mental diseases are observed in the relatives. Consanguinity of the parents has been noted in several cases. A toxicopathic heredity (alcoholism in the parents) has also been imputed.

Bouché treats the etiology in detail, *Mémoire couronnée*. Bruxelles, 1905.

The disease commences in childhood, during the seventh or eighth year or at the time of puberty, very seldom later. An acute infective disease has repeatedly acted as the exciting cause. Allan Starr lays special stress upon this factor.

The first symptom of the very slowly developing disease is *ataxia* of the lower extremities, which first becomes evident in the gait. The patient walks with the legs wide apart, and stamps—although this is not usually so marked as in tabes. The gait is uncertain, and very little makes the patient stagger. Charcot has named this disturbance the " démarche tabéto-cérébelleuse," because the unsteadiness resembles that of cerebellar inco-ordination. In standing it is likewise very pronounced, although it is not materially increased when the eyes are closed. In a few cases (Rütimeyer,[1] Soca,[2] and others), however, Romberg's symptom has been found to be present.

The ataxia becomes apparent even when the patient is lying in bed, both in movement and particularly in the attempt to maintain any position, in sitting, etc. (static ataxia). It spreads slowly, and sooner or later affects the upper extremities, sometimes also the muscles of the trunk, throat, and neck. Active movements of the arms sometimes reveal a combination of ataxia and tremor.

A certain degree of muscular weakness may also be associated with the ataxia, but this is neither severe nor general, and only in rare cases is there an early development of paraparesis. It is only in the last stages of the disease that the weakness generally becomes very marked, and it may then be combined with contracture and atrophy.

The latter may, however, be present in some muscles, even at an early stage, and sometimes to a high degree. It has been observed by Dejerine (and once by myself) in the muscles of the leg, by Whyte, Hodge, and Griffith in the small hand muscles. *Pseudohypertrophy* has also been noted (Bäumlin, Jendrássik,[3] Bing, Anciano), but it is not clear whether this sign belonged to the Friedreich's disease or whether there was a combination of this disease and pseudohypertrophy.

Another motor phenomenon frequently occurs: a *choreic* restlessness; tremor movements from time to time in the various muscles,

[1] *V. A.*, Bd. cx. [2] " Étude clin. sur la maladie de Friedreich," *Thèse de Paris*," 1888.
[3] *Z. f. N.*, xxii.

which are independent of any voluntary intention. When the patient is standing and walking, these may affect the muscles of the neck to such a degree that there is an obvious shaking and nodding of the head. Moreover, a sharp distinction cannot always be made between these choreiform tremors and the static ataxia.

The *tendon reflexes* usually disappear early or during the later course of the disease, although there are exceptions to this rule. Babinski's sign can frequently be elicited. The sensibility usually remains intact. Lightning pains are generally absent, but they may be a distressing symptom, as for instance in a case described by Bonnus.[1] Diminution of the tactile sense, which was objectively apparent, has been noted in a small number of cases only (Rütimeyer, Soca, Senator,[2] Stintzing, Kopczynski, Bäumlin,[3] and others). In one of the cases which I examined there was a distinct affection of the sense of position, so that even small objects in the hands could not be promptly recognised. That there may be a diminution of the sense of touch in the later stages was stated by Friedreich himself. Bladder weakness is an unusual sign. One of my patients—a boy of nine—lost urine only when laughing. Crises do not occur.

With regard to the *cerebral functions*, *nystagmus* and *speech disturbances* belong to the typical clinical picture. Speech is slow, difficult, indistinct, badly articulated, and irregular; some syllables and words are drawn out, others are shot out rapidly or even explosively. This symptom accompanies the ataxia, usually only in the later course of the disease.

Schultze mentions profuse salivation among the symptoms. Bert saw diabetes appear in the course of the disease. Vaso-motor disturbances occasionally appear. In a few cases there were marked *respiratory disturbances*; I have noted, for instance, jerky breathing, caused by the motor disturbances (ataxia, tremor) having involved the respiratory and especially the abdominal muscles. Fürstner mentions dyspnœa, which I also have seen, especially during movement of the body. Vertigo is sometimes complained of. Immobility of the pupils and oculo-motor paralysis do not belong to the symptomatic picture of Friedreich's disease, but the latter especially has been found in some cases. The results of ophthalmoscopic examination are always normal. There are no anomalies of the sensory functions.

The intelligence is almost always unaffected, but a combination with idiocy has been observed (Nolan, Pick, Degenkolb). Biro mentions epilepsy as a complication, and Collins, Menière's disease.

Scoliosis of the spinal column frequently develops. In many cases I have found a deformity of the foot, pes equino-varus, with marked hyper-extension of the toes, especially of the great toe, the basal phalanx of which is hyper-extended, whilst the end phalanx is in a flexed position (Fig. 104), a symptom which, nevertheless, is by no means pathognomonic of this disease (Cestan). Pes talipes cavus also occurs. Club-hand with claw-position has occasionally been described (Menaut). Dejerine attributes the deformities of the foot to atrophy of the muscles on the anterior surface of the leg and sole of the foot. Others regard it as a stigma of degeneration. One of our patients suffered from hypospadias

[1] "Contrib. à l'étude de la maladie de Friedreich," Thèse, 1898.
[2] *B. k. W.*, 1893.
[3] *Z. f. N.*, xx.

and microgнathia. Moravsik mentions congenital cataract, and Aubertin [1] a combination with congenital heart defect.

Abortive forms of Friedreich's disease may undoubtedly occur. In the brothers or sisters of the patient only isolated symptoms of the disease may be discovered. Thus I found in the brother of one of the individuals affected with the disease only disturbance of speech and nystagmus, and in the oldest sister also nystagmus presented the only sign. Gardner's observation (*Br.*, 1906) is very interesting in this respect; in some members of a "Friedreich" family, Westphal's sign was alone discovered—a remarkable illustration of the occurrence of this symptom as a stigma of degeneration.

The disease runs a very slow course, and may last from thirty to forty years. If the symptoms have attained their full development, the patient's walking will be more and more affected, until finally he is permanently confined to bed. The end is generally brought about by

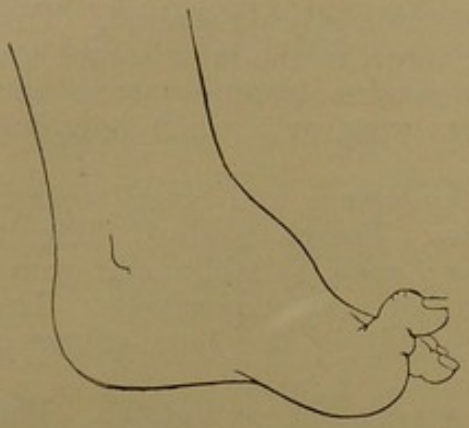

FIG. 104.—(After Brissaud). Position of the great toe and condition of the foot in Friedreich's disease.

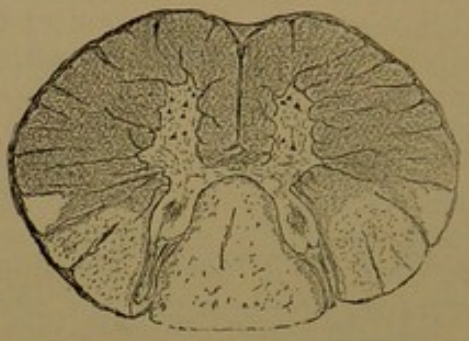

FIG. 105. — Transverse section through spinal cord in Friedreich's disease. Pal's method. (From a section prepared by Marinesco in Oppenheim's collection.)

an intercurrent disease. Lannois and Porot emphasise the frequency of heart affections.

The *prognosis* is wholly bad. No case of this kind has ever been known to recover.

With regard to the *pathological anatomy* of the disease, in spite of a great number of post-mortem results [2] and exhaustive examinations, opinions still differ in many points. The following are the conditions most generally found. The spinal cord as a whole is small and slender, and there is usually present a *combined disease of the posterior and lateral columns* (Schultze [3]), usually degeneration of the entire columns of Goll, of Burdach's column with the exception of some parts, of the lateral cerebellar tracts, the lateral pyramidal tracts and Clarke's columns, in which there is not only atrophy of the fibres but frequently also destruction of the ganglion cells. It seems still doubtful whether the lateral column affection involves the pyramidal tract, although recent observations (Mott and others) are strongly in favour of this view.

[1] *Arch. gén. de Méd.*, 1904.

[2] Vincelet has recently collected these in a monograph, which also contains valuable contributions of his own, whilst Soca and Ladame in particular (1889) have published a collective description of this disease. See also the exhaustive communication of Mingazzini-Perusini (*Journ. Nerv. and Ment. Dis.*, New York, 1904, vi.), the thesis of Guenot (Lyon, 1904), the papers by Mingazzini (*M. f. P.*, xviii., and *A. f. P.*, Bd. xlii.), Nonne (*A. f. P.*, xxii., xxvii., and xxxix.), and also Dejerine and Thomas (*R. n.*, 1907).

[3] *Z. f. N.*, v.

The degeneration frequently does not extend so far inwards as one would expect (Fig. 105). Gowers' tract may also be involved. Schultze found the anterior pyramidal tract affected in one case. Atrophy of the posterior roots and peripheral nerves does not form a constant and prominent condition. In the medulla oblongata no constant changes were found (Schultze, Pitt), but the degeneration of the spinal columns may be continued to corresponding areas in it also (Blocq-Marinesco,[1] Barker, Mott, and others). Philippe and Oberthür [2] mention ependymitis on the floor of the fourth ventricle, but much weight should not be laid on this. They also describe involvement of the vagus nucleus and the solitary bundle. Jelgersma and Raymond have found a similar condition.

Mott thinks he has found changes in the cells of the motor cerebral cortex, but these may well have been of secondary nature. Stcherbak (*N. C.*, 1900) has suggested the theory that the process of degeneration is localised entirely in the tracts which go from the spinal cord to the cerebellum, and in those which pass downwards from the cerebellum into the spinal cord. Thus in the posterior columns the fibres leading to the cerebellum would alone be affected, and the degeneration of the pyramidal tract would be merely an apparent one, as this area contains only scattered fibres coming from the cerebellum. Jelgersma and Raymond have expressed similar views.

Senator considered the conditions described in the spinal cord as inconstant and uncertain, and he suspected a congenital atrophy of the cerebellum, such as Menzel and Auscher have found, to be the real cause of this disease. In many recent cases, however (Meyer, Mackay, Vincelet, Rennie, Rainy), the cerebellum was found to be normal, whilst in a few (Raymond, Mott, etc.) changes were found in the form of hypogenesis (abnormal smallness) or of altered histological structure, chiefly of the nerve cells. Marie,[3] on the other hand, has isolated from Friedreich's disease a group of symptoms, to which he gives the name of *hereditary cerebellar ataxia*, and which he ascribes to an arrest of the development of the cerebellum. In this group he includes the cases decribed by Nonne, Menzel, Fraser, Klippel-Durante, etc., which show the following peculiarities: onset towards the age of twenty, normal or exaggerated tendon reflexes, oculo-motor paralysis (especially ptosis and abducens paralysis), in some cases immobility of pupils, affections of vision, optic atrophy, occasionally trouble in swallowing, ataxia of the pure cerebellar character, and finally a mixed form of motor ataxia and intention tremor, etc. Scoliosis and other deformities are absent, but later cases have shown that malformation of the foot and other stigmata may also occur.

Londe [4] gives a detailed description of the disease. More recently Miura,[5] Rossolimo, Spiller, Heveroch, Lemnalem, Switalski,[6] Thomas and Roux, Bäumlin, Barker, and others have made clinical and anatomical contributions to this question. Thus Miura has found in one of his cases an abnormal smallness of the cerebellum, pons and oblongata, which Raymond and Rose have also seen, but on the other hand Meyer and Sanger-Brown have not found this atrophy of the cerebellum, whilst Bourneville and Crouzon failed to observe Marie's group of symptoms during life in a case where the cerebellum was much atrophied. Switalski found, in addition to atrophy of the cerebellum, a degeneration of Goll's column, the cerebellar tract, and Gowers' column of the spinal cord, and the medulla oblongata, and also an ependymitis at the floor of the fourth ventricle. He lays great weight on the affection of the vessels. The pathological results were similar in the case investigated by Thomas and Roux (*Rev. de méd.*, 1901), but the

[1] *Arch. de neurol.*, 1890.
[2] *R. n.*, 1901.
[3] "Sem. méd.," 1893.
[4] "Hérédo-ataxie cérébelleuse," *Thèse de Paris*, 1895.
[5] *Mitt. d. med. Fakult.* Tokio, 1898.
[6] *R. n.*, 1900 and 1901.

cerebellum showed no real abnormality; whilst Barker, who examined one of the Sanger-Brown cases, found degenerative processes in the cerebellum, the bulb, and the spinal cord. Raymond also found this condition. Nonne has shown that a congenital smallness of the cerebellum may be the only cause of Marie's group of symptoms. He also found the cranial nerves abnormally thin.

The distinction between Friedreich's disease and Marie's cerebellar ataxia cannot be definitely drawn from the symptomatology any more than from their anatomical basis. Thus an onset of Friedreich's disease after the age of twenty-five is mentioned by Bonnus, an early beginning of the hereditary ataxia by Fraser, Bäumlin, Lemnalem, etc., exaggeration of the tendon reflexes in Friedreich's disease by Hodge, Starr and Gladstone, and hypotonia and diminution of the osseous sensibility in hereditary ataxia by Raymond and Rose, whilst oculo-motor paralysis and affections of the optic nerve have been repeatedly noted in Friedreich's hereditary ataxia. Vincelet regards as Friedreich's ataxia a case which was thought by Klippel and Durante to be hereditary ataxia, and was taken by Londe to be a transition form, etc., etc. Mixed and transition forms have also been described by Paravicini, Lenoble-Aubineau, Bäumlin, Thomson, Gardner, Raymond, Ballet-Taguet, Perrero, etc. Nonne has also shown that variations, both of the clinical and of the pathological conditions, may occur in the same family.

The view which was put forward in the earlier editions of this work, to which Marie himself had devoted some consideration and which Nonne had advocated in 1894, that hereditary ataxia cannot be absolutely separated from Friedreich's disease, is now accepted by most writers. Bäumlin, Seiffer,[1] Veraguth, Collins, Nonne, Mingazzini and Perusini, and Raymond[2] have stated that in their opinion there is only a single disease which corresponds in some cases more to the cerebellar, in others more to the spinal type.

Mingazzini would divide the various cases into three groups according to the special site of the disease: (1) the cerebellar (Marie's type); (2) the spinal (Friedreich proper), and (3) the cerebello-spinal. Raymond also distinguishes between a cerebellar, a bulbar, a spinal, and a general form. I very much doubt whether this can be carried into practice.

It should not be forgotten that the fact of an organ being unusually small is no proof that it is incapable of function, just as on the other hand a nervous system which appears anatomically and histologically to be quite normal may be very seriously impaired in its functions. It cannot, however, be doubted that a congenital inferiority of certain segments of the nervous system, and indeed mainly or exclusively of the cerebello-spinal region, is the primary cause of the disease, and that the parts thus constituted become first exhausted, a fact which I had previously stated (see p. 1094 of the fourth, and p. 941 of the third edition of this Textbook), and which Edinger and Bing (*Z. f. N.*, xxvi.) have recently emphasised. Mingazzini also lays stress on the importance of the agenesis. It is specially stated by Nonne and Raymond that the morbid process may begin at various segments of the cerebello-spinal system, and that the different clinical varieties are produced in this way.

The *differential diagnosis* from disseminated sclerosis is most difficult, as it occasionally commences in childhood. It does not, however, begin with ataxia, or if this is present it is usually associated with spastic paresis and with exaggeration of the tendon reflexes. Affection of the optic nerve, which is so common in sclerosis, is absent in hereditary ataxia, etc. A morbid condition may develop from hereditary syphilis which is closely allied to Friedreich's disease. I have seen cases of this

[1] *Charité-Annalen*, 1902. [2] *Nouv. Icon.*, xviii.

kind in which the diagnosis was uncertain. The acute or relapsing onset of the disease, the marked, remitting character of the symptoms, the frequency of affections of the optic and oculo-motor nerves, the spastic disorders, the apoplectiform and epileptiform attacks, etc., are usually, however, definite indications for the diagnosis between cerebro-spinal syphilis and hereditary ataxia. The processes which give rise to infantile cerebral paralysis (see corresponding chapter) may also be localised in the cerebellum, and may cause an acute development of a group of symptoms which are intimately related to those of Friedreich's disease and still more to those of so-called hereditary ataxia (Oppenheim, Nonne, Batten [1]).

There is no doubt that morbid conditions may arise as the result of arrests of development in the central nervous system, which are closely connected with Friedreich's disease, but which, on account of some individual symptoms, are not to be identified with it.

Combinations of and transition forms between Friedreich's disease and family spastic paraplegia, progressive muscular atrophy, etc., have also been described.

It is specially stated by Jendrássik [2] (and also by Higier, Kollarits, Gardner, etc.) that the hereditary nervous diseases in particular show no sharply defined clinical pictures, but that the associated symptoms to which they give rise are of many varieties and blend into each other. Jendrássik has also rightly pointed out that these congenital affections give rise to changes in the soft parts and the bones as well as in the nervous system.

Treatment is confined to careful nursing and the avoidance of injurious conditions. The ataxia should be treated as described on p. 172.

Acute Anterior Poliomyelitis, Acute Atrophic Spinal Paralysis, Infantile Spinal Paralysis

Literature: See especially the recent monographs of Wickman, "Studien über Pol. acuta," Berlin, 1905. Wickman, "Berträge zur Kenntnis der Heine-Medinschen Krankheit," Berlin, 1907. Harbitz-Scheel, "Pathol. anat. Unters. über akute Poliomyelitis," etc., Christiania, 1907.

This disease, described by Heine in 1840, affects mainly the first years of childhood. While in the first few months of life it is rare, it is more frequent in the sixth to eighth months, and most common during the second and third years of life. In the fourth year the risk of taking the disease is still very great; after this period the affection appears only in isolated cases; but from my experience I cannot characterise it as even very rare in children of from eight to twelve years of age. The middle-aged are not immune, while adolescents are only exceptionally attacked (see following chapter).

Among the causes of the disease, cold and trauma will be met with. Injuries, especially a fall from a chair or the like, are undoubtedly most often blamed by the relatives as the cause. But, notwithstanding that acute anterior poliomyelitis may be mistaken for traumatic hæmatomyelia in childhood, it seems to me that the fall, under those circumstances, is, as a rule, not the cause but a result of the suddenly developing paralysis. On the other hand, it must be carefully noted that recent experimental work has shown that injury provides areas of choice for the settling down of micro-organisms (Hoche).[3]

Of late, the view that an infective agent is here concerned has made

[1] *Br.*, 1905. [2] *Z. f. N.*, xxii. [3] *A. f. P.*, xxxii.

more and more headway. In favour of this view are the development and course, like that of an acute infective disease, and still more the fact that cases are more frequent at certain times, and that, again and again, an epidemic onset is observed. In particular Medin,[1] a Stockholm physician, has reported an epidemic where twenty-four cases of this kind came under successive observation, from one locality within one month. Other epidemics of similar character and sometimes of even greater extent have been described by Leegard,[2] Mackenzie, Macphail-Carsley and Lundgren.[3] While Medin believed in the simultaneous onset of neuritis, polyneuritis (and encephalitis), as pointing to an intimate relationship between those affections, Leegard has expressed his decided opinion against this attempt to identify them. In the year 1898, in Vienna, the incidence of the disease was "frankly epidemic" (Zuppert). Wickman gives a very recent account of the nature of the spread of these epidemics in Sweden; he believes the disease to be contagious. Sinkler and Baumann state that cases are especially frequent in the summer months. Smaller epidemics, such as several brothers and sisters or mother and child falling ill at the same time, have been frequently observed, by Auerbach, Bülow-Hansen,[4] Béclère, Boek, Packard, Griffith, Oppenheim (see below), and others. Whether disposition may play any rôle is uncertain; at all events I have seen a child with poliomyelitis, whose mother likewise in her childhood had had and recovered from this disease.

Further, it is admitted that an attack may follow in the wake of other infective diseases, such as measles, scarlet fever, and whooping-cough. The disease has occasionally developed in connection with vaccination (A. Schüller, Trömner[5]).

Of recent years, by the experimental introduction into animals of micro-organisms such as B. typhosus, B. influenzæ, B. diphtheriæ or its toxin, and also B. coli, anatomical changes in the spinal cord have been successfully produced, which, in their localisation and nature, call to mind those of acute poliomyelitis (Vincent, Bianchi, Crocq, Henriquez and Hoche, Marinesco; see the chapter on Myelitis). In chronic sulphonal poisoning a similar affection of the grey matter of the spinal cord has been noted (Helwig). Schultze's discovery (*M. m. W.*, 1898) of the Jaeger-Weichselbaum diplococcus in the cerebro-spinal fluid, obtained by lumbar puncture from this disease, is very important. Diplococci have also been found by Chapin, Concetti, Harbitz, Bülow-Hansen, Looft and Dethloft, Geirsvold (*Norsk. Mag. f. Laeger*, 1905), Harbitz-Scheel (*Journ. of Amer. Assoc.*, 1908). Others, such as Engel and Raymond, have obtained negative or uncertain results by this method. Schultze's view that poliomyelitis may be traced back to the same infective origin as epidemic cerebro-spinal meningitis, as yet lacks confirmation. Moreover, Schultze himself, who demonstrated the presence of diplococci in but one of his recent cases and failed to find them in others, refers with great reserve to the significance of his discovery. Raymond and Sicard (*R. n.*, 1902), it is true, have concluded from a case in which polynuclear leucocytes were present in the turbid cerebro-spinal fluid, that epidemic cerebro-spinal meningitis could show itself in the guise of poliomyelitis. In other cases an increase of lymphocytes has been found and attributed to an inflammatory irritation of the meninges (Brissaud-Londe, Achard-Grenet,[6] Guinon-Paris, Heubner, Tiedemann), but this condition is not constant (Guinon-Rist). In bacteriological examinations of the cord carried out post mortem, micro-organisms are not usually found, a fact, I admit, only striking when the *sectio* is made soon after the commencement of the disease, as the investigations of Homén[7] and others have shown that cocci tend to disappear quickly from the spinal cord. The case pointed out by

[1] "Verhandl. d. x. internat. med. Kongr," Berlin, 1890; *Nord. med. Ark.*, 1896; *A. d. med. des enf.*, 1898.
[2] *Norsk. Mag. f. Laeger*, 1901. [3] *Hygiea*, 1906. [4] *Zieglers Beiträge*, 1899.
[5] *N. C.*, 1907. [6] *R. n.*, 1903. [7] See *Zieglers Beiträge*, xxv., 1899, etc.

Marinesco of a collection of pneumococci in the sulcocommisural artery is of great interest; it deals, however, not with poliomyelitis, but with meningomyelitis. He expresses the probability that the entrance of micro-organisms into the sub-arachnoid space produces meningomyelitis, into the anterior sulcal artery, poliomyelitis. A case observed by Courmont and Bonne perhaps also belongs to this class.

Of recent years I have made a series of observations, all tending to illustrate the infective character of acute anterior poliomyelitis and its relationship to meningitis. They deal especially with involvement of brothers and sisters, such as the following: In the first instance a child was seized, while on a journey from Paris to Cologne, with symptoms of a fulminating meningitis and died; directly after, a nine-year-old sister was attacked by the same disease, but the meningitic symptoms subsided within a few weeks, and there remained a permanent atrophic paralysis of the legs and of the left arm. When I saw the child a few weeks later in Paris, it presented a typical picture of acute anterior poliomyelitis, with this peculiarity, that tenderness was very marked. The future progress confirmed the diagnosis.

In a record case two of the children of a Berlin doctor became acutely feverish; one recovered quickly, the other, a boy of ten, was taken ill with a severe acute anterior poliomyelitis, with paralysis of all four limbs, muscles of back and neck, diaphragm, involvement of the bulbar nerves, and with meningeal symptoms, and died after a week's illness.

In a third instance, two children of the same family in Berlin became acutely feverish; but while the one recovered quickly, the other developed a severe anterior poliomyelitis, with involvement of the legs, the right arm, the palate, and the diaphragm, and long persistence of pain. Gradual improvement followed, except for the paralysis of the legs.

Symptomatology.—The illness commences acutely, in most cases with the appearance of a *general fever*. The temperature rises to 39° or 40° C., and along with the fever there is vomiting, loss of appetite, stupor, even coma and delirium, and sometimes general convulsions.

This feverish stage lasts from a few hours to several days: only exceptionally does it persist for a longer period. During this stage the exact nature of the disease is not clear. Towards its close or after it has passed, the relatives first notice the paralysis, which, very characteristically, is fully developed and *most widespread at the very commencement.* Not infrequently the general disease is of so short duration that it is quite overlooked—perhaps it may not be present at all; the child goes to bed well the night before and wakes in the morning with paralysis. It also happens that during the first hours or even in the first few days—but not beyond that—the paralysis spreads over a wider area.

Most commonly one extremity is involved, a leg, less frequently an arm; fairly commonly both legs. Only in a small number of cases are the arm and leg of the same side, or of opposite sides, affected; it is also unusual for both legs and one arm or for all four extremities to be paralysed.

Duchenne and Seeligmüller give the following tables :—

	Duchenne.	Seeligmüller.
Paralysis of one leg	32 cases	42 cases
,, both legs	9 ,,	14 ,,
,, one arm	10 ,,	13 ,,
,, all four limbs	5 ,,	2 ,,
,, both arms	2 ,,	1 ,,
,, crossed extremities	3 ,,	2 ,,
,, resembling a hemiplegia	0 ,,	1 ,,

The experience of Medin and Oppenheim is in close agreement with these figures.

The paralysis has the following typical characteristics: it is always flaccid, always—and this is most important—degenerative, associated with degeneration of the muscles, which, however, is not always equally visible, and may be marked in the further course of the disease by proliferation of fat and connective tissue, although, on the other hand, it may always be recognised by the presence of the reaction of degeneration. These changes in the electrical excitability develop in the course of the first few days and become well established by the end of the first week.

Simultaneously and later another change occurs in the aspect of symptoms. By degrees some of the originally affected muscles regain a certain amount of power of movement; the whole extremity is no longer cut off from voluntary action, only certain groups of muscles remain in a paralysed condition, and the more extreme the paralysis was originally, the larger usually is the region of muscle still remaining paralysed. The improvement shows itself first in the muscles originally least affected.

If we take the commonest example of one leg being originally affected, motility may return in all the muscle groups, except the extensors of the foot and toes, and even then the tibialis anticus may recover. Again, it often happens that the peronei recover while the extensors and the tibialis anticus more especially remain paralysed. The extensors of the leg, with or without the tibialis anticus, may be those remaining affected. In this case the sartorius very frequently escapes. It may be the case that most of the muscles of the lower limb remain paralysed, only a few, such as the adductors of the thigh, the internal rotators or the hamstring muscles, recovering their function. If both legs were affected at first, one may recover more or less completely, while the other is partly or wholly paralysed.

In the arms, the deltoid, alone or in combination with the biceps, brachialis anticus, supinator longus (also infraspinatus, coraco-brachialis, etc.), are the muscles most frequently permanently paralysed. The clavicular portion of the deltoid only, along with the serratus anticus major, or its middle and hinder portions along with the infraspinatus and rhomboids, may be the parts picked out. In the pectoralis major also, its clavicular or costal portion only may be affected. I have seen an involvement of the clavicular portion of the pectoralis in a case where, along with a paralysis of the Erb group of muscles, the ext. carp. rad. was involved. In other cases it is the extensors of the forearm (the radial group, with exception of the supinators) or the small muscles of the hand which are the seat of a permanent paralysis; but it must be added that the distal muscles of the extremity are less often affected than the proximal (Seeligmüller, Zappert, Baumann).

Upon the whole the extent of the paralysis in the various types corresponds with the description first given by E. Remak, and subsequently found also by Oppenheim, by Cestan-Huet,[1] Dejerine, Brissaud, Dupré, Parhon-Papinian, Roussy-Gauckler,[2] Huet-Lejonne, and others in many cases. In the upper extremity it may resemble the upper or lower plexus or root type of paralysis, but more often the tendency is for the area of one or of several roots to be involved. In this case also some investigators distinguish between the spinal type and the root type (see p. 145).

Even in cases where the extension is diffuse, the grouping of the paralysed muscles indicates the affection of particular segments or roots. Finally there is a disseminated form of poliomyelitis which follows none of the usual laws of distribution and extension of the paralysis (Oppenheim).

[1] *Nouv. Icon.*, xv. [2] *R. n.*, 1904.

The trunk muscles, especially the muscles of the back, are not infrequently affected; in my experience an involvement of the abdominal muscles is not uncommon, even where the paralysis is otherwise limited to one or both legs.

Recently the occurrence of abdominal paralysis in this disease has received more attention, and corresponding cases have been published by Oppenheim, Ibrahim, and Hermann (*Z. f. N.*, Bd. xxix.), Cornelli, and Strasburger (*Z. f. N.*, Bd. xxxi.).

According to Ibrahim and Hermann the transversalis abdominis is specially involved, while the rectus usually escapes. Strasburger's case is especially interesting, as being one of isolated affection of the abdominal muscles.

On the other hand it is only exceptionally that one or more of the motor cranial nerves is involved: facial, hypoglossal or abducens and oculo-motor (Medin, Oppenheim, Béclère, Schulze, Erb,[1] Wickman). For long this symptom was observed almost only in epidemics where such complications as polyneuritis and Landry's paralysis were present (Medin, Wickman). Huet[2] has once seen aphonia appearing in this disease, and refers to a participation of the vagus, as does also Wickman. The involvement of the motor cranial nerve nuclei has only been demonstrated pathologically in isolated cases (see below).

Oculo-pupillary symptoms have been noticed in cervical cases by Loevegren,[3] Bruns, Clopatt,[4] and Oppenheim.

As a rule the *concentration of the paralysis* is accomplished within a few weeks, and its localisation may then be readily made out by the electrical reactions. Those muscles in which faradic excitability is not completely lost within the first week, or in which it soon returns, will recover their power of movement. But the process by no means ends here; further improvement takes place, although only very slowly, extending over months, or even a whole year (according to Remak, even in the following year).

The flaccid, degenerative paralysis is the keynote of this disease; all else is accessory and following thereon. The *tendon reflexes* associated with the affected muscles are lost. The knee jerk is at least diminished, even if the quadriceps be transiently affected, and it is completely lost if that muscle remains permanently paralysed. Similarly the Achilles jerk is lost when the calf muscles are paralysed, and loss of the Achilles jerk in the paralysed lower limb is a very common phenomenon in this disease.[5]

Pain is either quite absent or else present only at the commencement of the illness; only exceptionally is it so severe that the condition may be mistaken for an acute rheumatism. The account of the relatives, however, often points to a great sensitiveness to touch and pressure at the beginning, which shows itself especially on attempting to move the affected part. I have had occasion to confirm this sometimes even during the first weeks of the illness. The muscles also in later stages may become somewhat sensitive to pressure. If the painfulness

[1] *D. m. W.*, 1906 [2] *R. n.*, 1900. [3] *Jahrb. f. Kind.*, 1905. [4] *D. m. W.*, 1905.

[5] In one case of mine, in which the shoulder muscles of the left side, the right quadriceps, and also on the left leg, the calf muscles, peronei and ext. dig. comm., were affected (apparently thus a disseminated poliomyelitis), the tendon reflexes showed the following peculiar, but yet quite explicable, behaviour: on the right the knee jerk was absent, while the twitching of the Achilles tendon could be got distinctly, or even markedly; on the left the reverse was the case, *i.e.* the knee jerk was well marked, while the Achilles jerk was absent.

of the muscles and nerves is marked, then probably we have the complication of neuritis.

Sensibility is not diminished. A minimal blunting of sensation, however, is exceptionally noted. In the later stages this is probably only due to the lowered temperature of the skin. So also the galvanic current is not felt quite so painfully on the paralysed as on the sound extremity. As a rule it may be taken that *sensibility remains essentially normal.* The sphincters are usually not affected.[1]

As soon as the febrile stage is over, the urine is passed normally; but there may be difficulty for about a week.

The *skin reflexes* are only modified in so far that the contraction cannot extend to the paralysed muscles, so that *e.g.* on stroking the sole of the foot, only the flexors of the hip and knee contract if the foot-and-toe muscles are paralysed. In this way I have seen Babinski's sign produced sometimes, where the ext. halluc. long. alone of all the foot muscles remained intact, and it contracted in response to each stimulation of the sole. Fuchs and Schüller have also observed this phenomenon, but they interpret it otherwise. Similarly I found the dorsal leg-phenomenon present in a case where the flexors of the foot and toes were not completely paralysed, but yet were more affected than the extensors. Otherwise these reflexes always respond normally with plantar flexion.

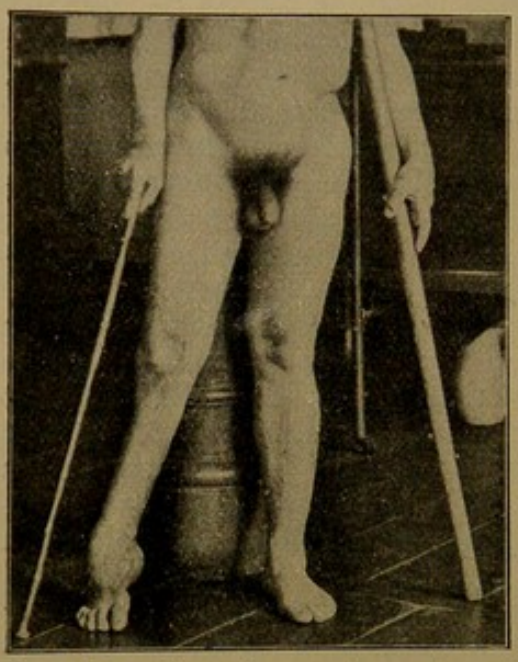

FIG. 106.—Shortening and atrophy of right leg. Pes varo-equinus from acute anterior infantile poliomyelitis. (Oppenheim.)

The *mind,* in uncomplicated cases, remains quite untouched.

We must now refer to a series of manifestations which develop out of the above, and are accordingly of secondary importance. *Arrest of growth, deformities* and *loosening of joint structures,* and also *disturbances of circulation.*

The affected limbs remain backward in development; if only one side of the body is affected, this shows itself in time as an evident shortening of the extremity whose growth is retarded, and produces

[1] I know one case in which there developed quite acutely in childhood an incontinence of urine, and in two others similarly an incontinence of fæces, without any other accompaniments. One might have thought of an unusually localised poliomyelitis of the conus terminalis, if it were not that, as a rule, those very functions remain intact in typical cases. However it can scarcely be doubted that a poliomyelitis of the lowest cord segments does occur, although very rarely. A communication just published by Frankl-Hochwart (*Obersteiner-Festschr.*, 1907) appears to justify the correctness of this assumption. On the other hand paralysis of the rectum in childhood has been observed as an isolated paralytic phenomenon from other conditions (after diphtheria, after long-standing rectal catarrh, from fissure of the anus, etc). The great rarity of sphincter paralysis in poliomyelitis is in favour of Müller's theory quoted on p. 120. Not long ago I saw a case in which retention of urine developed in a child after measles; this remained for a week as an isolated symptom, and then gradually recovered under the diaphoretic treatment employed at my suggestion. Ostheimer (*Univ. of Penn. Med. Bull.*, 1905) reports similar cases.

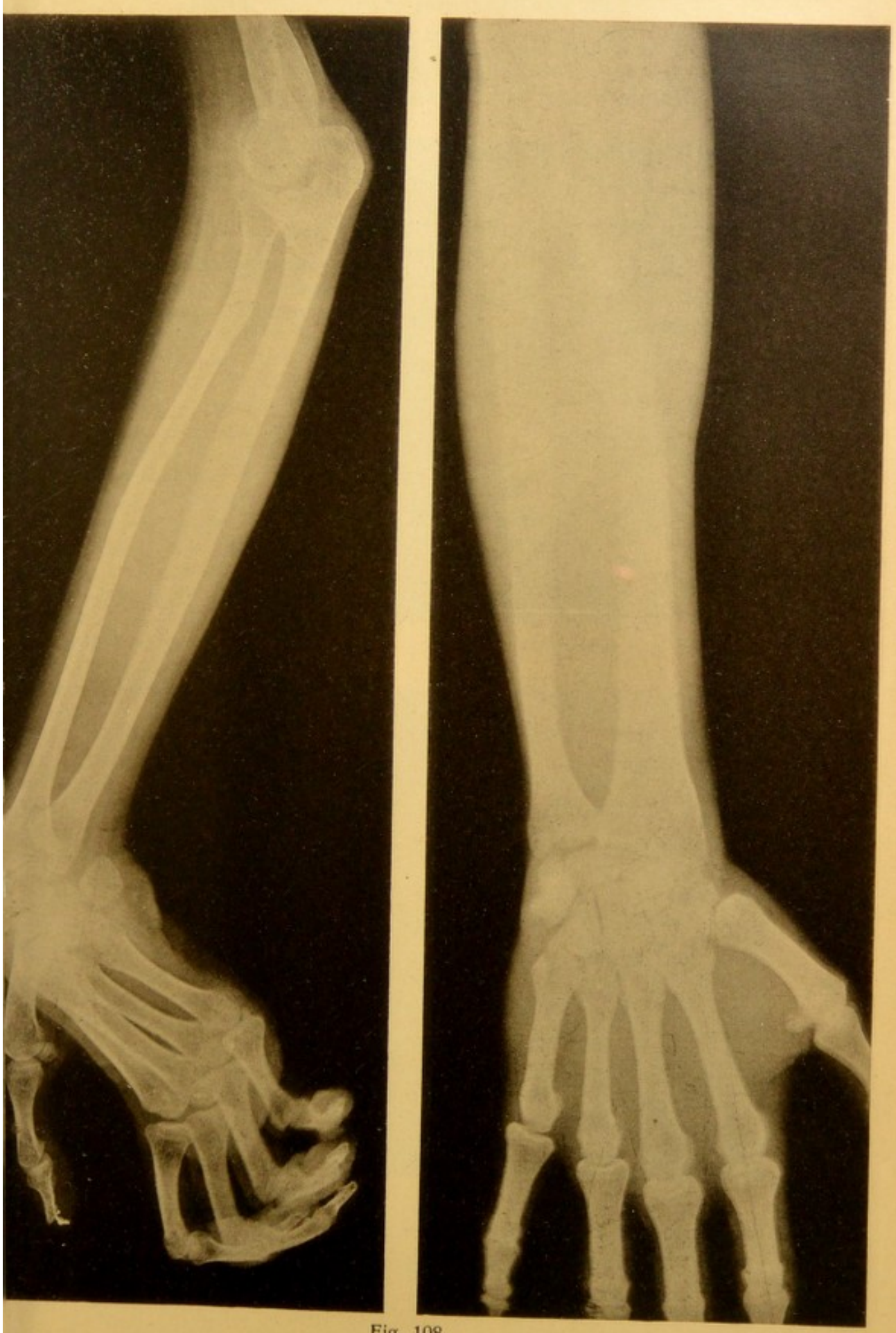

Fig. 108

Atrophy of Bones of Right Forearm and Hand in Acute Anterior Poliomyelitis.
(Author's case).

Otto Schulze & Co., Edinburgh.

more or less important difficulties in locomotion (Fig. 106). Besides this retarded growth of the bones in length, there ensues also an actual atrophy which is readily recognisable on examination with Röntgen rays (see Figs. 107A and B, Figs. 108A and B, Plate III.). The opposite condition may occur, but only very rarely —an excessive growth of the bones lengthwise (Seeligmüller). It is also regarded as a "trophic" disturbance, although Kalischer and Neurath attach a different significance to it. The latter thinks that a rachitis, dating from childhood, produces less result on the affected side. I have seen this elongation in several cases.

FIG. 107A.

The deformities which arise are very important, because, in consequence of the paralysis of whole muscle-groups the antagonists pass into a state of secondary or *paralytic contracture*. This may also come to pass when they themselves come within the scope of the disease, only their paralysis must not be a complete one. The contraction passes gradually into shortening; the muscular substance is replaced by fibrous tissue. The limb then becomes fixed in the position into which it was originally brought by the contraction of the muscles. Other factors, however, also play an essential part in this process (Volkmann, Reiche).

We meet with these contractures most frequently in the cases of deformities of the foot due to them. The commonest form is the pes equino-varus, which results from a paralysis of the extensors of the ankle and toes, and especially when the tibialis anticus is spared. If this muscle is involved while the peronei still functionate, then pes valgus ensues.

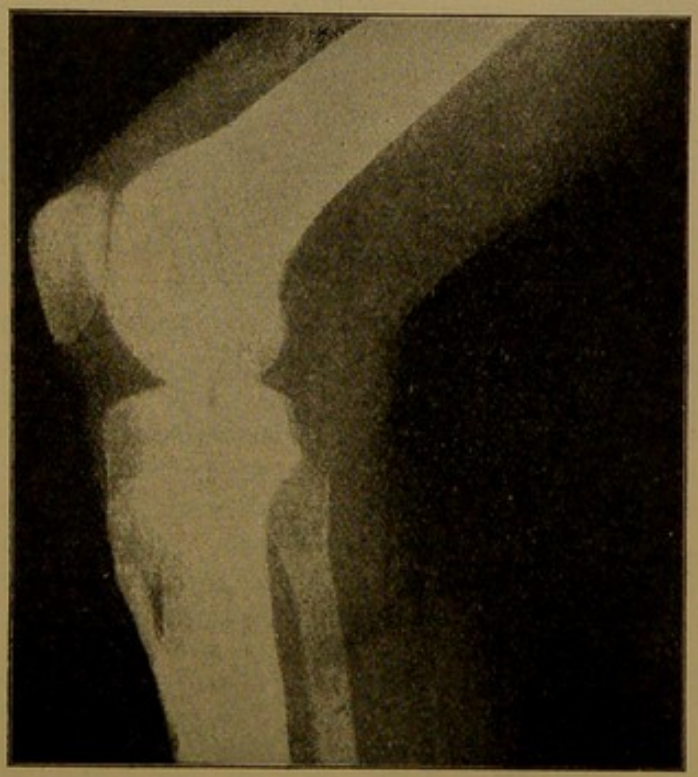

FIG. 107B.

(After Achard and Lévi.) Skiagrams of the lower limbs: A, in a case of infantile spinal paralysis; B, in a healthy person of the same age.

Paralytic flat foot results from a paralysis of the peronei and the plantar flexors ; a club foot may also develop in this form, if the weight of the foot counteracts the pull of the unaffected extensors. When the paralysis picks out the calf muscles, the contracture of the antagonists results in a pes calcaneus, eventually a pes calcaneus sursum flexus. Sometimes a flexor contracture develops at the knee-joint.

Corresponding deformities are much less frequently observed in the upper extremity, but a claw-hand may result from contracture.

Scoliosis and Lordosis of the vertebral column is likewise a frequent consequence of atrophic muscular paralysis (Fig. 109). Usually it occurs as a static, secondary scoliosis, but it may result directly from a paralysis of the back muscles (Laborde, Hoffa, Oppenheim).

These deformities may only develop in middle age, long after the disappearance of the poliomyelitis (Marie). A flail-joint results when muscles which support the structures of a joint are the seat of paralysis.

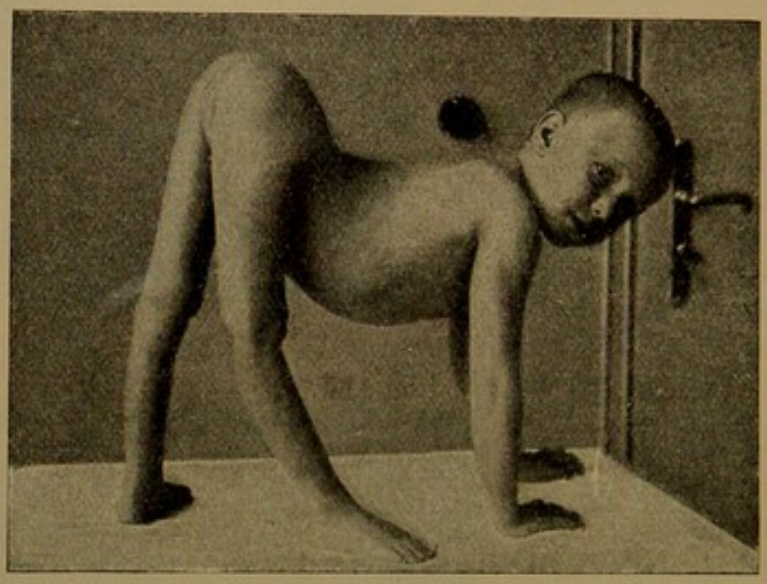

Fig. 109.—(After Zappert). Paralysis of the legs, flexion contracture of hip-joints, marked lordosis of lumbar spine. " Hand-walker."

These flail-joints are seen most often at the shoulder and hip. The disturbances of function in the limbs thus conditioned may be considerable. A rare consequence is an infrapubic luxation of the hip-joint ; a subluxation of the hand has also been once observed.

The skin of the affected extremity feels cold and is, as a rule, of a bluish red colour. The difference between the temperature of the skin of the sound side and of the paralysed may amount to 10° or 12° C. That this disturbance is merely the result of muscular inactivity is improbable ; without doubt there are *vasomotor influences* at work.

In rare instances, as a result of these, there may develop a swelling, a firm infiltration (hard œdema) of the soft parts, which may be so extensive that the circumference of the paralysed leg surpasses that of the sound one (Fig. 110). Disturbances of sweat secretion also occur (Higier) ; less commonly trophic lesions in the nails (Troisier, Heller, Rocher).

In some cases, in which the disease has resulted in a complete paralysis of the lower extremities, I have been struck by excessive development of the penis in youthful individuals. A growth of hair on the mons veneris of a five-year-old girl has likewise been observed.

Pathological Anatomy.—The pathological basis of this disease as first studied by Cornil, Prévost and Vulpian, Charcot and Joffroy, was thought to be an acute inflammatory process, mainly affecting the grey matter of the anterior horns. Charcot regarded it as a primary disease of the anterior horn cells. The majority of later investigators, however (Marie, Siemerling,[1] Goldscheider,[2] Redlich,[3] Wickman, and others), look upon the process as an interstitial, vascular one, while some, such as E. Schwalbe,[4] F. Schultze, Praetorius, and Neurath,[5] leave this question undecided or consider that the noxious agent acts on the nerve-cells, as well as on the connective tissue. When opportunity occurs of studying a recent case of this sort, one finds a hyperæmia of the anterior horns, a dilatation of the vessels, multiplication of vessels, and also thrombosis and hæmorrhage, later passing on to serous imbibition of the tissues and emigration of leucocytes. Accumulations of round cells are found particularly beside the vessels. The nerve elements are swollen, the ganglion cells turbid looking, with indistinct nucleus; the processes disappear first and granular cells accumulate in the tissue. Large epithelioid cells also appear and, in the later stages more especially, spider cells. This stage of inflammation is followed rapidly by one of atrophy, which leads to a more or less complete *destruction of the ganglion cells and nerve fibres* in some part of the grey matter. The process is most extensive at the very commencement; I have examined a cord from a case of this sort, where death ensued in the first stage—the inflammation extended in the anterior horns from top to bottom through the whole length of the cord and radiated out into the adjacent anterior and lateral columns. It appears from the investigations of Rissler,[6] Marie, Siemerling, Goldscheider, Redlich, Mathes, Philippe-Cestan, Wickman, and others, that the process is not sharply limited to the anterior grey substance,

Fig. 110.—Atrophic paralysis of left leg from an acute anterior poliomyelitis in childhood. Cyanosis and enlargement of the leg from swelling (hard œdema, état succulent) of the skin and subcutaneous tissue. (Oppenheim.)

[1] *A. f. P.*, xxvi.
[2] *Z. f. k. M.*, 1893, and "Handbuch der path. Anat. d. Nerv."
[3] *W. kl. W.*, 1894
[4] *Zieglers Beiträge*, Bd. xxxii.
[5] Obersteiner, xii., 1905.
[6] *Nord. med. Ark.*, xx.

but may encroach upon the posterior horns and the neighbouring white matter. The anterior horns are, however, the special seat of the disease, and definite changes are usually limited to this part. Their intimate relationship to the blood-vessels is especially insisted on by Marie. The meninges may also share in the inflammatory process (Schultze, Raymond-Sicard, Dauber, Fraenkel, and others). Analogous changes are occasionally found in the nuclei of the medulla oblongata, or actually in its substance (Eisenlohr, Medin, Redlich, Schultze, Wickman), but there is no occasion to suppose a similar extension of the process in typical cases. As a rule it is the grey matter of one of the enlargements that is affected, cervical or lumbar, on one side or on both; while definite subsequent changes are limited to the part most severely involved, usually only a section of the anterior horns in the cervical or lumbo-sacral cord, some 1-2 cm. in length. The majority of investigations deal

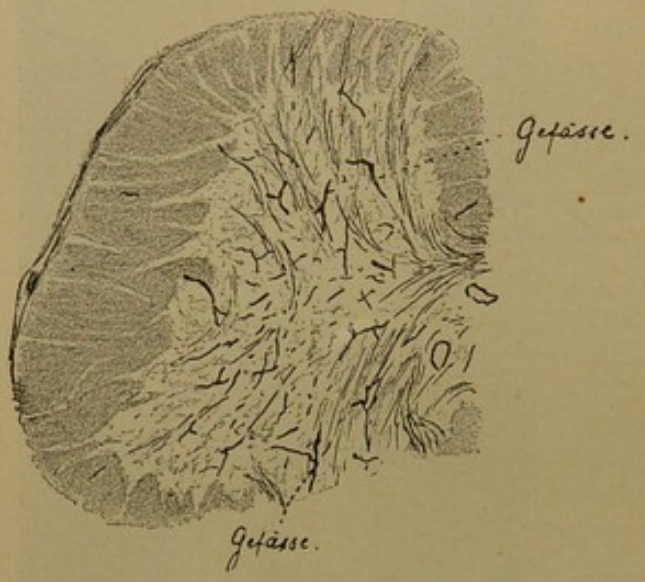

FIG. 111.—Acute anterior poliomyelitis. Anterior horn. Great increase of vessels (Gefässe).

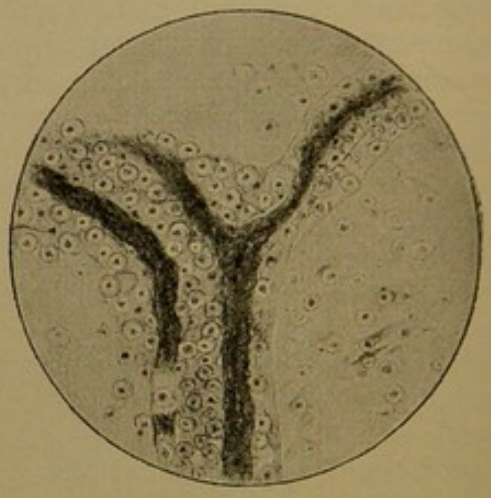

FIG. 112.—Accumulation of round cells in the neighbourhood of the vessels in acute anterior poliomyelitis.

with the further stages of the process, as the individuals affected usually die in later life of some other disease. There is found usually an atrophy of the anterior horn at some particular level of the cord, a marked diminution in size of the cord itself, a diminution in which the whole corresponding half may participate (Fig. 113). Microscopic examination shows an almost complete disappearance of the nervous elements from this anterior horn, while the glial tissue is increased in amount. The anterior roots are correspondingly atrophied, etc., and also the peripheral nerves, at all events the muscular branches going to the paralysed muscles. The muscles themselves are more or less markedly wasted; some may have completely disappeared. Instead of their normal tint, they appear of a rose-red, greyish red, yellowish, or yellowish-white colour, according as there is a simple atrophy, a fatty or fibrous degeneration of the muscular tissue. Different degrees and kinds of atrophy are usually found side by side, an observation which has been frequently confirmed of recent years in connection with operative treatment (see below).

Koch called attention to the fact that, even within one muscle, circumscribed areas of degeneration might occur, and that varying degrees of the same might be present side by side.

Such further accessory and rare charges as, for example, arrested development of the corresponding cortical motor centres (Edinger, Probst, *W. kl. W.*, 1898, and others) need not be gone into here.

With regard to the pathogenesis, the hypothesis has been put forward that infective material (an infective embolus) reaches the anterior spinal artery and its anterior commissural branches, which sink into the anterior horns. In addition to the pathological changes which are found in these arteries, the experimental investigations of Hoche, Marinesco, and others are in favour of this view. Hoche concluded from his observations that the still patent central canal of childhood may serve as a path of infection.

Batten looks upon a thrombosis of the anterior spinal arteries as the essential part of the process, and is of opinion that this may most easily develop in the vessels of the lumbosacral cord—those furthest away from the heart. Wickman ("Studien über Pol. ant. acuta," etc., Berlin, 1905) regards the condition as a disseminated myelitis or encephalomyelitis; a number

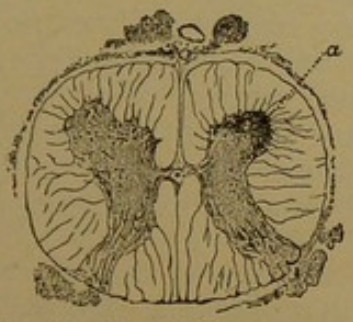

FIG. 113.—Atrophy of right anterior horn in acute anterior poliomyelitis of the lumbar region. *a*—focus of poliomyelitis. (Carmine stain.)

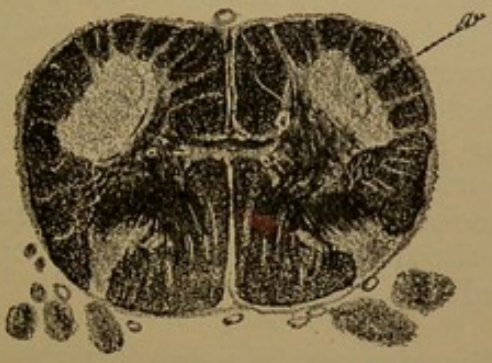

FIG. 114.—Bilateral acute anterior poliomyelitis of lumbar region. End stage. (From a section stained by Weigert's hæmatoxylin.)

of his cases, are, however, atypical. He supposes that the infective agent spreads by way of the lymphatics. Bing (*A. f. P.*, Bd. xxxix.) also is in favour of a hæmorrhagic myelitis as the foundation of the disease. See further F. Buzzard (*Br.*, 1907).

Differential Diagnosis.—In the first stage an error in diagnosis may be readily made. A feverish illness which causes pain in a limb and thus immobilises it, may give rise to the erroneous diagnosis of poliomyelitis. To this category belong, among others, acute rickets, coxitis, osteomyelitis, the so-called syphilitic pseudo-paralysis (separation of the epiphyses), etc. But it is always easy to show that it is only pain which prevents movement; wherever passive movement is attempted, evident muscular contractions will be observed, by means of which the little patient attempts to keep the limb firmly fixed in that position in which the diseased part is least irritated. Pain on pressure and on passive movement also point to one of those diseases; in poliomyelitis pain on pressure is, as a rule, absent or only temporarily felt. The electrical reactions and the condition of the superficial and deep reflexes will further prevent confusion.

It may be difficult to distinguish poliomyelitis anterior acuta from *multiple neuritis*. Although the latter is rare in childhood, it does occur: and I have seen several undoubted cases of it. The following points are of importance in the differential diagnosis: (1) The illness does not

attain its full development so quickly in multiple neuritis, it takes at least days, more often weeks before it reaches its height; while in poliomyelitis the paralysis is fully developed at the very start, and increase in its severity will, at the outside, only be noticed in the first day or two. An increase or extension of the paralysis up to the sixth day has been noted by Neurath, and a development by a succession of paralyses by Auerbach in one case. I have seen this, in adults or in quite atypical cases, similar to those which were observed by Neurath. (2) The feverish stage may last longer in multiple neuritis, and there may be exacerbations of the fever from time to time. (3) Pain and painfulness on movement are much more striking and remain longer, and the nerves and muscles are especially sensitive to pressure, and the former sometimes markedly swollen. (4) Disturbances of sensibility are almost always present in multiple neuritis, and can usually be discovered (but exceptions certainly occur). (5) Œdema is more frequent in neuritis. (6) An involvement of the cranial nerves is, in doubtful cases, more in favour of neuritis. (7) The distribution of the paralysis in poliomyelitis corresponds to the spinal-root type; in polyneuritis it follows that of the peripheral nerves as a rule (but the distribution in this respect follows no very fixed law). Although it is possible that in some cases of poliomyelitis the peripheral nerves are also affected, and certain that in polyneuritis spinal changes of a slighter degree are frequently present, it is absolutely essential to distinguish sharply between these two conditions, as the prognosis for complete recovery is much more favourable in a pure multiple neuritis.

In a case in which a young officer was suddenly seized with a flaccid, atrophic paralysis of the whole right arm and part of the left, I was led astray in giving my opinion because of the very marked sensitiveness to pressure of the nerve trunks, and diagnosed a polyneuritis, although objective disturbances of sensibility were entirely absent. Further progress showed that we were dealing with a poliomyelitis; the painfulness of the nerve trunks had arisen through the weight of the completely paralysed arm, which hung down limply, thus producing a dragging on the nerves.

There are cases of acute anterior poliomyelitis which present the picture of a *Landry's paralysis* (*q.v*); observed most frequently in epidemics of the disease (*e.g.* Wickman, Mackenzie, a house epidemic observed by myself, also F. Schultze[1]). These cases always appear to have an atypical character.

Infantile cerebral paralysis should not, on careful examination, be mistaken for spinal paralysis, but a combination of the two conditions has several times been observed (Oppenheim, Bayer,[2] Weber, Neurath). A well-marked case of this kind was thoroughly examined pathologically and described not long ago by Rossi.[3] The question has also been dealt with by Batten.[4] Marie has described the combination of Little's disease with poliomyelitis in one case. I once diagnosed an encephalitis pontis, while further observation showed that it was a combination of a right cervical and lumbar poliomyelitis, with a polioencephalitis affecting the left facial nucleus. This quite unusual localisation had presented the picture of an alternate hemiplegia, but the paralysis of the leg disappeared in a few days; that of the arm was a flaccid degenerative paralysis limited

[1] *Zieglers Beiträge*, etc., 1905.
[2] *N. C.*, 1895.
[3] *Nouv. Icon.*, xx.
[4] *Trans. Med. Soc. London*, vol. xxviii.

to the Erb group of muscles. Soon afterwards I observed another quite analogous case.

I have seen, likewise, a case of disseminated myelo-encephalitis acuta in childhood, in which the signs of a cervical poliomyelitis constituted a part of the symptoms. In another case, in addition to a flaccid paralysis of the leg muscles, which was quite of the poliomyelitic type, there was present a right-sided spastic hemiparesis, or at least the residue of one, besides a well-marked cerebellar ataxia. All these appearances resulted from a past disease of early childhood, which I was obliged to regard as a disseminated myelo-encephalitis.

Differentiation will be exceedingly difficult in the simultaneous epidemic onset of poliomyelitis, polyneuritis, and encephalitis, as, for example, in that observed by Medin.

Now and again the appearances of an acute atrophic spinal paralysis follow so directly on an accident, that the possibility of a hæmorrhage into the cord cannot be quite excluded. It is not improbable that *hæmatomyelia* in childhood may evoke a group of symptoms corresponding to that of poliomyelitis anterior acuta. At all events disturbances of sensibility have been missing in isolated cases of hæmorrhage into the spinal cord (Raymond, Murawjeff). On the other hand it seems to me, as I have already mentioned, that the fall of the child is, in many of these cases, a consequence of the rapidly developed paralysis, and not, although it gets the blame of being so, its cause.

Birth-Palsy is also in most cases a degenerative, flaccid paralysis. It affects the arm almost exclusively, and especially the parts supplied by the fifth and sixth cervical nerves. A mistake is not possible if it be known that the paralysis was present immediately after birth (difficult labour, breech presentation, dislocation of the arm, etc. etc.) If we do not see the patient until later life, and reliable accounts as to the time and development of the paralysis are lacking, the distinction may be impossible.

The rare form of birth-palsy, due to hæmorrhage into the spinal canal, may likewise closely resemble a poliomyelitic paralysis (Beevor).

Acute myelitis is very rare in early childhood, but may occur. If symptoms are present which indicate a lasting simultaneous involvement of the white matter (disturbances of sensibility, weakness of the bladder, spasms, ataxia, etc.), then one has no further right to speak of poliomyelitis, even if the atrophic paralysis be the main symptom of the disease. In such a case one is dealing with a myelitis. It is very difficult, however, if not impossible, to sharply distinguish between poliomyelitis and myelitis, and, as a matter of fact, there are cases of myelitis or disseminated myelitis, which in their onset and course remind one extraordinarily of an acute poliomyelitis.

I will quote one example from my own practice: a young woman became suddenly ill with fever and a paralysis of all four extremities, which was completely developed in a few hours; in the arms it was of an atrophic character, in the lower limbs spastic. At the same time there developed a sensory paralysis on the extremities and trunk. Involution followed within a few weeks, and nothing more remained of the disease than an Erb's paralysis of the right arm with R. D. and a therm-alg-anæsthesia or hypæsthesia of the extremities, and even this last shared in the involution process.

To this group also should belong some other cases, wrongly named by writers, as *e.g.* one described by Tedeschi.

There is a form of *lumbar spinal gliosis* which develops in early childhood, but this can be distinguished from poliomyelitis by its gradual

development and the combination of atrophy with partial sensory paralysis.

Congenital muscular defects can scarcely give rise to confusion, nor yet *delayed development* of particular groups of muscles which I have seen once or twice (see below). In one case of congenital defect of the tibialis anticus and extensor communis digitorum muscles the presence of other congenital developmental defects (webbing and deformities of the toes) prevented confusion with poliomyelitis.

Occasionally, it would seem, there occurs in children a simple congenital atrophy of a single limb, or segment of a limb, which only shows a quantitative diminution of electrical excitability. Klippel describes arrested development of this kind, following on accidents which have occurred in early childhood.

French literature also contains records of individual cases (Ballet, Charcot, Raymond, Guillain) of localised and diffuse muscle-atrophy of a non-degenerative nature, which developed after accidents and which was attributed to a kind of functional injury to the motor ganglion cells. If it be not the muscular atrophy following on a traumatic joint affection, then the nature of this atrophy is not quite clear, nor yet is that in a peculiar case of this kind described by Sabrazés-Marty (*Nouv. Icon.*, 1899).

Finally, in rickets conditions of muscular weakness have been described, which may be combined with atrophy. Characteristic histological appearances have been reported by Hagenbach and Bing; the latter speaks of a rachitic myopathy.

Some years ago I drew attention to the occurrence of a peculiar paralysis [1] in early childhood, associated with atony of the muscles, the symptoms of which closely resemble those of poliomyelitis. In this condition, which I term *myatonia congenita*, there is a striking flaccidity of the muscles of the lower, sometimes also of the upper limbs, much less often those of the trunk. The limbs may be moved like loose appendages; the tendon reflexes are weakened or lost. The muscles are not visibly wasted, but the electrical excitability is more or less diminished or may even be absent. Active movements are feeble, the child appears paralysed, yet on close examination one recognises that in most cases a certain amount of power of movement is present. In one of my cases, for example, while the legs lay as if almost absolutely paralysed, single movements could be obtained in response to the electrical stimulation, the muscle tonus increased a certain amount under one's hand, and the previously absent knee jerks could be feebly brought out. In another of our cases in which, at the time of observation, the leg muscles had again become capable of functioning, the Achilles jerk could be brought out, while the knee jerk was absent. I have established that this condition is capable of gradual improvement, and it is referable to a *delayed development* of the muscles, although a delayed development of the central parts, namely, the anterior horn cells, or of their functions cannot be excluded. The only case, that of Spiller, which, so far, has been examined post-mortem, although a somewhat doubtful one, confirms this supposition as a matter of fact, for only muscular changes were found, the nervous system being intact.

I have now had the opportunity of investigating eight or nine of these cases, two of which have been described by my pupils Kundt and Rosenberg. For further observations we are

[1] *M. f. P.*, viii., 1900, also *B. k. W.*, 1904. See also the works of my pupils: Kundt, "Über Myatonia congenita (Oppenheim)," Inaug. Diss., Leipzig, 1909; Rosenberg (*Z. f. N.*, xxxi.).

indebted to Muggia, Berti, Collier, Wimmer (*A. f. P.*, Bd. xlii.), Comby (*Archives de méd.*, 1905 and 1906). Jovane ("La Pediatria," 1906), Baudouin (*Semaine méd.*, 1907), Tobler (*J. f. Kind.*, Bd. lxvi.). Perhaps also some cases which have been otherwise interpreted (Sevestre, Hutinel) belong to this class.

The paralysis described by Vierordt in syphilitic pseudo-paralysis, and especially in rickets, as due to inhibition, has also a great resemblance to myatonia, but is absolutely different pathogenically. But that conditions of muscular weakness, possibly associated with atrophy, may be present in rickets, I have already pointed out in the second edition of this work. I do not consider that the degenerations shown by the Marchi method, which have been described by Zappert (Tiling, and others) in the anterior roots of young infants are related to this myatonia, as these appearances must be considered as quite physiological.

I saw a case in which there was a poliomyelitic paralysis limited to the lumbar extensors, which caused a loss of power of straightening the back, analogous to that seen in juvenile muscular atrophy. The rapid development and the presence of the reaction of degeneration enabled one to make the proper diagnosis.

If the anamnesis be lacking, it may be difficult to make a differential diagnosis from the Werdnig-Hoffmann form of progressive muscular atrophy (*q. v.*). Rapin (*Nouv. Icon.*, xix.) describes a hypertrophy of an extremity, either congenital or coming on in childhood, dependent on swelling of the skin and proliferation of the subcutaneous fat, which in its development and localisation reminds one of poliomyelitis. This, as he supposes, perhaps represents an abortive variety of poliomyelitis, in which the muscular atrophy is lacking; but it requires further observations and investigation before the relationship can be admitted.

Prognosis.—The prognosis is favourable as regards life. A fatal issue has only been observed in the first stage in exceptional cases. I have seen this once in a severe case with cervico-bulbar symptoms, which was quite atypical and was probably associated with cerebro-spinal meningitis. The danger to life is greater in that type of the disease which in its development and course is akin to Landry's paralysis. The involvement of the cervical cord, especially of its upper part, also appears to increase the danger to life. The prospects of complete recovery are exceedingly small, and yet in epidemics of the disease recovery may follow in no small percentage of the cases (Leegard). There are also isolated observations of this kind, which have contributed to the idea of an abortive form of the disease (Bury). In the great majority of cases improvement only can be looked for. *Ceteris paribus*, the more circumscribed the paralysed area at the onset of the attack, the less will be the permanent disturbance of function. Thus I have seen complete recovery follow in a case, in which at first the tibialis posticus was the only part involved. If a whole extremity be affected, some defect will always remain behind. Those cases are unfavourable in which both legs, or three or four limbs are affected, especially if there be simultaneous involvement of the trunk muscles. Yet Burckhardt has obtained complete recovery in a case of widespread paralysis—the disease came on in a fifteen-year-old girl after influenza—by a course of treatment which was mainly electrical, and which was begun early and carried out systematically.

We possess a valuable criterion for prognosis in the condition of the electrical excitability. Those muscles in which faradic excitability is not completely lost after the lapse of the first two to three weeks will probably

become again capable of function. The muscles in which a complete reaction of degeneration has appeared at the end of the first week will probably remain permanently affected; a certain amount of improvement, however, is not excluded, even in these. It is quite unusual, although I have seen cases of it, that in the muscles which are definitely paralysed there is a partial R. D. or merely a quantitative diminution in their electrical excitability.

The period of improvement extends over six or nine months, but may continue for a year or longer.

The prognosis as to recovery of function is further disturbed by secondary changes. Recovery of the power of walking may always be expected if only one leg is affected, and even if there be only partial paralysis of the other. Naturally, supporting apparatus will then be required. With the help of these walking is even possible in almost complete paralysis of the legs (Hoffa).

I once saw, while in the mountains, a young man with an almost complete flaccid paralysis of the legs, following on poliomyelitis, who had climbed a high mountain on his crutches, and got there entirely by the use of his arms.

The prognosis has become more favourable through operative measures (see below). But there are isolated cases in which all the muscles of the leg and back remain permanently paralysed.

In a certain connection the subjects of infantile paralysis are also endangered in later life, viz., in that they have and retain a *predisposition to atrophic paralysis*. At least it has been occasionally noted that in adult life, long after an infantile paralysis, there may develop a progressive muscular atrophy or a chronic anterior poliomyelitis (Charcot, Ballet-Dutil, Hayem, Raymond, Bernheim, Langer, Weber, Filbry, Cestan, Sarbó, Potts). Cassirer describes a combination of a poliomyelitis anter. acuta with dystrophy. Hirsch found a muscular atrophy of this kind, which developed in an adult who in childhood had suffered from poliomyelitis, and which was due to a diffuse cervical myelitis, which from all appearances had taken its start from the site of the old lesion. In one of my patients there occurred, four and six years after the onset of infantile paralysis, new attacks of paralysis which ran a course like a disseminated myelitis, and finally led to the production of a Brown-Séquard symptom. I have also seen cases of a less serious character, in which there set in, in adults who had suffered from infantile spinal paralysis, an *atrophic occupational paresis* which was capable of cure.

Treatment.—In the first stage our main aim is to prevent any extension of the process in the spinal cord. With this in view absolute rest is the first essential. Of course the child is kept in bed, all forcible active movements prevented, and coughing, straining, etc., diminished as far as possible. A moderate clearing-out of the intestine is recommended. A slight blood-letting may be of advantage at the commencement of the illness (application of several leeches to the back, according to the site of the disease). Nothing essential is to be gained through the application of an ice-bag, and it usually involves such an uncomfortable position for the child that it is better to do without it.

Diaphoretic treatment, on the other hand, is eminently desirable. When possible one tries to evoke sweating, not by baths, but by

wrapping up in woollen garments and giving warm drinks, or by hot-air applications. We have found the electric hot-air apparatus especially valuable. Children are thrown into perspiration most easily by it, and it saves the manifold handlings required in carrying to a bath.

The employment of *lumbar puncture* in the first stage would appear to be rational, if one were not afraid that the diminution of pressure might cause rupture of the congested and diseased vessels in the inflamed area. Finkelnburg [1] recommends it on the strength of one case; but further experience of it is desirable.

In one severe case of cerebro-spinal meningitis in my practice, at all events, it did not prevent the onset of a widespread poliomyelitic paralysis.

Medicinally, salicylate preparations may be recommended in the feverish stage, also belladonna.

As a rule children come first under our care after the disappearance of the general symptoms, after the time when the paralysis has developed and defined itself. Now comes the time for electrical and mechanical treatment. The child should be confined to bed, however, especially if the back muscles are affected, and should not be allowed to walk until the doing so is no longer likely to cause any increase in deformity. As soon as active movement returns in any part of the muscular system, it is advisable to aid this through electricity, gymnastics, and massage. One can begin this after the course of the first two or three weeks. This treatment applies also to the muscles adjacent to those still in a condition of paralysis. The method recommended for electrical treatment is the use of the *galvanic current* in such a way that the cathode is applied to an indifferent part, or else over the part of the back corresponding to the site of the lesion, while with the positive pole the affected muscles are stimulated, or the skin over them is stroked. In order to win the child's confidence, one begins with a weak current, or with practically none at all. The current can then be gradually increased till it brings out muscular twitchings. The galvanic stream will be directed, moreover, through the spinal cord. The muscles which as yet, or again, react to the faradic current (direct or indirect) may also be stimulated by it.

Gymnastics consist in getting the little patient to carry out those movements of which he has again become capable. One can then employ correspondingly slowly increasing resistance to the muscular power. As the so-called "habit-paralysis" appears to play no small rôle in childhood, and, according to my experience, in this disease also, great weight is to be laid on using the muscles. Hoffa recommends Krukenberg's pendulum apparatus for this exercise. In the case of definite atrophy of certain muscles, one must strive through purposive movements to strengthen others which can act in a compensatory manner for them, as *e.g.* Kron, who has succeeded in this way in compensating for loss of the deltoid by using its supplementary muscles (pect. major, supra- and infra-spinati, etc.).

Massage is of especial value as it counteracts any circulatory sluggishness, and places the muscles under favourable nutritive conditions. A gentle rubbing and stroking of the muscles is to be recommended; later they may also be kneaded. Particular care should be taken to prevent the formation of contractures by the early employment of the proper passive movements. The foot should be protected by a wire-cage from

[1] *M. m. W.*, 1904.

the pressure of the bedclothes. Children should not lie with their legs drawn up. If the tendency to contracture is marked, bandages and apparatus should be employed to bring the limb into a position opposed to that of the contracture; this can be achieved by strips of adhesive plaster, by Scarpa's shoe, or other appliances.

Warm baths at 90°-95° F. (or 32°-35° C.), with the addition of sea salt, Kreuznach lye, etc., also have a favourable influence on the course of the disease at this stage; the bath treatment of Nauheim, Kreuznach, Tölz, Kolberg, etc., may also be recommended.

To overcome the paralysis subcutaneous injections of strychnine may also be advised.

The treatment of any deformities which may develop comes under the domain of orthopædic surgery. *Tenotomy* of the shortened tendons is often necessary (eventually also section of the fasciæ), as well as forcible extension, further measures, such as the application of extension bands and splints, being premised. In the selection and construction of these great care is necessary; they must fit well, exercise no pressure, cause no interference with the circulation, and further one must take into account the constant modifications of the apparatus required by the growth of the patient. Hessing's shell splint and its modifications (Hoffa) have been recommended during the last few years, and personally I have found it do well. It acts on the one hand as a contrivance for giving a firm support to the limb, which is flail-like through joint relaxation, on the other as supplementary to the wasted muscles through rubber bands, which counteract the contraction or contracture of the antagonists. Accurate accounts of the various apparatus and their application to different parts of the body will be found in Hoffa's work ("Die Orthopädie im Dienste der Nervenheilkunde. Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie," Bd. v., 1900). Besides tenotomy, other operative procedures may be necessary. Thus the unserviceableness of the extremities from flail-joints, or even from secondary contracture, may be compensated to a certain degree by arthrodesis (Albert), *i.e.* by ankylosis of the joint and fixation of the limb in its normal position. Operations of this nature have been carried out at the various joints after the methods of Albert, Winiwarter, Dollinger, etc., even at all the joints of one, or at various joints of both extremities at the same time. At first this procedure was recommended chiefly for the shoulder, but now it is mainly employed for the lower extremity. Karewski has obtained the best results at the ankle-joint. Such measures are only indicated when spontaneous improvement can no longer be expected, and conservative methods of treatment are having no further effect (usually after one or two years). But an attempt should always first be made to obtain the requisite fixation of the extremity by good apparatus. Plastic operations on the tendons (see below) or tenodesis (Tilanus, Codivilla, Reiner) offer in many cases an alternative to arthrodesis. For great shortening of the leg Mikulicz recommends arthrodesis of the ankle-joint in the pes equinus position, so that the length of the extremity is artificially increased by the length of the foot. Infrapubic luxation may render operative reduction of the hip-joint essential (Karewski). Deutschländer refers to further bone operations. For severe paralytic flexor-contractures at the knee-joint Hoffa does mainly a supracondylar osteotomy.

For several years the long-forgotten operation originally suggested

by Nicoladoni and revived by Drobnik, viz. the *transplantation* of tendons or muscles [1] has come more and more into use.

The principle of the method lies in this, that an attempt is made to restore the function of a muscle useless through paralysis and atrophy, by this means, that its tendon is united with that of a neighbouring muscle, which is intact functionally and nutritionally. One may cut through the diseased muscle and stitch its tendon to that of the "strength-giver" (ascending, passive transplantation), or—and this is the favourite method—cut through the tendon of the healthy muscle and unite it with that of the paralysed one (active transplantation). The complete transplantation of a healthy muscle on a paralysed one is only admissible when the former is of no very great importance for the use of the extremity. That holds good, *e.g.* for the sartorius (and the biceps femoris), in paralysis of the quadriceps. The more usual operation consists in splitting off a part of the tendon, which is then stitched to that of the paralysed muscle or implanted on its point of insertion. Thus in pes equinovarus the Achilles tendon can be split lengthwise and stitched to the tendon of one of the extensors or abductors. Lange, however, objects on principle to tendon splitting, and also to the employment of the antagonists. Hoffa also favours total descending transplantation, and avoids wherever possible the union of a split-off muscle graft with an antagonist. With this procedure is combined, when necessary, the operative shortening of the relaxed and lengthened tendon, or, more rarely, the artificial lengthening of a shortened, contracted one. Naturally the operation is carried out most frequently on the leg or foot muscles.

Examples (after Hoffa).

Operation for pes equinus on the assumption of a paralysis of the ext. comm. dig. and tib. ant. : Lengthening of the Achilles tendon by Bayer's method, shortening of the extensors and transplantation of the peroneus brevis on the dorsum of the cuboid and of the ext. long. hallucis on the dorsum of the scaphoid or on the shortened ext. comm. dig. The peripheral end of the ext. long. halluc. is attached to the shortened ext. comm. dig. If the ext. long. halluc. is also paralysed, the flex. long. halluc. can be implanted on this or on the scaphoid. If that will not do it is recommended to split off two shoots from the Achilles tendon, of which the one is implanted on the tib. cant. and the other on the periosteum on the outer side of the foot.

Paralytic pes equinovarus, paralysis of the peronei and extensors : Lengthening of the Achilles tendon by Bayer's method, shortening of the extensor muscles, transplantation of the tib. post. and perhaps also the tib. ant. to the outer side of the foot, or to the dorsum of the cuboid bone. Other things failing, stitching of the shortened tib. post. to the peroneus brevis, etc. Further details will be found in the treatise quoted.

Atrophic paralysis of the extensor cruris quadriceps has given occasion in a number of cases for transplantation of the sartorius and biceps femoris on the patellar tendon, the patella or tuberosity of the tibia (Lange, Krause, Hoffa, Schanz, Magnus, and others). Lorenz has raised a protest against transplanting the flexors to the extensor side. In the upper extremity an extensor paralysis has most frequently been the indication for transplantation, but the deltoid or the biceps has also been

[1] For the full original and casuistic discussions see the works of Nicoladoni, Drobnik, Gluck, Milliken, Lange, Kunik, Franke, Hoffa, Gocht, Codivilla, Noehte, Vulpius, Jordan, Tubby, White, Hackenbruch, Heusner, Redard, and Wollenberg quoted here. For a minuter study of these questions I would recommend the writings of Vulpius, "Die Sehnenüberpflanzung und ihrer Verwertung in der Behandlung der Lähmungen," Leipzig, 1902 ; *ibid.*, *B. k. W.*, 1906. A. Hoffa, "Über die Endresultate der Sehnenplastiken," *Arch. f. klin. Chir.*, Bd. lxxxi. Auffret, "Transplantations tendineuses," etc., *Thèse de Paris*, 1905.

grafted on the triceps (Krause-Oppenheim), the trapezius on the deltoid, or the pectoralis major on the deltoid (Hildebrand, Sachs). The last operation has been done with the special object of fixing a flail-joint at the shoulder. According to the reports of orthopædic surgeons, especially those with a large experience such as Vulpius, Codivilla, and Hoffa, the results are excellent. Hoffa makes out with regard to it, that, although the operation does not always succeed in bringing back muscular function, yet it leads to reduction of the deformity; he lays stress on the necessity of skilful treatment before and after operation, avoidance of suppuration, etc. It is very important in these plastic procedures on tendon and muscle that the muscles which are turned to account as "strength-givers" should be healthy, or at least not much altered as regards their functional power and their reaction to electricity. In infantile spinal paralysis, in which these operations are most frequently carried out, there are often beside the quite atrophic muscles, others less atrophied, still capable of function, yet not strong enough to act as "strength-givers." From a large personal experience (I have investigated a particularly large number of cases of this sort for the Bergmann Klinik) I can state that it is not always easy to come to a decision on this question. Hoffa, it is true, believes that the restoration of the muscle to normal tension has a favourable influence on regeneration and function. Of course operative measures should only be contemplated when the disease has subsided and when no further spontaneous restoration of function is to be expected.

By some surgeons, especially Lorenz, the sphere of operations has recently been narrowed.

It is not necessary here to go into the further efforts and modifications of tenoplasty, the proposals of Lange and J. Wolff to unite the transplanted muscle, not with the tendon of the paralysed one, but to the place of insertion on the bone, or those of Gersuny and others.

It must be mentioned, however, that nerve grafting has also been recommended, *e.g.* the union of a slip of the posterior tibial nerve with the peroneal (Spiller-Frazier, Hackenbruch, and others). This was originally employed in peripheral paralyses (*q.v.*). I have seen the method tried without result.

Acute Anterior Poliomyelitis of Adults, Acute Atrophic Spinal Paralysis of Adults

This is a disease rarely observed, but still a considerable number of cases have been recorded and confirmed in several instances by autopsy (Schultze, Friedländer, Williamson, Strümpell-Barthelmes, Taylor, Hoch, Lövegren,[1] Gehuchten,[2] F. Schultze,[3] Wickman[4]). It is usually persons between the ages of twenty-five and thirty who are affected. It may follow on the acute infectious diseases, especially measles, or may occur during or after the puerperium. In one case which I saw it was preceded by a severe gonorrhœa. Gumpertz has assumed a typhoid causation in one instance. It would also seem as if a simple severe chill were sufficient to cause the disease. According to Rank's statistics this seemed

[1] "Zur Kenntnis der Pol. ant. acuta, etc," Berlin, 1904.
[2] "Névraxe," 1904.
[3] Ziegler's *Beiträge*, xxx.
[4] "Studien über Pol. ant. acuta," etc., Berlin, 1905.

to be the chief etiological factor in twenty-five of thirty-six cases. Over-exertion and trauma have also been blamed. I once saw an atrophic paralysis, probably of spinal origin, follow on a prolonged chloroform narcosis.

The illness commences, as in the infantile form, with general disturbances and with a rise in temperature, but the febrile stage has, in this instance, as a rule, a longer duration, and may last over one to two weeks. Pains may be present, especially pain in the back, which, as Strümpell has specially pointed out, is sometimes very severe; should severe persistent pain appear in the extremities, it indicates an involvement of the peripheral nerves, and in doubtful cases points to peripheral neuritis. In one case this symptom was referred by Raymond and Lejonne to a meningitis in the region of the posterior roots. With the disappearance of the fever, or more rarely at its height, the paralysis appears, in most cases involving a larger area of the body than in the infantile form. Both legs, both arms (diplegia brachialis), or even all four extremities are affected. A crossed type is described by Lejonne and Schmiergeld.[1] The paralysis has now all the characteristics described under the infantile form. By the fourth or sixth day after its appearance, the changes in electrical excitability—reactions of degeneration—can be demonstrated. The increase in galvanic excitability is in most cases only present for a short time. Partial reaction of degeneration has been found in individual muscles. Grawitz has described involvement of the diaphragm. Cranial nerves may share in the paralysis, but only exceedingly rarely, as instanced by Taylor and also Erb [2] (and referred to an involvement of the medulla oblongata). Within the first few weeks or months the area paralysed begins to diminish. Some of the muscles, which had become incapable of voluntary contraction, regain their power of movement, while others, usually whole segments of a limb, remain permanently paralysed and their muscles atrophy. The characteristic localisation-type of E. Remak is markedly brought out. It is found here, much more frequently than in infantile paralysis, that several or even all the extremities may continue to be paralysed, and that later a number of muscles may everywhere recover their power. But I have also seen all the muscles of one upper extremity become paralysed and atrophied, and remain so in full intensity, while an improvement appeared only in the other arm which was previously partially paralysed.

Here also complete recovery is rare, so rare that in those cases where the paralytic symptoms entirely disappear there must be a doubt as to the nature of the disease, and the possibility that there may have been multiple neuritis must be kept in view. Improvement begins usually after the course of one or more months; frequently, however, several months pass before the least trace of movement becomes evident, and it may be a year or more before that degree of improvement is reached at which the disease remains stationary. In one case I could still record after two years an increase of movement in certain muscles. Electrical excitability returns to normal later than the motor power. Not infrequently an excessive development of fat subsequently occurs in some of the atrophied muscles, so that a superficial resemblance to the pseudo-hypertrophic form of progressive muscular atrophy may ensue.

Differential Diagnosis.—With reference to this, confusion with multiple

[1] *R. n.*, 1907, and Schmiergeld, *Thèse de Paris*, 1907.

[2] *D. m. W.*, 1906.

neuritis is to be specially guarded against (see previous chapter). In hæmatomyelia there is a still more rapid development of paralytic symptoms, and the early febrile stage is absent. As a rule also there are sensory disturbances, and frequently the sphincter functions are interfered with. This fact is also the most important in the distinction of myelitis and specific spinal disease from poliomyelitis. Yet I have seen [1] a disease develop in a syphilitic patient undergoing energetic mercurial treatment which could only be differentiated from acute anterior poliomyelitis by the presence of a slight involvement of the sphincter vesicæ. See also the chapter on Landry's paralysis. That acute poliomyelitis in adults may present the clinical picture of a Landry's paralysis has been brought out especially by some of the cases described by American and Scandinavian physicians.

The *prognosis* as to life is very favourable. Only quite exceptionally does the condition lead to a fatal issue through involvement of the respiratory muscles. Complete recovery is very rare, and in those muscles which, after a period of four to six months, still show no trace of movement, none need be expected.

On account of the average greater extension of the paralysis, the prognosis is not so favourable as in the infantile form ; on the other hand the chances of restoration of function are better, in that the disturbances of bone-growth and the deformities caused by secondary contracture as a rule do not occur. Even then, where the movement of the legs in the dorsal position is reduced to a minimum, these persons learn usually to get about with the help of crutches and by a compensatory use of the non-paralysed or slightly affected pelvic muscles.

With regard to *therapeutics* there is nothing to be added to the remarks made under infantile spinal paralysis. Of the remedies recommended ergot must be mentioned ; it should be administered subcutaneously in combination with atropin. (Ergotin 10·0, atropin 0·01, $\frac{1}{3}$-$\frac{1}{2}$ injected twice daily.)

Subacute and Chronic Anterior Poliomyelitis (Subacute and Chronic Atrophic Spinal Paralysis)

Literature : Oppenheim, *A. f. P.*, xix. and xxiv. ; Nonne, *Z. f N.*, ii., *B. k. W.*, 1896 ; J. B. Charcot, "Arch. de méd. expér.," etc., 1895, and *Thèse de Paris*, 1895 ; Dejerine-Thomas, "Traité de méd.," etc., ix. ; Medea, *M. f. P.*, xxiii.

These rather rare forms of atrophic spinal paralysis are met with chiefly in elderly people. Their causation is unknown, but the great similarity of the clinical features to the various types of lead paralysis makes one suspect that some toxic substance is responsible. The observation of chronic anterior poliomyelitis in the course of a diabetes (Nonne) is quite in agreement with this assumption. On the other hand Erb [2] especially, followed by E. Meyer, Perrin, Starck, Pagenstecher, Nonne, and others, has pointed to the traumatic etiology of the disease, and Schmaus-Sacki have studied these questions more closely ; they showed that injuries or concussions lead above all to disturbances of the lymphatic circulation, to local lymph stasis and tissue necrosis, and have in this way approached a comprehension of the genesis of chronic cord diseases from trauma.

[1] *Z. f. N.*, xxiv. [2] *Z. f. N.*, xi.

Allied diseased conditions may develop from syphilis (Dejerine, Oppenheim, Eisenlohr, Schultze, Medea), and corresponding pathological conditions have been reported in some of these cases, but other cases with very different symptoms have been wrongly included in this group (*e.g.* Lévi and Wilson). Whether bodily over-exertion can bring on this disease (Raymond-Guillain) is doubtful. A tendency to affect members of one family can but rarely (Bruining [1]) be established.

The cases of subacute development are particularly rare; their frequency was rated too high, when multiple neuritis was less well understood; it is certain that the great majority of the cases brought together as subacute poliomyelitis belong to the category of polyneuritis. On the other hand, there are without doubt cases of pure poliomyelitis with a subacute or chronic development, which, as shown by cases with post-mortem examination, especially one described by myself, can be clinically differentiated sharply from multiple neuritis, in that the motor apparatus is exclusively involved, and disturbances of sensibility are entirely lacking.

The development of the disease is usually as follows: the patient, healthy up till then, becomes aware of a weakness in one extremity, which increases from day to day, so that after several weeks its usefulness is quite markedly diminished. The arm is as frequently affected as the leg. The weakness then involves the arm or leg of the opposite side, or it begins in one arm and then involves the leg of the same, or of the other side, so that in the course of several months a partial paralysis of both legs, both arms, or of all four extremities is produced.

If we examine the patient at this stage, we find a flaccid paralysis, which, although it has extended to several extremities, still shows a selective character, inasmuch as there are always individual muscles or muscle groups spared or less affected. In paralysis of the upper arm muscles, the triceps may remain unaffected; in paralysis of the whole arm, the flexors of the fingers, the adductor pollicis, and other finger muscles may be spared. In the legs the region supplied by the peroneal nerve may be paralysed completely, or with exception of the tibialis anticus or peroneus longus, while the remaining muscles are only slightly affected. The paralysis is an absolutely flaccid one; the tendon reflexes in the area of the affected muscles are diminished or lost, although, according to some observations, there appears to be an exaggeration at the beginning of the process, especially in the muscles that are not as yet atrophied. The paralysis, further, is always a degenerative one. Atrophy usually follows quickly on paralysis, and the patient observes that the muscles have not only lost their strength, but that they have also diminished in size. But even where atrophy is not evident, the presence of the reaction of degeneration points to the degenerative nature of the paralysis. The R. D. will be complete in certain nerve-areas, partial in others. In certain muscles which cannot be moved voluntarily, the electrical excitability may be only slightly diminished, while conversely in non-paralysed muscles the reaction of degeneration may be sometimes present. An exact parallelism between paralysis and degeneration is not therefore present in all the muscles.

Fibrillary twitching is almost always noted. Sensibility, in pure cases,[2] is in every respect normal. Slight rheumatic pains may be present

[1] *Z. f. N.*, xxvii.

[2] There have been described by myself, Schuster (*N. C.*, 1897), and others, cases in which, in addition to the anterior horn lesion, a slight degeneration of the posterior columns was found.

at the beginning and during the course of the disease, but they play a quite subsidiary rôle. Bladder troubles are absent throughout. The sexual powers remain preserved—in short, muscular paralysis and muscular degeneration form the sole signs of this disease.

According to the further course of the disease, one can distinguish different types of the same: There are cases in which the disease is stationary; after one arm or both arms (one leg or both legs) have come to a condition of incomplete atrophic paralysis, *i.e.* with not all the muscles involved, the process comes to a standstill. This stage may be reached in several weeks, and the disease remains stationary.

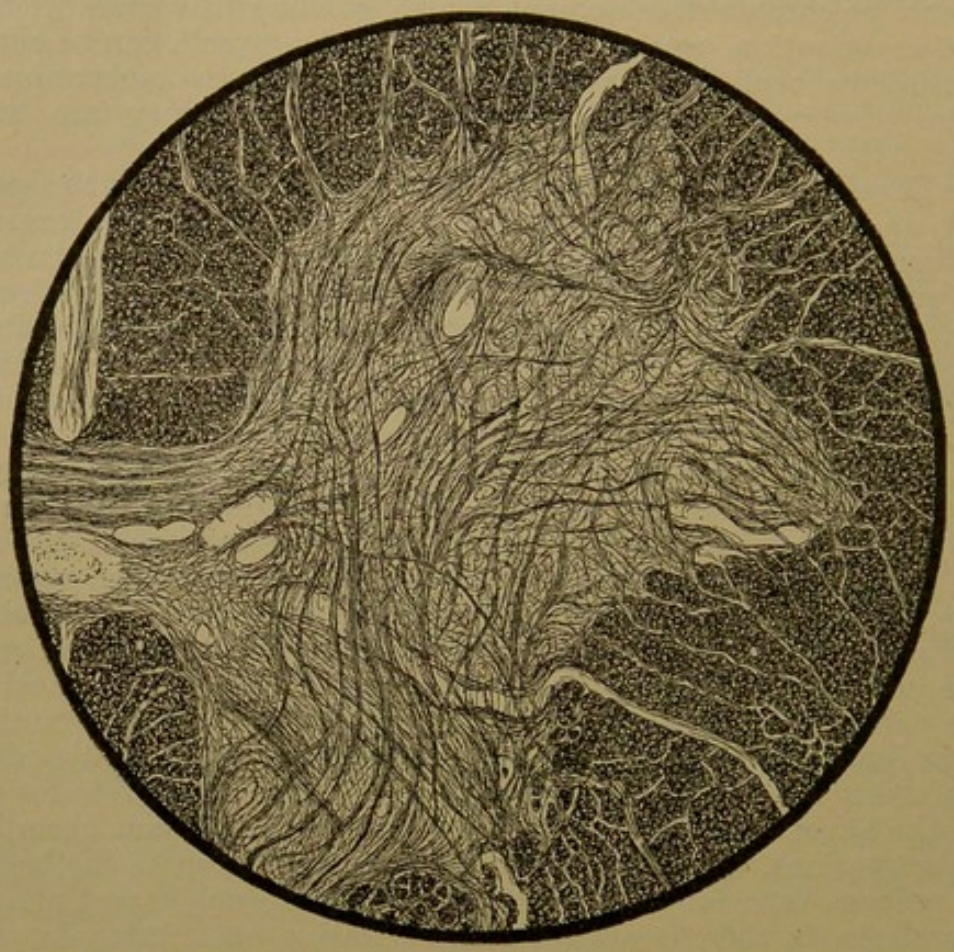

Fig. 115.—(Compare with Fig. 116.)—Atrophy of anterior horn, especially of ganglion-cells, in chronic anterior poliomyelitis.

We owe a very remarkable observation to Eversmann (*N. C.*, 1900). The atrophic paralysis, which had affected a large number of the muscles of the body, came to a complete and definite standstill after lasting eight years, so that the author speaks of a poliomyelitis adultorum decursa. The acute onset and the real hypertrophy of certain muscles were also unusual features in this case.

In a further series of cases, after the process has reached its height, even after six or eight months, improvement follows, and this is either

so that a sort of combined anterior horn—posterior column degeneration—was present. Pal has discussed the question fully, and states that a degeneration of the long posterior column fibres, as well as of the cerebellar paths may be combined with the anterior horn lesion. In atypical cases of this sort, slight disturbances of sensibility may be present. The combination of poliomyelitis ant. chronica with cavity formation has been referred to, especially by Rossolimo.

incomplete or it goes on to a cure. Probably, however, a multiple neuritis underlies some of the cases included in this group, or else one is dealing with mixed forms of spino-peripheral paralysis (see chapter on Multiple Neuritis).

Finally, there are cases with a chronic progressive course and fatal termination. As a rule in these cases the development is subacute, as described above, but now one region after another is affected, and the intensity of the paralysis in the muscles involved increases from day to day—till finally all four extremities and some of the trunk and neck muscles are included in the paralysis. The patient then lies motionless in bed with flaccid, atrophied limbs; he can no longer move himself from one position

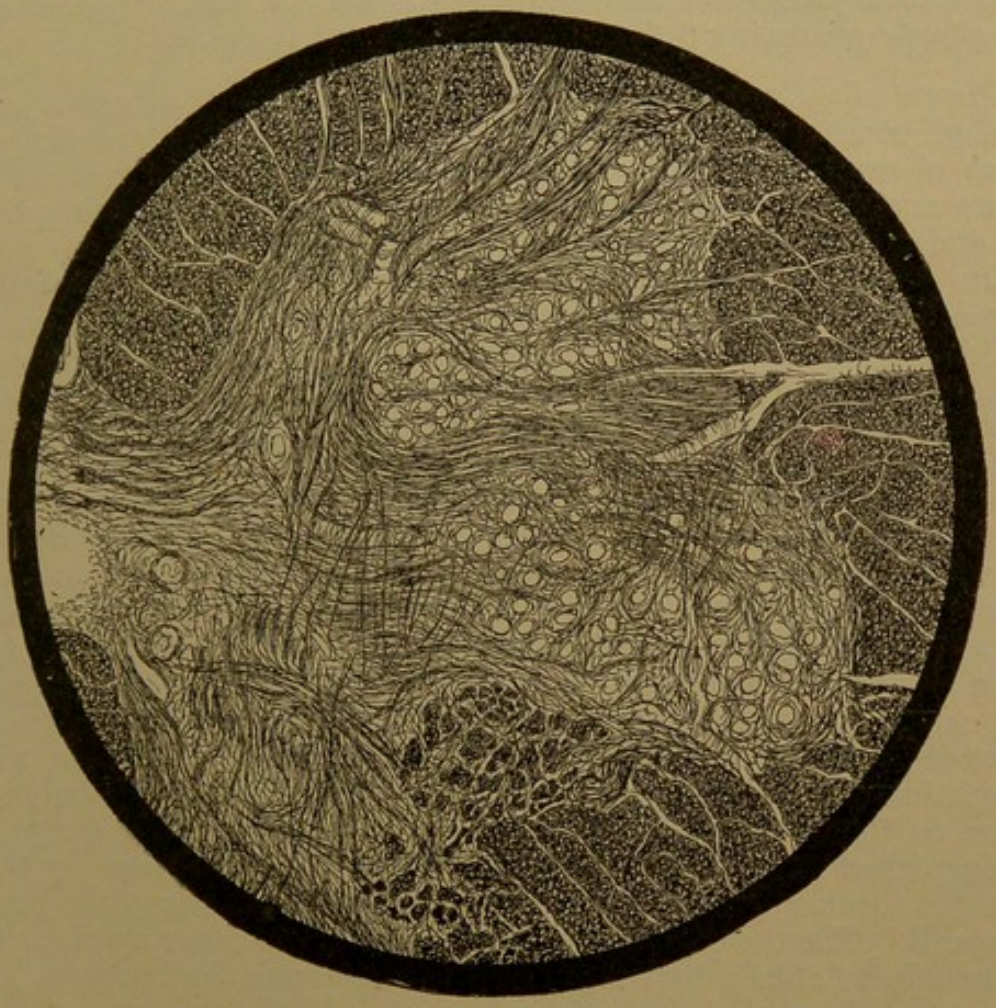

Fig. 116.—(Compare with Fig. 115.)—Normal anterior horn of lumbar enlargement. (Weigert's method.)

into another, he has no further complaints, no pain, and bed sores do not form. Now respiratory troubles, and often also bulbar symptoms, are added, and the patient dies from asphyxia, deglutition pneumonia, or some intercurrent illness—bronchitis even is dangerous on account of the weakness of the respiratory muscles. The duration of the disease varies from one to three years.

The pathological investigations carried out by Oppenheim, Nonne, B. Charcot, Dejerine, Grunow,[1] Starck, Aoyama,[2] Bruining, Lövegren,[3]

[1] *Z. f. N.*, xx. [2] *Z. f. N.*, xxvi.
[3] "Zur Kenntnis der Pol. ant. acuta und subacuta s. chronica." Berlin, 1904.

and Moleen show the presence of a primary chronic inflammation of the anterior horn terminating in atrophy, and specially with complete destruction of the nervous elements—the ganglion cells and nerve fibres (compare Figs. 115 and 116). The white matter is practically normal or only shows in isolated scattered fibres a limited atrophy, which appears in the neighbourhood of the grey matter, and perhaps may be attributed to the destruction of the column cells.

There seems therefore to be a disease of the anterior horns arising from the vessels, as well as a primary atrophy of the nerve cells.

A marked involvement of the vessels is shown in one of the cases described by Bielchowski (*Z. f. kl. M.*, Bd. xxxvii.), and also in one of R. Ewald's ("Inaug. Diss.," 1899). Marinesco gives a full account of the histological process in the ganglion cells (*C. f. N.*, 1898). The condition of the muscles coincides practically with that described under acute poliomyelitis.

Differential Diagnosis.—In the cases running a subacute course, confusion with multiple neuritis comes especially into question. The absence of sensory irritant and paralytic symptoms, of sensitiveness to pressure and swelling of the nerve trunks, of ataxia, of psychic disturbance, and of the known noxæ which bring about polyneuritis, point in doubtful cases to poliomyelitis. The distribution of the paralysis (see the preceding chapter) may also afford valuable evidence for the differentiation.

In the cases with a chronic progressive course, the conditions especially to be borne in mind, with regard to a differential diagnosis, are amyotrophic lateral sclerosis, progressive muscular atrophy, and gliosis. The lack of all spastic phenomena and the obliteration of the tendon reflexes point to chronic poliomyelitis. If they are markedly increased in the legs only, with a general flaccid condition of the muscles, and if in addition Babinski's or Oppenheim's sign is present, then we are dealing probably with an amyotrophic lateral sclerosis. The exaggeration of tendon reflexes alone is not a certain sign however, as that may also be of neurasthenic origin. Commencement of the paralysis and atrophy in the small muscles of the hand, an early appearance of weakness in the legs, without degeneration, and marked accentuation of bulbar symptoms, point likewise to this disease. Progressive muscular atrophy of spinal origin is distinguished from chronic poliomyelitis in the strict sense by the fact that in the former the atrophy is the primary symptom and the paralysis is its result. The development also proceeds much more slowly, and the atrophy affects chiefly individual muscles (atrophie individuelle), not a whole group of muscles (atrophie en masse). In the majority of cases, it is the small muscles of the hand which are first affected, while poliomyelitis begins more often in the legs or in the shoulder and upper arm muscles. This separation of chronic poliomyelitis from progressive muscular atrophy of spinal origin is, however, regarded as an artificial one, and it must be admitted that the division cannot be strictly maintained on the strength of the histological evidence.

Primary myopathies only exceptionally give rise to clinical pictures bearing a certain resemblance to pol. ant. chronica (Oppenheim-Cassirer, Dejerine-Thomas).

Gliosis is characterised by an extremely gradual development of the atrophy, by the presence of sensory disturbances, and frequently also of trophic changes in the skin and joints.

Prognosis.—This is always doubtful as regards complete recovery. It is the more favourable, the more rapidly the paralysis becomes defined, and the more incomplete it is. If we find only a partial reaction of degeneration (only slightly diminished nerve excitability), recovery may be hoped for. The purer the case is, and the more certainly neuritis can be excluded, the worse is the outlook with regard to complete recovery. If the degenerative paralysis proceeds gradually and slowly, seizes on one set of muscles after another, and if bulbar symptoms appear, the outlook is bad, and a fatal termination may be expected.

Treatment.—In the first stage diaphoretic measures are recommended. Any over-exertion of muscles, whether they be already affected or still intact, is to be avoided. Electrical treatment should be carried out on the lines laid down for the acute form. No objection can be urged against the employment of gentle massage. Strychnine is not of much use in this condition.

If the disease is stationary the therapeutic measures referred to in the chapter on acute poliomyelitis should be considered.

Amyotrophic Lateral Sclerosis

Literature: Leyden, *A. f. P.*, viii.; Kahler, *Z. f. Heilk.*, v., and *Vierteljahrschr. f. p. Heilk.*, 1879; Charcot, *A. de Physiol.*, 1870, "Leçons," etc., ii.; Duval-Raymond, *A. de Physiol.*, 1879; Strümpell, *A. f. kl. M.*, 1888; Charcot-Marie, *A. de Neurol.*, 1885; Oppenheim, *A. f. P.*, xxiv.; Strümpell, *Z. f. N.*, v.; Tooth-Turner, *Br.*, 1891; Raymond, "Leçons," etc., 1903, and onwards; Anton, *W. kl. W.*, 1896; Probst, *A. f. P.*, xxx., and *Sitz. d. k. Ak. d. W.*, Bd. cxii.; Pilcz, *Jahrb. f. P.*, 1898; Mott-Tredgold, *Br.*, 1902; Raymond-Cestan, *R. n.*, 1905; Spiller, "Univ. of Penn.," 1905; Rossi-Roussy, *R. n.*, 1906. See also Ballet in the "Handbuch d. path. Anat. d. Nerv.," ii.

Amyotrophic lateral sclerosis (Charcot) is a disease of middle life. It has been observed only in isolated cases in childhood, and then in brothers and sisters (Seeligmüller, Gee, O. Maas, Hoffmann, Holmes[1]), but unsupported by any pathological evidence.

The cause of the disease is still unknown. It has been attributed to chill, trauma (Clarke, Joffroy-Achard,[2] Hauck, Ottendorf, Giese[3]), over-exertion, and fright. In one case observed by myself the symptoms developed directly after a severe fright, whereby the affected person was at the same time compelled to exert all his strength in rowing.

There is much to be said for the assumption of Strümpell that a congenital predisposition—a weakness of the corresponding motor apparatus present from birth—is the most important factor in the etiology. Thus micrognathia was present in one of my cases; in another there was a familial malformation of the thumb.

It appears to me to be doubtful whether the disease can develop from pellagra (Testi).

Symptomatology.—In typical cases we find an association of three groups of symptoms: those of chronic anterior poliomyelitis, of spastic spinal paralysis, and of bulbar paralysis.

The disease shows almost always a chronic development. It begins with weakness and atrophy in the upper, or with weakness and stiffness in the lower extremities. Fibrillary twitchings, often involving a wide area, may precede the muscular atrophy and form the first sign of the disease.

[1] *R. of N.*, 1905. [2] *Arch. de méd. Expér.*, 1890. [3] *D. m. W.*, 1904.

Usually one arm or one leg is more markedly affected. A true hemiplegic type also occurs not so infrequently. The paralysis and atrophy gradually extend and intensify on the one hand, the muscular rigidity on the other, while other symptoms, especially pain, are absent or unimportant. Within a period of six or eight months both arms and legs may be in a state of advanced paresis.

Objective examination in this stage, shows a condition somewhat as follows. The upper arms are strongly adducted, the fore-arms flexed, the hands pronated, frequently also excessively bent, while the fingers take up a position more or less completely resembling the claw-hand. This attitude depends on muscular tonus, on active contracture which especially affects the pectoralis major and latissimus dorsi, the flexors of the forearm, hand and fingers, as well as on the atrophic paralysis. The spastic condition of the muscles can at first be overcome.

The *tendon reflexes* are markedly exaggerated. A slight tap on the tendons of the supinator longus or triceps, or on a bony part of the forearm or hand, causes contractions in a number of muscles. Sometimes tremor of the hand may be brought out.

Fibrillary twitching and *muscular atrophy* are striking symptoms. The latter appears first in the small muscles of the hand. Simultaneously or at a later date the shoulder muscles and those supplied by the musculo-spinal nerve are affected. Electrical investigation shows complete or partial reaction of degeneration, or in some cases simple quantitative diminution of excitability.

Motor weakness is combined with the atrophy and contracture. It is not simply a result of these conditions. Their independence is shown by the fact that the weakness affects muscles which are not yet atrophied and whose motility is not diminished by spasm. The paralysis may be almost a complete one in the arms at the time of examination, yet, in most cases, a slight power of movement is usually retained for a long time.

While the signs of a spastic-atrophic paralysis are present in the arms, the condition of the lower extremities differs for the most part from that of the upper, principally through the absence of atrophy, but there appears here early a *marked rigidity*, the recognised characteristic of spastic spinal paralysis. We have found that Babinski's sign and Oppenheim's sign may be either present or absent. Raymond and Cestan think that it is usually absent even in typical cases. The power of walking remains long preserved, the gait is altered in a typical manner, slow, with short steps and stiffness of limbs ; finally, the patient drags himself along with difficulty, his toes clinging to the ground. After the illness has lasted one to two years, seldom more, the patient is confined to bed, the more so as he has no longer the use of his hands to prop himself up, and help him along while walking. In the later stages the atrophy appears in the legs also, but it never reaches the degree that it does in the upper extremities. In only three of my cases was atrophy prominent in the lower limbs in the first stage, and since it involved the whole of the leg muscles, there was here complete atony with loss of Achilles jerks, while the knee jerks were increased.

Pain is usually quite absent. Paræsthesiæ may be present (the constrained position of the arms tends to subject the nerve trunks to pressure). Objectively, sensibility remains normal and bladder and rectal

functions are not interfered with (*vide infra*); in short, *atrophy*, *rigidity*, and *paresis* form the sole symptoms of this disease.

Later on the symptoms of *bulbar paralysis* appear. Although as a rule these only reach their full development in the last stages, yet some signs may come on quite early, simultaneously with the onset of paralysis in the extremities. The bulbar paralysis may even be the first to manifest itself. To begin with, the patient speaks somewhat indistinctly, and nasally. This gets gradually worse and worse, until there is well-marked *dysarthria*, and finally *anarthria*, along with the speech trouble, or somewhat later *dysphagia* will be noticed; solid food is swallowed only with difficulty, fluids regurgitate through the nose, etc. Mastication may also be interfered with. These disturbances of function depend on paralytic phenomena in the muscles of the *lips*, *tongue*, *palate*, and *jaw*. But just as in the extremities rigidity and atrophy are combined with the paralysis, so here also along with paresis are the signs of rigidity and atrophy. The rigidity may even be the earliest symptom of this form of bulbar paralysis, and shows itself especially in an exaggeration of the jaw jerk, or in *masseter-clonus*. Trismus may also be present (Schlesinger [1]). The atrophy is wont to become marked first at a later stage: the lips are thin, the tongue lies limp on the floor of the mouth, greatly wrinkled, with fibrillary twitching and spongy feeling. The result of electrical testing is partial reaction of degeneration, but it is not always present and often is first noticed just before death.

In the final stage the lower half of the face is quite rigid, the mouth stands open, saliva trickles out between the lips, the corners of the mouth are drawn down, the lips cannot be pressed up, nor a light blown out; the lower jaw sinks down, there is only a trace of power to put out the tongue, the speech has become an unintelligible lalling, swallowing is no longer possible, and there may be also *aphonia*. The soft palate no longer rises on phonation, laryngoscopic examination shows paresis of the adductors, etc. The patient readily laughs and weeps (the latter especially), and these expressions of emotion have sometimes a convulsive character. Meanwhile the paralysis on the extremities has also advanced more and more; with increasing atrophy the rigidity may diminish, so that the limbs, hitherto rigid, become flaccid.

In this last stage also all other functions remain undisturbed. Of the cranial nerves only the motor ones—and even then not those for the eye—are affected. Only in one atypical case of Strümpell's has there been an associated paralysis of the eye-muscles. Death occurs from asphyxia, inanition, and, most of all, from aspiration pneumonia.

The average duration of the disease is from two to four years (twenty-six months according to the observations of Raymond-Cestan), but it may be longer. Florand and Dancourt describe cases with a very protracted course.

Among the *modifications* which the clinical picture of the disease sometimes affords, there is one form especially in which the spastic features drop quite into the background. When they are lacking entirely, we have no longer any right to the diagnosis amyotrophic lateral sclerosis in a clinical sense. We are dealing then with a chronic atrophic spinal paralysis with bulbar paralysis; but it is remarkable that the patho-

[1] "Obersteiner," vii., 1900.

logical condition still corresponds to that of amyotrophic lateral sclerosis. To explain this remarkable fact—this absence of spastic phenomena, despite the disease of the pyramidal tracts—the supposition has been put forward that the process in the grey matter long preceded that in the white.

A rarer modification is that in which the spastic symptoms stand out prominently, while the atrophy is scarcely noticeable. Corresponding to this clinical condition the lesion in the anterior horn is then quite slight. Bulbar symptoms of a spastic character are also present in this form (compare with this the section on spastic spinal paralysis, with the corresponding observations of Strümpell, Mills-Spiller, and Ballet-Rose). An acute development of the disease has been once described (Oppenheim).

Schlesinger ("Obersteiner," vii.) brings forward a like observation and discusses atypical forms or symptoms in which, in one of his patients, he found loss of pupillary reflexes and bladder disturbances. Spiller mentions rigidity of pupil and affections of the optic nerve in one case. Slight disturbances of sensibility have likewise been occasionally found, *e.g.* by Egger and Lejonne-Lhermitte (*R. n.*, 1906), in whose case also there were particularly severe pains: Florand and also Kojewnikoff (*R. n.*, 1906) refer to painful muscular cramps. We are dealing here, however, with unusual symptoms, quite outside the usual clinical picture of the disease, or with quite doubtful cases (*e.g.* Redlich). But study of the pathological conditions teach us that, besides the pure typical forms of the disease, atypical ones occur with involvement of tracts and regions which, as a rule, are spared (see below).

Pathological Anatomy.—This definite clinical picture is associated with a sharply localised pathological lesion. In the spinal cord the *motor paths* and *trophic centres* of the muscles are the seat of a degenerative process, *i.e.* we find an *atrophy of the pyramidal tracts and anterior horns.* In the white matter the crossed pyramidal tract is the most severely involved. The direct pyramidal is also usually affected. Moreover we frequently find a slight diffuse degeneration in the rest of the antero-lateral ground bundle (see Fig. 117). The sensory tracts are always spared. Only once have I seen the process encroach upon a part of the posterior horn, and similar lesions have been recorded by others in isolated cases (see below). Only the anterior horns are affected in the grey matter, and they most markedly in the cervical enlargement. The nerve-cells have for the most part or altogether disappeared; the reticulum of nerve fibres is also more or less thinned out. I have usually found that the paths regarded as reflex-collaterals are spared (Fig. 118), (these are the fibres which I have found to be degenerated in tabes). The atrophy affects also the *anterior roots*, but they are not always involved (Oppenheim, Pardo).

We find the same changes in the medulla and pons: *atrophy of the pyramidal tracts* and of the motor nuclei of the *hypoglossal* (Figs. 119 and 120), (this is always very markedly affected), *facial*, *vagus-accessorius*, and *fifth* (motor root) nerves. An involvement of the nucleus ambiguus has been pointed out by Turner, Probst, myself, and others. The degeneration of the pyramidal tracts which appears to be a centripetally advancing one can be followed into the crura (Fig. 121), and in some case has been observed even in the cerebrum, in the internal capsule. Charcot and Marie, Koschewnikoff, Mott, Probst, Rossi-Roussy and others, found in addition an atrophy of the pyramidal cells in the paracentral lobes. The supposition of Marinesco, that we are dealing here with a retrograde

degeneration, such as he can demonstrate by Nissl's method in degeneration of the pyramidal tracts, should not be overlooked. Nonne and Kaes refer to a stunting of the fibræ propriæ and projection fibres in the motor region of the cerebral cortex; they assume a defective development of these fibres.

Campbell [1] has established the presence of cell atrophy in the anterior central convolution.

We would accordingly have before us a disease of the whole cortico-muscular tract.

With Marchi's method Hoche has found degeneration in the posterior longitudinal bundle. Probst and also Spiller refer in addition to an involvement of the motor cortex of the anterior central convolution—including the adjacent part of the frontal region—also a degeneration of corresponding fibres in the pons. Pennato found Lissauer's tract involved in one case; Pilcz, Gowers' tract; Pal, sensory tracts in the cord and fillet; Mott and Tredgold, the posterior columns; Sarbó, the direct cerebellar tract and Clarke's column; Rossi-Roussy, the cerebellar

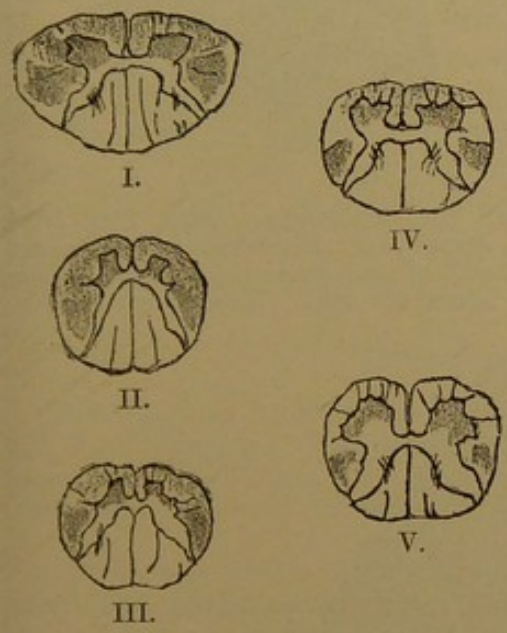

Fig. 117.—Transverse section through spinal cord in amyotrophic lateral sclerosis. The affected areas are shaded.

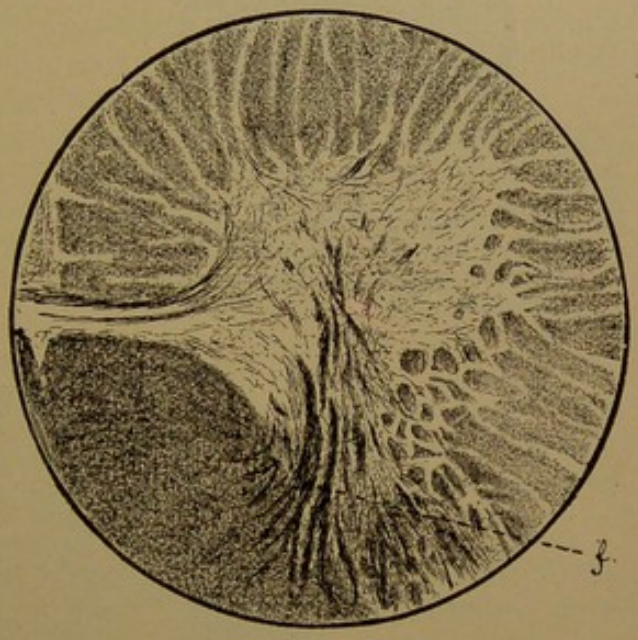

Fig. 118.—Atrophy of the anterior horn in amyotrophic lateral sclerosis, with integrity of the fibres (f) coming in from the posterior horn or the posterior roots. (Weigert's method.)

tracts of the spinal cord; Miura, the direct cerebellar tract and the superior vermis of the cerebellum, but these forms, where there are pronounced changes in the non-motor tracts, should be separated from the pure amyotrophic lateral sclerosis. This applies still more to some cases (Senator,[2] Haenel) which have been included here, and brought forward as evidence against the individuality and unity of the disease, but which agreed with it neither clinically nor pathologically.

Differential Diagnosis.—Distinction from chronic anterior poliomyelitis is, as we have already said, only possible in typical cases. Chronic cervical myelitis may lead to similar symptoms: atrophic paralysis of the arms, spastic paralysis of the legs—but here there are almost always disturbances of sensation and of the bladder functions, and in the subsequent course of the disease the atrophy does not extend to the lower extremities.

Cervical gliosis, when it spares the posterior horns, can give rise to a clinical picture corresponding to that of amyotrophic lateral sclerosis, but this is very rare, and in the later stages at least symptoms appear

[1] "Histol. Studies on the Localisation," etc. Cambridge, 1905. [2] *D. m. W.*, 1894.

indicative of an involvement of the posterior horns. This disease also tends to run a much slower course.

Multiple sclerosis may also assume the clinical features of amyotrophic lateral sclerosis, but there is very seldom such a marked degenerative atrophy ; further, there are practically always the characteristic cerebral symptoms of this disease (nystagmus, atrophy of optic nerve, etc.).

Syphilitic processes have occasionally given rise to similar appearances. Caries of the cervical vertebræ almost always betrays itself by

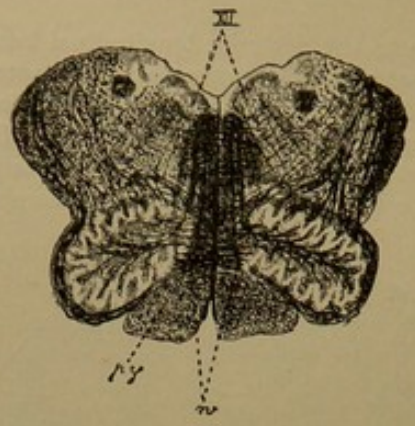

FIG. 119.—Normal medulla oblongata at the level of the hypoglossus. xii, hypoglossal nucleus ; w, hypoglossal roots ; py, pyramids. (Weigert's method.)

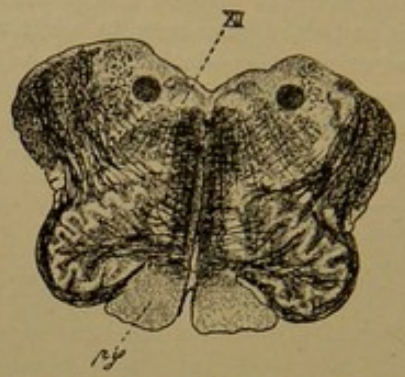

FIG. 120. — (Compare with Fig. 119.) Atrophy of xii nucleus and its roots, and of the pyramids in amyotrophic lateral sclerosis. (Weigert's method.)

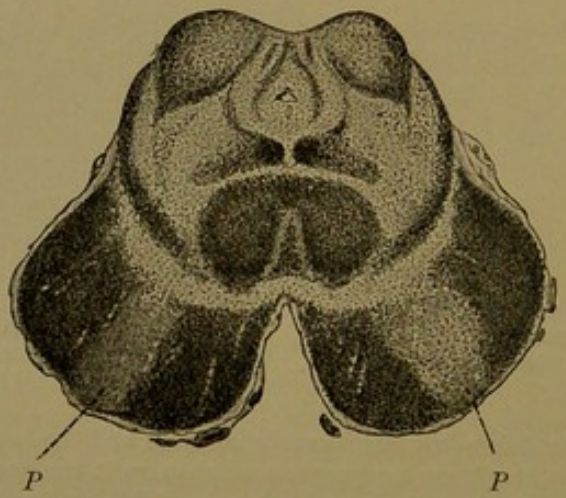

FIG. 121.—Degeneration of pyramids in the cerebral peduncle in amyotrophic lateral sclerosis. (Weigert's method.)

signs of a spinal disease ; moreover, the affection of the cord leads as a rule to sensory disturbances and bladder troubles in addition to the symptoms of atrophic spastic paralysis. The simple atrophy present in chronic joint affections, in which there is also an exaggeration of the tendon reflexes, can scarcely give rise to confusion.

I made the probable diagnosis of amyotrophic lateral sclerosis in one case which the further course showed to be completely erroneous : a young girl began to suffer from a slowly progressive degenerative atrophy of the small muscles of the hand. There were no definite spastic symptoms, but the tender reflexes were much exaggerated in both arms and legs. There were no objective sensory disturbances. I thought the diagnosis of amyotrophic lateral sclerosis must be right, but

an improvement set in and there remained only a circumscribed atrophy. Perhaps, in addition to general nervousness, which would account for the exaggeration of the reflexes, there was an occupation-atrophy, as the girl, who was delicate, was working up to the limits of her strength at typewriting. This interpretation is not satisfying, however. She had also suffered much from "cold hands," but although I have seen a simple atrophy result from spasm of the blood-vessels in the hands, I have very rarely seen a degenerative atrophy from this cause. The case therefore remains unexplained, but it teaches that one should be cautious in making a diagnosis of amyotrophic lateral sclerosis. That an atrophy of the small muscles of the hand can develop as a result of vasomotor disturbances is specially shown by the observations made in my polyclinic by Luzzatto.

In the cases in which the process commences on the medulla oblongata, the disease cannot be distinguished from progressive bulbar paralysis (*q.v.*).

The *prognosis* as to life is absolutely bad.

Treatment is limited to the reduction of the spastic rigidity of the muscles, for which prescriptions have already been given, and to galvanic treatment for the medulla and cord. It is advisable also, where there is much difficulty in swallowing, to place one electrode of the constant current on the back of the neck and to move the other about on the front of the neck, and in this manner to produce swallowing movements.

Massage and passive movements in a warm bath may act soothingly. Plenty of fresh air, good nourishment—which in the later stages must be given through a stomach-tube—are, of course, indicated. A trial of strychnine injections may be made, after the practice of Gowers and Sanger-Brown.

Progressive Muscular Atrophy

With regard to the literature we can only refer here to the comprehensive works of Erb, *Z. f. N.*, i.; Schultze, "Über den mit Hypertrophie verbundenen Muskelschwund," etc., Wiesbaden, 1886; J. Charcot, *Thèse de Paris*, 1895; Raymond, "Leçons," etc., 1903; Marinesco, "Maladies des Muscles," *Traité de méd.*, x.

Although only certain forms of progressive muscular atrophy can, from the pathological standpoint, be regarded as spinal diseases, it is desirable to consider the various forms together.

The conditions grouped under this heading are very numerous; a whole series of types have been described, but they are only varieties of the same disease. Taking as a basis of classification the characteristic signs from a clinical and pathological point of view, we are justified in establishing two typical forms: that of *atrophia musculorum* or *amyotrophia spinalis progressiva* (*Aran-Duchenne* type), and that of *primary progressive myopathy*. But even here, as we shall see, the separation is by no means a sharp one; transition forms establish a connection.

The Spinal Form of Progressive Muscular Atrophy
Amyotrophia Spinalis Progressiva (Aran-Duchenne)

The disease sets in as a rule in middle life. Its commencement rarely occurs before the twentieth year, although there is a familial form which begins in infancy. If one disregards this and isolated observations (Gowers, Strümpell, Hervouet, Etienne [1]), hereditary tendencies seem to play very little part in the etiology. Yet there has been described,

[1] *Nouv. Icon.*, 1899.

especially by Bernhardt, a hereditary form appearing in adults, which probably belongs to this group. Nothing certain is known about the causation ; trauma and chill have of course been blamed. Some observations (Ziehen, Hoffmann) point to an etiological significance of the former. Over-use of the muscles in many cases precedes the development of the disease, but there is a form of occupation-atrophy which, from its course, must be throughout separated from progressive muscular atrophy. Men are much more frequently affected than women.

Whether syphilis is capable of bringing about the typical form of this disease (Lannois) is doubtful. Though Dana (*Journ. of Nerv.*, 1906), on the ground of his statistics, maintained the importance of this cause, it must be borne in mind that he included far too much under the conception of progressive muscular atrophy.

On the whole this form of progressive muscular atrophy is a rare disease. Duchenne has over-estimated its frequency, as in his time gliosis and amyotrophic lateral sclerosis had not been recognised, and were included along with it, but Marie goes decidedly too far when he calls the existence of this affection altogether in question.

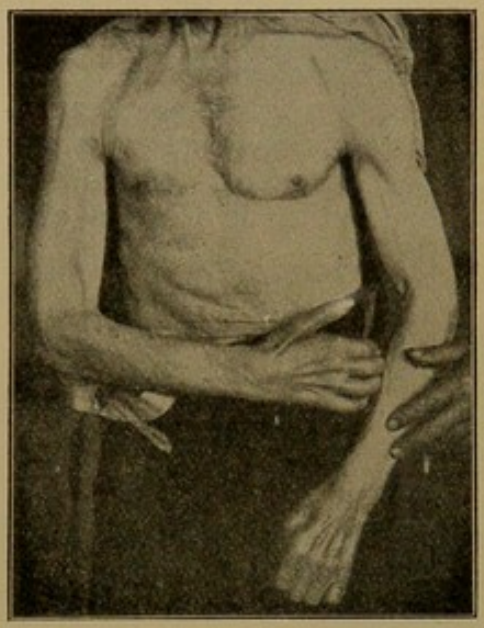

Fig. 122.—Localisation of atrophy in the spinal form of progressive muscular atrophy. (Oppenheim.)

Symptomatology.—The disease develops insidiously. Months or even a year may pass, before it gives rise to any marked symptom.

In the majority of cases the small muscles of the hand are first affected. The opponens pollicis and the first interosseus usually first succumb to the atrophy ; the ball of the thumb becomes gradually more and more flattened, the interosseous space sinks in, the other small muscles of the hand gradually follow, and some muscular function is encroached upon *pari passu* with the wasting ; a dropping out of certain movements, and an unusual position of the hand become evident. While the patient has his attention drawn to his condition first usually through the weakness, it is the altered appearance of the hand which reveals it often quite early to the physician. The *deepening of the interosseous spaces*, the *wasting* of the thenar and hypothenar eminences, the *claw-hand position* of the fingers (comp. Figs. 122 and 123), the attitude of the thumb—in the same straight line as the other fingers (monkey hand), or abducted and over-extended—are, as a rule, the first objective signs. The palm of the hand also shows this flattening sooner or later, while the atrophy of the lumbricals makes hollows appear between the tendons of the long flexors of the fingers. The subcutaneous layer of fat usually also shares in the atrophy. The weakness is only a result of the muscular atrophy ; paralysis of a muscle ensues first with its complete disappearance. The atrophy is regularly accompanied by *fibrillary twitching*, which may also be present in muscles which are not yet visibly wasted.

The muscles are never hypertrophied.

Electrical testing yields on the one hand a diminution of excitability corresponding to the disappearance of muscle substance, but in addition there is generally present a reaction of degeneration in individual muscles and muscle bundles. This reaction is consequently as a rule an incomplete one.

These appearances will be noted in both hands, but the process is usually more advanced in one than in the other, and it may happen that it is limited to this one for a considerable time.

The patient has no pain at all, or it is trifling. Paræsthesiæ are likewise absent or are quite in the background, and they are perhaps only a consequence of the abnormal attitude and unusual position of the limb. Objective signs of sensory disturbances are absolutely lacking.

I find the presence of arthropathies once referred to (Etienne [1]).

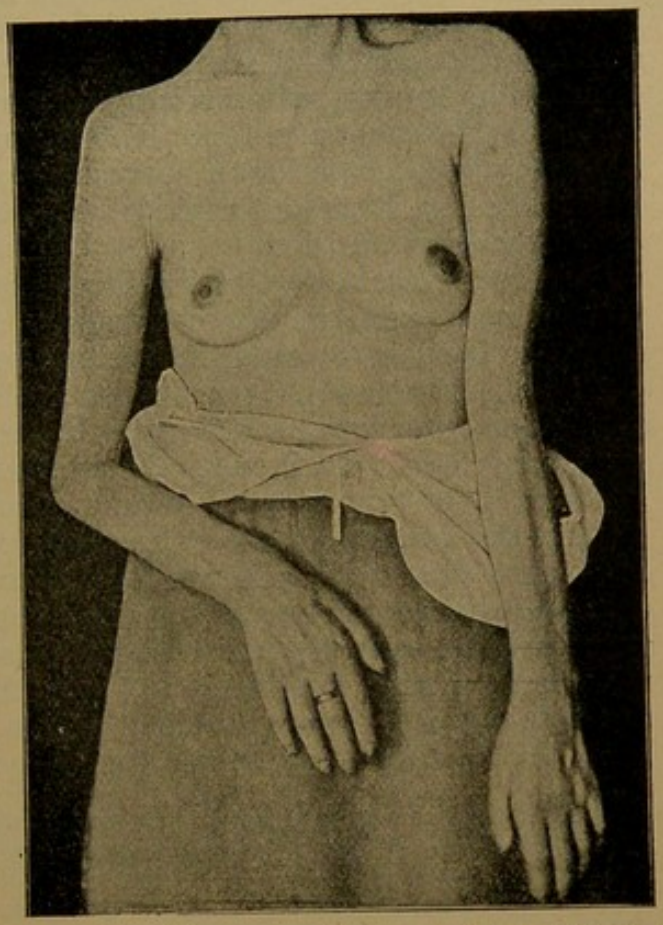

Fig. 123.—Almost complete cervical paraplegia due to progressive muscular atrophy. (Oppenheim.)

As the atrophy has slowly developed—within one or more years—so it advances slowly forwards, not indeed gliding from one muscle to its neighbour, but *by jumps*, so that it may pass over direct from the hand muscles to the shoulder girdle, especially to the deltoid. This march of the atrophy is not, however, always rigidly followed, as it frequently sets in in various places at the same time, and at the time of examination the muscles of the hand, some of the extensors and flexors of the forearm, as well as individual shoulder muscles, may already be affected. The disease has also been observed to appear first in the extensor muscles of the forearm and their atrophy to set in unusually quickly. In the later stages, the extremity first affected usually remains the most markedly so. After several years atrophy and paralysis will have developed over a greater part of the arm, shoulder, and even back muscles. In place of the muscle reliefs we find grooves and trough-shaped hollows; the contours of the head of the humerus and of the acromion can be made out absolutely sharply—like a part of the skeleton only covered by skin; the arms hang limply by the chest, but some muscles always retain a certain amount of motor power. The more slowly the disease advances, the more adept does the patient become at finding ways of making use of the muscles which are still capable of contracting and of serving as substitutes for the paralysed ones. For example, if the flexors of the

[1] *Revue de Méd.*, 1899; *R. n.*, 1901.

elbow are no longer operative, the patient may contrive to bend the joint by an exaggerated flexion of the hand and fingers, by pronation or by hyperextension of the hand. He slings the limbs until they reach a position in which, with a firm support, or, with the aid of a movable part of the body, they can be turned to a certain amount of use. The fact is noteworthy that the tendon reflexes in the arms may be diminished or lost.

In a fair number of cases the shoulder and back muscles are affected first: the deltoid, infraspinatus, trapezius, serratus anticus major, and others. The deformity is then markedly apparent only after undressing, and thus remains longer undiscovered. On viewing the exposed shoulder region, the wasting of the supra- and infra-spinatus fossæ of the back of the neck, and upper part of the back of the shoulder, and also the abnormal position of the scapulæ strike the eye. The first disturbances of function are noticed on raising the arm, and vary according as this or that muscle is most affected. The extensor muscles of the head are sometimes involved, even at an early stage; the head can only be supported with difficulty, it sinks forwards, and can scarcely be moved backwards out of the bent position.

If the disease sets in at the shoulder girdle the upper arm muscles are usually next affected in the progress of the case, and so the process advances further towards the periphery.

In this form of progressive muscular atrophy—disregarding certain rare types—the lower extremities do not share at all in the atrophy, or only very late. It is only in isolated cases (Hammond, Raymond-Philippe [1]) that the atrophy begins in them.

The disease may indeed show long remissions, but a complete arrest or a cure does not occur to my knowledge. Where they have been observed, there has been some confusion with subacute and chronic anterior poliomyelitis, with occupation atrophy or other affections.

Life is finally endangered when the *respiratory muscles*, especially the diaphragm, become involved in the atrophy, or when the symptoms of *bulbar paralysis* make their appearance.

Sluggish pupils have been reported in a few uncertain cases. Remak (*N. C.*, 1906), who noted a difference in size and loss of reflexes in the pupils in an otherwise typical case, is inclined to diagnose syringomyelia.

A general involvement of the non-striped muscle of the bowel in the atrophy has only once (Léri [2]) been recorded. The combination of the disease with infantile spinal paralysis has been already referred to. I have once seen in the Bernhardt type of this affection in addition the signs of a severe neurasthenia, symptoms of myoclonus, or of one of its allied conditions. A combination of spinal muscular atrophy with scleroderma has been recorded once or twice.

Differential Diagnosis.—A number of conditions which may easily be confused with progressive muscular atrophy must be taken into consideration. As far as chronic anterior poliomyelitis is concerned, it must be admitted that there is no sharp distinction, that the two conditions are closely allied. Nevertheless chronic poliomyelitis develops faster at first, affects from the onset a number of muscles, a whole segment of a limb, and at once brings about paralysis and indeed extensive paralysis,

[1] *Arch. de Neurol.*, 1902; *R. n.*, 1902. [2] *R. n.*, 1902.

while the atrophy follows this. It starts frequently in the shoulder girdle and in the lower extremities. Before genuine atrophy occurs, marked changes in the electrical excitability may be detectable. These are, at least in part, the main points of distinction, which make a separation of the two forms of disease possible, despite their pathological similarity, although there are cases which bridge over the transition between them. Amyotrophic lateral sclerosis is distinguished from progressive muscular atrophy only by the presence of spastic phenomena in the former. In the rare cases in which these are lacking throughout, the preponderance of paralysis, which may affect muscles not atrophied at all or not markedly so, still gives a certain clue for differentiation, although it is by no means a definite one. We can thus understand why many authors would like to class together chronic anterior poliomyelitis, progressive muscular atrophy, and amyotrophic lateral sclerosis as one disease. Since, however, in typical cases one can carry out a symptomatological separation, it seems good to adhere to it.

Other diseases of the spinal cord, gliosis and hypertrophic cervical pachymeningitis, as well as caries of the lower cervical vertebræ, may give occasion for confusion. Gliosis is sufficiently characterised by the sensory disturbances and the trophic disturbances of the skin, etc.; the atrophy also is on the whole less symmetrically distributed, and may be for a long time, or even permanently limited to one extremity. Hypertrophic cervical pachymeningitis sets in with signs of sensory irritation over a certain area, and goes along with objective sensory disturbances in the area of the ulnar and median nerves. In its later course a mistake is practically impossible.

Caries of the lower cervical vertebræ may lead to muscular atrophy in the hands which may be mistaken for the similar progressive form of muscular atrophy. Sensory symptoms are usually present; there is also sensitiveness to pressure on the vertebræ; if an acute-angled kyphosis is present, the diagnosis is thereby made certain. Skiagraphy may also be employed in the diagnosis. As a rule there soon appear symptoms of so-called compression myelitis, particularly spastic paralysis of the legs, bladder troubles, etc. According to several observations (Lannois-Levy,[1] Dana) syphilis may give rise to clinical features resembling those of Aran-Duchenne's disease. It is much more important to refer to certain less harmful forms of circumscribed muscular atrophy, which according to my experience are common and are wrongly confused with progressive spinal muscular atrophy. These are the *occupation-atrophies* or *professional pareses*, *i.e.* atrophy dependent on over use of certain muscles and simultaneous compression of the same, or of their nerves (compare the corresponding chapter). This atrophy is as a rule unilateral, and is usually accompanied by slight *paræsthesiæ* and *blunting of sensibility* in the corresponding nerve areas. Obviously, neuritic changes mostly underlie this, or they are present incidentally; but there exists also a pure motor form which is perhaps of myopathic origin. The distinction of this occupation-atrophy from progressive muscular atrophy must be made with special care because they have an absolutely different prognosis; *cessante causa*, according to my experience the atrophy practically always recovers, if it has not already lasted too long. If one considers on the other hand that progressive muscular atrophy may commence in over-used

[1] *Echo méd. de Lyon*, 1900.

muscles—Dana would even make a special type of this variety—one can make this diagnosis in doubtful cases only, when the disease shows itself to be a progressive one after the cause has ceased to act.

Arthritic muscular atrophy can scarcely come into consideration in the differential diagnosis. This accompanies the acute and chronic joint inflammations, affects especially the quadriceps in diseases of the knee-joint, the glutei in hip affections, the triceps or deltoid in diseases of the elbow or shoulder-joint. It only exceptionally extends to a whole extremity or segment of a limb. The wasting may develop quickly, and even within a week, but it is always a simple[1] atrophy, characterised by diminution, never by qualitative changes in the electrical excitability. On recovery of the joint affection or soon afterwards the muscular atrophy tends also to disappear. It has been supposed—and it has been made probable experimentally (Charcot, Krause)—that the joint affection brings about the atrophy by a reflex action on the anterior horns (Charcot, Vulpian, Halipré); but this has been contradicted, and the wasting attributed to inactivity (Strasser, Sulzer, Bum). Recent researches dealing with the reaction of the anterior horn cells in experimentally produced joint-lesions have yielded mutually contradictory results (Klippel, Mallet, Hartmann). Only exceptionally does an inflammatory joint affection extend directly to the adjacent nerves.

See also on this question Sudeck, ref. *N. C.*, 1907. *Vasomotor* disturbances in the hands may lead to an atrophy of the small muscles (Oppenheim; Luzzatto, *Z. f. N.*, xxiii.), usually a simple one, but exceptionally qualitative changes in the electrical excitability may also be present. These atrophies may be recovered from.

The diagnosis of progressive muscular atrophy must never be based on *fibrillary tremors* alone. These are present under the most varied circumstances: in health after excesses, in emaciated persons under the influence of cold, and particularly in neurasthenic and hypochondriac individuals. In these particularly the tremor occurs not infrequently in the small muscles of the hand, the orbicularis palpebrarum and the muscles of the lower extremities. The higher grades of this tremor, it is true, occur chiefly in spinal muscular atrophy.

Finally, we must point out, that not a few cases have been described which, from their clinical features, appear to be related as well to the spinal as to the myopathic and the neurotic forms of progressive muscular atrophy, and can be completely included neither in the one nor the other group (observations of Strümpell, Pick, Abundo, Cassirer, Haushalter, Cohn, K. Mendel, and others).

Pathological Anatomy.—The right to classify the disease as a spinal one depends on this, that a *lesion of the grey anterior columns* is constantly found, a lesion which consists essentially of an atrophy of the nerve elements (nerve-cells and nerve-fibres). This forms the sole substratum, or there may be at the same time slight changes in the antero-lateral columns, either merely a scattered fibre atrophy, especially in the lateral limiting layer, which is of purely secondary significance, or a degeneration

[1] From recent researches of Loewenthal, Stier, Hauck, F. Pick, Jamin ("Exper. Unters. zur Lehre v. d. Atrophie gel. Muskeln," Jena, 1904), it appears as if the distinction between "simple" and "degenerative" muscular atrophy cannot be drawn with the earlier rigour, and as if the atrophy of muscle in spinal and neuritic processes were essentially a simple one. Further experience in required, however, before we yield the views based on numerous careful older investigations to the new teaching.

of the pyramidal tracts. Although the combined degeneration of the anterior horns and pyramidal tracts forms the anatomical basis of amyotrophic lateral sclerosis, yet even in pure cases of progressive muscular atrophy (without spastic phenomena) this affection of the white matter is sometimes found. The supposition is, that it develops subsequently to the disease of the anterior horns, and so has no clinical expression.

The *anterior roots*, *motor nerves*, and even the *muscles* share in the atrophy. The roots may be distinguished, even by the naked eye, from the white posterior roots, by their grey colour and their diminution in size.

The muscles are markedly wasted, show, instead of red, a pinkish, reddish-yellow, or pure yellow colour, and are traversed by fatty streaks. Histologically we find : diminution in size of the fibres, decomposition of the muscle-substance into a mass of granular and fatty material, after absorption of which only the sarcolemma envelope remains, studded with nuclei.

Where the appearances of bulbar paralysis are exhibited, corresponding changes in the medulla oblongata will be found.

We consider we may disregard here the impure and complicated cases in which the pathological process in the cord has encroached on other columns, *e.g.* on the posterior columns, as in a case of Placzek's.

Treatment.—All the remedies recommended for this disease are, alas, powerless. Gowers, however, strongly recommends strychnine, with which he has in many cases brought about an arrest or even improvement. It should be employed subcutaneously in doses of $\frac{1}{120}$ to $\frac{1}{40}$ grain, once daily, after some time only three or four times a week. Sanger-Brown likewise speaks in favour of this treatment. Arsenic may also be ordered by way of trial. In one case thyroidin preparations were employed with some result (?).

Avoidance of over fatigue of the muscles is the chief requisite. The muscles still unaffected should of course be kept in action by regular exercise, but any forced muscular activity is to be avoided.

Electricity may be employed in such a way that the spinal cord is put under the influence of the galvanic current only, while the muscles are stimulated directly with the faradic or galvanic. Strong stimulation should be avoided, as it may do harm, while the efficacy of treatment is questionable.

Nothing is known unfortunately as to how far we can influence the disease by qualitative changes in diet. Smoking should be forbidden. No objection can be taken to a moderate use of alcohol.

Treatment by change of climate has not proved of much service.

Appendix. Hereditary or Familial (Infantile) Form of Progressive Muscular Atrophy of Spinal Origin

Werding [1] and more especially Hoffmann [2] (and also Bruns [3]) have observed cases of this sort in which the disease attacks several members of one family, and is further distinguished by appearing in early childhood. The children fall ill in the second half of the first year of life, subacutely or chronically, with weakness and atrophy of the thigh, pelvic and back muscles, which in the course of months or years extends to the muscles

[1] *A. f. P.*, xxii., xxvi. [2] *Z. f. N.*, iii., x., and xviii. [3] *Z. f. N.*, xix.

of the upper part of the trunk and upper extremity. At first the atrophy is well concealed by the adiposity of the subcutaneous connective tissues, but there is no pseudo-hypertrophy and still less any real hypertrophy. Reaction of degeneration is present in the atrophied muscles. The tendon reflexes usually disappear. There are no sensory disturbances. Bruns especially has referred to deformities of the vertebral column (kyphoscoliosis). The process goes steadily forwards with a symmetrical distribution, and causes death in from one to six years. Werding has seen bulbar symptoms set in.

Anatomical investigation shows *degeneration of the nerve-cells in the anterior horns* of the cord as the principal change; the peripheral nerves may also take part in the degeneration. The slight degeneration which has occasionally been found in the antero-lateral columns is only a secondary process. Of recent years corresponding pathological conditions have been described by Ritter (*Jahrb. f. Kind.*, ix.), Bruce-Thomson (*Edinb. Hosp. Rep.*, 1893), Bruns (*N. C.*, 1906), and also Armand-Delille and Boudet (*Nouv. Icon.*, xix.). A clinically allied case described by Fletcher and Batten, cannot, from its anatomical basis, be included here.

The disease therefore corresponds in regard to its familial character and commencement in childhood to the myopathies described in the next section, on the other hand to Aran-Duchenne's disease from the nature of the atrophy and the pathological lesions, and may be set up as a connecting link between these two main forms.

The relationship to myatonia congenita (*q.v.*) assumed by Winner is very doubtful.

A hereditary appearance of the Aran-Duchenne form has also been observed in rare cases (Gowers, Bernhardt,[1] Hammond, and others). Finally there appears to be an infantile familial form of this disease, in which the bulbar muscles are chiefly affected (Fazio, Londe [2]).

We shall not go into other complications (*e.g.*, cases combined with mental weakness), and clinical cases not resting on a sure histological basis, which have only provisionally been classed with this group (observations of Hoffmann,[3] Thomas, Bruce, and others).

Primary Myopathy. Progressive Muscular Dystrophy

Under the heading of progressive muscular dystrophy Erb has comprehended the forms, which were hitherto separated, of *pseudo-hypertrophic*, *juvenile*, *hereditary* (Leyden, or the type of Leyden-Möbius and Zimmerlin), and *infantile* (Duchenne) primary myopathy, which, as he points out, form a clinical and pathological unity. The accuracy of this conception is now generally recognised, and it is corroborated by Raymond in particular, who has exhaustively studied the disease.

These primary myopathies are distinguished from progressive spinal muscular atrophy by the following characteristics :—

1. The onset of the disease *at an early age*.
2. Its *hereditary* or familial occurrence in most cases.
3. The commencement of the dystrophy in the *muscles of the trunk* and the adjacent parts of the limbs, indeed usually in the *pelvic girdle* and the muscles of the *lumbar vertebral column*, of the thigh, or of the shoulder, girdle, and upper arm.
4. The combination of the atrophy with *true hypertrophy* and *pseudo-hypertrophy*. Whilst the atrophy from the first affects certain muscles, the hypertrophy makes its appearance in others, so that there is in this respect a remarkable similarity in the various cases.

[1] *V. A.*, Bd. cxv. [2] *Revue de Méd.*, 1894. [3] *Z. f. N.*, vi.

5. The absence of fibrillary tremors.
6. The simple *quantitative diminution* of the electrical excitability, reaction of degeneration being very seldom, if ever, present.

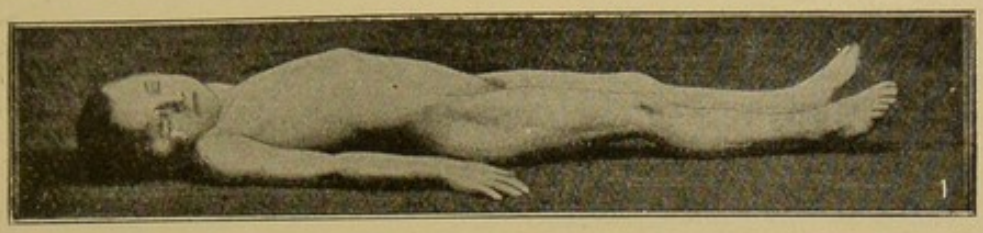

Fig. 124.

Figs. 124—132.—Mode of rising from the horizontal position in myopathy. (After Dejerine.)

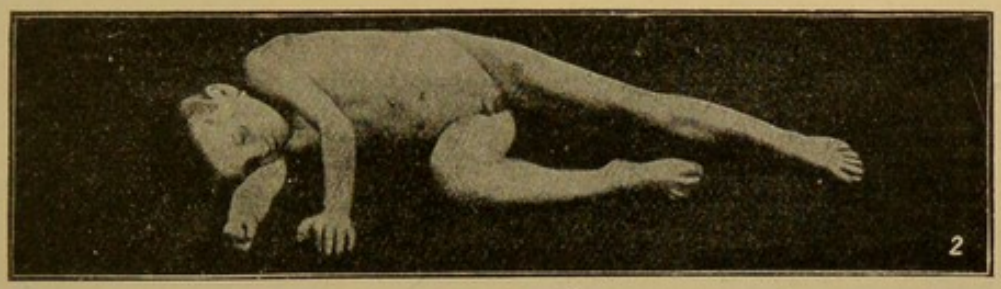

Fig. 125.

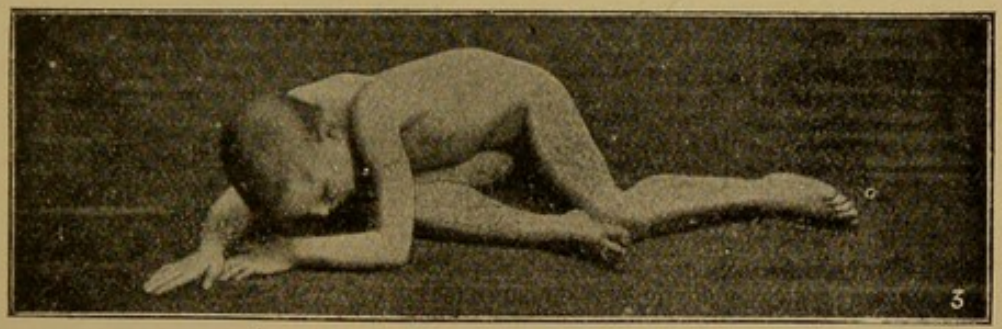

Fig. 126.

Fig. 127.

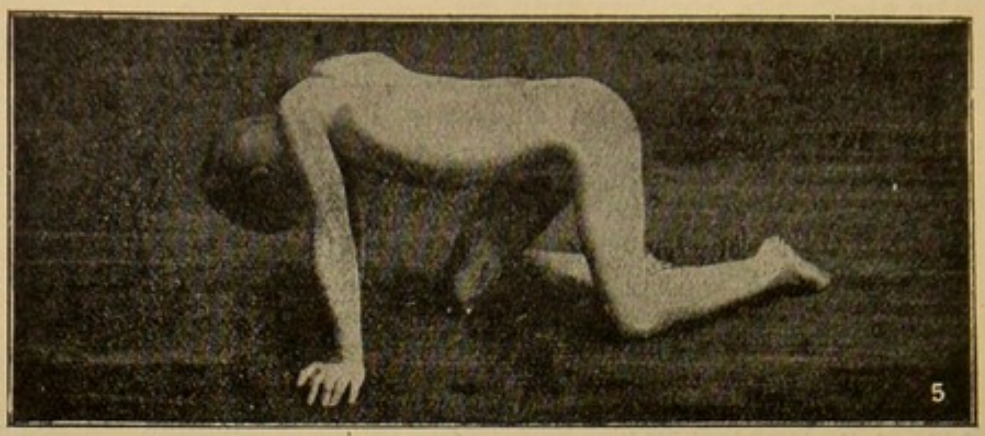

Fig. 128.

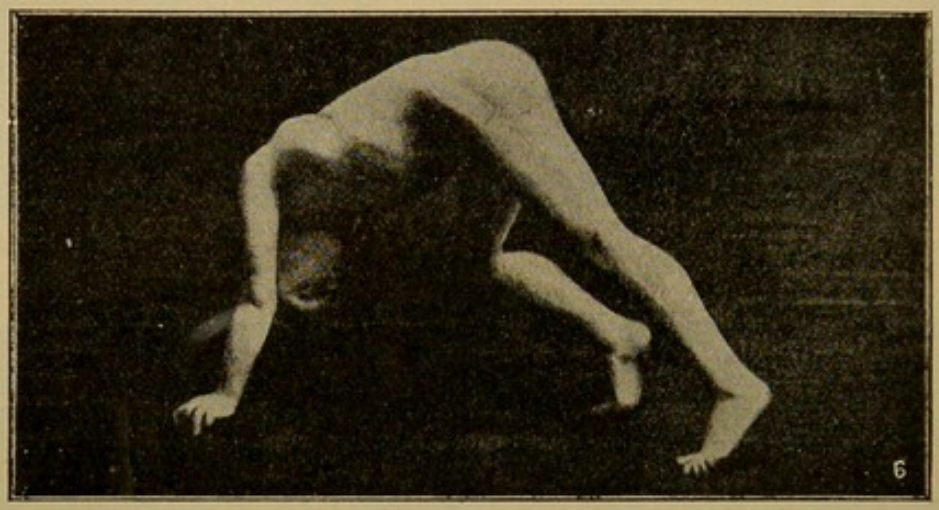

Fig. 129.

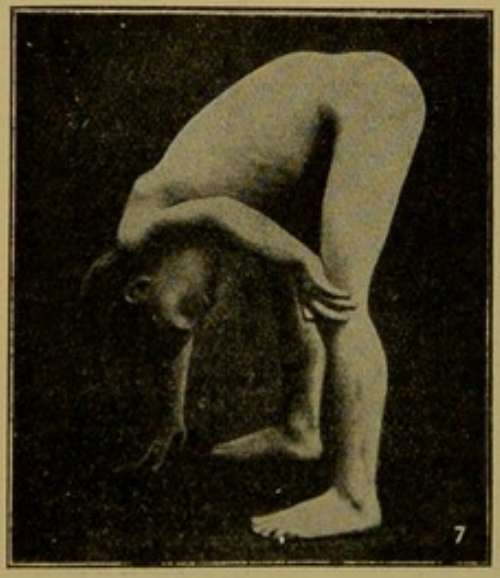

Fig. 130.

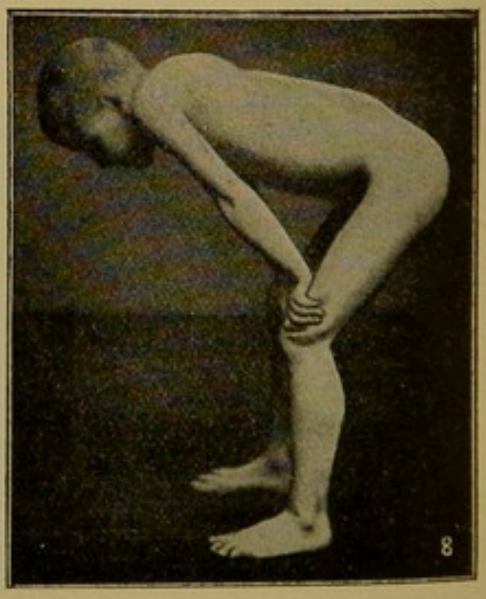

Fig. 131.

Clinical Picture.—The disease commences in the first or later years of childhood, at the age of puberty or in adolescence. It rarely appears during the third or fourth decades, and still more rarely at a later period.

As a rule several members of the same family—brothers and sisters—are affected, and the disease is often inherited through several generations. A sporadic onset is not, however, uncommon. I think I have seen almost as many isolated as family cases. The disease is specially inherited through the mother.

The symptoms develop very slowly and are often unnoticed for a long time. In the cases where the muscles of the pelvis and thigh and the extensors of the spinal column are first affected, the disorders first become apparent in walking and in raising the trunk. The gait is *waddling*; in walking the pelvis is excessively raised and lowered; the

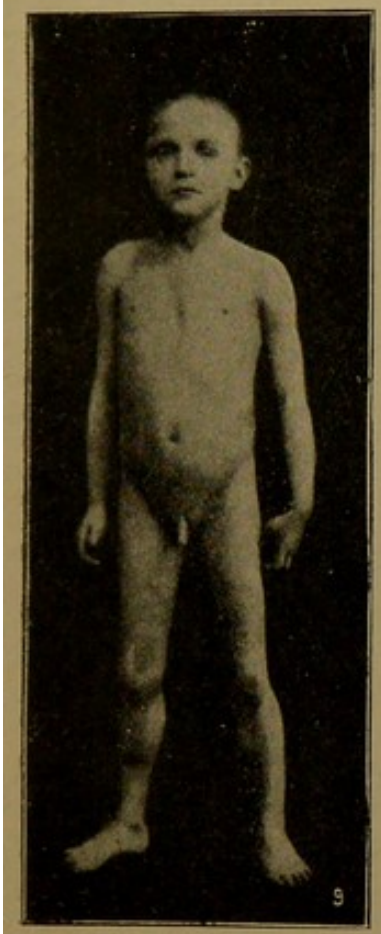

Fig. 132.

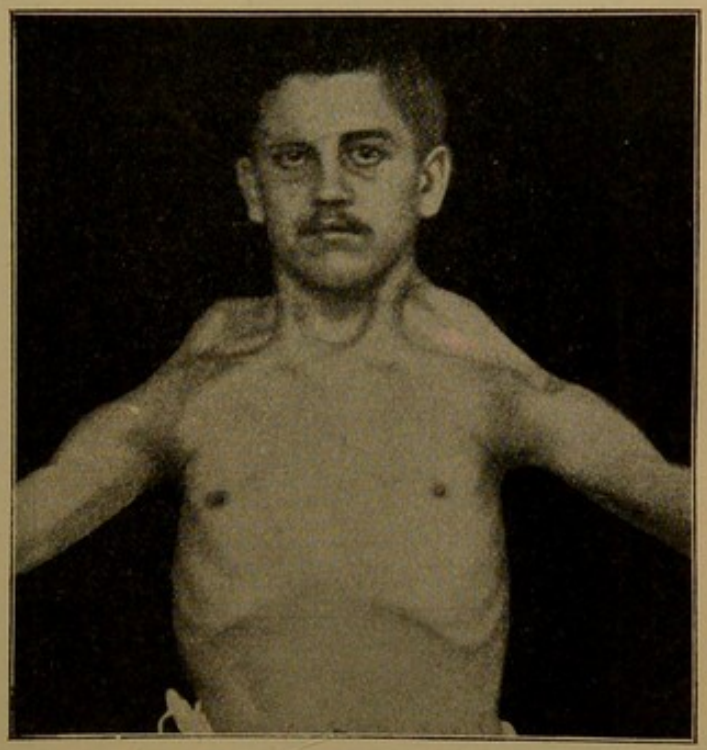

Fig. 133.—"Loose shoulders." High position of the inner upper angle of the shoulder-blade in progressive muscular dystrophy. (Oppenheim.)

patient finds it difficult to go up stairs and he falls easily. In raising himself from the sitting posture, he makes use of his arms; he leans with his hands upon his thigh or knee, and thus brings his body into an erect position. The way in which he rises from the recumbent position is particularly characteristic (Figs. 124 to 132). If he is lying on his back, he first turns round on to his abdomen, and then, resting his hands upon the ground, he raises the trunk until he finds himself in a kneeling posture; he then lifts the knees from the ground until only his hands and feet rest upon it and support him; then one hand is placed upon the knee of the same side, and the body is brought up with a jerk; or the patient works his hands up his legs, trying to find a support for them at successively higher points on the thigh. In the end he cannot raise himself without assistance from the ground, or can only do so if he can hold on to some firm

object (a table, etc.). These peculiar movements are rendered necessary by the involvement, more or less marked, of the function of the muscles which extend the thigh on the pelvis and the leg on the thigh. The difficulty is greatest when the whole weight of the trunk has to be lifted at the same time.

Another prominent feature is the lordosis of the *lumbar spinal* column (Figs. 134, 135). The abdomen is greatly protruded, and the upper part

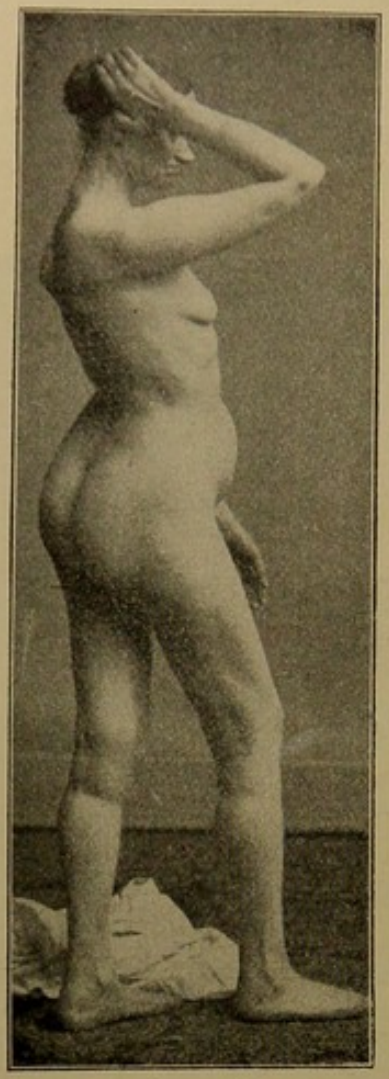

FIG. 134.—Progressive muscular dystrophy. Lordosis of the lumbar spinal column. (Oppenheim.)

FIG. 135.—Progressive muscular dystrophy. Lordosis from atrophy of the abdominal muscles, etc. (Oppenheim.)

of the trunk is thrown backwards. This symptom is practically caused by the weakness of the muscles which produce extension at the hip-joint; this gives rise to an abnormal inclination of the pelvis, and a forward curve of the lumbar vertebræ, so that the upper part of the trunk is instinctively thrown back in order to bring the centre of gravity of the body above the feet. The lordosis thus produced disappears when the patient sits down, as the pelvis then rests upon a firm support. Under other conditions it may also occur in the sitting posture, as in a case described by Souques,[1] where it was present to an excessive degree (Fig.

[1] *Nouv. Icon.*, 1894.

136). Lordosis may also be caused by paralysis of the abdominal muscles.

Paralysis of the abdominal muscles may also cause some difficulty in respiration, because the abdomen is thrust forwards in a globular form, instead of being contracted during inspiration, as I have found in several cases. The atrophy of the trunk muscles may produce a peculiar configuration of the body which Marie has described as the wasp-waist (*taille en guêpe*), see Fig. 142.

The weakness of the shoulder muscles gives rise to an abnormal attitude of the shoulder-blades and to motor symptoms which become specially evident when the arms are raised (see Figs. 133, 137, 138). As a result of the atrophy of the muscles which fix the shoulder blade — the *trapezius*, *pectoralis major*, *latissimus dorsi*, *serratus magnus* — it becomes abnormally mobile, and follows the movements of the upper extremity like a loose appendage. If we try to raise the patient by placing the hands below his axilla, it is not the body which is lifted, but the shoulders (loose shoulders) (Fig. 133).

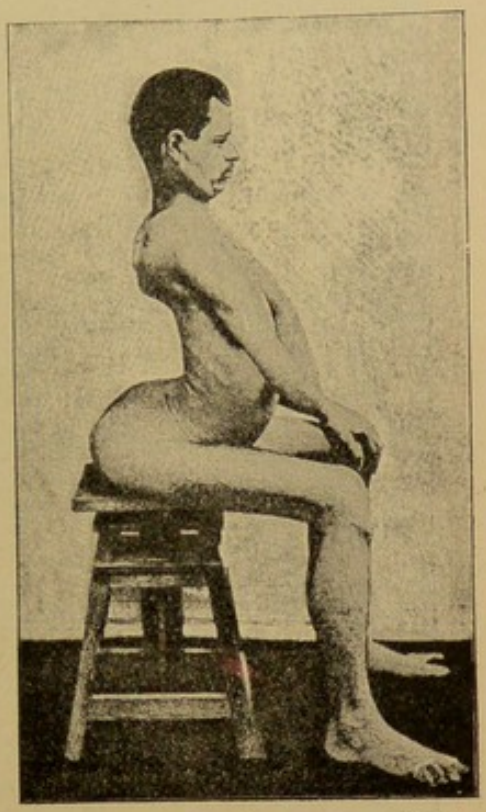

Fig. 136.—(After Souques and Brissaud.) Very advanced progressive myopathy. Marked lordosis in sitting.

During rest, the shoulders fall downwards and forwards; the acromion is lower than the internal upper angle; the shoulder-blades stand out from the spinal column and away from the thorax like wings; when the arms are raised, the anomalies of position and the functional symptoms which are characteristic of serratus paralysis become apparent (compare Figs. 137, 138). "If the horizontally raised arm be forcibly pressed downwards, the point of the shoulder-blade gives a strong outward movement, and becomes approached to the arm by the powerful action of the teretes and the infraspinatus, and the absence of fixation by the rhomboids and the trapezius" (Erb).

Close examination shows that the following muscles are as a rule involved: the *trapezius* (with the exception of its upper portion), the *serratus magnus*, the sterno-costal portion of the *pectoralis major*, the *latissimus dorsi* (these may be congenitally absent) the *rhomboids*, *infraspinatus*, *deltoid*, *biceps*, *brachialis anticus*, and *supinator longus*; in the spinal column, the *erector spinæ*; then the *pelvic muscles*, the *glutei*, *quadriceps*, the *adductors*, and finally the *muscles of the calf* and some of those of the peroneal region.

The movements of the proximal parts of the extremities are naturally most affected, whilst the distal portions, especially the hand and fingers, the muscles of which are almost without exception spared, are freely mobile.

The way in which the nutrition of the muscular tissue is affected is

very important and characteristic. As a rule it is only some of the muscles involved that show an appreciable atrophy; the others present a more or less considerable increase in size, due to a *proliferation of the fatty and connective tissue*, and partly also to a *true hypertrophy* of the muscle fibres. The configuration of the muscles is essentially altered by this process. Some of them are flattened, others markedly increased in size, and as the hypertrophy may be limited to certain portions, the

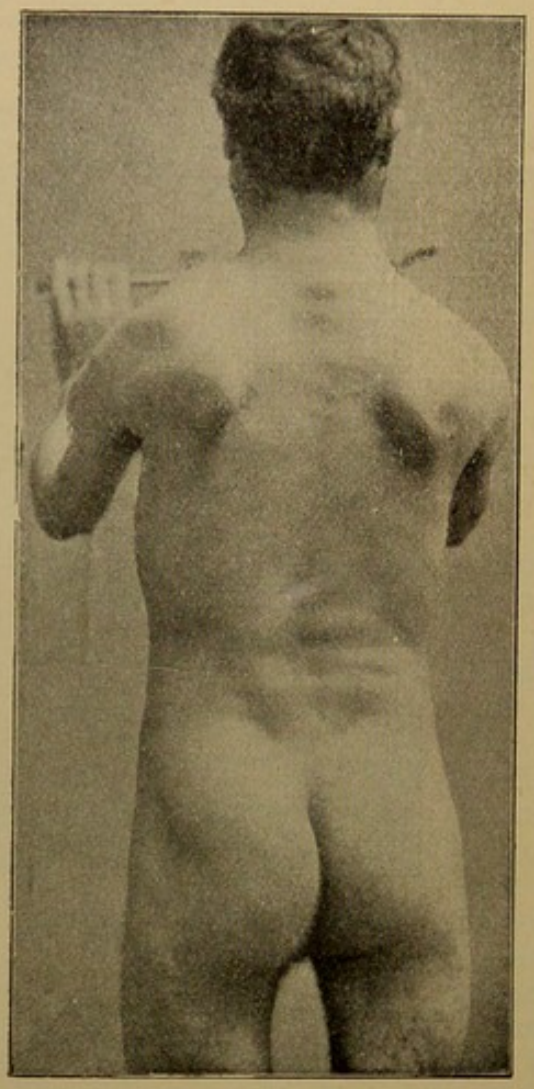

FIG. 137.—Progressive muscular dystrophy. Juvenile form. Position of the shoulder-blades from atrophy of the trapezius and serratus magnus. (Oppenheim.)

FIG. 138.—Side view of Fig. 137.

result is a kind of tumour growth in the muscle (see for instance the characteristic condition of the right deltoid in Fig. 139). The atrophy may be confined to the longitudinal segment of a muscle, to that adjacent to the tendon, for example (Roth, Marinesco). I have seen this occasionally in the quadriceps. The atrophy specially attacks the pectoralis major, the trapezius, serratus magnus, latissimus dorsi, biceps, brachialis anticus, quadriceps femoris, the adductors, etc., whilst the real or spurious hypertrophy affects preferably the infraspinatus, deltoid, triceps, sartorius, glutei, and especially the calf muscles. In addition to the peculiarity

of the figure and attitude, this co-existence of atrophy and hypertrophy is the most prominent symptom, and from these characteristics the diagnosis can usually be made at a glance.

In not a few cases the *muscles of the face* are involved, especially the *orbicularis oris and palpebrarum*. The mouth is slightly open, there is usually pseudo-hypertrophy of the lips, or at least of some part of them, the

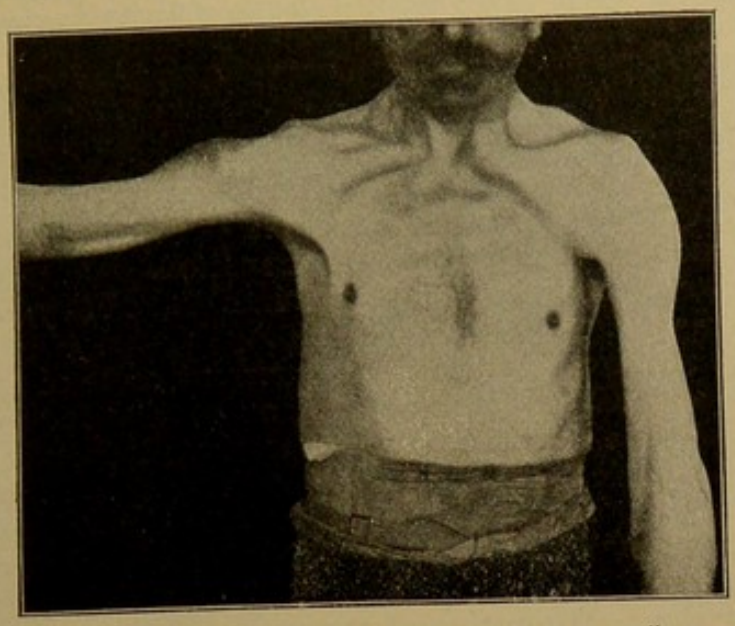

Fig. 139.—Peculiar conformation of the deltoid muscle in juvenile muscular atrophy. (Oppenheim.)

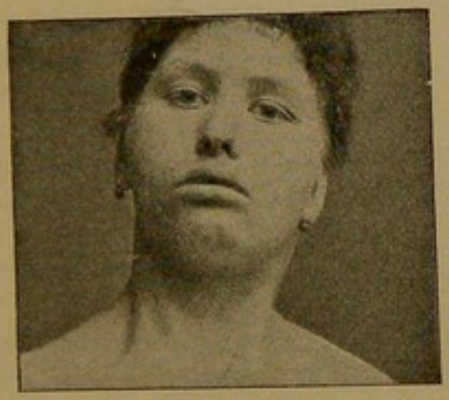

Fig. 140. — Myopathic facies. Patient cannot pout the lips, which are much swollen (pseudo-hypertrophy). (Oppenheim.)

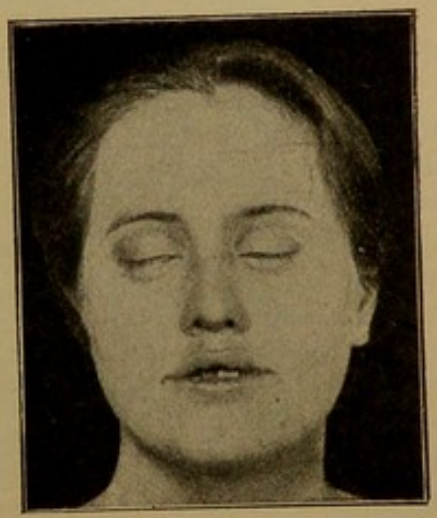

Fig. 141. — Myopathic facies. Attempt to close the eyes and pout the lips. (Oppenheim.)

middle portions or the lower lip being abnormally swollen, and the patient being unable to pout the mouth or to whistle (Fig. 140, 141). The weakness of the orbicularis palpebrarum is shown by the incomplete closing of the eye-lids; every degree of this may occur, from a feeble closing of the lids to complete lagophthalmus. In advanced cases the face may be as rigid as a mask (myopathic facies). Ballet [1] speaks of a "facies de sphinx."

[1] *R. n.*, 1902.

There is a rare type of this disease, described by J. Hoffmann (*Z. f. N.*, x.), which the author has named the "bulbar paralytic" form, on account of the marked involvement of the muscles of the face, tongue, palate, and jaw. This form does, it is true, clinically resemble that described by Fazio (*Rif. med.*, 1882), and Londe (*Rev. de Méd.*, 1894), as a familial, infantile bulbar paralysis, but differs from it to a certain extent, especially in the response of the muscles to the electric current and in the existence of a real dystrophy of the muscles of the body. I have already shown in one case (*Charité-Annalen*, xiii.), that progressive muscular dystrophy may involve the muscles of the tongue, palate, and larynx. Marie mentions affection of the jaw muscles, which I, Wendenberg, and Kollarits have also seen in a condition of pseudo-hypertrophy. Involvement of the ocular muscles has been observed in exceptional cases (Gowers, Lombroso, Oppenheim, Marie, Bäg, Jendrássik). It is stated by some writers (Ross, Hammond, Coste, Stembo, Marinesco), from their clinical and anatomical observations, that the heart muscle may be involved; but this is doubtful.

The affection in rare cases commences in the muscles of the neck, the movements of the head being then early impaired.

This muscular affection involves both sides of the body, but not always in a symmetrical way.

The muscles still react to the electric current, some of them, however, with a lessened excitability corresponding to the degree of atrophy. There is hardly ever a reaction of degeneration; it is only found at circumscribed spots in a few, usually atypical cases (Erb, Eisenlohr, Hoppe, Abadie, K. Mendel). Some isolated cases are regarded by French writers (Brissaud, Allard [1]) as dystrophy, in which the electrical excitability is practically unaltered, but I am very doubtful as to these cases.

There is usually a diminution of the mechanical excitability and also of the tendon reflexes, which are mostly entirely absent in advanced dystrophy. They may, however, be conserved and even exaggerated.

The sensibility is absolutely normal, and so is the function of the sphincters.

We sometimes find congenital anomalies in the formation of the jaw and skull and of other parts of the skeleton. The deformities of the spinal column are indeed partly of this origin. Atrophy of the bones has been observed by Friedreich and Schultze,[2] by Lloyd, Clarke, Spiller, Noica, and Schlippe.[3] Marie and Crouzon [4] were able to trace a spontaneous fracture to this cause. It has been specially stated and demonstrated by Jendrássik, that the congenital nature of the disease is the cause of its combination with the other anomalies of development in the soft parts, the skeleton and various organs.

The development and course of the disease is extremely protracted. From insignificant beginnings it very gradually increases; years may pass before there is an appreciable aggravation of the symptoms and an extension of the dystrophy to other segments of the limb. The illness may extend over a period of thirty to forty years. Indeed, I saw in the Berlin Hospital for Incurables, a woman of fifty-eight, who had had the disease from her earliest childhood, some of the symptoms being present at her birth, but the patient had, notwithstanding, been able to move about a year before, although with some difficulty. It is conceivable that when the development is so insidious, the power of the intact muscles to assume the function of the affected ones is cultivated to the highest degree, so that one is often astonished at the activity of which the patient is capable, in spite of his muscular weakness.

[1] *Soc. de Neurol. de Paris*, 1901, and *Nouv. Icon.*, xv.
[2] *Z. f. N.*, xiv. [3] *Z. f. N.*, xxx. [4] *R. n.*, 1903.

Paralytic contractures occasionally appear in certain muscles, such as the biceps, the flexors of the knee, and most often in the calf muscles. The pes equinus position may be so pronounced that the patient can only walk on his toes and with his heels unduly raised.

These contractures develop late as a rule, but they may appear at an early stage. There are cases in which they extend to the areas of many muscles, and thus, by the fixation of the parts of the skeleton, which are otherwise excessively mobile in this disease, give rise to an essential

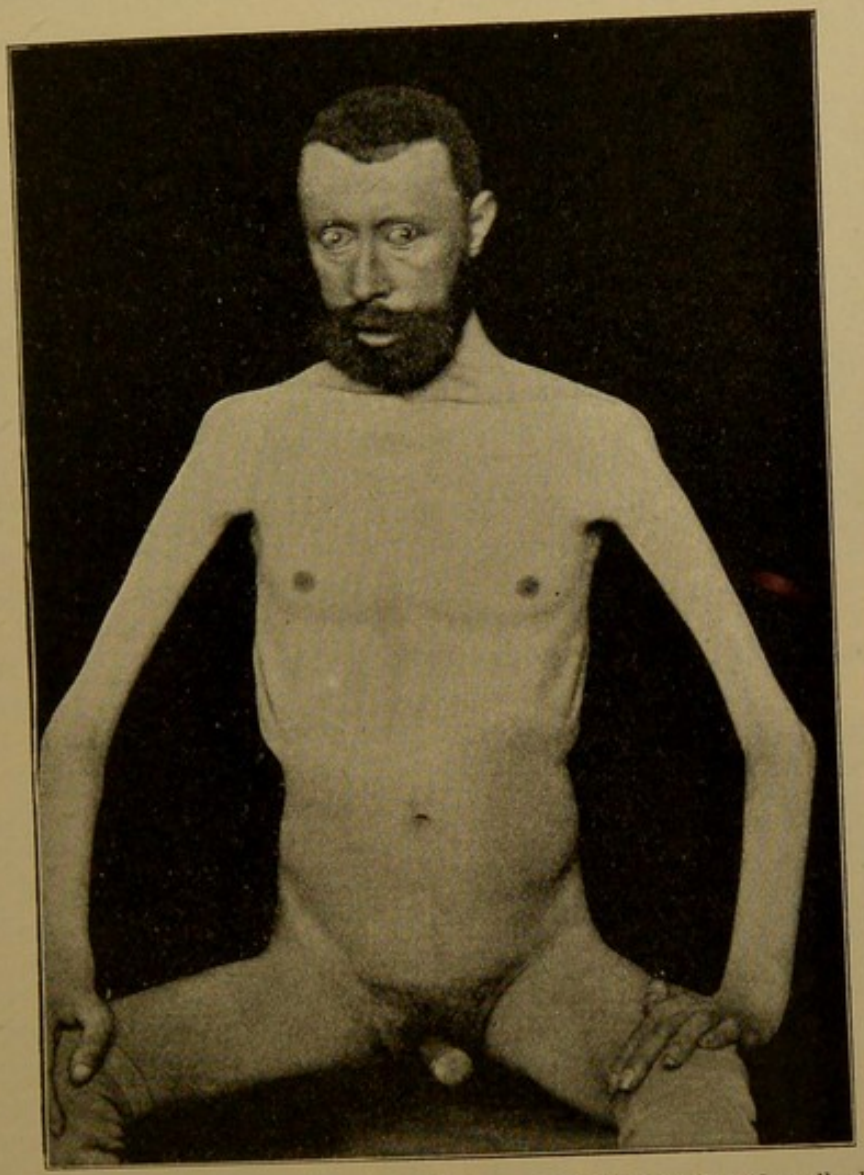

FIG. 142.—Deformity of the thorax in progressive muscular dystrophy, described by Marie as "taille de guêpe" = wasp-waist. (After Marie and Marinesco.)

modification of the clinical picture (Friedreich, Hahn, Cestan-Lejonne,[1] Dreyer,[2] Schlippe). A familial occurrence of this form has also been observed. Myosclerotic processes are apparently the essential cause of these contractures.

Varieties.—Within the main groups of the dystrophies, it is possible to mark off certain sub-species, provided that we always bear in mind the fact that the classification does not rest upon any essentially distinguishing characteristics.

The *juvenile* form is differentiated by its onset in youth and middle

[1] *Nouv. Icon.*, xv.; *R. n.*, 1901.

[2] *Z. f. N.*, xxxi.

age, by the preponderant and early involvement of the muscles of the shoulder girdle and upper arm, and by the limitation of the real hypertrophy and pseudo-hypertrophy to certain muscles.

Pseudo-hypertrophy makes its appearance in earliest childhood, affects chiefly the male sex, involves mainly the muscles of the pelvis, of the lumbar region, the thigh and the calf, and the hypertrophy extends over large areas of muscle, whilst the atrophy is limited rather to the muscles of the upper half of the body (Fig. 143).

The *infantile* form (Duchenne-Landouzy-Dejerine type) is characterised by the primary involvement of the facial muscles.

The so-called *hereditary* form is usually strikingly hereditary, develops as a rule from the eighth to the tenth year or later, and commences with weakness in the sacral region and lower extremities. The localisation is similar to that in pseudo-hypertrophy, but the muscles are not pseudo-hypertrophic. The so-called Leyden-Möbius type and the Zimmerlin type are specially included in this group, the affection in both cases involving mainly the lower extremities.

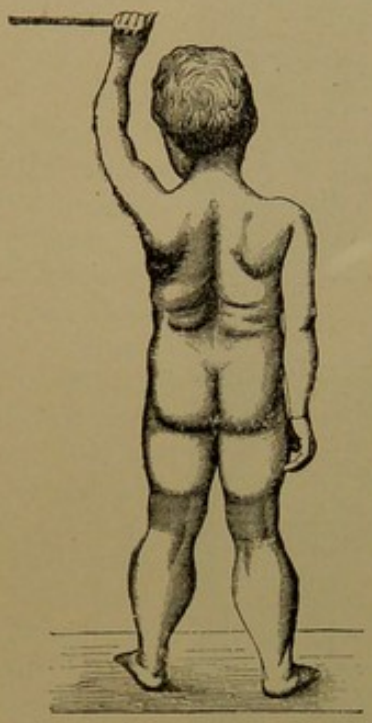

Fig. 143.—Progressive muscular dystrophy. Pseudo-hypertrophy. (After Erb.)

Pathological Anatomy.—This points to a primary disease of the muscles, as the nervous system (spinal cord and peripheral nerves) is found in the majority of cases to be practically intact. Even the investigations which have lately been made by means of the newest methods (Spiller-Dejerine, Sachs, Marinesco) have confirmed this fact of the integrity of the central and peripheral nervous system. There are indeed a few cases in which positive changes were found in the spinal cord (Dejerine-Thomas, Port, Rocaz-Cruchet), but these were atypical and complicated cases, or cases in which there were comparatively insignificant changes in the anterior horns.

Marked changes are found in the muscles—co-existing atrophy and hypertrophy of the primary fibres, increase of the muscle nuclei, proliferation of the internal perimysium, and a deposit of fat cells in the latter which may entirely supplant the muscle fibres, formation of fissures and vacuoles in the muscle fibres, entire disappearance of individual muscles, etc. The primary fibres have been found to show an enlargement of 230 μ. Figs. 145, 146, and 147 show the essential changes.

The size of the primary fibres certainly varies within wide limits, even in health, and there are great differences between the corresponding masses of various muscles in the same person, and of the same muscle in different persons, but we may take it that the average size of the muscles of the extremities is 30 to 50 μ, with a maximum of 108 μ. Even in normal muscles, however, there are scattered fibres which are of unusual diameter. The investigations of Schiefferdecker (*Z. f. N.*, xxv.) show that in estimating the breadth of the fibres we must keep in mind the influence of the rigor mortis, of the hardening fluid, etc. The number and size of the nuclei usually increase with the growth of the diameter of the fibre, so that the "relative proportion of nuclei" is approximately constant.

The pathological process, however, gives no definite indication for the differentiation of the various kinds of progressive muscular atrophy (Cramer,[1] Pick [2]). Erb thinks that the hypertrophy of the fibres is a preliminary to their atrophy.

There is no unanimity of opinion as to the condition of the terminal nerve plates and of the so-called neuro-muscular bundle.

Volume X. of the "Traité de Médecine" contains a careful, exhaustive investigation by Marinesco of the writer's reliable description of the histopathology of this morbid condition.

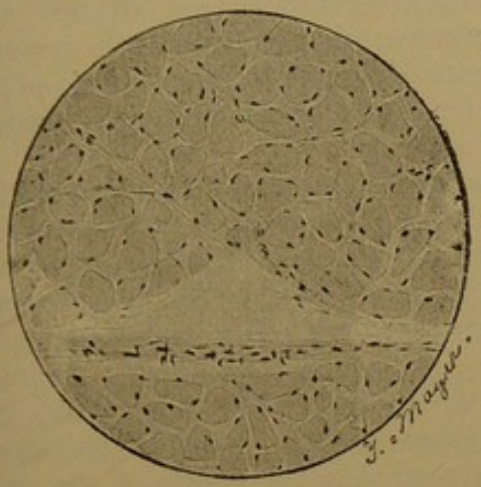

FIG. 144.—Transverse section through normal muscle. (Stained with alum-hæmatoxylin.)

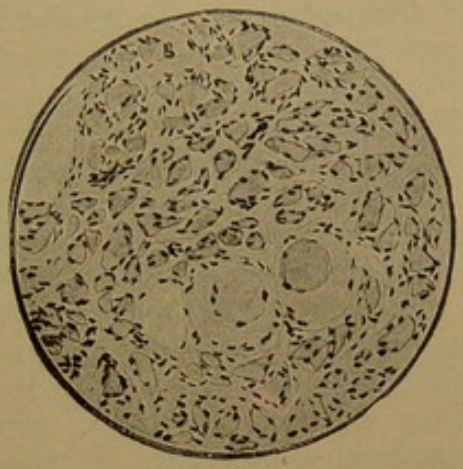

FIG. 145.—Transverse section through atrophied muscle.

The staining and enlargement is the same in Figures 144 to 147.

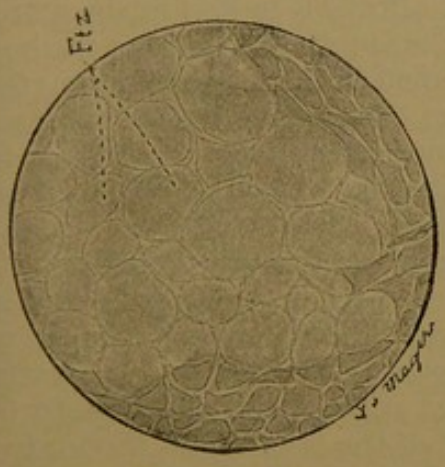

FIG. 146.—Progressive muscular dystrophy. Transverse section through a muscle showing lipomatous degeneration. Ftz—fat-cells.

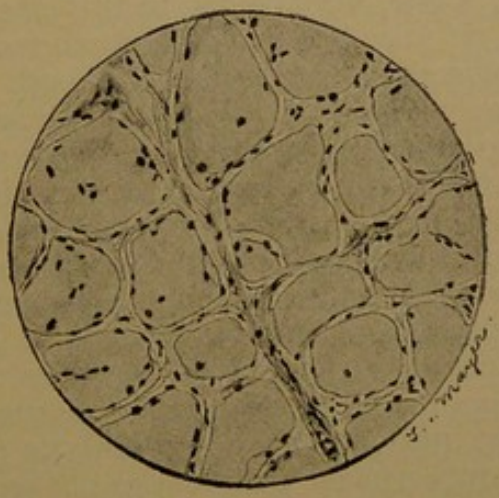

FIG. 147.—True hypertrophy of primary fibres and widening of the internal perimysium in progressive muscular dystrophy. (Transverse section.)

There can, indeed, hardly be any doubt that congenital anomalies of development in the muscular system are the cause of this condition. It has been demonstrated in a few cases that certain muscles have been absent from birth. I myself have found the presence of abnormal muscle bundles, especially in one of the sternal muscles. We must be very

[1] *C. f. path. Anat.*, vi.

[2] *Z. f. N.*, xvii.

cautious as regards the view that trauma may produce this affection (Lion-Gasne, Joffroy, Cramer).

Differential diagnosis.—In fully developed cases, the disease cannot be confused with any other. Certain difficulties may arise in cases where the disappearance of muscular tissue and the proliferation of fatty and connective tissue so equalise each other that the size of the muscles is at no point essentially altered. I have seen this in several cases of the infantile form. The diagnosis can, however, be made from the functional symptoms and the changes of electrical excitability; the normal contours of the muscles are to a certain extent effaced, and the affected segments of the limb have a rounded, tumour-like appearance. Developments of fatty tissue, causing deformities resembling those in myopathy, may, according to Richet, be found occasionally in healthy individuals.

It has already been mentioned that there are transition and intermediate forms of myopathic and spinal muscular atrophy. Thus it has happened in several instances that a case which from its clinical symptoms was classed with the former, had to be transferred to the latter group on the pathological evidence, and *vice versâ*. The affection also closely resembles the so-called "neurotic" form, still to be discussed; indeed, there is a myopathic type of the latter. There is, further, a localised and diffuse muscular atrophy, which arises from a polymyositis (Schultze, Oppenheim-Cassirer[1]). It resembles dystrophy in the mode of response of the affected muscles to the electrical current (for the most part simply quantitative diminution of the excitability), but there is no pseudo-hypertrophy, and the history reveals the fact that pain has accompanied the development of the disease. A case described by F. Pick, and another by Klarr, may belong to this class.

It may be very difficult to recognise the affection in its first beginning and especially in early childhood. The peculiar method of raising the body may be observed, though in a somewhat modified form, in any inflammatory process of the vertebræ or the muscles of the back which makes extension of the trunk painful. An error in the diagnosis is usually averted by the painfulness, by the definite tenderness of the lower part of the back, by the local condition of the vertebræ, by rise of temperature, etc. I have seen this mode of rising in a patient suffering from traumatic neuroses after contusion of the dorsal region. I have once seen an acute poliomyelitis limited to the lumbar muscles, causing inability to extend the spinal column, and simulating the picture of commencing dystrophy. In another case post-diphtheritic paralysis had mainly affected, in addition to the cranial nerves, the muscles of the lower part of the trunk, and the lordosis as well as the way in which the child raised himself from the horizontal position, entirely corresponded with the type described above. The pathological examination made by Sano in my laboratory confirmed the diagnosis.

As regards the diagnosis from Friedreich's disease, Thomsen's disease, and myasthenic paralysis, the chapters on these subjects should be consulted. A pseudo-hypertrophy of isolated muscles has occasionally been found in syringomyelia. The "waddling-gait" also occurs in osteomalacia, but this disease is so well characterised by its special symptoms that the differentiation is usually quite easy. I have, it is true, seen

[1] *Z. f. N.*, x.

two cases in which the differential diagnosis presented great difficulties. Rickets may also affect the gait and the mode of rising in a similar way (E. Remak).

A localised hypertrophy or pseudohypertrophy of the muscles has occasionally been found in venous obstruction (venous thrombosis), (Hitzig, Berger, Eulenburg, Lorenz, Schultze, Lesage, Bechterew,[1] etc.). Babinski saw it follow typhoid. It can hardly give rise to diagnostic errors.

Congenital defects in the muscles [2] are confined to certain muscles and are not progressive; Erb has shown, however, that they may occasionally confuse the diagnosis. The condition of "congenital high shoulder blade" should also be borne in mind (as to its origin, consult B. Keyser,[3] Ehrhardt,[4] etc.)

Complications.—The disease may be associated with hysteria, mental weakness, and epilepsy. Mental weakness or imbecility are by no means infrequent accessory symptoms (Vizioli). I have once seen a complication with tabes, and Cassirer with poliomyelitis. In one case the dystrophy supervened on an old spastic infantile hemiplegia. Bernhardt [5] describes the association with periodic paralysis of the extremities.

There was evidently a combination with scleroderma in a case reported by Ballet-Delherm, and with myxœdema in Schlesinger's case. Clarke's explanation of a swelling of the salivary glands in one of these cases is unsatisfactory.

I saw a case of this kind in one of Dr Jellinek's patients (San Francisco), who apparently presented a combination of the clinical features of Little's disease and those of dystrophy, and there was probably a similar association of symptoms in a case described by Jendrássik.

The congenital nature of the disease explains the frequency of its combination with other anomalies of development and clinical symptoms, as the communications of Jendrássik and Kollarits [6] have specially shown.

Prognosis.—Life is not as a rule in danger, but the respiratory muscles and the diaphragm may be involved in the dystrophy, and owing to the weakness of the expiratory muscles, death may result from affection of the breathing or from an intercurrent chest affection. The patient does not as a rule live long, and the earlier the onset of the illness, the shorter will his life be. The prognosis is therefore, in this respect, better in the juvenile than in the infantile form and in pseudo-hypertrophy. That exceptions to this rule may occur is proved by the case cited above, in which the patient lived to the age of fifty-eight, and by another in which the illness lasted for thirty-four years. Further, the disease may come to a standstill. There are also abortive forms in which some particular muscular area alone is affected—such as that of the shoulder girdle—the process showing no tendency to extend to other areas. One man of thirty, whom I treated, declared that his disease had not made the slightest progress since he was ten or twelve.

I have seen one man who presented the typical picture of the Erb-Landouzy-Dejerine type of dystrophy. Closer examination showed, however, that there were no functional disorders, with the exception of weakness in the muscles for closing the eyelids and lips, and it was ascertained that this symptom, as well as the anomalous attitude of the shoulders and trunk, were congenital, and that they had in no way advanced in the course of thirty years. He desired to

[1] *Z. f. N.*, xxxi. See bibliography here.

[2] For the literature up to 1902 consult Bing, *V. A.*, 170, and for more recent references, see Steche (*Z. f. N.*, xxviii.), and Capelle, *ibid.*

[3] *D. m. W.*, 1904. [4] *Z. f. kl. Chir.*, Bd. xliv. [5] *Z. f. N.*, viii. [6] *Z. f. N.*, xxx.

know whether he might marry, and out of regard to his posterity I felt constrained to advise him against this step. Jacquement (*R. n.*, 1905) has reported an extraordinary improvement in one case.

Treatment.—Excessive strain on the muscles should be absolutely avoided, but gentle movement and systematic exercise is, on the other hand, very necessary. Residence in a congenial climate and nourishing food, without much of the fat-forming elements, should be recommended. Electrical stimulation, especially galvanic, is perhaps of some value; a few cases are known, and I myself have seen two, in which considerable improvement followed electrical or gymnastic treatment. Sachs and Brooks have found this even more frequently. On the other hand, Marie especially disapproved of treatment by electrical stimulation. Hydropathy may also be tried.

Nothing has of late been heard of the results said to have followed injection of muscle-juice (Allard,[1] Tordeus). Rossolimo has obtained improvement in one case with thyroid preparations, but this does not seem to have been confirmed by any other evidence. Marinesco found no benefit from thymus extract.

When the Achilles tendon is greatly shortened and the power of walking is otherwise unimpaired, tenotomy is indicated. I[2] was formerly opposed to the use of tendon transplantation in this disease on account of its progressive character, but I must admit that it may be of transient benefit in cases which have a lingering course. It is advocated by Hoffa, Doberauer, Kuh,[3] etc. (see next chapter). Eiselsberg has sometimes fixed the two shoulder-blades to each other, and has in this way restored the power of movement to the arms.

Raymond also reports benefit from surgical fixation of the shoulder-blade.

Supporting apparatus for the shoulder girdle or the upper extremities, is recommended by Hager and others.

The So-called Neurotic or Neural Form of Progressive Muscular Atrophy. (Peroneal Type of Progressive Muscular Atrophy of Tooth. Charcot-Marie Type.)

Literature: Charcot-Marie, *Rev. de Méd.*, 1886; Hoffmann, *A. f. P.*, xx.; *Z. f. N.*, 1; Dubreuilh, *Rev. de Méd.*, 1890; Bernhardt, *V. A.*, Bd. cxxxiii.; Tooth, *Br.*, 1898; Marinesco, *Arch. de Méd. expér.*, 1895; Oppenheim-Cassirer, *Z. f. N.*, x.; Dejerine-Sottas, *Prog. méd.*, 1893; Siemerling, *A. f. P.*, xxxi.; Sainton, "L'Amyotrophie type Charcot-Marie," *Thèse de Paris*, 1899.

The disease commences as a rule in the second half of childhood, sometimes later, and may even appear in the third and fourth decades. Several members of a family are almost always affected. It may be directly transmitted to the children (through the father) or may miss a generation. Herrington[4] reports the affection of twenty-six individuals in one family. Interesting cases of this kind are also described by Eichhorst and Stiefler. On the other hand, the hereditary element was wanting in cases observed by Charcot-Marie, Oppenheim, Siemerling, Lähr,[5] and others. Men are much more often affected than women.

The development is insidious. The atrophy begins in the great majority

[1] *R. n.*, 1898. [2] *B. k. W.*, 1905. [3] *Prag. med. Wchnschr.*, 1905.
[4] *Br.*, 1888. [5] *Charité-Annalen*, 1894.

of cases in the *muscles of the foot*, in the peronei, the extensor communis digitorum, and the small muscles of the foot. This atrophy is accompanied by the development of a club-foot, a pes varus, equinus, or equino-varus, and usually with a claw-position of the toes. The ankle-joint is usually ankylosed in this position, but it may hang loose. The leg is correspondingly wasted (Fig. 148). The calf muscles are atrophied later. After some years the *upper extremities* become involved, the small muscles of the hand—thenar, hypothenar, and interossei—being always first and most affected. A claw-hand is thus developed. The proximal segments of the extremities are usually spared.

Fibrillary tremors are observed. In one of my patients there was a true rapid tremor. Electrical examination reveals *partial reaction of degeneration*, and also a sluggish faradic response and impairment of the excitability in the non-paralysed nervous areas. It may thus happen that the crural, posterior-interosseous nerves, etc., react only to the galvanic current. I have seen a case of this kind in which this marked modification of the electrical excitability extended almost over the whole body, whilst the muscular atrophy was present in the lower extremities alone. In certain cases there is merely a considerable quantitative diminution of the electrical excitability. The nerves are usually not specially tender to pressure.

The knee jerks are as a rule absent.

In one case, atypical also in that the disease had commenced in the first year of life in the arm, and showed, in addition to the characteristic condition of the leg, etc., a pseudo-hypertrophy of the thigh, the Achilles jerk and the tendon reflexes in the arms were absent, whilst the knee jerks were increased.

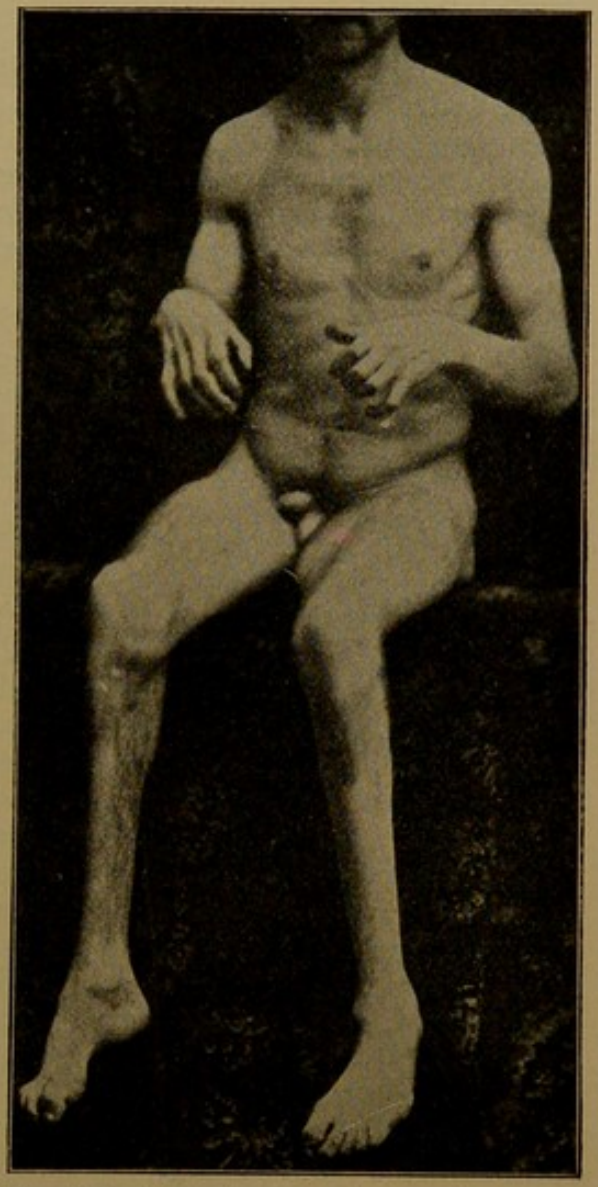

FIG. 148.—Neurotic muscular atrophy in an advanced stage.

Sensory disturbances sometimes occur (Sainton thinks very rarely); there is pain and even slight diminution of the sensibility at the ends of the extremities. In a few cases the pain was distressingly severe, and the hypæsthesia in one case was very marked (Marinesco). Vaso-motor symptoms are specially common in the legs.

I have once seen perforating ulcer in this affection. The occurrence of rigidity of the pupils and mental disorders is mentioned by Siemerling (the former also by Schulz, the latter by Sainton), atrophy of the optic

nerve by Vizioli, by Ballet-Rose, and by Krauss.[1] Spasms (epilepsy, narcolepsy) have also been observed in the course of the illness. These, however, are not symptoms of the disease itself, but are to be regarded as complications. All the other functions are unimpaired.

The affection runs a very slow course, and there may be long remissions and perhaps an arrest of its progress.

The extremely gradual course, especially in the later stages, the comparatively mild character, and the possibility of an arrest have lately been emphasised by Stiefler (*Z. f. Heilk.*, xxvii.), from his own experience.

There is not necessarily any pronounced limitation of the movements of the arms or hands, in spite of the atrophy (Guillain). The patient may live to a good age and may continue to be able to work. Surgical-orthopædic treatment (tenotomy, etc.) has given good results in some cases. In two such cases described by Hoffa and J. Fraenkel, I was able to satisfy myself that the tenoplasty had removed the deformity and had thus improved the function. Wimmer [2] makes a similar statement. The use of thyroidin seems to have been beneficial in one case (?).

Pathological Anatomy.—In some of the older cases (Virchow, Friedreich) a lesion has been found in the peripheral nerves and in Goll's column of the spinal cord. Hoffmann laid chief stress on the process in the nerves and proposed the term, "neurotic or neural form of progressive muscular atrophy." According to this view, we are dealing with a chronic hereditary form of multiple neuritis. This is in accord with the fact that the clinical picture was in a few cases (Oppenheim, Dercum) very similar to that of a chronic polyneuritis. Bernhardt preferred the term "spinal-neuritic form of progressive muscular atrophy." This term seems to be in accordance with the results of the post-mortem examination recently reported by Dubreuilh, Marinesco, Siemerling, Sainton, Dejerine, and Armand-Delille. These show a degeneration of the posterior columns, and later also of the lateral columns, and an atrophy of the anterior horn cells (and Clarke's column), the anterior roots, the spinal ganglia, and the peripheral nerves. The difficulty of reconciling the clinical symptoms with the pathological condition has not been sufficiently insisted upon by these writers. There is no unanimity of view as regards the site of origin of the pathological process. Siemerling, Sainton, and Raymond do not attach so much importance to the affection of the peripheral nerves as Hoffmann does. Dejerine and Armand-Delille found a simple atrophy of the motor nerves. Further, I have been able, along with Cassirer, to prove that the clinical picture may arise from a primary disease of the muscles (and for some of these cases at least, the term "*myositic form of progressive muscular atrophy*" is well adapted).

Thus, on account of the variety of the pathological processes, the disease cannot be regarded as a well-defined unity. Hoffmann points out from his own experience and a case described by Brossard that it may clinically be very closely allied with myopathy. A case published by T. Cohn may be interpreted in this way. Hoffmann, however, maintains that myopathy does not commence at the distal parts of the extremities.

The atrophy very rarely begins in the upper extremities. In the case illustrated by Fig. 148, the hands were as severely affected as the feet. In one of my patients the disease first affected the right hand and then

[1] *Z. f. Aug.*, 1906.

[2] *A. f. P.*, Bd. xlii.

passed to the right leg. The condition produced by the muscular atrophy of the lower extremities is well shown in Fig. 149. This case was also characterised by a malformation of the jaw and congenital nasal speech.

Cases have been described by Dejerine and Sottas (*Soc. de Biol.*, 1893; *Rev. de Méd.*, 1896) which are closely related to this affection, although they lead us still further from the class of progressive muscular atrophy. They show an onset in childhood with muscular atrophy in the end segments of the extremities (as already described). This gradually increases and is associated with severe lightning pains, marked sensory disorders, ataxia, especially in the arms, myosis, sluggish reaction and rigidity of pupils, nystagmus, Romberg's sign, kyphoscoliosis, marked quantitative diminution of electrical excitability, and no reaction of degeneration. Marked hypertrophy and hardening of the nerve trunks can be recognised on palpation. The pathological condition showed chronic interstitial neuritis, diminishing from the periphery to the centre, and present also in the posterior roots, sclerosis of Goll's and Burdach's columns in the lumbar cord, atrophy of the anterior horn cells and the anterior roots. In another case examined by Dejerine and Thomas (*R. n.*, 1902), the hypertrophy of the nerves and spinal roots was very considerable and extended to the cranial nerves and the sympathetic.

This affection, described as "névrite interstitielle hypertrophique et progressive de l'enfance," has also been observed in members of the same family. Strümpell thinks it represents a special form of hereditary systematic disease, a combination of Friedreich's disease and the hereditary peroneal form of progressive muscular atrophy. Marinesco and Raymond are inclined to class this affection which Dejerine describes with the Charcot-Marie form. It represents a systemic disease of the spinal cord, a combined degeneration of the anterior horns and posterior columns, with involvement of the corresponding roots and nerves. In the Charcot-Marie form the symptoms of affection of the anterior horns are most prominent, while in the "névrite interstitielle" it is the ataxic element which predominates. Raymond, it is true, recognises that the thickening of the nerve trunks forms a new factor. Dejerine himself (*R. n.*, 1906) is emphatically opposed to this classification and to Marie's attempt to differentiate the disease as a special type. It is doubtful whether Long's case should be included with this group.

Among German writers M. Brasch (*Z. f. N.*, xxvi.) has described cases apparently of this type.

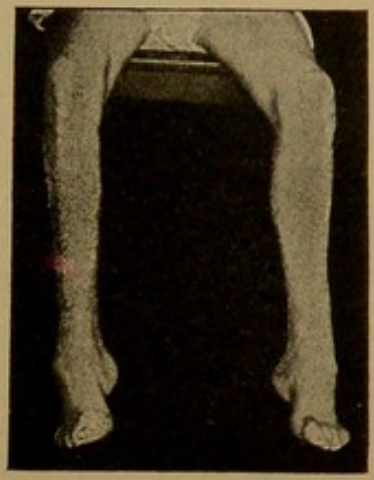

Fig. 149.—Legs and feet in a case of neurotic muscular atrophy. (Oppenheim.)

Supplement

I shall here discuss a clinical condition which is but distantly allied to progressive muscular atrophy, but which should not really be classified along with diseases of the spinal cord. My justification for considering it at this point, is that it represents a disease (primary or secondary) of the muscular system, a kind of myopathy, although it is a form which must be distinctly differentiated from those described above.

Thomsen's Disease (Myotonia Congenita) [1]

This is a disease which usually affects *several members of the same family* and is transmitted from *generation to generation*. Over twenty cases of the kind occurred in four generations of the family of Dr Thomsen, to whom we owe the first detailed description of the disease. Blood-relationship in the parents has in certain cases been the cause of a marked

[1] Literature: Erb, *Die Thomsensche Krankheit*, 1886; *A. f. kl. M.*, 1889; Pelz, *A. f. P.*, Bd. xlii., gives a very comprehensive bibliography.

predisposition, whilst in a few other cases no hereditary disposition could be ascertained.

The affection usually becomes evident *in earliest childhood*, but it may only be noticed about the period of puberty or later, and may give one the impression of an acquired condition. Thus it appears occasionally to have developed after a mental excitement such as a severe fright, but it is not impossible that the shock has merely increased the symptoms, and thus roused the affection out of a latent existence. We may in this sense speak with Pelz of a myotonia congenita adultorum.

Patients with this disease have generally well developed, and even *excessively large muscles*. The muscular force is, however, somewhat diminished. The first movements after a period of rest may be especially feeble (Mann). There may also be a "Herculean" condition of the muscles limited to certain regions. In one of my cases this appeared in the lower extremities and jaw muscles, whilst the muscles of the arms were by no means hypertrophied. The essential symptom is the inhibition of voluntary movements by *stiffness of the muscles*. If, after a long rest, the patient tries to set a group of muscles into activity, they pass into a condition of *tonic* contraction, which at first cannot be voluntarily relaxed. After five, twenty, or thirty seconds, the spasm begins to yield, and the movement becomes smoother and easier with every repetition, until eventually it can be executed without any difficulty. The patient is therefore able to undertake long walks, to dance, etc.

The *myotonic affection* becomes specially evident when the patient *suddenly* attempts an energetic, forceful movement, when, for instance, he rapidly and powerfully clenches his hand, flexes the elbow, or presses his jaws tightly together. The arm, extended towards a glass, may remain stiffly stretched out, the hand with which he grasps another may remain firmly clenched round it like a clasp, until in time the tension relaxes. A powerful reflex movement may pass into a condition of tonic spasm; thus, in severe cases the patient may, on making a movement of any kind, fall to the ground and lie there *absolutely rigid*, until the muscles relax and his power of free movement returns. One of my patients severely injured himself in this way.

All the muscles of the body are as a rule more or less involved, but the affection may be very pronounced in certain parts, in the limbs, for instance, and very slight in others, such as in the muscles of the face and jaw. The ocular muscles may be involved; in one of Charcot's cases when the patient looked upwards, the eyeballs remained in this position for a considerable time. The tongue is not infrequently affected, but the muscles of the throat and respiration very rarely are. An involvement of the heart has been assumed in a few cases, but this seems to me very doubtful. It may happen that the arms are apparently free, whilst the legs are affected, and *vice versâ*. There are even isolated cases in which the disease is confined to *certain groups of muscles*. I have treated one man in whom the myotonic affection was distinctly marked in the orbicularis palpebrarum only, whilst the other symptoms were present in the other muscles of the body also.

Cases of this partial myotonia are also described by Gaupp (*C. f. N.*, 1900); Schott (*Z. f. N.*, xxi.), and Curschmann (*B. k. W.*, 1905). They are atypical in that they are often associated with atrophy of the muscles (see below).

The myotonia is aggravated by the influence of *emotion*, and especially so if the patient thinks he is being observed. It is also more intense after a long rest and on forced efforts, after feverish illnesses, and particularly in cold weather. In some cases the muscular stiffness becomes evident *only during cold weather* (as in a case of intermittent myotonia described by Martius and Hansemann [1]), but these must represent a special variety of the disease. Warmth on the other hand has a beneficial effect, and so have mental rest, the use of moderate quantities of alcohol, and particularly the frequent repetition of a movement.

Objective examination always reveals certain symptoms in the muscular system, which had been noted by earlier observers and have been carefully studied by Erb. These are—

1. *Exaggeration of the mechanical excitability of the muscles.* While the mechanical excitability of the nerves is not increased, but is if anything diminished, percussion of the muscles gives rise to a slow, tonic, persistent contraction of the affected part. The belly of the muscle protrudes like a tumour, or shows local bifurcations and depressions. As a rule pressure with the finger upon the muscle is sufficient to produce the symptom.

2. *Alteration of the electrical excitability : the myotonic reaction* (MyR). The faradic excitability of the nerves is on the whole unchanged. Stimulation of the nerves with strong currents causes a *tonic muscular contraction, which persists*, whilst single opening induction shocks give rise to a short jerk. Even feeble currents produce a *tonic contraction of long duration.* In continuous faradic stimulation there is sometimes a wave-like contraction of the muscles stimulated. The *galvanic excitability* of the nerves is also somewhat diminished from an early stage. When the nerves are stimulated by a labile current a persistent contraction is produced. The *direct* galvanic excitability of the muscles is increased ; closing contractions alone appear, and the ACC usually predominates. *The sluggish, tonic character of the muscular contractions* and their persistence are specially remarkable. The stabile galvanic current produces a *rhythmic* undulation of the muscles, and the wave of contraction passes from the cathode towards the anode. Strong currents (up to 20 MA) are necessary to produce this phenomenon. An electrode of some size is placed in the region of the neck or sternum, the other of medium size on the palm of the hand, and after a time the undulatory movement sets in. In some cases the current has to be used repeatedly before the symptom appears (Erb).

The spark discharge of the *static current* produced simply *single contractions* in a case which I examined, and the reaction for static electricity was thus seen to be practically unaltered.

The clinical picture is represented by the symptoms described above. The other functions of the nervous system are not affected ; there is in particular no change in the sensibility, the sensory functions, reflexes, etc. A diminution or even an abolition of the knee jerk has been often noted (Seeligmüller, Buzzard, Pelizaeus, Bernhardt, Erb, Hoffmann, H. Curschmann). In one of my cases there was nystagmus and Graefe's sign (the latter of which has also been observed by Mann). The patient also complained of bulimia or polyphagia. Disturbances of metabolism have been reported by Bechterew, Karpinski, Wersiloff,[2] and Ballet

[1] *V. A.*, Bd. cxvii.

[2] *N. C.*, 1897.

(increased secretion of creatinin, in particular, though Zülzer has not been able to confirm this). Guillain found increase of the blood pressure.

The disease is not uncommonly complicated with *mental symptoms*, *epilepsy*, hemicrania, etc. A combination with multiple neuritis (Hoffmann), with tabes (Nalbandoff,[1] Hoffmann), and with tetany (Bettmann) has been described. The combination with muscular atrophy (Hoffmann, Jolly, Delprat, Bernhardt, Schönborn, Frohmann,[2] Cassirer, Lortat-Jacob, Curschmann, Lannois, etc.), with pseudo-hypertrophy (Charcot) or dystrophy (Nonne) is particularly frequent. Hoffmann[3] has studied this question carefully and has investigated a great number of cases ; he thinks that progressive muscular atrophy may itself develop from the myotonia. Rossolimo and Schott agree with him. In one case there was a congenital defect of the muscles in the shoulder-girdle, and in a case under my observation there were anomalies of formation in the fingers, which were inherited.

There are severe and slight cases of this kind. In the latter case the motor affection is sometimes so insignificant that it is not noticed by other people, and the patient can follow any calling. One of my patients was a celebrated professional violinist. His arms were quite unaffected, and there was merely a slight degree of myotonia in the legs, which was aggravated under certain conditions. He used himself to relate as a joke that at a public meeting at which an order was to be bestowed on him, at the very moment when he should have advanced to receive it, he stood as if rooted to the ground and could not move from the spot. The disease was very pronounced in two of his sons. Men suffering from Thomsen's disease are generally speaking not fit for military service.

The *diagnosis* can be easily made. A condition somewhat similar to the myotonic reaction has occasionally been found in other diseases ; electrical stimulation with a strong faradic current causes painful, persistent contractions, but these were probably identical with muscular cramp.

An affection resembling myotonia has been described by Eulenburg (*N. C.*, 1886), under the name of *paramyotonia congenita* ; a spasm and rigidity of the muscles of the face and neck, of deglutition and of the extremities coming on in cold weather, which made the patients more or less incapable of movement. When this rigidity relaxed (in a quarter to several hours), there followed a condition of paralytic weakness, which sometimes lasted for days. The orbicularis oris and palpebrarum were chiefly involved. The mechanical excitability of the muscles was not increased. Electrical examination showed diminution of the excitability. Observations of a similar kind have since been reported by Sölder, who also found a pathological condition corresponding to that of myotonia (*W. kl. W.*, 1895). Some recent cases have been regarded as a combination of myotonia and paramyotonia. It has also been found that some of the members of a family were suffering from Thomsen's disease, the others from paramyotonia (Delprat, Hascovec). A case described by Martius-Hansemann of *intermittent congenital myotonia* (*V. A.*, cxvii.) represents an absolutely atypical transition form. Weichmann describes a similar condition.

Jacoby (*Journ. of Nerv. and Ment. Dis.*, 1898) makes a sharp distinction between the congenital, acquired, and transitory forms.

Talma (*Z. f. N.*, ii.) describes as *myotonia acquisita* an acquired disease which is closely allied to Thomsen's disease in so far that it presents the various symptoms of the myotonic reaction ; but here, so far as one can gather from the short history of the case, the condition is a

[1] *N. C.*, 1899.

[2] *D. m. W.*, 1900.

[3] *Z. f. N.*, xviii. Magneval's *Thèse de Lyon*—"Des Myotonies atrophiques," 1904—is also a contribution to this question.

transient and curable one, the muscular rigidity persists to a certain degree even during rest, and the spasms chiefly follow prolonged exertion. A condition observed by Fürstner (*A. f. P.*, xxvii.) is still further removed from Thomsen's disease, as there was no myotonic reaction and the muscular spasm came on spontaneously during rest; the course, moreover, was a favourable one. Bechterew also noted the absence of the myotonic reaction.

The characteristic changes in the mechanical excitability were absent in a few instances (Seeligmüller, Strümpell, Pantoppidan, etc.). Jolly shows (*N. C.*, 1896) that the myotonic reaction may become gradually less marked on repeated stimulation. A modification of the myotonic reaction is described by Pässler.

Gowers describes by the name of *ataxic paramyotonia* an acquired condition in which a persistent tonic muscular contraction, which made all the movements stiff and slow, was associated with ataxia and sensory symptoms; its nature is undetermined. It has certainly no connection with myotonia congenita. Cases which are difficult to classify have been described by Stein, Wichmann (*N. C.*, 1897), Seiffer and Dercum, and by Lannois.

The intention spasms (Kasparek) which occur in tetany are sufficiently identified by the other symptoms of the disease. Hoffmann, however, has found the myotonic reaction in a case of tetany with myxœdema due to removal of the thyroid.

Lundborg (*Z. f. N.*, xxii.) has drawn attention to the relation of *myotonia* to *myoclonia*, and he has especially noted a myotonic affection of the movements under the influence of mental excitement in the family form of the latter. This question requires further elucidation, however. Oddo distinguishes between myotonia and periodic paralysis of the extremities (*q.v.*), which he names myoplegia, and endeavours to find relations between these two affections.

The atypical forms of Thomsen's disease have been exhaustively discussed by A. Pelz. The paper by Mingazzini and Perusini (*Riv. di Patol. nerv.*, 1904) is also a contribution to this subject. I have seen the following case, which I found it impossible to classify: F., a doctor of sixty-five, had been conscious for about ten years of motor disorders in his legs, and upon these there supervened pain in the shoulders and back, and eventually a moderate degree of dysarthria and dysphagia. In the arms there was weakness, especially of the extensors, myotonic affection of the movements and myotonic reaction to mechanical stimulation, whilst there was simply quantitative diminution of the electrical excitability. There was diminution of the tendon reflexes in the legs, but no marked affection of sensibility. There was paresis, most pronounced in the distal areas, especially of the peronei, which also showed mechanical myotonic reaction and quantitative electrical diminution The muscles were somewhat tender to pressure; the sense of position was dulled at the toes. The pupils and palpebral fissures were narrow, but reaction was conserved. Slight dysarthria and dysphagia were present, and a trace of Romberg's sign.

The cases just cited teach us that myotonia congenita is an affection presenting numerous varieties, and that it is very apt to be associated with other groups of symptoms, and especially with other diseases of the muscular system. Myasthenic symptoms have lately been observed in combination with myatrophic myotonia (Steinert, Curschmann, Oppenheim).

The *prognosis* as to life is favourable, but up to the present no case of recovery or material improvement has so far been reported. The affection persists till the end of life, although it shows no sign of being progressive in character. Remissions are not uncommon.

Pathological Anatomy.—Erb and others have found in excised portions of muscle, a *hypertrophy of the primary fibres* (which may be twice the ordinary size), an increase of the sarcolemma nuclei, and a slight increase of the interstitial tissue (see Fig. 150). Some investigators (Ponfick, Ballet) have failed to find these changes. I[1] have been able along with Siemerling to demonstrate that in portions of muscle excised from a living patient an apparent hypertrophy was produced by the contraction

[1] *C. f. d. med. Wiss.*, 1889.

of the fibres, which can be avoided if one relaxes the contraction. Erb's results have been explained by the exaggeration of mechanical excitability—that there was marked contraction because of the mechanical irritation during excision—and this suggestion has been made by myself and later by Jacoby.[1] However, Dejerine and Sottas [2] have found the same changes in the muscles in a case examined post mortem, the nervous system being absolutely intact.

According to Jacoby's investigations, the sarcous elements of the muscle are increased, diminished in amount, or increased in consistence. Atrophic as well as hypertrophic fibres have been found by Hoffmann and Pelz.

Schiefferdecker (*Z. f. N.*, xxv.) makes the following statements: the primary fibres are broader than normal, but this hypertrophy is not very pronounced and there are also many small fibres. The nuclei are increased in number and size in correspondence with the size of the transverse

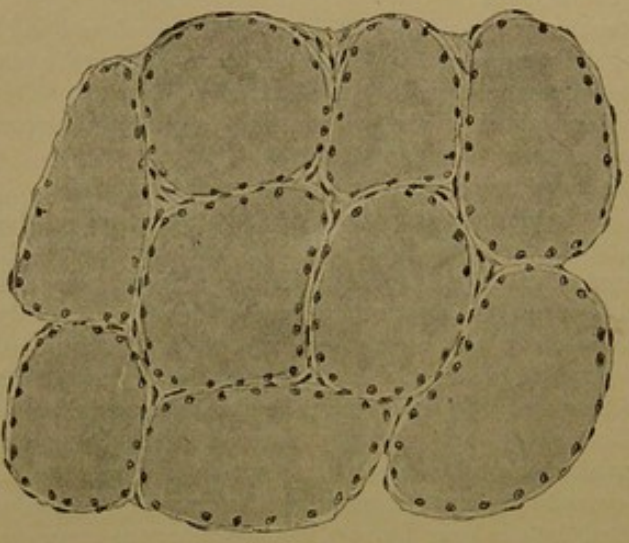

Fig. 150.—(Compare with Fig. 151.) Transverse section of muscle in Thomsen's disease. (Alum-hæmatoxylin.)

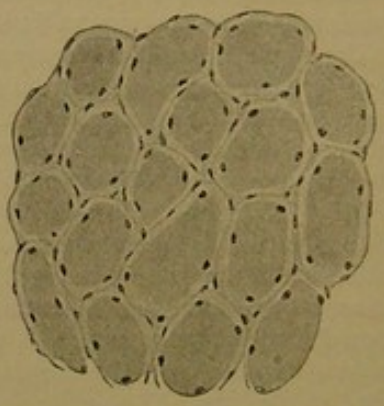

Fig. 151.—Transverse section of normal muscle. Staining and enlargement the same as in Fig. 150.

diameter of the fibre, the "relative proportion of nuclei" being unaltered. The sarcoplasm, *i.e.* the substance lying between the muscle fibres, is shown, by a certain process of fixation in formol, to contain granules which are not found in normal muscles. The fibrils are condensed, especially at the margins of the fibres, and cohere with neighbouring fibrils.

Symptoms resembling myotonia have been produced in animals by veratrin and creatinin poisoning. Ioteyko in particular has assumed that the toxins of metabolism may affect the sarcoplasm in the same way. Lévi,[3] Pässler, and Buzzard attribute the origin of myotonia to an *exaggerated excitability of the sarcoplasm*. Bechterew is also inclined to trace the disease to autointoxication. In my opinion, however, everything seems to point to the fact that in the typical form we are dealing with a disease which is due to an abnormal development and disposition.

H. Curschmann has recently declared his opposition to the theory of the myogenic nature of the affection and his belief in its central or supranuclear origin.

No means of cure has yet been discovered. From all our experience we should expect, as I pointed out in the first edition, and as Bechterew

[1] *Journ. of Nerv.*, 1898. [2] *Rev. de Méd.*, 1895. [3] *R. n.*, 1905.

has since emphasised, that *systematic gymnastics* would prove of greatest benefit. Bechterew also believes in the use of massage. Seiffer [1] rightly rejects the proposal of Gessler to bring about an atrophy of the muscles by stretching the nerves.

DIFFUSE DISEASES OF THE SPINAL CORD

Many of these affections originate not in the spinal cord itself, but from the membranes surrounding it, or from the spinal column. Even morbid processes which take place outside the spinal column, and at a considerable distance from it, may make their way through the intervertebral foramina or by means of erosion of the vertebral bodies into the spinal canal, and so involve the spinal cord. It is well known that aortic aneurisms may affect the spinal cord after erosion of the vertebral bodies, and that malignant tumours arising from the kidneys (sarcoma, cysto-sarcoma) or from the retroperitoneal lymph glands may sometimes break through the spinal column and compress the spinal cord. Bedsores also, penetrating deeply, may give rise to pus and ichorous matter, which find their way into the spinal canal and produce a condition of septic inflammation in the meninges and spinal cord. Pus from a psoas abscess much less often finds its way into the spinal canal.

Other diseases of this class originate in the spinal cord itself. Although these for the great part are due to organisms which penetrate from without into the organism or come from an infective process at any part of the body, they should from a pathological point of view be regarded as independent primary diseases of the spinal cord, and should be distinguished from secondary affections. It will be expedient to describe the latter first.

A.—Affections of the Spinal Cord due to Diseases of the Vertebræ

In considering the diseases of the spinal column which may involve the spinal cord, we shall merely glance at the subject of trauma, such as fractures and dislocations, as a detailed description of these would find a more fitting place in a surgical text-book.

Dislocations and Fractures of the Spinal Column

Dislocations occur most frequently in the cervical region of the spinal column, and especially between the fifth and sixth and the first and second cervical vertebræ. They are comparatively rare in the thoracic region, and very rare in the lumbar.

Dislocations are generally bilateral (flexion-dislocations or a total dislocation). Unilateral (or rotation) dislocations are less common, and they may be complete or incomplete. In flexion-dislocation the articular process of the upper vertebra moves over the lower one. If the articular processes remain with their margins in contact, the condition is called flexion-dislocation with riding of the articular processes, but if they are displaced still further forwards it is called flexion-dislocation with impaction. In the unimpacted form, there is always a marked kyphosis and separation of the spinous processes (Stolper). A simple separation of the vertebræ, without

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[1] *N. C.*, 1900.

displacement in the horizontal direction, may be brought about by rupture of the ligaments. The upper vertebra, which is termed the dislocated one, is almost always driven forwards. The dislocation is generally caused by indirect violence, excessive flexion of the head and neck, a fall or a blow upon the head, or by traction on the latter. Unilateral dislocation is generally produced through excessive lateral movement. Distortion, according to Kocher,[1] most frequently occurs in the middle cervical region.

The most important of the direct symptoms of dislocation is deformity of the spinal column. This is but rarely absent.

In bilateral dislocation in the cervical region, the head is usually inclined forward ; the spinous process of the vertebra immediately below the dislocated one forms a projection, while that of the latter is displaced forwards. Abnormal prominence may also, in spare individuals, be palpated laterally in the neck, and in the case of the upper cervical vertebræ, from the pharynx. (Palpation succeeds as far as the third, and, according to Stolper, even as far as the fifth cervical vertebra.) The spinal column is kept rigid through tension of the muscles. Every attempt at movement is painful. In dislocation of the upper cervical vertebra, patients tend to fix the head with the hand and anxiously avoid all movement. Dislocations between the atlas and axis are generally associated with fracture of the odontoid process.

In *unilateral dislocation* (rotation-dislocation) the head is inclined towards the opposite shoulder, while the chin is turned towards the corresponding one. The spinous process of the dislocated vertebra is directed towards the side of the dislocation, so that the cervical column forms a curve with the convexity directed towards this side. The neck muscles and the sterno-mastoid of the same side are firmly contracted.

Fracture of the vertebræ may occur at any part of the spinal column, most frequently in the middle cervical and upper thoracic, and especially in the region between the tenth thoracic and the first lumbar. Thus in 250 out of 383 of Menard's cases, the lower thoracic and upper lumbar vertebræ were concerned. Most of these fractures are due to indirect violence, a fall on the head, on the buttocks, the fall of a heavy body upon the head or neck, and through these causes it is mainly the body of the vertebra that is fractured. By a fall upon the head, the upper thoracic vertebræ are most frequently fractured ; in falls upon the feet or upon the buttocks, the last thoracic or the first lumbar. Even violent muscular effort, a sudden turning or movement of the head and neck, have, in rare cases, produced fracture. Direct violence may lead to a forcible separation of the laminæ and of the spinous processes, without injury to the body of the vertebra. This however, is almost entirely limited to the cervical vertebræ. In the upper part of the spinal column, several vertebræ are frequently affected, while fracture of the lower thoracic and lumbar column tends to be limited to single vertebræ. Dislocation is frequently associated with fracture. The majority of total fracture dislocations occur in the lower thoracic vertebræ (Kocher).

The symptoms of fracture are so akin to those of dislocation that it is often impossible to make a definite differential diagnosis. Marked displacement with fixation of the upper part of the cervical column points to dislocation. In fracture this may be entirely absent, although some deformity (kyphosis and separation of the vertebræ) is usually produced. Crepitation indicates fracture. The fragments can seldom be directly

[1] *Mitt. aus d. Grenzgeb. d. Med. u. Chir.*, Bd. i., 1896.

palpated from the neck or pharynx. The deformity may only develop after a secondary displacement.

We may now consider the *symptoms arising from the nervous system.* From this point of view, the fact should first of all be noted, that in fractures as well as in dislocations of the spinal column, symptoms pointing to implication of the spinal cord and the nerve roots may be entirely absent.

This applies particularly to *partial* injuries of the vertebræ, amongst which Kocher includes contusions and distortions of the spine, fractures limited to the vertebral arches and spinous processes or bodies, dislocations limited to the lateral articulations. In total dislocations and fracture dislocations, on the other hand, the cord is almost always destroyed, or at least injured.

As a rule, however, the *spinal cord* and the roots are involved. In many cases the cord is permanently compressed by the displacement of the vertebræ, or by fragments of bone that have been dislocated into the spinal canal. The contusion, crushing, or tearing of the cord is often merely the result of violent dragging or of the displacement of the portions of the column, which has been distorted at the moment of the injury, and has subsequently become readjusted. Thus the spinal cord may be injured without there being a permanent solution of the continuity of the spinal column (as in the interesting cases of Jolly, Wagner, Stolper,[1] Spiller, Hartmann,[2] Schäffer, Fischler, and the experimental investigations of Fickler[3]), or the injury of the spine and of the spinal cord may be co-effects of the same violence, without a permanent compression being caused by the displacement of the parts of the spine. Thus, Bowlby has found that compression from spinal displacement was entirely absent in many cases of spinal fracture which terminated fatally. Further, compression of the spinal cord may also be caused by hæmorrhage into the epidural space (rupture of the veins). Subarachnoidal hæmorrhages are less frequent. Stolper,[4] however, attaches no great significance to extra-medullary hæmorrhages as a cause of symptoms, as they almost always represent an accessory factor, and the compression which they produce hardly comes into account as compared with the direct injury to the cord. It should, however, be noted that they may be of great extent and may spread beyond the focal lesion. Injuries of the spine, especially of the lower cervical region, even when they do not affect the vertebral column, fairly often cause central hæmatomyelia (Thorburn,[5] Minor,[6] Kocher, and others). They occur specially in the cervical region, and are caused by hyperflexion of the cervical column. Stolper attributes such hæmorrhages to *traction* as contrasted with those due to *contusion.* These hæmorrhages usually extend upwards and downwards far beyond the site of the lesion, and may take the form of a column within the cord. Fischler[7] notes that violent dragging of the caudal roots on the conus may destroy the latter, with hæmorrhage, etc., without the vertebral column being necessarily injured. Finally, compression of the cord may be caused by reparatory processes at the site of the fracture—by callus and the formation of callosities of connective tissue. The spinal roots

[1] Wagner-Stolper, "Verletz. d. Wirbelsäule u. d. Rückenmarks," *Deutsche Chirurgie*, Bd. xl., Stuttgart, 1894.
[2] *Jahrb. f. P.*, 1900.
[3] *Z. f. N.*, xxix.
[4] *L. c.* and *Mitt. auf d. xii intern. med. Kongress Moskau.*
[5] "Contribution to the Surgery of the Spinal Cord," London, 1889, and *Br.*, 1888.
[6] *A. f. P.*, xxiv., xxviii., and "Handbuch d. path. Anat. d. Nerv.," ii.
[7] *Z. f. N.*, xxx.

are compressed either close to the cord and along with it, or are crushed at their passage through the intervertebral foramina.

Fractures and dislocations of the *two first cervical vertebræ* are usually immediately fatal, but in many cases death may be postponed for some time. Besides the dislocation (the displacement, inclination, and twisting of the head), the rigidity of the neck—which is here absolute—and severe local pains, there are also observed as symptoms of lesions of the roots and spinal cord, *radiating pains in the region of the upper cervical nerves*—especially in the region of the great occipital nerve—*dyspnœa*, and, usually during the further course, marked bulbar symptoms along with symptoms of paralysis of the muscles of the trunk and extremities. In dislocation life is in very rare cases preserved by reduction, but death usually follows, if it does not take place immediately, in a few days or weeks, from some rash movement of the head, or as the result of myelitis of the uppermost cervical cord or the medulla oblongata.

A frequent symptom of contusion or laceration of the cord, especially in the cervical region, is *erection of the penis*; this is ascribed by Kocher, as well as by Müller and Lerchenthal,[1] to paralysis of the corresponding vaso-motor nerves. Ejaculation may take place at the time of the injury,

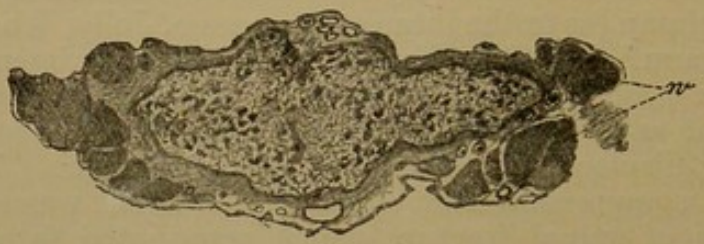

FIG. 152.—Spinal cord in a case of fracture of the spinal column. Complete destruction of the cord. The meninges adhere to each other and to the roots. (From Oppenheim's collection.)

and under these circumstances the erection may often persist for a considerable time. *Marked rise of temperature, rapidity or slowness of pulse*, and *vasomotor disturbances* are observed in these lesions of the cervical cord.

In fractures and dislocations of the *third and fourth cervical vertebræ*, life is specially endangered by involvement of the phrenic nerve. Death takes place at once or within a few days or weeks, except in a small proportion of cases.

In general the spinal symptoms which accompany fractures and dislocations observe the following rules: The cord is usually so affected at the level of the injury, by compression, laceration, hæmorrhage, and, at a later stage, by inflammation, that conduction is more or less completely suspended (see, for instance, Fig. 152). Thus all those muscles, the motor conduction-tracts of which are contained in the spinal cord below the lesion, are paralysed. Therefore, in disease of the upper cervical cord, all the four extremities and the muscles of the trunk, and in affections of the thoracic cord the muscles of the leg and some of those of the trunk are affected. Sensation is also lost in those areas of the skin the sensory nerves of which enter the cord in the segments below or at the level of the focus. That is to say, the area of the distribution of the sensory disturbances corresponds with that of the paralysis. We must not, however, expect that its upper margin would correspond with the level of the affected vertebra. In the majority of cases it does not extend

[1] *Z. f. N.*, xxxi.

so far up, as the roots which arise from the segment of the cord involved in the lesion belong not to the same, but to a deeper level. We have also to take into account accessory conduction by means of anastomoses (see section, Localisation in the Spinal Cord, pp. 125 *et seq.*).

Since in a compression or contusion, which simultaneously involves the cord and the roots, the latter frequently remain uninjured, whilst the more delicate structure of the cord is affected, the paralysis and anæsthesia may extend upwards only as far as the area of the root which arises from the affected segment of the spinal cord. Thus, in injury of the fourth thoracic vertebra, it may not reach higher than the region of the sixth intercostal nerve. The difference of level of the lesion and of the paralysis and anæsthesia will be the greater the lower down in the vertebral column or cord is the site of the injury, since the distance between the origin of the root and the site of its emergence from the spinal canal increases from above downwards. If, on the other hand, the injury involves not only the cord, but also the roots which lie alongside of it but have a higher origin, then the distribution of the paralysis and anæsthesia will extend upwards as far as the area of distribution of the injured roots. It is further to be noted, that frequently it is not the vertebra the spinous process of which projects backwards, which causes the compression, but the one either above or below it.

It not infrequently happens that there is a marked want of agreement between the distribution of the symptoms and the site of the spinal injury which cannot be explained by the above-mentioned facts, since sometimes in an injury of the highest thoracic region, the sensory disturbance extends only as high as the lumbar region, if so far. In such cases the cord is usually only partially affected. Frequently there is a central hæmorrhage. In a compression of the cord the soft, vascular tissue of the grey matter is injured, while the white remains more or less intact. Therefore, as the peripheral areas of the spinal segment are unaffected, the sensory conduction in the cord is retained for the posterior roots which enter at this level, and are continued upwards in the white strands, near the surface of the cord. However, the experimental observation of Flatau, described on p. 105, is opposed to this explanation. It much more rarely happens that the limits of the anæsthesia and paralysis extend upwards beyond the part of the cord corresponding to the level of the vertebra. There is usually in such a case a hæmorrhage, which has bored its way upwards within the grey matter beyond the part of the cord directly injured, and this may later result in a myelitis. The total anæsthesia may be bounded by a zone within which the sensibility for pain and temperature is diminished (Kahler, Pick, Minor). At the upper limits of the anæsthetic area, there is sometimes a hyperæsthetic zone. Herpes zoster may also occur.

In these transverse lesions of the spinal cord, wherever their site may be, the *function of the sphincters* is always affected. There is retention of urine and incontinence, as well as incontinence of fæces (see also p. 118), and bedsores usually form sooner or later. It is only when the lesion in the cord is not a completely transverse one that these functions remain undisturbed. Stolper mentions affections of the bladder and kidneys, which he does not ascribe to infection of the urine, but regards as trophic disturbances. We (Borchardt and I) have so frequently found these

affections after excision of tumours lying outside the cord, that we have come to the same conclusion. Arthropathies also have been observed in isolated cases (Chipault).

Further, it is the rule that, in severe injuries of the spinal cord which cause total interruption of conduction, even when they have their site in the upper segments of the cord, the paralysis of the legs is flaccid, and is accompanied by loss of the deep and usually also of the superficial reflexes. This certainly applies to the period immediately following the injury, in which, according to the prevailing view, the effect of the shock extends over the whole of the spinal cord. But from numerous observations, especially of recent times, it appears that under these conditions the atony and the absence of reflexes may continue to exist even later. Indeed, the same conditions have been repeatedly described in non-traumatic diseases of the spinal cord, in which the element of shock is entirely absent. The explanation which was formerly given, that in such cases a myelitis or a hæmatomyelia had spread through the whole cord as far as the lumbar region, is not sufficiently supported by the results of post-mortem investigation. It has been observed that in compression of the cord, in addition to the main foci and the secondary degenerations, there are found outlying foci at a distance, but these are small and few in number, and usually lie in the neighbourhood of the main focus. Bastian lays it down as a rule (see p. 115) that when a disease or an injury causes a *total interruption* of conduction in the cord, and is therefore equivalent to a complete transverse section, it abolishes all the reflexes, superficial and deep, in the region of the lower segments of the spinal cord. Therefore in a completely transverse lesion of the cervical cord, in addition to a total paralysis and anæsthesia, the plantar reflexes and the knee jerks are absent. According to Thorburn, the reflex functions of the bladder and rectum are also abolished under these conditions, so that evacuation of the bowels cannot take place by reflex means. This is, however, disputed by Kocher[1] and others. The majority of earlier observations are inconsistent with this view, and it has already been shown (p. 115) that Bastian's theory, in spite of the confirming opinions of Bruns, Collier, and others, can no longer be maintained, and that the observations on which it is founded can be better explained in other ways.

In partial lesions of the spinal cord, situated above the lumbar enlargement, the tendon reflexes may certainly be absent; but this is so only during the first period, the spastic symptoms which appear later being associated with exaggeration of the tendon phenomena and cutaneous reflexes (Babinski's sign, etc.). With regard to the differentiation of total from partial lesions of the cord, the following points should be taken into account: If sensibility is not completely abolished, the interruption of conduction may be only partial. When the lesion is situated in the cervical or thoracic regions, then paræsthesiæ, pains, and especially hyperæsthesia in the lower extremities as a rule indicate a partial destruction. (Hyperæsthesia may be found also in complete transverse lesions, but only at the upper margin of the anæsthetic zone.) This is the case also as regards integrity or even exaggeration of the sensation of desire to pass water or fæces, etc. A spinal injury which has produced sensory dis-

[1] This writer draws attention to the fact that the erection-reflex (sometimes that for ejaculation also) is retained, and may be elicited in various ways—touching of the genitals, pressure on the bladder, etc. He further described a special testicle-reflex, a contraction of the abdominal muscles on the same side, from pressure upon the testicle.

turbances only, leaving the motor functions intact, is, as a rule, due simply to an injury of the nerve roots.

Conversely, we are not justified in assuming from symptoms of total interruption of conduction that there is a complete destruction of the cord, since simple compression of the spinal cord may entirely inhibit conduction. There is indeed no symptom which can justify the diagnosis of an irreparable lesion of the spinal cord (Walton). It is only when this interruption has persisted for a considerable time unaltered that it becomes probable that there is a more or less complete transverse lesion.

Spinal injuries at the level of the *cervical and lumbo-sacral enlargements* and of the *cauda equina* deserve special consideration.

Compression of the nerve roots at these sites causes marked symptoms. Affection of the *posterior* roots produces radiating pains, felt in the lines of the nerves of the extremities, hyperæsthesia, and usually anæsthesia in cutaneous areas corresponding to the affected roots. Involvement of the *anterior* roots is shown by atrophic paralysis of the muscles innervated by the affected spinal roots ; and symptoms of irritation—tremors, spasms, and persistent muscular hypertonicity—may be the result of compression of the anterior roots.

Injuries in the region of the cervical enlargement, which damage the cord to such a degree as to cause complete interruption of conduction, produce a total paralysis of the legs and of the muscles of the trunk, whilst those muscles in the arms which derive their motor fibres from the portions of the cord lying above the focus are usually spared. If the compression takes place, for instance, below the origin of the fifth and sixth cervical root, then amongst the arm muscles the deltoid, biceps, brachialis anticus, and the supinators are more or less intact, whilst the muscles of the fore-arm and hand are paralysed. In these non-paralysed muscles a spastic condition may appear—probably as the result of irritation—which causes a certain position of the arms : abduction and external rotation of the arm and flexion of the elbow (Thorburn, Oppenheim, Wagner-Stolper, Müller-Lerchenthal). The lower the site of the disease in the cervical cord, the greater is the number of muscles which escape paralysis, so that from the data given above as to the innervation by the various roots the different localisations can be ascertained. Partial paralysis of the arm muscles is usually degenerative (atrophic), since the anterior roots or their trophic centres are degenerated. The sensory disturbances show a condition analogous to the motor-paralysis. These always extend on the trunk as far up as the second intercostal space, whilst in the arm those areas which derive their roots from the cervical cord above the lesion retain their sensibility. If the lesion is situated at the level of the eighth cervical and first thoracic roots, then the anæsthesia is practically limited to the ulnar region of the hand and fingers, to the inner surface (ulnar side) of the fore- and upper-arm ; it extends further towards the radial side, the higher the site of the affection of the cervical cord. In injury at the level of the fifth and sixth cervical roots, it may be limited to the region of the circumflex nerve and to the external surface of the arm and fore-arm, if it is merely a root symptom and if the sensory conduction in the cord is not materially affected (see pp. 129 *et seq.*).

Oculo-motor symptoms are specially to be expected in a lesion of the first thoracic segment. Myosis and narrowing of the palpebral fissure have been observed even where the cord has been injured after fracture or

dislocation of the fourth to the sixth cervical vertebræ. Indeed, according to Kocher, they are to be expected in every severe injury which affects the cervical cord, above the first thoracic segment. Here the effect of the shock extending even to the lower sections of the grey matter in the cervical cord may play a part (*cf.*, however, p. 117). The loss of the light reflex of the pupil observed in exceptional cases (Brassert) are probably due to complications.

Vasomotor disturbances are frequently observed in injury of the thoracic cord. Kocher ascribes even priapism to this cause. Thorburn, to whom we owe excellent observations upon these conditions, has occasionally found alterations in the fundus of the eyes, in traumatic diseases of the cervical cord. Taylor and Collier have seen this also.

Kausch (*Mitth. aus Grenzgeb.*, vii.) mentions that in a case of complete destruction of the cord in the lower thoracic region, he found at the upper margin of the anæsthetic zone an area in which stimulation of the skin caused no local redness. He also found marked dilatation of the stomach in a lesion of the ninth and tenth thoracic segment.

Injury of the twelfth thoracic and first lumbar vertebræ may involve both the lumbo-sacral cord and the lumbar and sacral roots which here pass downwards and are applied to the cord on both sides. Contusion or compression of this segment tends to affect the cord more severely and persistently than the roots. The cord may show a condition of hæmorrhagic inflammation and softening, whilst the roots remain unaffected. Thus, after fracture of the first lumbar vertebra there may appear symptoms which are caused by *injury to the conus terminalis* (see corresponding chapter): paralysis of the bladder, rectum, genital regions, with anæsthesia in the region of the third and fourth sacral nerves and normal power of movement of the lower extremities, as I [1] have been able to show in a case with autopsy. If, however, the roots surrounding the conus are involved at the same time, there is also atrophic paralysis of the lower extremities and absence of the knee jerks.

In fracture dislocation between the tenth and eleventh, frequently also between the eleventh and twelfth thoracic vertebræ (sometimes also in fracture of the twelfth thoracic vertebra), the two highest segments of the lumbar enlargement are also injured, and there is therefore complete paralysis of the lumbar and sacral plexus (Kocher).

If the upper lumbar cord, *i.e.* the part from which the first to the third lumbar roots arise, is uninjured, which is the rule, for instance, in fracture of the twelfth thoracic vertebra, and also in dislocation between the eleventh and twelfth, then sensation in the region of the ileo-hypogastric and ileo-inguinal, etc., is retained, and the flexors of the hip-joint as well as the adductors may partially retain their function, whilst the other muscles of the lower extremities are paralysed and more or less completely atrophied. The integrity of the parts innervated from the upper segment of the lumbar cord is only to be expected if the compression has the affected cord alone and has left the roots intact. If the segment from which the third and fourth lumbar roots originate, and these roots themselves have remained intact, then the knee jerks are retained, as well as sensibility in the region of the obturator and crural nerves. The motor disturbance is limited in this case to the sacral and coccygeal plexus. In fracture of the first lumbar verebra, it may happen that the compression may affect the third

[1] *A. f. P.*, xx.

and following sacral segments, whilst the first and second escape (Oppenheim, Kocher); the anæsthesia then assumes the saddle-form distribution described on p. 134. The sensibility of the testicles is also conserved. The patient feels the desire to pass urine and fæces, but these functions are not under voluntary control. There may also be sexual desire and erections, whilst coitus cannot be performed.

Injuries of the third (or even the second) and the following lumbar vertebræ affect the cauda equina only. If this is totally compressed, the central parts, *i.e.* the lowest sacral roots and the coccygeal nerve, may in some cases be more severely injured than those in the periphery of the cauda, but this does not seem to be the rule. The lower down the affection of the cauda, the smaller is the number of the root fibres which it involves, and therefore the smaller the region to which the symptoms are limited.

Injuries of the *cauda equina* can hardly be distinguished from those of the conus, because the conus is surrounded by the roots of the cauda (see corresponding chapter on Diseases of the Cauda Equina, etc.). In lesions confined to the cauda, symptoms of irritation—radiating pains in the region of the sciatic and pudendal nerves, etc.—are present as a rule, whilst they do not appear when the affection is limited to the conus. Paralytic symptoms are usually less complete and less symmetrical in injuries of the cauda equina than in diseases of the lowest segments of the spinal cord. The course of the disease is more favourable in lesions of the cauda equina.

Differential Diagnosis.—Distortion, destruction of the *intervertebral discs* (Kocher) and *spinal concussion* have to be specially considered.

Concussion may simulate the symptoms of a severe injury to the cord, but these rapidly disappear. Kocher maintains that there is no such thing as spinal concussion, in the sense of a severe affection of the spinal cord unaccompanied by any definite lesion in it. There may be in such cases hæmorrhages, traumatic necrosis (Schmaus), or other injuries of the cord, or symptoms of concussion of the brain or psychoses. Page, Thorburn, and especially Stolper take a similar standpoint. Although it must be admitted that organic changes of this kind are often present where a simple concussion has been diagnosed, yet we have no reason to abandon the idea of spinal concussion altogether. Further, it has long been known that functional disturbances of *cerebral* origin occur after concussion of the spine (see chapter on Spinal Concussion and Traumatic Neuroses). On the other hand recent writers are inclined to assume an organic basis for cerebral concussion—fine pathological changes, such as Kalberlah[1] has lately described. Fickler[2] has recently studied the question exhaustively, and has come to the conclusion that concussion of the spinal cord, in the old sense—of purely molecular origin—is not admissible, that much more likely it is a case of minimal crushing of the cord upon the bones and of oscillations of the axo-plasm in the nerve fibres, the result of the jarring movement of the spine, and that these mechanical processes cause the transient functional disturbances. Hartmann also distinctly shows that we can draw no certain conclusion from the clinical picture of a traumatic spinal cord affection as to the pathological nature of the process.

Spinal injuries may also give rise to the development of a *tubercular* and a *simple spondylitis*. Kümmel[3] has recently drawn attention to cases in which, after a considerable time simple (not suppurative) spondy-

[1] *A. f. P.*, xxxviii. [2] *Z. f. N.*, xxix. [3] *D. m. W.*, 1895.

litis developed, and gave rise to pain, stiffness, and deformity, as a rule not very marked. Symptoms of compression also occur. Cases of this kind have been published by Heidenhain, Schulz, Lissauer, De Ahna, and others. According to Henle they are caused by softening of the bones (traumatic spondylomalacia). Schede also states that after fracture of a vertebra, the affected portions of bone may undergo softening, and that this may lead after a long interval to visible deformity and to compression of the cord. Reuter gives another explanation. Kocher, Trendelenburg, Oberst, E. Fraenkel, and other surgeons, however, ascribe the symptoms observed by Kümmel to compression-fracture of the vertebral body, and Kümmel himself is said to adhere to this view. Nonne (as well as Verhoogen) shows that a similar picture may be simulated by muscular contracture in functional neuroses. We should not forget that the signs of a spinal cord disease may develop after injury, as the result of hæmatomyelia and meningeal hæmorrhage, even when the spinal column is uninjured. The fact that a simple contusion may be the cause of a severe spinal disease, by means of "traumatic necrosis" or softening, has received less attention (Schmaus, F. Hartmann, Fickler). Not a few cases, however, have been described (for instance by Wagner-Stolper, A. Westphal, Jolly, Spiller, Hartmann, Raymond-Cestan, Lohrisch, Schäfer, Nonne, and others) in which a diffuse transverse disease of the cord has followed injury, and the spinal column has shown no evidence of solution of continuity. Evidently, therefore, it cannot always be denied that trauma may have directly caused displacement and momentary compression.

Radiography may be of the greatest value in the diagnosis of spinal injuries, as by its means a compression fracture has sometimes been recognised even in the absence of any deformity (Sudeck-Nonne). Stempel, for instance (*M. f. U.*, 1904), has shown the forensic importance of this question. This method may also be employed to distinguish fracture from dislocation (Kienböck).

The prognosis in all cases of severe injury to the spinal column is very grave. Life is specially endangered in fractures and dislocations of the cervical vertebræ. Total lesion of any of the upper four cervical segments always causes sudden death, and one of the lower segments has almost always a fatal issue. Out of 150 cases of spinal fracture collected by Courtney, fifty affected the cervical vertebræ, and in all these the disease ran a rapidly fatal course. An observation of Lloyd, however, shows that even dislocation of the atlas need not necessarily be fatal. Steinmann also has shown that the spinal cord is not always severely injured in such cases. When death does not at once supervene, paralysis of the bladder and intestine, cystitis and bedsores, constitute the chief danger. As to dislocation, the prognosis is in general rather more favourable, since compression of the cord is sometimes absent or incomplete, and artificial support is often of more service than in fracture. The direct danger to life is least in injuries of the lumbar vertebræ. The prognosis is *ceteris paribus* the more favourable the less the cord and roots are affected. Recovery follows rapidly, within a few days or weeks, or not at all. According to statistics by Gurlt, spontaneous improvement is not to be expected if the paralysis of the bladder and intestine does not improve before the end of eight to nine weeks.

Treatment.—Great care whilst the patient is being examined, removed,

and put to bed, and avoidance of all unnecessary manipulation of the injured parts are the most important points to be attended to. The patient should be so placed that the injured part is well supported and fixed, all active movement being avoided, as it might result, even a considerable time after the accident, in further displacement.

Attempts to reduce the dislocation have sometimes had wonderful immediate results. As, however, they are not without danger, they should only be undertaken when signs of compression of the cord are present. As to the methods to be used, the text-books on surgery should be consulted. In fractures of the cord, no attempt whatever at reduction should be made.

Treatment is otherwise limited to measures for prevention of the development of bedsores. The patient should be laid on a water-bed, which, however, should not be so large that the body is moved about upon it; the heels and trochanters should be protected by small indiarubber rings, pads of wadding, etc. The greatest cleanliness in the use of the catheter is of course requisite. Kocher recommends prolonged drainage of the bladder, allowing the urine to flow by means of Nelaton's apparatus into a glass filled with antiseptic fluid and placed at a lower level. He considers the emptying of the urine by expression of the bladder (after Wagner) to be very dangerous when the lesion lies above the lumbar region. It is advisable during the first few days to give opiates, in order to prevent all movement of the bowels, as this necessarily involves movement and disturbance of the patient.

Even in early times, but especially within the last two or three decades, Macewen, Brown-Séquard, Chipault, Horsley, and others, have made numerous attempts to effect a cure by means of operation, by direct opening of the spinal canal, by removal of the arches (laminectomy), removal of the fragments, etc. Unfortunately, the results have hitherto been very discouraging. Out of 167 cases with operation, which Chipault has collected from the literature, only twelve were cured and twenty-four improved. Schede, however, draws attention to the fact that in cases with operation since reported a higher percentage of recovery, and especially of improvement, has been attained (Macewen, Lauenstein, Schede, Thorburn, Lücke, Sick, Munro, Hinsdale, Welford, Quercioli, and others). Chipault's later statistics, founded on 140 observations of his own, do not make this clear, but it should be noted that in two old cases improvement took place after resection of the callus which had penetrated into the spinal canal. Out of sixty-four cases of laminectomy, which F. Hahn [1] collected from the literature, nineteen resulted in recovery or substantial improvement, twelve showed slight improvement, whilst eight were treated without result, and twenty-five ended fatally. Munro [2] mentions that out of thirty cases of injury of the cervical and upper thoracic spinal column, in which there was no operation, only one survived, while three completely recovered after laminectony. There is great difference of opinion as to the indications for operation and the time at which it should be performed. The majority of writers are against early operation, since, immediately after the injury, it cannot be determined with any certainty how far the symptoms are caused by compression, how far by intramedullary hæmorrhage, and especially how far they are due to a simple concussion. Even at a later period this factor has such a

[1] Résumé in *C. f. Grenzgeb.*, 1898.

[2] *Journ. Amer. Med. Assoc.*, 1904.

confusing influence on the data upon which we would base our indications that it deprives us of any definite criterion by which to decide whether such symptoms of motor and sensory paralysis as are present are due to a compression that still continues or to one that has passed away. There is no doubt that operative measures are out of place in a complete transverse destruction of the cord at any level. Where, therefore, the signs of complete interruption of conduction do not pass away after an interval of several weeks, an operation is not advisable, even although there are a few cases in which simple compression has produced similar symptoms without causing any organic damage to the cord.

The statements of Stewart and Harte, which they base upon the results of suture of the spinal cord in its total transverse destruction in man, must be taken with the greatest scepticism; as well as those of Fowler (*Ann. of Surgery*, 1906), of Haynes (*New York Med. Journ.*, 1906), and of Shirres (*Lancet*, 1905), who claims to have caused a regeneration of fibres by transplantation from the spinal cord of a dog.

On the whole, operation is only suitable in cases which show signs of *incomplete* interruption of conduction. But as in such cases spontaneous improvement is frequently considerable under expectant and conservative treatment, it is usually advisable, even here, not to decide too soon upon operation, but only when no improvement becomes apparent, or the condition becomes aggravated. It is quite possible that improvement or recovery may be brought about by surgical removal of a fragment, of a cicatricial growth, etc. Lauenstein recommends operation if the sphincter paralysis does not disappear after eight or nine weeks. Schede thinks this too late in many cases. Walton and Horsley declare themselves in favour of early intervention. The general view is that operation should not take place before the sixth week or after the third month.

In fractures of the laminæ (which are of rare occurrence) early laminectomy may be advisable. We may conclude from the data accessible to us that surgical operations give most satisfactory results in injuries of the cauda equina, as the nerve fibres affected are capable of regeneration (Thorburn). Even in irreducible dislocations, associated with merely partial lesion of the cord, operation is advisable.

A communication by Krauss makes it clear that a fragment of bone which has penetrated into the cord may escape observation even during surgical treatment.

Goldschneider,[1] after a review of the existing data, comes to the following conclusions: (1) In early cases all operative intervention is contra-indicated. Those cases are exceptions in which there is a comminuted fracture of the arch, with obvious penetration of fragments into the spinal cord (which does not often occur on account of the thickness of the posterior longitudinal ligaments). (2) If the paralysis does not improve, and if there is a deformity which indicates a fracture of the arch, then operation may be advisable. (3) Surgical intervention is most hopeful, and is most clearly indicated in fracture of the lower lumbar vertebræ. (4) Hæmorrhagic exudation into the spinal canal is not an indication for laminectomy.

Schede and Chipault, in view of the evidence of the unfortunate results of expectant treatment, do not draw the limits of surgical treatment so

[1] *D. m. W.*, 1894.

narrow as Goldscheider. Horsley especially gives it a much wider range.

In any case it is apparent from this discussion that we are still groping our way in a condition of uncertainty.

Appendix

The symptoms which appear after injury of the spinal column by shooting or stabbing, study of which has hitherto been practically left to the surgeon, resemble in many points the conditions following on fracture and dislocation described above, with this difference, that as a rule the injury to the cord is usually only partial and unilateral. Thus it is that Brown-Séquard paralysis (*q.v.*) plays a great part in these cases. On the other hand, the symptoms of compression of the cord produced by gunshot so closely resemble those in extra-medullary tumour, that we shall return to the question when dealing with the latter.

A more detailed consideration of the conditions cannot be entered upon in this text-book, but references are given to communications and contributions to this question which have appeared recently and may serve for further information, namely, those of Schmidt, Rumpf (*D. m. W.*, 1903), Perthes (*D. m. W.*, 1904), Wilde, Sforza (*R. n.*, 1904), Engelmann (*M. m. W.*, 1904), Peugnier-Philippe (*Arch. d. Neurol.*, 1903), Weisenburg (*Amer. Journ. Med. Sciences*, 1904), Oddo (*Marseille médical*, 1904), Federmann (*D. m. W.*, 1905), Amberger (*Bruns Beitr.*, Bd. xlviii.), Couteaud (*Gaz. des hôp.*, 1905), Faure (*R. n.*, 1906), the particularly interesting case of Raymond and Rose (*R. n.*, 1906), and the review by Strauch (*Viertelj. f. gerichtl. Med. xxvii Suppl.*), G. Flatau ("Die Stichverletzungen des Rückenmarks," *C. f. Gr.*, 1905), Rosenstein (*Inaug.-Diss.*, Berlin, 1906), Krajewski (ref. *N. C.*, 1907).

A notable case, which I had the opportunity of observing along with Roth and F. Krause, has recently been published by the latter (*B. k. W.*, 1906). It shows that paralytic symptoms may appear or may be aggravated a considerable time after the gunshot wound by necrosis of the bone, suppuration, and serous meningitis.

The prognosis depends to a certain extent on the site of the injury, but especially on its severity and the possibility of infection of the wound.

For treatment, which is mainly surgical, surgical text and handbooks should be consulted. It is well known that X-ray photography is of great service in revealing bullets and other foreign bodies.

Spinal Caries, Spondylitis Tuberculosa (and other Forms of Spondylitis)

Spinal caries is almost always tuberculous in nature, but a simple spondylitis is sometimes met with, *e.g.* after injury. The disease usually starts in the bodies of the vertebræ, less frequently in the joints and ligaments, and still less frequently in the vertebral arches and their processes. Tuberculous osteitis of the body of a vertebra arises as a localised development of soft spongy granulation tissue which leads to a softening of the bone and the formation of caseous or purulent material. This process is generally limited to one vertebra or one joint, and only rarely does it attack several neighbouring vertebræ or vertebræ remote from each other. Cure may occur, new bone being formed at the site of that which has been destroyed by a process of ossifying osteitis. If the disease advances to the stage of resorption or necrosis of the body of a vertebra with consequent collapse and falling together of the neighbouring vertebræ, or if the soft caseous purulent material of the abscess presses backwards towards the spinal canal, then the spinal cord is endangered and in the majority of cases is actually implicated.

Injury to the cord from direct bony pressure by a vertebra is of rare occurrence. It does, however, occur in cases where there is a sudden

giving way in a diseased vertebral column, as when, through some injury, a carious vertebra suddenly collapses, leading to an immediate and definite dislocation. Moreover, compression of the cord may occur from a projecting edge of bone or from a sequestrum without actual displacement of the vertebra. The latter point has of late been emphasised by König, Long, Guibal, Taylor, and others, and has been observed by the author.

Usually the narrowing of the spinal canal is of slow occurrence and is brought about not by the vertebræ themselves but by caseous material or less frequently by purulent matter. This in the first place, as it presses against the dura mater, lights up in its outer layers a chronic inflammatory process which may extend far beyond the confines of the diseased vertebra (pachymeningitis externa, epidural tuberculosis); eventually, however, it bulges into the canal so far that it comes to press on the cord and the nerve-roots.

In such a case the dura is often three to four times its normal thickness; rupture of the dura is, however, quite the exception.

The compression and obstruction of the blood and lymph vessels in the spinal membranes produces in the corresponding segment of the cord an œdema which may exist for a long period without producing any appreciable alteration in the nerve elements. Possibly the œdema may depend to some extent on an irritative factor, and may be in fact a collateral inflammatory œdema, the result of toxic action (Schmaus). In consequence of the œdema the nerve fibres first become thickened (swelling of both axis cylinders and medullary sheaths); sooner or later there follows destruction of the nerve elements and softening (myelin globules, granular cells, etc.), and eventually a sclerosis or a true inflammatory process, *i.e.* an interstitial transverse myelitis. It is, however, an undoubted fact that œdema may remain for a long time the only change in the cord. Only in a small number of cases does a myelitis exist from the outset. When it does so it is usually tuberculous in nature and is the result of a direct spread of the tuberculous process from the affected meninges. The tubercular process may by attacking the vessels, and more especially the arteria sulci, extend thus to the substance of the cord, or by setting up an obliterative arteritis produce softening (Schmaus [1]). Serous meningitis may in rare cases play a part in the compression of the cord (Oppenheim [2]).

The œdema, softening, or myelitis produced as above described, are limited to a localised portion of the cord of about $\frac{1}{4}$ to $\frac{1}{2}$ inch or more, *i.e.* to one or less frequently to several segments of the cord.

Frequently smaller scattered foci of softening are found round the main focus; usually they are close to the main focus, but rarely are they found at some distance from it.

In paralytic cases of some standing one finds in addition the signs of secondary degeneration. At the seat of pressure the cord may be swollen from œdema, but usually it is compressed and looks constricted and diminished in size.

Sometimes this diminution in size from pressure is a striking feature. Occasionally one may find patches of disseminated myelitis, and these are

[1] "Die Kompressionsmyelitis bei Caries," Wiesbaden, 1890; further, Schmaus-Sacki, "Pathol. d. Rückenmarks," Lubarsch-Ostertag, 1898, and "Vorles. über d. path. Anat. d. Rückenm.," 1901.

[2] *Mitt. aus. d. Grenzgeb.*, xv.

sometimes of independent tuberculous origin and unassociated with pressure.

The nerve-roots usually participate in the morbid changes, being subject to pressure in the spinal canal or in their passage through the intervertebral foraminæ with consequent inflammation or atrophy.

Of the recent papers on this subject those of Von Fickler (*Z. f. N.*, xvi.), Ménard ("Étude sur le Mal de Pott," 1900), and Alquier (*Nouvelle Icon.*, xix.), are especially noteworthy.

This disease may attack any part of the spinal column. In the experience of some authors (Rey, etc.) the lumbar vertebræ are those most frequently affected, whilst other writers consider that the dorsal region is by far the commonest site of caries. This diversity of clinical experience is, however, readily explained by the fact that caries dorsalis affects the spinal cord more frequently than lumbar caries, and consequently comes more frequently before the physician and neurologist.

Tuberculosis or scrofula is the cause of this disease. The spinal caries may be the only apparent manifestation of this constitutional disease, but certainly this point has been established only in a few cases, *e.g.* those of Schmaus and Westphal. Generally it is found along with other evidences of active or healed tuberculosis or scrofula, such as caries of other bones and joints, the scars of former suppurating glands, phthisis, etc.

Children are most frequently affected, but there is no age limit for this disease. Even in infancy it is by no means rare (Froelich). It may develop spontaneously; frequently a trauma, such as a fall or blow on the back or the lifting of a heavy weight, may act as the exciting cause.

Acute osteo-myelitis of the vertebræ is rare, but it also may secondarily affect the cord by compression.

A simple traumatic spondylitis may also occur; Kümmel has laid stress on its occurrence and the symptoms associated with it (*cf.* the foregoing chapter).

Further Quincke has described a "Spondylitis typhosa," an inflammatory condition of the spine occurring during or after an attack of typhoid fever (*Mitt. aus d. Grenzgeb.*, iv.). Since then Schanz, Könitzer, Freund, Neisser, and Taylor have reported cases of this nature. Nonne once diagnosed a spondylitis and pneumonia. The rare syphylitic affections of the vertebræ will be described in a separate chapter. Very exceptionally actinomycosis extends, *e.g.* from the lungs to the vertebral column (Martens).

Symptoms.—Fever may be present but is by no means a constant symptom. The general condition of the patient is not always affected, but in longstanding cases and when general scrofula is present loss of strength is a constant feature.

Besides these constitutional symptoms one finds signs which are the direct results of the vertebral disease, and in many cases though not in the majority (about 30 per cent., in caries dorsalis, however, up to 80 per cent.) the symptoms of disease of the spinal cord.

The vertebral disease betrays itself first by pain which is felt in the region of the affected vertebra, and which is aggravated by movements, and usually also when pressure is applied. The pain may be very intense, and causes the patient to shun all movement of the diseased part, and to hold it stiff. When he wishes to pick something off the ground, instead of bending his back, he kneels down, keeping his body erect. In cases of caries of the cervical vertebræ stiffness of the neck is the earliest symptom.

Tenderness on pressure over the affected vertebra is a symptom of very uncertain value. In dealing with nervous hysterical patients one frequently finds that pressure on the spinous processes of vertebræ elicits pain. In the latter, however, tenderness can usually be elicited over several vertebræ or even over the whole vertebral column, and frequently a light stroking of the skin over the vertebræ is sufficient to cause pain. These facts, together with the dependence of the symptoms on the watchfulness of the patient and the other psychical phenomena, allow of a definite diagnosis.

In cases of caries the tenderness on pressure is limited to one or to two neighbouring spinous processes (rarely to two processes at any distance from each other); it is always the same spinous process, and pain is elicited particularly by deep pressure, by percussion, or by attempts to jolt or to displace the spinous process. In the cervical region where the transverse processes are accessible, pressure on these produces pain.

It is, moreover, of value to pass a sponge wrung out of hot water or the cathode of the galvanic current over the skin along the line of the vertebral spines.

Pain is produced the moment the hot sponge or cathode touches the skin over the affected vertebra. These methods are, however, less certain than the others. Another diagnostic test which appears to me to be not without danger is as follows: The patient sits on a chair, the examiner places his hands on the patient's shoulder and attempts by applying considerable force to compress the vertebral column. This test may elicit great pain (in the back or round the thorax).

The most important symptom of the spinal disease is the deformity, the angular kyphosis, or Potts' curvature, which—if trauma be excluded—is almost pathognomonic of spondylitis. As usually only one vertebra is diseased, a localised curvature occurs when it collapses or becomes displaced, and the spinous processes of one or two (seldom more) neighbouring vertebræ project prominently; they may in addition be laterally displaced. This deformity occurs most markedly in caries of the dorsal vertebræ, still it is by no means an invariable sign and may be absent through the whole course of the disease.[1] Its absence is particularly noteworthy when the carious process is limited to a vertebral arch, in which case a depression may form and the diseased focus may sometimes, though seldom, be palpable (crepitation).

The vertebral disease frequently discloses itself by some secondary lesion, *e.g.* a burrowing abscess. The pus which forms at the tuberculous foci usually presses inwards towards the spinal canal; it may, however, find its way outwards and make an appearance at various places, such as the neck or back, reaching even the subcutaneous tissues, though the latter is of rare occurrence.

In caries of the upper cervical vertebræ the pus frequently burrows between the posterior pharyngeal wall and the spine (retropharyngeal abscess); in caries of the lower cervical or of the dorsal vertebræ it may burrow in the mediastinum and thence burst into some thoracic organ; usually, however, it gravitates further down, and following the line of the psoas and the great vessels, reaches the groin and appears in the region of

[1] Among twenty cases of this disease in adults, nine showed no spinal curvature (Touche). This type was also discussed in a thesis by Moussaud, Paris, 1906.

Poupart's ligament, or in the front, or occasionally in the back of the thigh as a "gravitation abscess."

Only rarely does it burrow to the surface at a higher level than the primary focus, as occurred in a case of Joachimsthal's.

The view held by several authors that a gravitation abscess occurs in 25 per cent. of cases does not tally with my experience, for I have not found this complication to be of such frequent occurrence.

The involvement of the nervous elements in the spinal canal is indicated by *root symptoms* and *cord symptoms*. The former usually precede the latter but both may appear simultaneously or the cord symptoms may be first. In caries of the dorsal vertebræ as a rule the pressure symptoms refer only to the posterior roots; thus we find pain which is referred round the chest or abdomen in the form of a girdle or half girdle, corresponding to the distribution of a pair or of one intercostal nerve. These pains may exist for a long time before any objective sensory disturbance is demonstrable in the affected nerve zone. Hyperæsthesia may be the first sensory disturbance, but usually hyperæsthesia or anæsthesia is the first alteration that can be made out. In a few cases one may find herpes zoster or œdema in the affected nerve area.

When the spondylitis affects the lower dorsal vertebræ or involves the dorsal roots viii.-xii., then diagnostic significance may be laid on the state of the abdominal reflexes and on any evidence of paralysis of the abdominal muscles of one or both sides.

In caries of the cervical or lumbar and lowest dorsal vertebræ, the root symptoms are of much more moment. When the vertebræ in relation to the cervical enlargement of the cord are affected, the roots of the nerves forming the brachial plexus may be involved, either in the spinal canal or in their passage through the intervertebral foraminæ, with resultant irritative or paralytic phenomena in their area of distribution.

When the eighth cervical and first dorsal roots are involved the root symptoms consist of pain and sensory disturbance in the ulnar nerve area, atrophic paralysis of the small muscles of the hand, etc., and oculo-pupillary symptoms (usually contraction of the pupil and of the palpebral fissure).

The latter symptom is more frequently absent when compression affects the cord than when it involves the nerve roots (Krausz).

When the disease is situated higher up involving say the fifth and sixth cervical roots, we find atrophic paralysis of the deltoid, biceps, brach. ant., supinator long., etc., and anæsthesia in the region over the deltoid and on the outer surface of the upper arm and forearm. Sometimes some of the muscles supplied by the musculo-spiral nerve partake in the paralysis. The root symptoms, particularly the atrophic paralysis confined to individual muscles of the upper extremity, may be the first evidence of cervical caries. The symptoms associated with involvement of the spinal cord vary according to the level where the compression takes place. In the commonst form, dorsal caries, if the conductivity of the cord be almost completely interrupted, we get the following conditions :—

1. Paraplegia with rigidity and exaggerated tendon reflexes. The Babinski sign and the Oppenheim phenomenon can usually be elicited.

2. Anæsthesia corresponding to the nerve roots arising from the cord

up to the seat of pressure. At the upper limit of the anæsthetic or hypoæsthetic region, there is a zone of hyperæsthesia.

3. Girdle sensation.

4. The skin reflexes in the lower extremities are present and may be greatly exaggerated, even a breath of air or a light touch on the sole of the foot may be sufficient to excite strong reflex twitchings.

5. Disturbances of the functions of bladder and rectum.

In addition trophic phenomena often appear in the form of bedsores over the sacrum, the trochanters, and other points of pressure.[1] On the other hand the muscles remain in good condition and react normally to electrical stimuli.

As was already described on pp. 115 *et seq.*, one may find under certain conditions, even in cervical and dorsal caries, muscular atony and absence of reflexes instead of or succeeding the spastic paralysis. If the caries involve the eleventh or twelfth dorsal or first lumbar vertebræ, and if the compression arrest the conductivity or produce a transverse area of disease in the lumbar enlargement or lumbo-sacral cord, then the clinical picture is different, for instead of a spastic we find from the first a flaccid atrophic paralysis of the lower limbs, and the reflexes, instead of being exaggerated, are weakened or are altogether absent. I have, however, seen one or two cases where compression in the region corresponding to the peroneal muscles produced a degenerative paralysis of these muscles, whilst the tone of the calf muscles was so increased that a definite foot clonus could be elicited. The lower the level of compression of the cord the larger is the area of the lower extremities which escapes interference with its innervation, till finally, when the disease affects the conus terminalis, the symptoms are confined to the area of distribution of the sacral nerves (third sacral *et seq.*), always provided that the roots are not involved. Lately, Alquier has reported cases of this kind.

Carious disease of the sacrum, especially of the sacro-iliac joint, does not affect the cord, but the cauda equina. This disease is well known to surgeons (Hahn, Ollier, Delbet ,Sayre, Wolff, and others have worked at this subject). The nervous phenomena have only lately, however, attracted much attention (Naz, Cestan-Borbonneix, and more especially Bartels). Recently, Rossi has recorded an interesting observation (*Arch. d. Neurol.*, 1905). The symptomatology is described in the section devoted to disease of the cauda.

In caries of the lower cervical vertebræ, disease of the cord manifests itself by symptoms which differ from those described for dorsal caries in that one finds along with spastic paralysis of the lower limbs an atrophic paralysis of the upper extremities. This depends not only on an affection of the nerve roots, but also on an involvement of the cord, and in particular the grey matter of the cervical enlargement. Moreover the paralysis extends to the thoracic and abdominal muscles, and leads to respiratory difficulties, especially to weakness of the muscles of expiration, which may, if bronchial catarrh exist, have serious consequences.

When the cord is compressed above the cervical enlargement spastic paralysis of all four extremities usually results; it may, however, for some time be limited to the arms or, less frequently, the legs. The sensory disturbance has a corresponding distribution. The atrophy may then, as happened in a case of my own, be limited to the muscles supplied

[1] Chipault has drawn attention to the occurrence of arthropathies, but they are certainly very rare.

by the spinal accessory. When the phrenic nerve or its region of origin is involved, inspiratory dyspnœa results. A. Westphal demonstated, in such a case, the loss of electrical irritability in these nerves.

In the above description of the cord symptoms we have taken for granted that the compression caused a more or less complete interruption of conductivity. This is, however, by no means always the case. Usually the interruption of conductivity is incomplete, and the symptoms are likewise imperfectly developed. The motor disturbances are almost always more pronounced than are the sensory ones, indeed the latter may for long be absent or be very slight. A single sensation or all sensations may be affected. Fickler always found hypæsthesia for tactile and thermal impulses, whilst involvement of sensibility to pain was less common, and the sense of position was still more rarely interfered with. Partial sensory paralysis in the shape of analgesia and thermanæsthesia occur occasionally however (Minor, Oppenheim). The bladder troubles may be slight, they usually appear late, but it is exceptional for the disease to run its whole course without some vesical symptom. That the sphincteric and genital functions may remain intact, even in a severe case with paraplegia and anæsthesia, is proved by that recorded by Schilling.[1] When the disease is situated in the cervical or dorsal region, the constant symptom is therefore spastic paresis; should this advance to total paralysis, then the other symptoms of a breach of conductivity, namely anæsthesia and sphincter paralysis, are seldom wanting.

The sequence of events in, and the spinal symptoms exhibited by a case where a slowly advancing compression of the cord is taking place, likewise the course of a case where recovery is taking place, have been accurately described by Collier (*Br.*, 1904); in its main points his description tallies with my own experience.

The Brown-Séquard syndrome is rarely seen in caries; I have seen it several times however, in one case in particular where the bony displacement narrowed the spinal canal in such a manner that it led to a large accumulation of cerebro-spinal fluid above the site of compression of the cord. In this case the spastic paralysis of the limb of the same side preceded the development of the anæsthesia of the opposite limb by a considerable time—similar observations are recorded by Luce and by Alquier.

Caries of the uppermost cervical vertebræ and of the atlanto-occipital joint, the malum sub-occipitale, merits special description. The caries may involve the atlas, the odontoid process, the condyloid processes of the occipital bone, or the whole region round the foramen magnum; single parts, such as the odontoid process, may be undermined, loosened, and knocked off, and the separated odontoid may press directly on the medulla oblongata or be pushed far forwards and upwards, etc. The first symptom is usually pain in the head or back of the neck. Vertigo, and even nystagmus sometimes occur (Bergmann [2]). There is also stiffness of the back of the neck added to the above. The head is kept fixed in a certain position and all rotatory movements (in disease of the odontoid or the joint between atlas and axis), and all nodding of the head—in caries of the atlanto-occipital joint—are avoided. Such patients nod, and rotate not the head but the whole body; they support the head with their hands, or pull it up by the hair, when rising from the recumbent position. Also

[1] *A. f. kl. M.*, Bd. lxxxiv. [2] Volkmann's "Samml. klin. Vortr.," I. N. F. 1.

a marked resistance is exhibited to all passive movements, which are very painful. Occasionally crepitation may be present: a light blow on the head or on the sole of the foot elicits pain. The most important and usually the earliest root symptom is unilateral or more frequently bilateral occipital neuralgia. Later on, anæsthesia develops in the area supplied by the occipital or upper cervical nerves. In addition paralytic phenomena not unfrequently develop, and are referable to disease of the spinal accessory or hypoglossal nerves; thus, I have observed unilateral atrophy of the tongue in two cases of this nature. Dercoly records a case with bilateral atrophy of the tongue. Finally, the cord itself is compressed, and according to whether the medulla oblongata or the upper cervical region of the cord be affected one finds the symptoms of bulbar paralysis (especially difficulties of respiration and deglutition) or of a myelitis cervicalis superior. In the latter case usually first the arms and then the legs become paralysed, but the converse order has also been observed. If the observations of Flatau, described on p. 105, be correct, one must accept the statement that in a case of compression the central parts of the cord usually suffer earlier than do the peripheral parts, and this view is supported by several actual observations. Sensory paralysis of corresponding distribution also appears, etc. At any moment death may suddenly occur from compression of the medulla oblongata by a separated fragment of bone (particularly the odontoid process) or by the spongy caseous debris, or a retropharyngeal abscess suddenly bursting inwards may obstruct the air passages.

The diagnosis presents no difficulty if the signs of the vertebral disease be evident. Should deformity be absent, then the diagnosis must be made from the presence of tenderness over a certain vertebra, from the cautious carriage of the trunk or head, or from the root and cord symptoms.

In some cases of caries of the upper cervical vertebræ I have succeeded by means of the Röntgen rays in demonstrating the vertebral destruction or displacement, and this method has frequently been successfully employed by others (Kümmel, Leyden-Grünmach, Sick, Sudeck-Nonne, Redard). Very often, however, has it failed me (*cf.* Fürnrohr, "Die Röntgenstrahlen im Dienste der Neurologie," Berlin, 1906). The secondary osteomalacia or rarefying osteitis found in cases of primary myelitis may simulate a caries with compression, as was apparently shown by an observation of Raymond-Alquier.[1] When no sign of a vertebral lesion is present the following points may help to a diagnosis—the youthful age, tuberculosis of other organs, fluctuations of temperature, symptoms of a slowly increasing compression of the cord and the nerve roots. In any case, it is necessary, when the symptoms of spastic paresis, with great exaggeration of the reflexes, and girdle pain, etc., slowly develop, to think of vertebral caries.

The use of tuberculin-injection to verify the diagnosis is so uncertain in its results that one cannot recommend it. The recent results obtained with the cutaneous and ophthalmic reactions Pirquet, Calmette, Wolff-Eisner, and others) appear to establish its use as of diagnostic value, still, at least in the case of the ophthalmic reaction, its use is not free from danger. Much investigation is still required to determine what diagnostic value may be laid on the results of lumbar puncture (*cf.* the remarks relative to this in the chapter on Meningitis Cerebralis). In a case described by Léri and Catola lumbar puncture decided in favour of a meningeal neoplasm, whereas the symptoms had indicated caries. An observation of Raymond-Sicard (ref. *C. f. Gr.*, 1906) is of interest in this connection.

[1] *R. n.*, 1906.

In caries of the vertebral arches kyphosis is absent or is but slight; another evidence of disease in this situation is the formation of a gravitation abscess in the back round the vertebral spines (Wieting), and, moreover, the caseous focus may sometimes itself be palpable. According to Wieting's statistics it occurred nine times in seventy cases.

When deformity is absent tuberculous myelitis or meningo-myelitis may be mistaken for this disease. In a case of solitary tubercle of the cord the vertebral symptoms are absent. Oberndörfer[1] refers to several other criteria.

Simon and Schultze have described a so-called granular celled myelitis of tuberculous nature. Observations by Oppenheim (*B. k. W.*, 1891), Raymond,[2] Goldscheider, Dupré-Delamare, Philippe-Cestan, Oddo-Olmer, Marie, Ransohoff, Dupré-Hauser,[3] Dana-Hunt, Clément (*Lyon méd.*, 1905), and others have shown that in tubercular subjects a myelitis may develop, and that on the other hand tuberculous affections of the spinal membranes may occur, having no connection with bone disease. Henneberg and Rossi (*Arch. d. Neurol.*, 1906) describe softening of the cord caused by epidural granulations, the bones being normal (perhaps healed ?).

It must be remembered that a gravitation abscess may produce paralytic symptoms from pressure on a plexus. I have seen an Erb's paralysis produced in this manner in a case of cervical caries; Engelken records a similar observation.

It not infrequently happens that caries is mistaken for some other vertebral disease, *e.g.* for tumour of the spinal column, and I have on one occasion mistaken for caries a renal sarcoma invading the vertebral column. In the latter type of case the pain is as a rule much more severe, and spinal curvature is usually a later development. Primary sarcoma, however, may cause no definite symptoms.

Aortic aneurysm also may, after evading the vertebral bodies, lead to a syndrome which closely simulated that of caries, as happened in a case recorded by Burr.

In this connection, an observation of Bálint and Benedict, where an aneurysm of the int. iliac artery had eroded the sacrum, is worthy of mention (*Z. f. N.*, xxx.).

After a spinal trauma a simple spondylitis or bone softening may develop, the symptoms of which, *e.g.* kyphosis, pain, compression effects, may not appear till after a clear interval of months or even one to two years (Kümmel, Henle[4]). Usually, in such a case, arrest or cure of the disease occurs. Lately, Kümmel's teaching, that as rule a fracture may be demonstrated as the cause of the deformity, has lost ground.

Caries may be confused with gliosis. In the latter the Potts' curvature is certainly wanting, but if the start of the trouble dates back to childhood, and if to the localised deformity secondary curvatures have been added, the apparently diffuse kyphosis or kyphoscoliosis may simulate that found in gliosis. Moreover in a case into which this point did not enter, the difficulties of differential diagnosis were so great that one of the ablest clinicians in this department diagnosed it as gliosis, and I on the other hand diagnosed caries of the upper cervical vertebræ with compression. The further course of the case decided in favour of my view, the patient recovering under treatment with extension.

Spiller and likewise Alquier-Lhermitte (*R. n.*, 1906) have recorded observations of a similar nature.

In childhood confusion may arise between caries and dystrophy.

I have seen cases of caries in which at the outset the diagnosis neurasthenia or hysteria was given. The patient, though on testing for tenderness his answers were indefinite and vacillating, complained of pain in

[1] *M. m. W.*, 1904.
[2] *Revue de Méd.*, 1896.
[3] *R. n.*, 1903 (see Bibliography).
[4] *Arch. f. kl. Chir.*, Bd., lii.

the back and of weakness in the legs; the latter was not pronounced, and was accompanied by exaggeration of the tendon reflexes (the value of the Babinski sign was then unknown); in other respects the malady had a distinctly hypochondriacal aspect. In a case of this kind the following facts weighed with me: the patient instinctively held his back stiff even when his attention was distracted; further, in spite of markedly exaggerated reflexes and weakness in the legs, the arms were not affected, which fact did not come into line with the diagnosis of neurasthenia—in which the general increase of reflex irritability is generally demonstrable in all situations. Since it had been preceded by a suspected lung disease, I diagnosed caries dorsalis, and this was confirmed by the subsequent course of the disease. Conversely, I have seen a case of hysterical stiffness of the back of the neck in which caries was diagnosed, and in which Glisson's apparatus had been applied for many weeks.

I have pointed out[1] that there is a congenital (sometimes hereditary and familial) form of kypho-scoliosis which forms a stigma of degeneration. In the past few years these congenital forms of scoliosis have elsewhere received attention (*s.* Nau, *Thèse de Paris*, 1904). Should, as frequently happens, hysteria or neurasthenia later develop in such a case, one may suspect caries. The nature of the deformity should protect one from this mistake, however. The intercostal neuralgias which sometimes occur in kypho-scoliosis of high degrees, should not, considering the character of the deformity, lead to errors in diagnosis.

Caries of the upper cervical vertebræ may, on superficial examination, be confused with torticollis.

Osteomyelitis vertebralis, we know, from the observations of Chipault,[2] König, Riese, Ferrio, Wiesinger, Schönwerth, Weber, Labeyrie,[3] and others, is an affection which arises and runs its course acutely, with high fever and severe constitutional disturbances. Boys are chiefly affected. Along with the focus in the vertebra there are frequently foci of osteomyelitis in other bones. In Riese's case it accompanied an onychia. From boils also and from throat troubles the causal organism (staphylococcus pyogenes) may be carried to the vertebræ. The local symptoms (pain, tenderness, and later œdema, fluctuation, contraction of the muscles of the back) are usually well marked. Signs of compression of the cord and nerve roots have been made out in several cases. The vertebral arch is in this disease more frequently affected, and it shows a preference for the lumbar region. Tubby distinguishes a mild, a severe, and a fulminating, usually multiple type. Israel has observed a case the course of which was subacute and remittent; Labeyrie has reported a similar one and has described this type as one the symptoms of which resemble caries. I diagnosed one such case and saw the diagnosis confirmed by operation.

Spondylitis typhosa is a very rare disease We are indebted to Gibney, Osler, and especially to Quincke,[4] for observations on this subject. The pathological anatomy has been studied by Ponfick and E. Fraenkel, who have demonstrated in the diseased vertebra an invasion by typhoid bacilli. A case described by Raymond-Sicard, where typhoid bacilli were found in the pus obtained by lumbar puncture, appears to have been of this type.

Quincke describes as characteristic: (1) the unusual severity and

[1] *Deutsche Ärztezeitung*, 1900.
[2] *Gaz. des hôp.*, 1892.
[3] *Arch. prov. de Chir.*, 1905.
[4] *Mitt. aus Grenzgeb.*, iv.

duration of the pains; (2) the visible swelling of the soft parts; (3) the feverish course; (4) the rapid disappearance of the spinal symptoms.

Flusz, in a review of the literature (*C. f. Gr.*, 1905), has treated very fully of this disease. It appears that after other infective diseases a similar process may develop.

Course of the Disease.—Caries almost always runs a chronic course. The signs of the vertebral disease may exist for months and even years before any spinal symptoms develop. Cases have been observed in which the kyphosis was present from childhood, but the paralytic symptoms did not develop till late in life. Thus, I have seen the signs of an acute compression of the cord in a man thirty-five years of age, in whom the kyphosis had developed when he was four years of age. Not infrequently, however, some symptom of spinal disease is the first warning of the malady, the deformity developing later, or the vertebral disease remaining latent. Sometimes the signs of spondylitis and of compression of the cord appear simultaneously. The disease of the cord develops slowly; the paralytic symptoms appear and progress gradually over a period of time of from several months to one year. The paraplegia may, however, appear suddenly, namely, when the kyphosis develops suddenly or when a necrosed piece of a vertebra is detached.

The further course is generally a protracted and not infrequently an intermittent one. The disease may at any time be arrested, the paralytic symptoms may disappear in spite of the continuance of the caries; on the other hand, they may persist whilst the caries is cured. It is not unusual for the paralysis to disappear when the abscess bursts outwards, and it is a striking fact that this spontaneous cure may result even when the paraplegia has existed for one-half to one year. The description of the nature of the pathological process, given above, explains this. I have indeed[1] treated a young man who improved so much from a paraplegia which had lasted for seven years, that he again learnt to walk; this improvement, however, only lasted in all for seven to eight months.

The explanation of the recovery of function, even after a long period of the disease, has been made clear to us by the histological researches of Schmaus. From these we learn that the purely mechanical results of the compression (œdema congestion with lymph) may exist for a long time before the irreparable inflammatory and sclerotic changes take place. Another very interesting observation has been recorded from Strümpell's clinique by Fickler.[2] He found bundles of delicate nerve fibres which, coming from the region of the pyramidal tract, entered the grey substance and thence passed outwards along the collaterals which join the central with the pial veins, so passing downwards in the anterior sulcus. In his opinion these bundles of fibres arose from an area above the site of the disease, passed over it, and when below its level they re-entered the cord and joined the grey matter. A similar view had been previously brought forward by Saxer. Fickler considered the process as one of regeneration of nerve fibres—newly formed outgrowths from the pyramidal fibres which had been cut off from their destination—and he credits the human spinal cord with the capability of recovering its function in this way, even after complete breach of its conducting paths, provided always that the blood-vessels remain intact. Before we can feel justified

[1] *B. k. W.*, 1896. [2] *Z. f. N.*, xvi.

in accepting Fickler's conclusions, further researches in this matter are very necessary.

Meanwhile Bielschowsky has opposed Fickler's view; he had also seen those bundles of fibres and taken them for an aberrant part of the anterior pyramidal tract or for a long commisural tract. Likewise the researches of Dercum and Spiller appear as evidence against Fickler's view. Hellich in particular has shown that the bundles described by Fickler occur in the normal cord; he considers them to be sensory nerve fibres of the pia mater, like those already shown by Bochdalek to exist in the region of the pons. Touche, Thomas, and Lortat-Jacob have likewise described them.

Fickler has, however, in his most recent paper on this question (*Z. f. N.*, xxix.), maintained his view, except in so far that he now considers that the newly formed fibres are derived not from the pyramidal tract, but from the ganglion cells of the grey substance and of the spinal ganglia. Lately Marinesco-Minea (*Nour. Icon.*, xix.) have likewise worked at the subject of regeneration of the spinal cord, and in spite of some positive results, they could not ascribe any great importance to these in regard to the recovery of function. *Cf.* also Henneberg (*Charité-Annalen*, xxi.), containing the literature on the subject.

The condition often remains stationary and continues for years without any change. In other cases the vertebral disease heals and the spinal affection improves so much that only a certain degree of paresis remains and the patient is able to walk about again. Sometimes recurrences take place, and not infrequently an accident is the exciting cause. Pregnancy may in this sense be a causal factor (Charpentier).

In many, indeed in the majority of cases, the disease is progressive, and bedsores develop; cystitis, pyelonephritis, septicæmia, or general tuberculosis may determine the fatal issue.

The prognosis is on the whole favourable in young patients, in cases where the spinal symptoms are slight or are absent, where the disease is situated in the dorsal region, or where nutrition is good and the patient of powerful build. It is essentially more unfavourable when the caries appears in a patient of more advanced years, of broken-down constitution, or in one showing the signs of general tuberculosis. Caries of the upper cervical vertebræ is always serious; still, I have seen three cases of this kind run a favourable course. Further, the prognosis is bad in cases of long duration, when the paralysis is of flaccid type, with loss of reflexes and the presence of reaction of degeneration.

Even should the disease run a favourable course and the treatment be successful, a long time is necessary to effect a cure.

Statistics from Billroth's clinique show that out of 97 patients suffering from vertebral caries, 48 died, 22 were discharged cured, and 11 left without a cure being effected. More recent statistics from the Tübingen Hospital (Reinert) show a mortality of 60 per cent., and 30 per cent. of cures. Fickler saw, among 14 cases with paralysis from compression, three recoveries; only in one case however, was the restitution of the functions of the cord complete. The lasting effect of the cure was demonstrated in several cases (and in two cases of my own), even after eighteen to twenty years. In a case of Loison's a spontaneous cure resulted from the effects of an attack of erysipelas.

Gowers has made the interesting observation that in a patient who, in childhood, had suffered from caries without any cord symptoms, there developed later in life the symptoms of lateral sclerosis.

I have seen a very interesting case of this nature. A lady, fifty years of age, complained of severe pains in the legs, and as after lasting for many years no objctive abnormalities developed,

the case was diagnosed as hysteria by many distinguished physicians, whilst others diagnosed sciatica. I myself, on my only examination of the case, could find no definite cause, and was inclined to view the case as one of functional disorder. The patient departed when I advised that further observation was necessary. A year later, I saw her in Wiesbaden again with the signs of a compression paralysis, and her doctor (E. Coester) had found out that she had in the upper dorsal region a kyphosis, the result of a healed caries from which she had suffered in childhood. She had concealed this fact because she considered that it had no bearing on her present trouble.

I have seen in a boy of eleven years, who, when four years of age, had suffered from caries, no sign of which however was at this time to be seen, incontinence of urine and fæces develop.

Treatment.—The treatment of this condition must fulfil two objects; it must improve or maintain the general health of the patient, and it must protect the vertebral column from all shocks and movements, and the affected vertebra from pressure.

Besides a liberal diet rich in fat and albumen (cod-liver oil is specially recommended), rest in bed is essential, for the majority of patients, particularly in the active stage of the disease. If possible, it is still better to have the patient carried out into the open air, the diseased part being kept absolutely at rest by the apparatus described below. Fresh air (particularly residence at the seaside) is undoubtedly a very important factor in treatment. In those cases where the patients must remain lying on their backs for months or even longer, especially in those with marked compression paralysis, one must from the first guard against the formation of bedsores by having a good smooth bed, by frequent washing of the gluteal region, by great cleanliness, and also by the use of air or water cushions. Should this not suffice, one may try by means of extension and counter-extension to take all weight off the diseased vertebra. Extension is of most value in cervical caries; the pull is applied to the head by means of the Glisson suspender or some similar apparatus, whilst the body acts as the counter-extension. The latter factor may be enhanced by raising the head of the bed and by applying weights to the patient's feet. In caries of the dorsal verebræ the extension for the upper part of the body is carried out by means of a band which grips the patient round the axillæ. Of the various forms of apparatus which have been recommended for taking the weight off and straightening the vertebral column, besides the Volkmann extension apparatus, the Rauchfuss suspender (the value of which has lately been upheld by Schilling) and Bonnet's wire basket have been widely used; although recently they have been largely replaced by Lorenz's plaster cradles. In using extension by weights, it is advisable to begin with a light weight (3 to 4 pounds) and not to exceed 12 pounds (for the head) and 10 to 20 pounds (for the extremities).

In some cases the prone position has proved useful. It can be kept up for a long time, and pressure may be applied to the kyphosis by means of a sand-bag laid on the back or some similar device (Bouquet). Taylor, Wullstein, and Hoffa have recommended forms of apparatus for correcting the kyphosis, the patient sitting or being suspended in the erect attitude.[1]

For the slighter cases and for after treatment, portable forms of supporting apparatus and corsets may be used, so that the patient may walk about; naturally they are suitable only for cases in which there are no signs of compression or where such signs are very slight, or have almost

[1] See also Helbig, *B. k. W.*, 1905, and Wollenberg, "Die tuberkulöse Wirbelentzündung und die moderne Behandlung derselben," *Berliner Klinik*, 1906, H. 217.

disappeared, and in which the tuberculous process has healed. For this purpose the plaster of Paris jacket may be employed, being applied according to Sayre's or one of the other methods, where the patient is suspended and the vertebral column straightened. In order to relieve the diseased vertebra from pressure, the weight of the upper part of the body may be conveyed to the pelvis by means of a steel rod (Sayre's jurymast, Nebel's head support, Schede, Heusner, Dollinger's jacket, etc.). Wolff obtained good results with his step-bandage, likewise Maass with celluloid gauze bandages. A further modification of the use of extension is described by Picart and Calot—Hoffa strongly recommended the Hessing supporting apparatus.

To secure not only immobility and straightening or extension of the vertebral column, but also a sufficiency of fresh air for the patient, Phelps and Lorenz have recommended the so-called extension plaster beds. Lorenz's plaster bed is highly spoken of by surgeons (König, Karewski, Redard, Hoffa, Vulpius, and others). Beuthner has reported on the favourable results obtained in Bergmann's clinique, and recently Vulpius has recorded very satisfactory results with this form of treatment. Karewski describes a plaster jacket which encircles the whole trunk, and wearing which the patients may walk about. Hoffa also devised a similar apparatus of his own.

The results of the treatment by extension have of late been very favourably commented on by Reinert, from the study of some very instructive statistics. Its value is sometimes very strikingly shown. Thus Schede has seen paralysis of all four extremities disappear in a night, under extension treatment. Hoffa had seven recoveries out of eight cases of spondylitic paralysis treated by this method : the favourable results obtained by extension were recorded again recently by Helbig and Wollenberg, two of his pupils. Likewise Dollinger reports success by his correction and fixation method ; he had thirteen recoveries in fifteen cases. Vulpius showed seven cures among fifteen patients treated with the plaster bed. The results obtained by Ménard at Berck-sur-Mer are well known. A few surgeons (Trendelenburg, Kraske) have, however, opposed the use of extension treatment.

Difference of opinion exists as to the value of counter-irritation as a method of treatment. Modern opinion opposes the use of the cautery, the issue, and the seton ; nevertheless in those cases where rest and mechanical treatment have failed, and where direct operative interference is not indicated, one might employ these measures, and keep up a moderate suppuration in the back over the diseased vertebra. I have given up this practice in the last few years for I do not feel justified in using it, in face of the condemnation which it receives from many surgeons. Kapesser, Diruf, and Hoffa have recommended repeated energetic rubbing with soft soap. Concerning the technique of this treatment, *cf.* Helbig.

Concerning the medicinal treatment, I can from successful practice recommend the iodide of iron preparations ; one must watch, however, that the appetite does not suffer. Calcium phosphate, and also creosote, have been recommended. Recourse should be had to iodide and mercury only in cases where a syphilitic factor is probably present (see the chapter bearing on this).

In the year 1888, Macewen recorded several cases in which operative treatment for vertebral caries (with compression paralysis)

was successfully carried out. This suggestion was received with enthusiasm. The experience of surgeons in this line of work has since then, however, not been so encouraging, so that the indications for this method of treatment, even in the hands of surgeons, have once again become more limited. Above all, one must guard against premature interference, for a spontaneous cure may take place even after a whole year, and in one of my cases, certainly an exceptional one, astonishing improvement occurred after seven years. Moreover, we must remember that the focus of disease in the body of the vertebra can seldom be completely removed, and frequently all that can be done is to remove the spongy, caseous, or breaking down purulent material or the callous thickened deposit on the dura (Macewen, Ménard, and others), or the intradural abscess (Trapp). By such means a complete cure would only be affected after the vertebral disease had itself healed. Could these facts be determined, the opening of the spinal canal would be undertaken with greater hopes of success. Horsley, however, advises surgical treatment even for the tuberculous bone disease, just as this would be carried out for tuberculous disease of other bones (oral communication). Thorburn and Chipault have collected statistics with regard to the fate of cases undergoing operation. Of 103 cases on which laminectomy was performed, forty-three died soon after the operation, whilst in fifteen a definite cure was effected. Frequently when the spinal canal has been laid open it has been demonstrated that the disease was inaccessible to the surgeon's knife (Fürstner, Raymond, and others). Lately an attempt has been made to gain access to the vertebral bodies and the spinal canal, from the side, by resecting the transverse processes and head of the ribs (costotransversectomy of Ménard). Tillmans recommends this measure under certain circumstances. Sick also by this means succeeded in gaining access to and draining a prevertebral abscess with successful results. We may, along with the majority of competent workers at this subject, lay down the following as the indications for operative treatment : operation is indicated

1. In the case of that rare form of caries in which the vertebral arches are affected,[1] when it is not arrested by conservative treatment (see Péan's results).

2. If the opening of a secondary abscess leads directly to the focus of disease in the vertebra.

3. In those cases in which, after the disease has existed for a long time, and the spondylitis is apparently cured, the paralysis persists in spite of treatment by extension (Trendelenburg). In any case, during the past few years a considerable number of cases have been recorded in which cure was effected by operative treatment (Macewen, Trendelenburg, Tillmans, Chipault, Sick, Höftmann, Israel, Selberg, Krause, Wieting, Cotterill, Sultan,[2] and many others). Payr,[3] in a case of malum suboccipitale, brought about a cure by operative measures on the atlas. Lexer effected marked improvement in one of my cases by operation; I have, however, also met with failures in this respect.

In several cases which I have seen operated on by Krause and Horsley, the cord and dura had to be lifted up and the body of the vertebra probed before the operator reached the tuber-

[1] Of late Wieting has fully discussed the subject of caries in this situation, its peculiarities and its diagnosis (*A. f. klin. Chir.*, Bd. lxxi.).

[2] *Z. f. Chir.*, Bd. lxxviii.

[3] *D. m. W.*, 1906.

culous focus, out of which caseopurulent material flowed. Horsley then cleaned out the cavity in the bone with concentrated corrosive solution, afterwards washing it out with a very weak solution of the same.

Schede says that after all the other measures have been tried, laminectomy remains as the ultimum refugium.

In osteomyelitis vertebralis operative treatment is clearly indicated, for a cure cannot be effected by other means, and it has come about in several cases as the result of surgical interference (Chipault, Riese, Wiesinger, Weber).

We have still to mention a method of treating Potts' curvature which some years ago attracted great attention, namely Calot's forcible correction: the patient being suspended in the prone position and assistants applying forcible extension to the head and legs, the operator by exercising great force attempts to press in or to break the projecting vertebra. The position of parts attained by this means was kept up for some months, by applying at once a plaster jacket covering the whole trunk. Calot considered that his method was suitable for recent as well as for old-standing cases, and also reckoned the presence of compression of the cord as no contraindication, and to all appearances his success with this method was considerable. German surgeons and also the majority of surgeons in other countries condemn this method of treatment. König drew attention particularly to the lessons of morbid anatomy, showing how seldom there is any effort at formation of new bone in cases of caries. J. Wolff has criticised the method most severely, and has pointed out the great dangers and the by no means small number of deaths already known to have taken place from it (Ménard, Malherbe, Schede, Braun, and others). Numerous modifications have been suggested, by which the effect desired by Calot has been accomplished in a less dangerous manner, and Calot[1] himself now seems to prefer these.

Goldscheider saw a patient, who, after treatment by Calot's method, had again developed paraplegia, cured by treatment with extension.

A form of forcible correction which was recommended some years ago by Chipault, has likewise not proved to be a method of value. This, however, is not the place to deal more fully with these or any of the other operative methods.

The treatment may be summarised as follows:—

1. For the majority of cases of caries, conservative treatment should be adopted.

2. During the active stage of the disease the best treatment is absolute rest, the patient lying on his back in bed, or in a Lorenz's plaster bed.

3. Later in the course of the disease, the essential part of treatment consists in fixation, extension, and counter-extension of the diseased vertebral column.

4. Operative interference is called for only in a small percentage of cases when the disease is confined to the vertebræ, and generally only comes up for consideration when a compression paralysis has resisted conservative methods of treatment.

5. Calot's method in its original form should be altogether avoided, but under certain circumstances, one of the later modifications of it may be justifiably used to overcome the kyphosis.

[1] "Die Behandlung der tuberkulösen Wirbelsäulenentzündung." Übersetzt von Ewald, Stuttgart, 1907.

Concerning the treatment of gravitation abscess, *cf.* the surgical text-books, the papers by Helbig (*B. k. W.*, 1905), Mende (*Therap. Monatsh.*, 1906), and others.

If the patient is able to get about, he may have warm baths containing sea-salt or Kreuznach mother-lye, or take a course of baths in Nauheim, Kreuznach, Tölz, etc. Warm baths and the massage and gymnastic exercises carried out in the baths (kineto-therapeutic baths) are especially valuable in combating the spastic conditions.

Electrical treatment is not of much value. In the acute stage of the disease it should not be employed at all. In cases of dorsal or cervical caries with spastic paresis, electrical stimulation of the muscles is contra-indicated, as thereby violent reflex twitchings are produced, the rigidity is increased, and harm may be done to the vertebral lesion by the twitching movements. When the skin is anæsthetic and liable to trophic lesions, the application of the galvanic current may readily produce ulceration. Electrical treatment should be reserved for the old cases, in which the results of the disease, in the shape of a flaccid paresis or an atrophy of the muscles, form the chief residual symptoms.

Direct galvanic treatment of the back is apparently of value in isolated cases, and I have seen it effect distinct improvement in one case in which the paralysis had existed for seven years. In cases of healed caries where contractures have been left behind, and where there is difficulty in walking, massage and other orthopædic measure, and also tenotomy, have to be considered. These conditions are only rarely met with however.

To avoid a recurrence of the disease the patient should live in the most hygienic surroundings possible, and should moreover carefully guard against any fall on the back ; it is also advisable, I think, to protect the diseased vertebra from injury by means of a small soft cushion or pad.

Carcinoma and other Tumours of the Vertebral Column

Literature : See in Schlesinger, " Beiträge zur Klinik der Rückenmarks- und Wirbeltumoren," Jena, 1898, and L. Bruns, " Die Geschwülste des Nervensystems," i. Aufl., Berlin, 1897 ; ii. Aufl., Berlin, 1908.

Carcinoma of the vertebræ is almost invariably of secondary metastatic nature. The primary disease may be in the stomach, uterus, prostate, or other region, but very frequently it is a carcinoma of the breast. In eighteen out of the twenty-two cases of vertebral carcinoma which I have had occasion to see during the past few years, the primary site of the disease was the mamma. In seventeen cases the mammary cancer had been removed by operation, whilst in one case the scirrhus was only discovered after the spinal trouble appeared. Two of my patients were sisters, and in them the carcinoma had spread from the breast to the vertebral column in exactly the same manner.

According to Petrén's statistics (*Mitt. aus d. Grenzgeb.*, xiv.), in 402 cases of mammary cancer, metastasis occurred in the vertebral column fourteen times, in the pelvis ten times, etc. Usually it occurs within one year of the removal of the mamma, but seven to eight years and even eleven years may intervene. One or two cases of primary vertebral cancer have recently been recorded (Péhu-Coste); its existence, however, is still open to doubt.

Sarcoma and osteosarcoma may originate in the vertebræ; more frequently they arise in the neighbourhood and then invade the vertebral column. Moreover, myelomata, osteomata, enchondromata, and cystic

tumours, more especially hydatids, may develop in the region of the vertebral column. A very rare form is a thyroid metastasis, a case of which was described by Dercum.

The age incidence of this disease is in the later years of life; sarcoma, however, may attack young people. These tumours seldom affect a single vertebra, but usually involve several adjacent vertebræ, and indeed carcinoma may involve a large portion or exceptionally even the whole vertebral column, as it did in a case of Bruns. It may spread by the development, in the first place, of isolated foci in the bodies of the vertebræ; as a rule, however, there is from the outset a diffuse infiltration of the vertebræ accompanied by necrosis and softening. The growth may invade the transverse processes and the adjacent portions of the ribs and spread in all directions, and extending backwards through the muscles may appear under the skin. The diseased vertebræ may readily collapse and be pressed together without producing any marked kyphosis; the latter, however, as a rule results, but it is usually less pointed and angular than in the case of vertebral caries, because several vertebræ participate in the deformity and the growth itself forms part of the prominence.

Frequently the metastatic process is not limited to the vertebral column, but affects other parts of the skeleton, more especially the pelvic bones.

The compression of the cord and the nerve roots leads to changes similar to those found in the case of caries, only the destructive processes are usually more marked in the malignant cases. At first there is compression of the blood and lymph vessels, interfering with the circulation in the cord and leading to œdema and softening. Moreover, formation of cavities may take place. The nerve roots become not only compressed but also invaded by tumour growth. Finally, the disease grows through the dura and presses on the cord itself. The cord has often been found encircled and constricted by tumour growth (Nonne). Very rarely metastatic foci develop in the medulla (Chiari). The symptomatology is influenced, however, not only by the compression and infiltration, but also by toxæmia, for this causes not only chemical and dynamic (Oppenheim), but sometimes real organic changes (Nonne, Siefert).

This disease is very painful. The pain is always aggravated by movement, by pressure, or by any jolting of the diseased vertebræ; often, though the spontaneous pain be very severe, there is no tenderness on pressure. The most acute pain, however, occurs when the posterior nerve roots are compressed by or involved in the tumour growth. Then violent neuralgic pains set in, being referred along the lines of the affected nerve paths; thus one gets intercostal neuralgia or neuralgia along the course of the nerves of the extremities. When carcinoma affects the lumbar vertebræ, severe double sciatica may be one of the earlier symptoms of the disase. The symptoms of irritation, hyperæsthesia, and local muscular spasms, are usually very striking. In one or two cases (Buckley, Oppenheim), however, the pains have been surprisingly slight. Herpes zoster (also bilateral) may likewise occur. Schlesinger has observed œdema of the parts supplied by the compressed nerve roots.

The other root and cord symptoms correspond to those described for caries. In one of our cases, bilateral Erb's paralysis was the first symptom of a tumour of the cervical vertebræ; in another case, where the neoplasm

involved the seventh cervical vertebra, oculopupillary symptoms were among the first developments.

Paraplegia may develop here with great rapidity. The tumour, growing into the spinal canal, compresses the cord and produces a condition of softening which quickly spreads. This is in many cases evidenced by a rapid increase in the paralytic symptoms. The course of the disease may, however, be long drawn out. As a rule, the pains persist in their full intensity after the limbs have become paraplegic.

The diagnosis is based on the patient's relatively advanced years (naturally this holds good only for carcinoma), on his loss of strength, on the evidence of a primary growth elsewhere (a cicatrix in the mammary region, etc.), on the great pain in the vertebra elicited by pressure or by movement, on the presence of an angular or rounded kyphosis, on the shortening of the trunk, the result of a sinking in of several diseased vertebræ —this shortening is sometimes very striking—and lastly on the root- and cord-symptoms. I have also often been helped in coming to a diagnosis by percussion over the vertebral column, the note over the diseased part being markedly impaired. This method must be employed, however, with great care and judgment. Frequently one is able to palpate the growth in the vertebræ or in the adjoining parts. There are often also metastases in other organs (in the brain, etc.), in the bones, lymphatic glands, etc.

An X-ray photograph may clear up the diagnosis in a doubtful case. That great care is necessary in interpreting the X-ray findings has been well brought out in Nonne's [1] interesting communication, in which he cites a case where the vertebra, which appeared from the X-ray to be the site of a metastasis, turned out to be the only one not affected by foci of the disease. In hydatid disease the diagnosis may be clinched by puncture, as happened in a case of Wilms'.

The differential diagnosis from caries can often not be made with certainty, although the greater age of the patient, the great pain associated with the trouble, and the other features above mentioned, generally lead one to a right diagnosis. Signs of tuberculosis favour a diagnosis of caries, whilst the presence of a primary growth in another organ verifies the diagnosis of tumour of the vertebral column. Lastly, the more recent forms of tuberculin reaction may be employed to aid a decision.

It is noteworthy that in these cases of vertebral tumour also, the spinal symptoms may be the first evidences of the disease, whilst the vertebral trouble may remain latent for a long time. I have seen a case in which the first complaint was pain in the back and in the abdomen. Some weeks later, there appeared signs of paralysis in the legs, bladder trouble and sensory disturbances, while the pains became more severe. There was as yet nothing abnormal to be made out in the spine. As there was a previous history of syphilis I made the diagnosis of spinal syphilis, and commenced the appropriate treatment. Only after several months did an angular curvature appear in the lower dorsal region, and it turned out that a sarcoma which had arisen in the neighbourhood of the vertebral column, probably from the retroperitoneal glands, had eroded a number of vertebræ and had compressed the cord over a considerable distance. Nonne has recorded some interesting cases of this nature.

One must remember in regard to the differential diagnosis that as the

[1] *N. C.*, 1903.

result of the cachexia of malignant disease there may develop a simple myelitis, *i.e.* inflammatory or degenerative changes in the cord (Oppenheim, Lubarsch, Meyer, Homén, Nonne, Buck, Ballet-Laignel-Lavastine), in the meninges (Siefert, Raymond), as may also a multiple neuritis (Oppenheim-Siemerling, Miura, Francotte, Mousseaux, and others.) The latter condition may also develop as the result of a direct invasion of the nerves and muscles by foci of disease in cases of miliary carcinomatosis (Raymond [1]). Moreover, in the subjects of malignant disease, pains are sometimes complained of in various parts of the body, including the vertebral region, for which no malignant focus is directly responsible.

My experience has shown me (*Charité-Annalen Jahrg.*, xiii.) that the signs of nerve irritation and involvement may be produced by the toxin of carcinoma, and Nonne and Spiller-Weisenburg have also described this condition; still we must not forget that there is a form of metastatic spread which can only be revealed by microscopic examination (Siefert,[2] Saenger [3]).

Diagnosis.—One must not diagnose carcinoma of the vertebral column from vague symptoms, but it should be strongly suspected when, in a person who has suffered from a carcinoma and particularly a mammary carcinoma, pains set in which are localised to the region supplied by one or several adjoining spinal roots and which are persistent. Even when for a long time one can detect no objective sign of vertebral disease, carcinoma of the vertebræ is often the root of the trouble. These facts, which were described in the former editions of this book, have been confirmed and more fully explained in a valuable paper by Petrén. He also emphasises the fact that the subjective troubles—the pains and the consequent restriction of movement—may exist for a long time before any objective sign is visible, and that they are very varied and inconstant in their order of appearing and their distribution. Carcinoma of the meninges, as has been shown by the researches of Siefert, is deserving of consideration.

The course run by a case of vertebral carcinoma is usually acute and sometimes is very rapid, but on the other hand some of these tumours are of slow growth. In two of my cases there were such marked intervals of freedom from symptoms that I was misled into thinking that the condition was benign in nature.

During the past few years I have repeatedly seen this, and am no longer led to form a false opinion from the presence of even pronounced improvement. In cases of multiple spread of carcinomatous or sarcomatous disease especially, it happens that the pains and the restriction of movement in a certain region may almost disappear, giving one the impression of a localised cure. Thus the paralytic condition produced by a malignant tumour in the lumbar region of the spine may for weeks at a time disappear or undergo marked improvement.

On the other hand I have seen a case in which, on account of the insignificance of the signs, the diagnosis of hysteria had been made elsewhere, but in which there followed a few weeks later absolute paraplegia, with incontinence of urine and fæces, and demonstrable metastases in various organs, with a fatal issue a week later.

Moreover, in a case of intercostal pain occurring in a very hysterical woman, I myself attributed the pain to hysteria, and by treatment on suggestive lines and by placebos, at first caused the pain to disappear for several months; it then returned, however, with increased severity, and was accompanied by all the signs of compression myelitis. In another

[1] *A. d. Neurol.*, 1904. [2] *A. f. P.*, xxxvi. [3] *N. C.*, 1901.

case, I could not, in spite of repeated examination, come to a definite diagnosis for a long time.

In cases of sarcoma the disease may linger for several years.

The prognosis is almost always a gloomy one. Operative treatment has so far accomplished little, and this is natural, considering the malignant and metastatic character of the majority of vertebral new growths. According to H. Schlesinger the malignant tumours are thirty times as common as the benign. In a case of osteosarcoma of the vertebræ under my care, a trephining operation on the vertebræ performed by Sonnenburg and Horsley achieved nothing, as the growth had already not only involved a considerable number of vertebræ but had extended deeply towards the abdomen. In recent years, however, surgical treatment has accomplished several good results in this field. Thus Büssem has recorded a successful operation for a vertebral tumour performed by Witzel; in this case, certainly, they had to deal with an osteoma of the vertebræ, which is not only a rare tumour but is one which very seldom leads to signs of compression. In a case of enchondroma operated on by Sick [1] the growth recurred four years later, and was again removed, but without affecting a complete cure.

In addition Kümmel [2] published a case of successful removal of a sarcoma of the spine, likewise Israel,[3] whilst Thomas succeeded in removing a myeloma, thereby giving the patient complete relief for six months. Walton and Paul also record a case of successful operation for a vertebral myeloma. None of these authors, however, conceal the fact that as the tumour is usually malignant, recurrence is to be feared. Personally I [4] know a case where a sarcoma which had affected the cervical vertebræ and extended through these into the vertebral canal, was excised with good result, the patient being still free from recurrence nine years later. Hydatid cysts of the vertebral column have been repeatedly subjected to operative measures (Ransom-Anderson, Hahn, Wood, Lloyd, Beltzer; *cf.* the chapter on Tumours of the Spinal Cord).

We may here disregard the very rare cases (observations of Bruns, Buckley) in which the carcinomatous metastases affect not the vertebræ but the dura. Mention has already been made of carcinomatous invasion of the meninges.

In the majority of cases medical treatment is confined to combating the pain by injections of morphia, and by carefully attending to the patient's position, so that the least possible amount of pressure is brought to bear on the diseased vertebræ. Sick has recorded a case, certainly unique, in which a sarcoma of the spine disappeared under treatment by subcutaneous injections of arsenic.

Syphilitic Disease of the Vertebræ

The incidence of syphilis in the vertebræ is as rare as it is common in such other bones as the tibia, the clavicle, and the skull. However, we sometimes find the formation of exostoses on the bodies and processes of the vertebræ; these may attain great size and compress the nerve roots or the cord. Gummata may also arise in the substance or on the surface of the

[1] *D. m. W.*, 1904. [2] *A. f. kl. Chir.*, 1895. [3] *B. k. W.*, 1903.
[4] *Mitt. aus d. Grenzgeb*, xv. See here some further reports of temporary operative success.

bone; also a syphilitic form of spinal arthritis has been reported on several occasions. In a case under my care there were large, irregularly shaped exostoses of the upper cervical vertebræ which could be palpated in the back of the neck, and there was paralysis and sensory disturbances in all four extremities, the signs of an advanced myelitis cervicalis superior. The patient had undoubtedly had syphilis. An energetic and prolonged course of mercurial inunction effected a complete cure. In another case I was able to palpate a sound exostosis, the size of an apple, on the tenth or eleventh dorsal vertebra; there were definite signs of compression of the cord—here again treatment with mercury led to a cure of the condition. In recent years Leyden, Dejerine, Fournier-Loeper, Strubell, Staub, Zak, Sternberg, and Fry have described cases of this or of similar nature.

A case reported by Brown (*Journ. of Amer. Assoc.*, 1904), in which, after the spontaneous extrusion of a necrosed portion of the atlas, and the formation of a retropharyngeal abscess healing occurred, leaving some restriction of movement, was possibly of syphilitic nature.

On the whole, however, syphilitic affections of the vertebræ are very rare, and practically never involve the spinal cord. Nonne[1] in particular has emphasised this point.

In cases of multiple exostoses one has seen signs of involvement of the spinal cord arise, which could be traced back to vertebral exostoses.

Arthritis Deformans and Allied Chronic Joint-Affections of the Vertebral Column (Ankylosis of the Vertebræ, Chronic Ankylosing Inflammation, Stiffness of the Spinal Column, Spondylitis Deformans, Ankylosing Arthritis, Spondylose Rhizomélique, etc.)

Every form of arthritis may attack the vertebral column. Most frequently, though even then it is rare, it is arthritis deformans which affects this part of the body. It may involve the whole joint apparatus of the spine, with ossification of the intervertebral discs, and the ligamenta subflava and bony outgrowths on the vertebral processes, so that bony clasps bind the separate vertebræ to each other, and a complete ankylosis of the whole vertebral column results.

The disease may, however, be limited to a definite part, *e.g.* the cervical region, the head and neck being fixed and the chin possibly bent toward the chest. Sometimes the marginal bony outgrowths can be palpated through the skin on the back or from the pharynx.

As a rule the disease is a painful one. The pain is situated in the vertebral column and is aggravated by all attempts at movement. *Root symptoms* are not uncommon; as the intervertebral foraminæ become narrowed by the formation of new bone, the roots are exposed to a very slowly increasing degree of pressure: this leads to shooting pains,[2] (*intercostal, brachial, crural neuralgia*) and to *atrophic paralysis* in the muscles of the extremities. The latter is almost never complete.

When arthritis deformans of the other joints is at the same time present, it is not easy to decide whether the muscular atrophy is dependent on an inflammatory condition of the nerve roots, or whether it is simply determined by the local joint affection. In a doubtful case which I saw, an

[1] "Syphilis und Nervensystem," Berlin, 1902. See here the literature.

[2] Babinski has seen a clinical picture develop in this way, simulating a case of tabes.

electrical examintion decided the matter : the discovery of partial R.D. showed that there was a degenerative atrophy and therefore one dependent on a root-neuritis. Herpes zoster has also been observed. Only very rarely is the cord itself compressed. Nevertheless Bechterew [1] and especially Lépine [2] have drawn attention to the occurrence of meningeal and spinal troubles in chronic arthritis of the vertebral joints. A case has also been described in which the arthritis led to such marked excrescences on the odontoid process that compression of the medulla oblongata occurred.

As a result of the immobilising of the vertebral column a characteristic attitude of the body is produced (*cf.* Fig. 153).

The disease runs a *chronic* course, usually with remissions and exacerbations.

The *diagnosis* is based chiefly on the evidence of arthritis in other joints, on the immobility of a large part or the whole of the vertebral column—an ankylosis which persists when the patient is under chloroform —and on the root symptoms. Sometimes palpation may reveal positive evidence. Ludloff makes use of auscultation of the vertebral column to determine whether the crepitation associated with this disease be present. In recent years, moreover, radiography has become a potent factor in the diagnosis of such conditions (Simmonds,[3] E. Fraenkel,[4] Schlayer, and others).

The facts described above, although well known to the older authors, and fully recognised by Braun (1875), and even in 1824 by Wenzel (according to Sänger), had been to some extent forgotten, until again brought into prominence by papers from Strümpell,[5] Bechterew, and Marie,[6] on the chronic affections of the vertebral bones and joints.

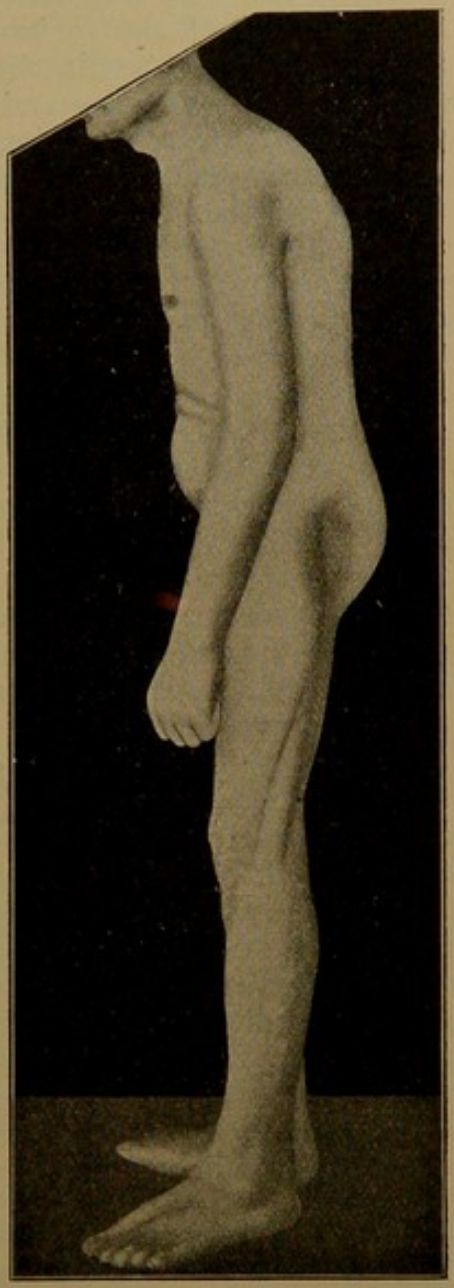

Fig. 153.—Spondylitis deformans. (Oppenheim.)

Several special points have been described in which these observations differed from the well-known pictures of chronic joint rheumatism and arthritis nodosa, and from each other, and the result is that this disease has been made the subject of a large number of papers and critical summaries by Marie, Kirchgässer, Heiligenthal,[7] Schlesinger,[8] Valentin, Zeri, Hoffa, Hartmann, Dana, Mayet, Simmonds, Ossipow, Cureio, Fraenkel, and others.

Undoubtedly there has been too much differentiation of particular

[1] *N. C.*, 1893 ; *Z. f. N.*, xi., xv., and *M. f. P.*, xxi.
[2] *Lyon méd.*, 1906.
[3] *F. a. d. G. d. Röntgen*, vii.
[4] *Ibid.*, vii. and ix.
[5] *Z. f. N.*, xi.
[6] *Revue de Méd.*, 1898.
[7] *Sammelreferat, C. f. G.*, 1900.
[8] *Mitt. aus Grenz.*, vi.

forms of the disease. Strümpell and Marie lay particular stress on the fact that the condition is limited to the vertebral column and larger joints (hips or shoulders), whilst arthritis deformans attacks usually the smaller joints, appears earlier in life, and is not generally associated with great pain or with root-symptoms; moreover, that the vertebral column is usually straightened, and only its cervical part undergoes kyphotic bending, etc. On the other hand, Bechterew describes a disease under the term "fusion of the vertebræ or chronic rigidity of the spine," in which ankylosis likewise results, but with a kyphosis of the dorsal spine, which is accompanied by pains and root symptoms, and the large neighbouring joints (hip, shoulder) escape. Whilst chills, infective diseases (particularly gonorrhœa), gout, etc., are said to cause the Strümpell-Marie type of this disease, heredity and traumatism appear to play an important part in the causation of the Bechterew type. Marie also describes a heredito-traumatic kyphosis, giving the post-mortem appearances in such a case; he distinguishes this type from "spondylose," the cause of which he considers to be infective or toxæmic. E. Fraenkel, on the contrary, believes the traumatic causation to be of most importance. A more extended experience has now shown that the points of difference between the Strümpell-Marie and Bechterew types are not essential in character, and that anyone of the signs said to be characteristic of the one type may be absent in this type, whilst it may be well developed in a case of the other type, and moreover, that the disease may affect other joints, even the small ones, as *e.g.* in cases recorded by Popoff, Jacobi Chmielewski, Anschütz,[1] and myself. Also the attempt of Bechterew on the one hand, and Marie-Léri,[2] on the other, to differentiate the types on the basis of the pathological changes found postmortem, cannot be described as successful.

We must not *à priori* conclude that there are diseases peculiar to the vertebral column which do not occur in other bones and joints.[3] In the meantime, however, we may, as Senator admits, include all these conditions in the category of rheumatism, gout, and arthritis deformans. Stress should be laid on the fact that the tendency to ossification and to the formation of new bone in these conditions is particularly prominent when they affect the vertebral column. Kirchgässer, Magnus Levy[4] and especially Anschütz, as the result of extensive observations, confirm this view. Even now, however, there are numerous investigators who uphold the nosological independence of this disease, and particularly its disjunction from arthritis deformans (Marie, Léri, Müller, Valentin, Dana, Glaser,[5] M. Müller, Joachimsthal, and others).

E. Fraenkel, from the results of pathological and X-ray examination, distinguishes an arthritis chronica ankylopoetica from arthritis deformans of the spinal column. In the case of the former the primary and essential changes in the ankylosing process occur around the *articular processes*, whilst the bodies of the vertebræ undergo no essential change. Through this process bony

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[1] *Mitt. aus Grenz.*, viii.

[2] Léri, "La Spondylose rhiz. Revue de Méd.," 1899; also *Nouv. Icon.*, xix.

[3] Some remarkable and puzzling cases have been described in which ankylosis of more or less every joint in the body occurred. Apert recorded such a case under the term spondylose olomélique. Voltz (*Mitt. aus. d. Grenzgeb.*, xvi.) has seen a case of congenital synostosis of the spine. Thaon cites another case of this kind in which the condition commenced in childhood. The affection usually starts at the distal parts of the extremities and progresses centrally. Raymond and also Berger (*Bull. méd.*, 1905) describe a "maladie ankylosante progressive et chronique." See also Mme. Jacobsohn's thesis, "Maladies ankylosantes," etc., Paris, 1906.

[4] *Mitt. aus d. Grenz.*, ix.

[5] *Mitt. aus d. Grenz.*, viii.

clasps may develop between the vertebræ. In spondylitis deformans, which is frequently limited to the lower dorsal or lumbar regions, the essential feature is the deformity of the vertebral bodies by the formation of exostoses, the unilateral bony bridges, and, in addition, changes in the intervertebral discs almost invariably occur. A similar view is held by Sivén (*Z. f. kl. M.*, Bd. xlix.), likewise by Nonne (*N. C.*, 1904), Ehrhardt (*Mitt. aus d. Grenzgeb.*, xiv.), Schlayer, Rumpel, and others.

Simmonds likewise differentiates the deforming from the ankylosing type of spondylitis, as in the latter there is no deformity from the formation of exostoses, the typical feature being the marked ossification of the ligaments; he calls the condition *syndesmitis ossificans*. He admits, however, that intermediate types frequently occur. On several occasions an X-ray examination has revealed quite distinctly during life some of these bony changes. As the result of personal observations (Cassirer,[1] who saw the cases with me, has at my suggestion recorded them fully) I must here note that chronic muscular rheumatism may give rise to a clinical picture, which is very similar to that described above. This is a form in which the muscles of the back, pelvis, and thighs are chiefly affected. The muscular pain leads to muscular contracture and a stiffness sets in, presenting the same attitude and limitation of movement as were described for the articular affection. But whereas in the case of spondylitis deformans, the rigidity persisted under chloroform, this does not occur in the case of the muscular affection. Moreover, I found the muscles very tender on pressure, and there was a marked increase in the mechanical muscular irritability and a pronounced tendency to fibrillary twitchings in my patients. Beer has recorded a case of this nature. Senator,[2] Dorendorf,[3] and Barg have described similar cases and confirmed the presence of muscular disease by pathological examination.

It appears to us very doubtful that a primary chronic meningitis occurs, in the course of which similar deformities of the vertebræ develop; such a condition is described by Bechterew and Winokurow.

From the point of view of differential diagnosis mention should be made of senile kyphosis and paralysis agitans, but one need not enter into the various differential signs. Schultze once observed a combination of spondylitis deformans and pseudo-hypertrophy of the muscles.

The *prognosis quoad vitam* is good; the treatment also is not always by any means hopeless. Massage, the local application of tincture of iodine, and baths are especially to be recommended.

Improvement results in many cases from a course of baths in Oeynhausen, Nauheim, Teplitz, Wildbad, Gastein, etc.; in other cases sulphur baths prove of value. The bath is made up with 50 to 150 grms. of sodium hyposulphite and 30 to 60 c.c. of vinegar or 50 to 150 grms. potassium sulphide, and 20 to 30 c.c. crude sulphuric acid. Where the conditions allow, one may advise a course of sulphur baths at Aix-la-Chapelle, Baden, Nenndorf, Lenk, Kainzenbad, Pistyán, etc. Also potassium iodide and the salicylic preparations, and particularly the prolonged use of salol, are recommended.

A few authors have reported good results from orthopædic treatment —plaster bandages, with gradual correction by stages—(Deutschländer). Marie and Léri recommend forcible correction of the deformity in its early stages.

[1] *B. k. W.*, 1902. [2] *B. k. W.*, 1903. [3] *Charité-Annalen*, xxv.

I have only seen one case in which severe root symptoms (atrophic paralysis) developed as a sequel to an attack of acute rheumatism which had involved the vertebral column. Since then Jaksch has[1] recorded a similar case (with pathological report).

B. Affections of the Spinal Cord arising from the Meninges

Spinal Meningitis

The inflammations of the external surface of the dura mater are almost always of a secondary nature, and are of no special clinical interest. I shall therefore confine my discussion to internal spinal meningitis, which usually arises in the form of leptomeningitis from the pia arachnoid, and frequently extends to the inner surface of the dura mater. Perimeningeal suppuration is also a very rare occurrence (cases of Mollière, Deléade, Chipault, Buck), although in one case the suppurative process extended to the perimeningeal tissue throughout almost the whole extent of the spinal cord.

Acute spinal leptomeningitis is seldom a primary, isolated disease of the spinal membranes. When it is not of traumatic origin, it usually belongs to the epidemic cerebro-spinal form, which is sometimes confined to the spinal cord, or it takes the form of a suppurative meningitis due to septicæmia, and occurs during the puerperium, after a suppurating wound, or after some acute infective illness. The membranes of the spinal cord are often, indeed almost always involved in tubercular inflammation of the membranes of the brain. A tuberculosis limited to the spinal membranes and involving the cord is much less common. A traumatic suppurative meningitis of the cerebral meninges may also spread to the spinal cord. Otitic meningitis, in particular, has the tendency to extend to the spinal meninges, and indeed it may even affect these almost exclusively (Abercrombie, Lichtheim, Jansen). Surgical treatment of diseases of the spinal cord has occasionally been followed by a meningitis which was confined to the spinal meninges or had its origin in them. *Lumbar anæsthesia* with cocaine, stovaine, novocaine, etc., has in a few cases (Walther, König, Sonnenburg, Trautenroth, etc.) been the cause of an inflammatory process in the spinal meninges which involved the spinal cord. In a case described by F. R. Fry an acute purulent spinal meningitis is said to have resulted from a furunculosis.

Pathological Anatomy.—The stage of hyperæmia is rapidly followed by one of serous, fibrinous, and suppurative exudation. The exudate is deposited in the meshes of the pia and arachnoid and makes the cerebro-spinal fluid turbid. The meninges are covered with a semi-solid or suppurative exudate, and are in parts stuck together or quite coalescent. The microscopical condition is shown in Fig. 154. As a rule there is no pus in the *tubercular* form. The exudate is scanty and gelatinous. Eruptions of miliary tubercle are found on the arachnoid and the inner surface of the dura mater. In rare cases there is a very marked deposit on the inner surface of the dura mater, which produces a true hypertrophic pachymeningitis. These changes are very seldom limited to a certain segment of the cord; they are much more apt to affect the meninges in their whole extent. In the forms which are propagated from the brain, however, the process may not extend beyond the lower margin

[1] *Prag. med. Woch.*, 1900.

of the cervical cord. The posterior portion of the spinal membranes is generally more severely affected than the anterior. The roots are also covered with masses of exudate. The cord itself, especially its peripheral layers, is usually the seat of a *marginal myelitis*.

Symptoms.—As a rule the brain symptoms are so very prominent as to mask entirely those of the spinal meningitis. Isolated disease of the spinal membranes, so seldom observed, shows the following symptoms: onset with rigor and more or less high fever of the irregular type; acute *pain in the back*, which is aggravated by every movement of the trunk; *radiating pains* in the extremities, *stiffness in the back, opisthotonus, tonic contraction of the muscles of the abdomen, thorax, and extremities*, increase of the pain and stiffness or clonic tremors when the patient is touched or attempts to move, *hyperæsthesia* of the skin and of the deep parts of the

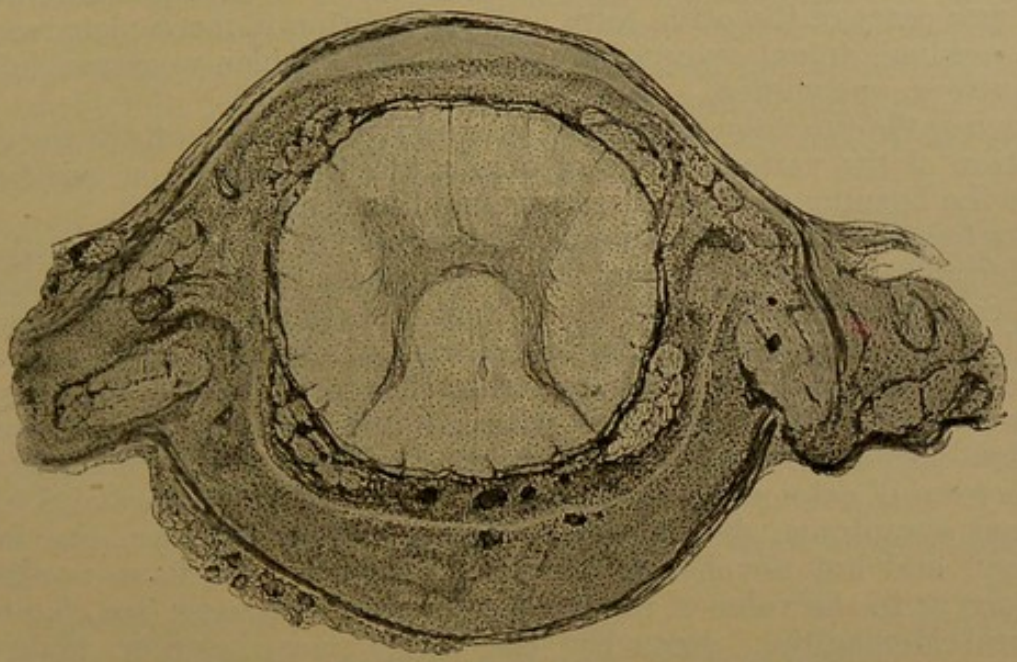

FIG. 154.—Cross section through spinal cord and meninges in purulent spinal meningitis. Stain: carmine-alum-hæmatoxylin.

trunk and extremities, exaggeration of the cutaneous, especially of the abdominal, and of the tendon reflexes. Tapping of the lumbar muscles elicits a sudden inward jerk of the lumbar spine, a symptom which I have termed the "back phenomenon." Kernig's sign, *i.e.* the inability in the sitting position to extend the leg on account of contracture of the flexors, is present. Also when the patient is lying on his back, and the hip-joint flexed to a right angle, the knee-joint cannot be fully extended. There is frequently retention of urine or strangury.

If death does not occur at this stage *symptoms of paralysis* often appear. A paraparesis, or even a paraplegia, may develop, with diminution of sensibility and weakness of the bladder. The tendon reflexes may also disappear, as one so often observes, in epidemic as well as in tubercular and otitic cerebro-spinal meningitis. Respiration and the action of the heart may be affected, and oculo-pupillary symptoms occasionally appear.

In a case under my observation, in which the infection had its origin in a surgical wound from a laminectomy for the removal of a tumour,

the meningitis ascending from the cord to the brain, I was greatly surprised to find that there were no brain symptoms, no loss of consciousness, headache, or convulsions, until the end, or until shortly before death. At first, the symptom of a general infection predominated—fever, rigor, restlessness. These were followed by very marked tachycardia and tachypnœa (of bulbar origin (?) from the ascension of the meningitis), accompanied by great rigidity of the neck, and later by optic neuritis and nystagmus. The general symptoms could at first be benefited by drawing off the excess of cerebro-spinal fluid. In another case of this kind, I could only attribute the transient inequality of pupils and the nystagmus to an ascending, serous, spino-cerebral meningitis of very short duration.

The course is acute, in many cases fulminating, and the prognosis is grave. Death occurs as a rule in a few days or after one or more weeks. Recovery takes place, however, in some cases of the epidemic, and in very rare cases of the otitic form. A termination in incomplete recovery with residual spinal symptoms (paraparesis, bladder weakness, etc.) is not quite so uncommon.

As regards the value of *lumbar puncture* or *cyto-diagnosis* in the differentiation of the various forms of meningitis, the section on this subject under the heading of brain diseases should be consulted.

For *treatment* and further details, the chapter on cerebral meningitis may be referred to. During the first stage the essential points are absolute rest and a comfortable position for the patient. As far as possible diaphoretic and derivative treatment should be carried out by the use of wet packs, or the application of dry cupping and leeches to the back. Ice-bags may be used if they are compatible with an undisturbed and comfortable position. Inunction with grey ointment, and even the internal use of Hg in the form of calomel, is recommended. In the later stages and for the residual symptoms, derivatives to the back (cantharides, the button cautery) and hot douches should be prescribed. We have no definite evidence as to the value of lumbar puncture in such cases (see chapter on Cerebral Meningitis). Recovery has taken place, however, under this treatment, as in Phelps' case, for instance. Laminectomy, opening of the dura, and washing out the subarachnoid space with antiseptic fluid has been tried in some cases. Rolleston found recovery in one case after this procedure. Amberger has also obtained a cure by early operation in spinal meningitis due to a stab, and he recommends this method in similar cases. At all events, this method of treatment should be tried, when the disease has resulted from a spinal operation.

Chronic Spinal Meningitis

Simple chronic spinal meningitis is a disease which, so far as our experience goes, is not clinically of any real importance. We have, at least, no typical cases confirmed by autopsy, which present a definite clinical picture. On the other hand, this condition is not infrequently discovered, usually unexpectedly, during sectio in cases which have succumbed to diseases of the spinal cord or of other organs. Thus it may be observed as an incidental change in tabes, myelitis, and other affections of the cord. Alcoholism, senility, concussion of the spinal cord, the prolonged effect of cold and physical exhaustion, have been regarded as factors which may produce chronic inflammation of the spinal membranes.

Thus a chronic spinal meningitis and meningomyelitis were regarded by earlier writers as the organic basis of "railway spine." That this view is erroneous will be shown in another place.

It has been already mentioned that an acute meningitis may develop into a chronic one; the meninges become opaque, thickened, and firmly adherent to each other and to the cord. The changes may be localised or may extend through the whole cord. A connection has been traced between calcareous plates frequently found in the arachnoid and which give rise to no symptoms, and the chronic inflammatory processes in the spinal meninges. Further, a circumscribed spinal meningitis may be the permanent residue of a general (epidemic) cerebro-spinal meningitis.

The most prominent symptoms, of which we can only give a very brief sketch, are pain and stiffness in the back, radiating pains in the region of the spinal nerves, slight symptoms of paralysis, and eventually atrophy and affections of co-ordination. I have been driven in a few cases by exclusion to the diagnosis of chronic posterior spinal meningitis, but I have been fully aware that this was a vague and provisional diagnosis. There is no doubt that in the earlier report of cases the condition was often confused with neuritis, myelitis, and hysteria.

Some diagnostic interest is attached to a very rare *circumscribed form of tubercular meningitis*, which is localised in the membranes of the spinal cord, and which, in a case thoroughly investigated by Goldscheider (*B. k. W.*, 1891), gave rise to severe symptoms of irritation, and to a persistent contracture in the muscles of the upper extremities. Cases of this and similar kinds have been described by Raymond, Dupré-Delamare, Brissaud and Brécy (*R. n.*, 1902), and by Marie. Henneberg and Rossi speak of an epidural tuberculosis. Jacobaeus (*Z. f. kl. M.*, Bd. xxxv.) describes a meningeal-tubercular process in the cauda equina, and Hobhouse saw a circumscribed internal pachymeningitis persist after an epidemic cerebro-spinal meningitis. Ziehen also gives a clinical analysis of a case of this kind.

There remain two kinds of chronic meningitis which we must carefully consider :—

1. *Hypertrophic cervical pachymeningitis.*
2. *Chronic syphilitic pachymeningitis and leptomeningitis.*

Hypertrophic Cervical Pachymeningitis (Charcot and Joffroy)

This is a condition of chronic inflammation which affects chiefly the inner layer of the dural mater, producing in them a laminated *deposit of fibrous tissue*, and thus causing the dura mater to be considerably thickened. It may be five to ten times its normal diameter. It usually becomes adherent with the periosteum. The new-formed membranes, arranged in layers like those of an onion, are firm and partially ossified, and cause the meninges to adhere to each other, as well as to the roots and to the spinal cord. The latter may be so severely involved that it becomes locally completely atrophied and sclerosed. The leptomeninges play a very important part in the inflammation, which indeed frequently commences in them. In the beginning it is only the peripheral segments of the cord that are as a rule involved, but the affection gradually spreads more or less through its whole thickness, sometimes on account of the compression, sometimes as the result of the propagation of the inflammation to the interior of the cord through the vessels and the pial septa. A lymph stasis may also play a part. There is occasionally a formation

of cavities in the cord. In one of Fischer's cases the affection had not extended to the cord.

The process may be confined to the portion of the dura mater surrounding the *lower part of the cervical enlargement*, and may then give rise to a characteristic clinical condition; but it may also have a much wider distribution in the cord and may even extend to the neighbourhood of the pons and the medulla oblongata (Adamkiewicz, Wieting[1]) and to the meninges of the cerebrum [2] (Probst [3]).

Nothing is definitely known as to the *etiology*, but chill, over exertion, trauma, alcoholism, tuberculosis (Brissaud-Brécy) and *syphilis* in particular have been included among the causes. There is no doubt that syphilis has been at the root of a great number of the cases observed. Hereditary syphilis may also be a cause, in which case the disease is merely a part of a cerebro-spinal syphilis (Probst). Other supposed cases are really due to supposed syringomyelia. There has been some doubt as to whether we are justified in regarding the morbid process as an independent one. Foulon[4] maintains that it may have a rheumatic origin.

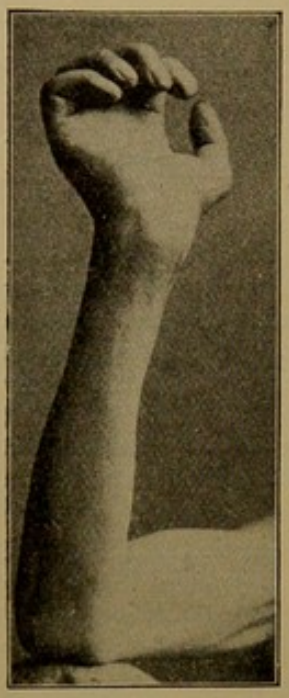

Fig. 155.—Position of hand in paralysis of the muscles innervated by the median and ulnar nerves. Type of "preacher's-hand" incompletely developed. (Oppenheim.)

The first *symptoms* are caused by the meningitis itself, and by the compression of the posterior roots. These are pain in the region between the shoulders, in the neck, and even in the occiput, a feeling of tension, and it may be of actual stiffness, tenderness of the cervical vertebræ to percussion, paræsthesia, and neuralgiform pains which specially follow the *course of the ulnar and median nerves*. There may sometimes also be symptoms of motor irritation, *e.g.* tremor and slight spasticity of the muscles of the upper extremities. This neuralgic stage may extend over weeks and months; it is followed by the stage of paralysis, which is at first of *neuritic* origin. But as the compression of the roots abolishes conduction and finally causes the roots to atrophy, it gives rise in time to symptoms of paralysis in the parts supplied by the lower cervical roots, *i.e.* in the region of the ulnar and median nerves. Hypæsthesia and even anæsthesia thus develop in the corresponding cutaneous areas, along with atrophic paralysis of the muscles supplied by these nerves or roots, in particular therefore the *small muscles of the hand and the flexors of the hand and fingers*, while the muscles supplied by the posterior interosseous nerve are almost or entirely intact. The preponderance of the extensors gives rise in most cases to a peculiar and almost pathognomonic position of the hands: hyperextension at the wrist-joint, extension of the basal and flexion of the middle and terminal phalanges (*main en prédicateur*, preacher's hand, Fig. 155).

From the cases reported we cannot gather much as to the presence

[1] Ziegler's "Beiträge," etc., xiii. and xix.

[2] Cases in which the pachymeningitis spread through the whole length of the cord have been described by Clarke and by Mills and Spiller, but the case of the latter can hardly be included in this group.

[3] *A. f. P.*, xxxvi.

[4] *Thèse de Paris*, 1900.

of oculo-pupillary symptoms, but we should expect to find the well-known changes in the pupils and palpebral fissures (dilatation followed by narrowing).

In a third stage, which we cannot sharply distinguish from the others, spinal symptoms occur—signs of an interruption of conduction in the lower cervical cord, viz., spastic paralysis of the legs, anæsthesia, bladder disorders, etc. The pain tends to become less severe during the later course.

The disease, which may last for years, may come to a stage of *arrest*. Complete recovery has also been observed (Charcot, Berger, Remak). In general, however, the *prognosis* is a grave one and the danger to life is great.

Modifications of the clinical picture are caused by the localisation of the disease in other parts of the spinal cord, in the middle or higher sections of the cervical cord, or in the dorsal portion. In the latter case the first symptom is pain extending over the region of several intercostal nerves; this is followed by anæsthesia of similar distribution, and eventually by paraplegia. The diagnosis, especially from tumour, is then naturally very uncertain. If the process extends to the pons and medulla oblongata, the symptoms will correspond. In one of my cases, for instance, there was bilateral deafness and tachycardia, and in one of Wieting's cases there were bulbar symptoms. The symptoms may also point to an involvement of the cerebrum.

Cases have been observed in which the pain was very slight (Köppen [1]).

If the lesion occupies its usual site, the *diagnosis* is not a matter of difficulty. Caries of the lower cervical vertebræ may of course give rise to very similar symptoms, but the affection of the vertebræ will be recognised, if not at the onset, at least during the further course, by the characteristics already described. It appears to me to be doubtful whether lumbar puncture may, as Widal maintains, be of value in the differential diagnosis of these two conditions. The disease may also be easily confused with tumours arising from the meninges of the cervical cord or from the cervical cord itself, as in the cases described by Collins and Blanchard, Soltmann, and by Schultze.[2]

Treatment.—Derivatives are of special value: *painting with iodine*, the *button cautery* to the neck, at the level of the fifth to seventh cervical vertebræ. Iodide and mercury should be specially prescribed if there is any suspicion of a syphilitic origin, and it is advisable to use these remedies in the other cases also. In the "rheumatic" form, salicylates are suitable (Foulon). Warm baths and diaphoretic treatment may also be beneficial. In one of the cases published by Remak, which I also saw, treatment by the galvanic current was of great service.

Chipault thinks that the cicatricial tissue should be removed by surgical operation, an opinion which does not seem very plausible in view of its adhesion to the spinal cord and roots. I have myself, however, seen, along with F. Krause, a case in which removal of a meningeal adhesion led to marked improvement.

[1] *A. f. P.*, xxvii.

[2] *Mitt. aus d. Grenzgeb*, xii.

Chronic Syphilitic Meningitis

(Gummatous Arachnitis, Syphilitic Meningomyelitis, etc., and other Diseases of the Spinal Cord of true Syphilitic Origin)

Literature: Consult Nonne, "Syphilis und Nervensystem," Berlin, 1902.

Syphilis of the spinal cord originates in the majority of cases from the meninges. The description of syphilitic meningitis is not, it is true, identical with that of spinal syphilis, but we think it expedient to treat all the syphilitic affections of the spinal cord in the same chapter.

Syphilis plays a most prominent part as a cause of diseases of the spinal cord. We shall not, however, here discuss those diseases of the spinal cord which have merely an *etiological* relation to syphilis, but shall consider only those which are *specific* from a pathological point of view.[1]

General *syphilitic meningitis* is the prototype of these affections. It arises from the lepto-meninges of the spinal cord, or less often from the internal surface of the dura mater. A granulation tissue develops in these, which spreads superficially, and leads to an opacity and thickening of the membranes, which become adherent to each other, to the roots, and to the spinal cord. In marked cases the whole extent or the greater part of the meninges are seen by the naked eye to be thickened and to be infiltrated by patches of suet-like, gelatinous, or fibrous tissue. Before the dura is opened, it appears in many cases to be swollen, either entirely or to a great extent. If an attempt is then made to separate the dura, a difficulty often arises on account of the adhesions. When the dura has been successfully separated, greyish-yellow patches, some of soft and some of firm consistence, are seen. These are distributed irregularly either as diffuse flat layers, or in a more circumscribed manner suggesting a tumour formation. On transverse section the cord with the adjacent membranes and roots shows changes which can be recognised even by the naked eye, but require *microscopical* examination to demonstrate their real nature.

Transverse sections through the spinal cord at various levels show a meningeal affection of varying intensity (Figs. 156-158). In some places the meninges are considerably thickened by a very vascular tissue, consisting of cells closely packed together. In other places the thickening is very slight and is due to a dense fibrous tissue. The process is by no means equally developed at all parts of the meninges; in many cases it is specially marked in the neighbourhood of the posterior columns. In the new formed tissue circumscribed portions are seen here and there in which there is a local *formation of gummata* (Fig. 158). This is often absent, however. The *spinal roots* are embedded in the new growth; in some parts they have a normal appearance, in others they are atrophied or infiltrated (Figs. 156, 157).

The spinal cord itself shows in some parts only slight changes in a narrow rim, *at its periphery*; in other parts it is severely affected, the

[1] Erb objects to this distinction on the ground that there are no criteria which definitely prove the specific nature of a morbid pathological process, and that we have therefore almost as much justification for regarding as syphilitic those simple inflammations and degenerations in the nervous system which develop in individuals who have had syphilis, as we have with regard to its so-called specific lesions (gummata, etc.). Further experience must show in how far the discovery of the *spirochæta pallida*, and the advances in cytodiagnosis and sero-diagnosis (Wassermann) will elucidate this question. We should also refer here to the theory of "syphilis à virus nerveux" (Lavallé, Fischler, *Z. f. N.*, xxviii.).

granulation tissue usually sending out processes from the periphery into the white matter in the form of wedge-like cones and buds. These penetrate more or less deeply into the cord and produce a condition of inflammation and atrophy in the surrounding nervous substance. In

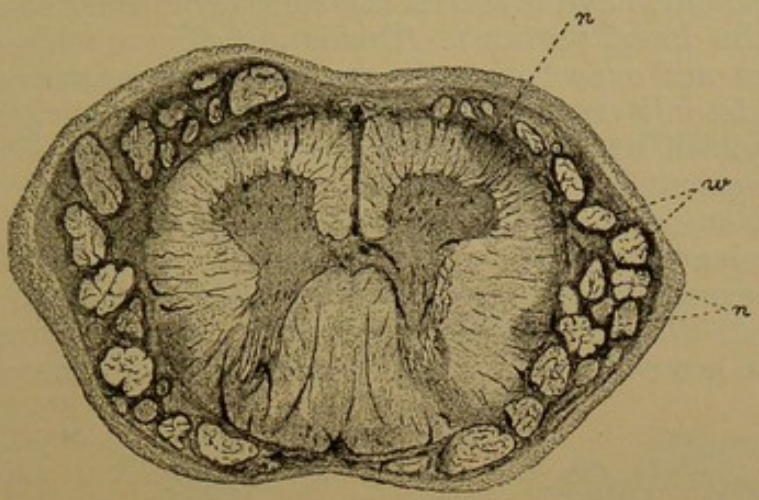

Fig. 156.—Syphilitic meningo-myelitis. *n*, new growth, *w*, roots. (From a section stained with carmine.)

other parts the cord is simply *softened*. The *vessels* are almost always involved; indeed, this vascular affection seems to be of essential importance, and in many cases to form the starting-point of the process (Raymond, Lamy,[1] Schmaus, Rosin, Singer, Preobrashenski). The walls of the arteries are thickened; indeed, there may be complete *obliteration*,

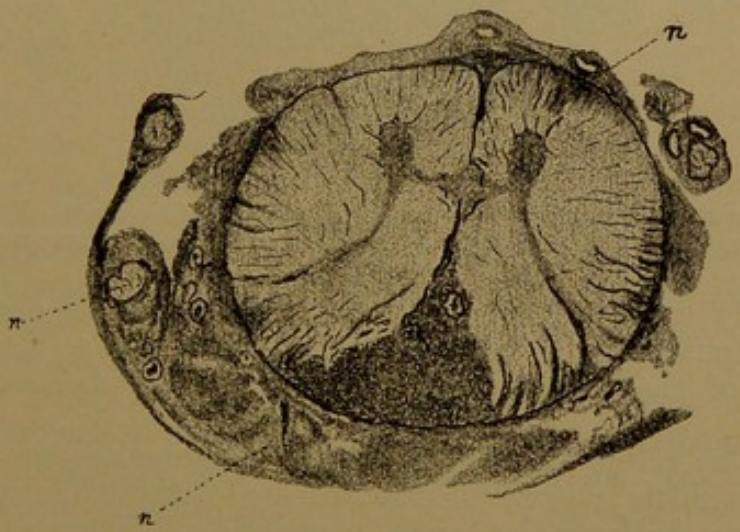

Fig. 157.—Syphilitic meningo-myelitis. *n*, new-growth which surrounds the extra-medullary roots and penetrates into the cord at various sites. (From a section from Oppenheim's collection stained with carmine and alum-hæmatoxylin.)

and the small vessels of the cord may be entirely consolidated. Changes pointing to *obliterating phlebitis* (Greiff, Rieder) are found in the veins. *Softenings* may develop from the vascular affection. The involvement of the circulation of the blood may lead to atrophic processes (Long, Wiki). The vascular disease seldom gives rise to large hæmorrhages (Williamson). The formation of cavities has also been observed.[2]

[1] *Nouv. Icon.*, 1893.

[2] By Eisenlohr, Oppenheim, Nonne, Schwarz, Wullenweber, Japha, Nebelthau, etc.

In some cases the affection of the meninges is found to be slight, whilst that of the spinal cord is severe. It is not impossible, however, that in such cases the meningitic process may have been materially improved under treatment. The meningeal affection may also, in spite of a universal development, have involved the spinal cord only at certain levels (specially in the dorsal portion). This is probably much more often the case than we should expect from the results of post-mortem examinations which are made only on the most severe cases. Finally, the meningeal affection may itself be limited to a certain portion of the cord—to a few consecutive segments.

Simple disseminated and diffuse myelitis, myelomalacia, poliomyelitis (?), and a disseminated formation of tumours may also develop from syphilis. In exceptional cases an *isolated gumma* has been found in the cord (M'Dowel, Wagner, Wilks, Osler, etc.). It very rarely represents the only lesion, however. Many cases described as gummata of the spinal

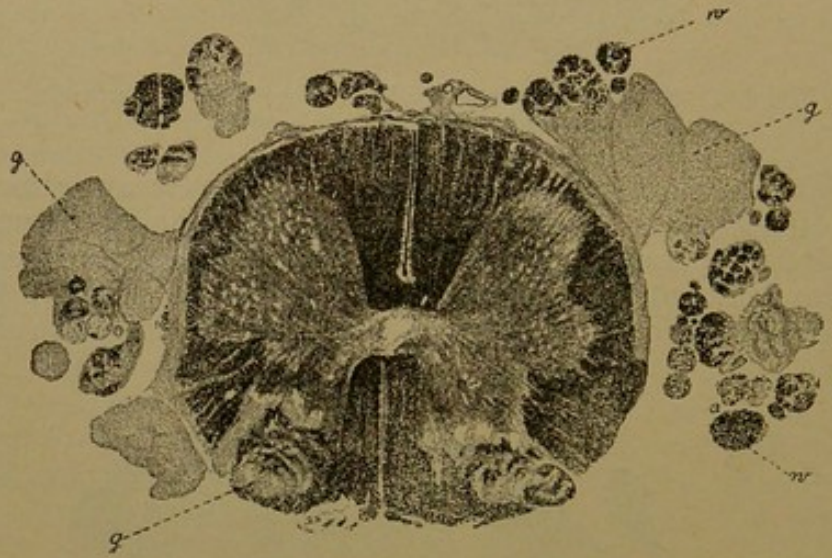

Fig. 158.—Gummatous meningitis and gummatous neuritis of the roots. Weigert's method. (From a preparation by Siemerling in Oppenheim's collection). *g*, gummatous tumour.

cord are really cases of the form of meningomyelitis described above (Hanot and Meunier [1]). Finally, a *gummatous neuritis* (Kahler [2]) limited to the roots of the cranial and spinal nerves has been described (see Fig. 159).

It has to be admitted that all these pathological processes do not prove the syphilitic nature of the disease, although, especially when combined, they indicate its great probability. There is in particular no definite histological characteristic to distinguish the disease from tuberculosis (Böttiger, Schamschin, Nonne, Flatau-Kölichen), but the presence of the tubercular bacillus on the one hand and of the spirochæta pallida on the other would set all doubt at rest.

Symptoms.—The symptoms of spinal syphilis follow somewhat rapidly on the infection. In not a few cases the disease appeared before the end of the first year; it has occurred within three months of the primary infection (Nonne), and in the great majority it develops within six years. *Hereditary syphilis* may also lead soon after birth or even in

[1] *Nouv. Icon.*, 1896. [2] *Z. f. Heilk.*, 1887.

later life to the development of a lesion of the cord. When treatment has been insufficient, spinal syphilis is specially apt to develop early. Chill, trauma, and infective diseases may also be exciting causes.

From the description of the pathological conditions the impossibility of describing a clinical picture which would apply to every case of spinal lues may be inferred. It will naturally vary according to the extension, intensity, and more or less rapid development of the process, to the involvement of the spinal cord at one or more sites, and even to the localisation of the point at which it has penetrated into the substance of the cord.

Certain symptoms and certain peculiarities of course are, however, as I [1] pointed out in 1889, sufficiently characteristic to serve as valuable diagnostic indications. The affection of the meninges gives rise to *pain* in the region of the back, neck, and sacrum. This pain is sometimes very acute and persistent, sometimes less severe, and is by no means always accompanied by any real hyperæsthesia.[2] According to Charcot it tends to be aggravated at night. The compression and infiltration of the posterior roots produce *radiating pains* in the area of certain nerve tracts: *e.g. girdle pain, neuralgiform pains in the extremities*. These may be very severe and may be localised at such different sites that they indicate a diffuse or disseminated distribution of the pathological changes. In many cases and stages of the disease, however, they are slight or absent. If compression of the anterior roots occurs only at the level of the superior and middle dorsal cord, it does not usually give rise to any prominent symptoms. Compression of the anterior roots of the inferior dorsal cord may cause symptoms of degenerative paralysis of the abdominal muscles (Kahler, Oppenheim). If the anterior roots of the cervical and lumbar enlargement are involved, there is *atrophic* paralysis of the arms and legs; this is almost always *partial*, being limited to certain muscles or groups of muscles in one extremity, as only single root bundles are severely affected by the pressure of the granulation tumour. Thus Dejerine and Thomas describe a syphilitic spinal meningitis originating in the eighth cervical and first dorsal root, the clinical picture being that of Klumpke's paralysis (*q.v.*). I have also often seen this form, as well as Erb's paralysis, and have lately observed a case in which the affection was limited to the extensor carpi ulnaris, the extensor communis digitorum, and the long muscles of the thumb, and was therefore due to a lesion of the seventh root. A strong rapid tremor, which I have observed in a few cases, is perhaps also due to this affection of the anterior roots and may be regarded as a symptom of irritation (although it may have been a purely accidental, neurasthenic symptom). Specific root disease may also develop at other sites, and may persist, although no symptoms appear which point to involvement of the spinal cord.

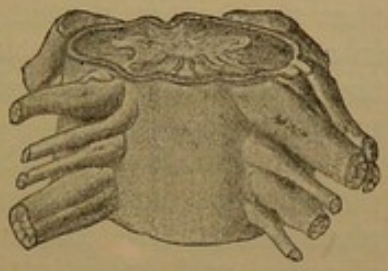

FIG. 159.—Syphilitic neuritis of the roots of the spinal cord. (After Buttersack.)

The most important symptoms, however, are caused by the *involve-*

[1] H. Oppenheim, "Zur Kenntnis der syphilitischen Erkrankungen des zentralen Nervensystems," Berlin, 1890.

[2] Symptoms of a simple meningeal irritation may appear as a transient occurrence in the secondary stage of syphilis. Recent investigations, especially cytological examinations, have pointed to the occurrence of an acute syphilitic meningitis. See also the *Thèse de Paris* of Drouet (1904).

ment of the spinal cord ; in many cases the disease only becomes apparent when this involvement takes place. As there is usually merely a partial transverse lesion of the cord, the symptoms are generally those of *incomplete interruption of conduction*, and consist in paralysis of one leg, *spinal hemiplegia*, *Brown-Séquard paralysis*, or *paraparesis* with excessive weakness of one leg.

After I had drawn attention to the great frequency of Brown-Séquard's paralysis in its incomplete, partial form in syphilis of the spinal cord, numerous cases of this kind have been described, especially by French writers (Lamy, Brissaud, Gilles de la Tourette, Raymond, Dejerine, Lépine, etc.). The symptoms of a " bilateral unilateral lesion "—double syndrome of Brown-Séquard—due to this cause have also been observed (Hanot-Meunier, Brissaud,[1] Oppenheim). Jolly,[2] however, in a case of this kind found a simple myelitis instead of the specific process which he suspected.

This paralysis is sometimes *spastic* (and, according to Erb, the spasticity is not necessarily severe though the tendon reflexes are greatly exaggerated), or it may be flaccid and atrophic. It is quite intelligible that the former should be the more usual, but the extremities which are in a condition of spastic paresis may show a few atrophied muscles. In these cases the Babinski and the Oppenheim signs are as a rule very pronounced.

The functions of the *bladder* and *intestine* are affected in the great majority of cases, even when the symptoms indicate merely an incomplete transverse lesion ; there may indeed be complete incontinence of urine and fæces, although in the extremities the paralytic symptoms are comparatively slight. *Sensibility* is almost always impaired. Paræsthesiæ are practically constant, whilst the objective diminution of sensibility is usually inconsiderable ; it may extend to all the qualities of sensation or to some of them only (*e.g.* there may be simply diminution of the temperature sense).

This *co-existence of meningeal, root, and cord symptoms*, this *multiplicity* and *incompleteness* of clinical signs are in themselves somewhat characteristic, but the most convincing evidence of the specific nature of the process is furnished by the course of the disease, the *inconstancy*, the remissions, and the fluctuations in the severity of the symptoms. Thus there may be a sudden onset of paresis in one or both legs, or the paresis may, without warning, develop into paraplegia, which again may as rapidly change back into paraparesis or to paresis of one leg. I have, for instance, seen one case in which, during the course of a few weeks, there were four attacks of paraparesis, and another in which the spinal hemiplegia came and went three times during a short period of time. As one examines the case marked fluctuations in the intensity and extent of the motor disorders may be found from day to day. The bladder and sensory symptoms also show the same variability. This *fluctuation* is particularly characteristic with regard to the *tendon reflexes* ; thus the knee jerks were in some cases quite absent at certain times, present a few days later, and at other times were markedly exaggerated. In one case of this kind I found the posterior roots in the upper lumbar region embedded in granulation tissue and partially atrophied, and I thought that this variability of the symptoms might be due to alterations of pressure within the membranes. Although this variability may occur in other diseases, such as sarcomatosis of the spinal cord (Nonne,

[1] *Prog. méd.*, 1897. [2] *A. f. P.*, xxxvii.

Mamlock [1]), it has been very pronounced in many cases of spinal syphilis. *Ataxia* is a not uncommon symptom, and it may also show the same inconstant character.

Finally, we must refer to one important factor. In many instances a similar disease develops in the brain, *i.e.* in the *basal meninges* and *vessels*, the symptoms of which may accompany or precede the spinal form. This *cerebro-spinal meningitis* represents in my opinion (my cases being supported by those of Siemerling, Eisenlohr, Sachs, Gerhardt, Pick, Homén, Henneberg, Nonne, Kopczynski, and many other writers) one main form of syphilitic disease of the nervous system. The brain symptoms are, it is true, usually so very prominent that the spinal symptoms are masked and overlooked. Gerhardt [2] draws attention to the triplegia which may exist, *i.e.* the paralysis of three extremities, which arises from the union of a hemiplegia and a paraplegia. Williamson also mentions this symptom.

We need hardly expound in any detail the fact that the clinical picture varies naturally according as this or that segment of the spinal cord is chiefly involved. As *dorsal* foci are common, *spastic paresis* occurs frequently. A dorso-lumbar localisation is by no means rare.

We shall only refer to a few special varieties of spinal syphilis. In the first place there are cases which correspond throughout to the picture of an acute, subacute, or chronic *myelitis* (*q.v.*), which have a progressive and often rapid course, but which may become arrested or even improved (Leyden, Schultze, Oppenheim, Goldflam, Pick, Singer, etc.).

The pathological process in such cases may also correspond to that of a simple myelitis, or the sudden supervention of a myelomalacia upon the hitherto latent meningeal and vascular changes may manifest itself merely as an acute paraplegia of very sudden onset, etc. (Brissaud).

Preobrashenski (*Korsakoff's Journ.*, iii.) attempts to draw a clinical picture due to a thrombosis of the anterior spinal artery. Dejerine (*R. n.*, 1906) has pointed out that these conditions may correspond to a kind of "intermittent claudication" of the spinal cord, as the spastic-paretic phenomena only develop during walking, and are absent or very slight during rest. Sollier (*Presse méd.*, 1906) also reports cases of this kind.

Under the name of *syphilitic spinal paralysis*, Erb [3] has classified a group of cases which he describes as follows: There is a gradual development of a spastic paresis of the legs, with great exaggeration of the tendon reflexes. The gait is markedly spastic, but the muscular hypertonicity is slight. The functions of the bladder are almost constantly impaired. The sensibility is affected, though as a rule but slightly. The course is usually chronic, showing a tendency to improvement, to remissions, and to a complete arrest. The disease may extend over many years, ten or more.

There can be no doubt, from our own experience [4] and that of many writers such as Gerhardt, F. Pick, Sachs,[5] Muchin,[6] and others, that this clinical picture has very often a syphilitic origin, but it is simply the picture of a partially developed or incomplete dorsal myelitis, and therefore of itself presents no feature characteristic of syphilis. It undoubtedly, however, often represents merely the first stage or an incomplete picture of the clinical form of cerebro-spinal syphilis, which I have described

[1] *Z. f. kl. M.*, Bd. xliii. [2] *B. k. W.*, 1893. [3] *N. C.*, 1892.
[4] H. Oppenheim, "Über die 'syphilitische Spinalparalyse,'" *B. k. W.*, 1893.
[5] *Br.*, 1893. [6] *N. C.*, 1894.

above, and receives in such cases from the accessory, especially from the brain symptoms, the headache, oculo-motor paralysis, pupil anomalies, etc., a stamp characteristic of syphilis. The combination with immobility of the pupils, for instance, was noted by myself, F. Pick,[1] Cestan, and Nonne. Such cases are mostly due to syphilitic meningo-myelitis, the process being localised chiefly in the dorsal region, or they may represent a certain stage of the disease.

Nonne,[2] however, has shown, in view of a few earlier observations (Westphal, Minkowski, Eberle) and some of his own, that the clinical symptoms of syphilitic spinal paralysis may be caused by a combined systemic disease of the pyramidal tract, the cerebellar tract, and Goll's column. A case of Harder's belongs to this form. Nonne agrees with Trachtenberg [3] that these are cases of systemic disease produced by the toxin of syphilis, but he admits that the clinical picture may also arise from other causes. He [4] himself found diffuse or disseminated processes and vascular changes along with the systemic disease. Erb [5] has practically accepted Nonne's view.

In other rare cases the resemblance of spinal syphilis to tabes dorsalis is, as I [6] have shown, so great, at least in one stage of the disease, that I considered the term of *syphilitic pseudo-tabes* justifiable. This is practically due to the fact that the specific process extends from the meninges mainly to the region of the posterior columns, and to the posterior roots, so that Westphal's sign, ataxia, lightning pains, and bladder troubles, etc., represent the chief symptoms. Brain symptoms analogous to those in tabes were also prominent in these cases—oculo-motor paralysis, reflex immobility of pupils, paralysis of the laryngeal muscles, nervous deafness (Eisenlohr), etc., anæsthesia in the trigeminal region, caused partly by basal meningitic and gummatous-neuritic processes, partly by simple atrophy of the corresponding cranial nerves or their roots. Cases of this and similar kinds are reported by Eisenlohr, Brasch, Valentin, Ewald, Collins, Garbini, Camp, etc. There is no doubt, however, that a true tabes may be associated with a syphilitic disease of the spinal cord (Dejerine, Sachs, Hoffmann-Kuh, Dinkler) and may perhaps precede the latter.

Guillain and Thaon [7] draw attention to these combinations and mixed forms.

The clinical picture of spinal syphilis may remind one of that of gliosis, of amyotrophic lateral sclerosis, etc., but as a rule there are other symptoms which are foreign to these affections. I have seen two cases in which the combination of a (unilateral) atrophic paralysis of the hand muscles with partial sensory paralysis entirely corresponded to the picture of gliosis, and which completely recovered under treatment with inunctions and iodide. Bechterew describes a cerebro-spinal focal sclerosis of syphilitic nature, but he admits that it is not identical with disseminated sclerosis (*vide infra*). Catola,[8] on the other hand, has described a few cases of syphilitic etiology, which presented the clinical picture of disseminated sclerosis with some peculiarities (such as aniscoria, sluggish reaction of the pupils), and could not be distinguished in their pathological anatomy from true focal sclerosis.

[1] *Prag. med. Woch.*, 1898, and *Z. f. Heilk.*, xiii. [2] *A. f. P.*, xxix., and *Z. f. N.*, xxxiii.
[3] *Z. f. kl. M.*, 1894. [4] *Z. f. N.*, xxix. [5] *Z. f. N.*, xxiii.
[6] *B. k. W.*, 1888. [7] *Soc. de Biol.*, 1905 : *R. n.*, 1906. [8] *Nouv. Icon.*, 1906.

Symptoms have occasionally been observed in syphilitic persons which corresponded to the picture of acute and chronic poliomyelitis (Dejerine, Eisenlohr, Reynolds, Nonne, and Oppenheim). Although we have no definite proof that these affections may arise from specific causes, we cannot *a priori* deny that both the true syphilitic processes and the toxins may occasionally be limited in their action to the anterior grey matter.

Finally, we may mention a special localisation of specific meningitis —namely, in the neighbourhood of the *cauda equina*. In the cases of this kind hitherto observed (Westphal, Eisenlohr, etc.), the roots had become adherent to each other and to the meninges. The symptoms were radiating pains in the region of the sacral nerve, especially of the pudic nerve, therefore in bladder, intestine, perineal region, penis, etc. This was followed by anæsthesia of similar distribution, paralysis of bladder and intestine, and impotence. I have seen these symptoms disappear in one case under treatment by inunction, whilst in another (described by my then assistant, G. Koester[1]) considerable improvement, almost recovery, followed the same treatment. Here, however, there was probably a specific disease of the conus terminalis.

In another severe case under my care, in which the gluteal muscles were also affected, the paralysis of the anal sphincter had led to marked prolapse of the rectum.

The same anatomical changes in the cord take place in hereditary as in acquired syphilis (Money, Jürgens, Gasne, Collet, etc.). The symptomatology is also essentially the same as in the acquired forms. The typical clinical picture therefore corresponds to that of syphilitic cerebro-spinal meningitis (cases of Siemerling,[2] Bury, Boettiger,[3] Oppenheim, Richon, etc.). The cerebral symptoms are as a rule the most prominent. A syphilitic disease of hereditary origin limited to the spinal cord is apparently very rare, but clinical cases of this kind have been published (Gilles de la Tourette). I have seen one case of this kind in which, in addition to the symptoms of spastic spinal paralysis, there was merely rigidity of the pupils, and another where there was also a degree of mental weakness.

Peters describes paresis in the upper extremities corresponding to plexus paralysis, in which a kind of "swimming attitude" was produced by the paralysis of certain groups of muscles (extensors of hand, supinators, etc.), and the excessive action of others. He does not, however, appear to have taken Parrot's pseudo-paralysis sufficiently into account as regards the diagnosis. Further, both congenital spasticity of the limbs (Little's disease) and the diseases corresponding or allied to tabes dorsalis and occurring in childhood and later life have been ascribed to hereditary syphilis. With regard to the former disease we should specially keep in mind the observations of Friedmann, Moncorvo, Vizioli, Gallois, etc. I have notes of similar cases. Some affections resembling cerebro-spinal insular sclerosis may also be due to hereditary syphilis (Moncorvo, Carrier, De Sanctis), and a disease allied to Friedreich's has been noted by myself and by Bayet in hereditary syphilis.

That symptoms corresponding to syringobulbia and syringomyelia may also develop on this basis and be considerably improved by anti-syphilitic treatment is shown, or at least made probable by a case under my own observation.

Differential Diagnosis.—When no history is obtainable, the diagnosis can seldom be made from a single examination. It usually requires consideration of the course of the disease or prolonged observation. On the other hand, the possibility of a syphilitic basis should be kept in mind in every diffuse—one might almost say, in every—disease of the spinal cord. The tendency to remissions during the course of the disease, the

[1] *Z. f. N.*, ix. [2] *A. f. P.*, xx. [3] *A. f. P.*, xxvi.

incompleteness of the paralytic symptoms, the variability of the individual symptoms, the intercurrent onset of cerebral symptoms, and the circumstance that the symptoms cannot usually be traced to a single focus, are particularly characteristic. These signs do not occur in simple myelitis, nor in compression myelitis. But we must not expect them to be present in every case of spinal syphilis. The relapsing course is common to this disease and to disseminated sclerosis, but in the latter meningeal and root symptoms are absent, and in spinal and cerebro-spinal syphilis the characteristic tremor and the scanning speech do not occur, and nystagmus is unusual. It has been amply shown, however, by myself, Sachs,[1] Cassirer,[2] Pini,[3] Krewer,[4] Blumenau, Widal, Sicard, Babinski, Bélètre, etc., that the differentiation between these two diseases may occasionally present great difficulties. *Cytodiagnosis* does not definitely determine the question, as lymphocytosis has been found by Carrière and others in disseminated sclerosis also. On the other hand, Wassermann's seroreaction may be regarded a definite proof of syphilis. The observations of Bechterew and Catola already mentioned seem to prove that a process closely allied to multiple sclerosis may develop as the result of syphilis.

Spinal syphilis has in many cases a great resemblance to combined disease of the posterior and lateral columns, but in the latter the course is as a rule steadily progressive. In spinal syphilis symptoms of degenerative paralysis are not infrequently absent, and the cerebral symptoms are exclusively those of tabes (rigidity of pupils, oculo-muscular paralysis, atrophy of the optic nerve, etc.), whilst in cerebro-spinal syphilis neuritic processes (optic neuritis) and the signs of a focal brain disease are also observed.

Diffuse sarcomatosis of the spinal cord and brain or of the cerebro-spinal meninges may also give rise to a morbid picture which is almost entirely identical with that of cerebro-spinal syphilis. The tendency to remissions and to abrupt, violent exacerbations is, however, much less pronounced.

That tubercular cerebro-spinal meningitis may occasionally give occasion to confusion is shown by a case of Hensen's (*Z. f. N.*, xxi.). A feverish course does not exclude the syphilitic basis, as seems to be shown by a case reported by Dorendorf and by the cases quoted by Drouet in his thesis Feverish attacks are certainly extremely rare in syphilitic meningitis.

It is not yet certain whether serous spinal meningitis (Oppenheim-Krause) may be caused by specific disease ; this affection should in any case be included among those for diagnostic discussion.

Course and Prognosis.—There are cases of spinal syphilis which run their course within some weeks or months and then terminate in death or in complete recovery. These form the minority. The course is usually *chronic, remittent* ; the condition requires some months or even years for its development, but from time to time there are spontaneous improvements or even arrests, until a relapse occurs, with or without any apparent exciting cause.

The prognosis is decidedly more favourable than in the other, non-syphilitic, diffuse diseases of the spinal cord. Complete recovery takes place, it is true, in only a small percentage of cases. This may be looked for especially in those cases which show only meningeal and root symptoms. I have seen complete recovery in a great proportion of such cases. Involvement of the cord does not of itself exclude perfect recovery, if there is merely an incomplete transverse lesion from the penetration of the

[1] *New York Med. Journ.*, 1891, and *New York Med. Assoc.*, 1898.
[2] *D. m. W.*, 1896. [3] *Z. f. N.*, xxiii. [4] *Z. f. kl. M.*, 1901.

granulation tissue along the vessels. But if atrophy has taken place in the neighbourhood of the focus, or if a secondary myelitis or softening has developed, we can no longer expect a perfect recovery of the damaged part ; a softening or a cicatrix will in any case be left behind, even should all the rest be absorbed. From a clinical standpoint we may say that the prognosis is the more favourable the less marked the spinal symptoms of paralysis are and the shorter the time they have existed. Recovery is not impossible, however, in cases showing symptoms of a more or less complete interruption of the conduction, or in other words where there is paraplegia. But the symptoms must not have existed for any considerable time, for many months. Even should we ultimately succeed in curing the paralysis due to a primary transverse lesion, a secondary degeneration which it is impossible to arrest will have meantime developed.

During the process of cicatrisation the disappearance of some symptoms will be accompanied by the increase of others. Thus, in one case I have seen a Brown-Séquard paralysis disappear under treatment, whilst the girdle pains increased considerably in intensity.

Inflammation or softening of the lumbo-sacral cord gives rise to severer symptoms than when the dorsal cord is affected.

Trevelyan saw an almost complete recovery apparently under the influence of an erysipelas which was treated with anti-streptococcic serum.

In the majority of cases the improvement attained stops more or less short of complete recovery. The meningeal and root symptoms disappear under the influence of treatment, but there remain certain signs of spinal lesion, most frequently a spastic paresis, weakness of the bladder, impotence, etc. Relapses are always to be expected, and although the illness may extend over ten to fifteen years or more, life is generally shortened.

In not a few cases the disease has a progressive course and terminates in death after a comparatively short interval. These include cases in which there has, from the commencement, been a diffuse myelitis or an extensive softening in the cord, as well as severe cases of disseminated cerebro-spinal syphilis in which the medulla oblongata (the vagus) or the cerebrum are involved.

The hereditary form of cerebro-spinal syphilis affords on the whole a less favourable prognosis, although I have obtained most satisfactory results in a few cases.

Treatment.—In every case anti-syphilitic measures should be instituted without delay. According to the urgency of the case we should use inunctions of 3-5 g. of ung. hydrarg. cinereum, and give iodide of potassium in increasing, or from the first large doses (40 to 50 or even 200 to 300 grains per day). There is no doubt that we can attain the same results with other perparations of mercury and with subcutaneous injections. I have seen much good lately from the use of enesol. I have used iodipin internally, and especially in subcutaneous injections (10-20 g. of 25 per cent. solution daily for eight to ten days) with success, even in a few cases in which Hg and iodide of potassium had failed. As regards sajodin and iodglidin I can as yet speak only with reserve ; these require further testing. If no special incidents occur, the treatment should be continued until recovery takes place. This is not usually feasible, at least not

without interruptions ; the improvement continues up to a certain point, beyond which it does not go. We can, however, prescribe about 250-300 grammes of the ung. hydrarg., and more, even in the first period of treatment. Then an interruption is usually necessary, but the treatment may be resumed after a few months, and when relapses or fresh symptoms appear it must be repeated. If recovery or a permanent arrest has been attained, it is necessary to repeat the treatment at least once every year. I know cases of spinal syphilis in which several thousand grammes of grey ointment were used in the course of a few years without causing any marked effect upon the health.

An objection to the energetic mercurial treatment of spastic paralysis in syphilitics, which it is difficult for me to understand, has been raised by Brissaud and Marie. Of late years the attempt has often been made to inject mercury and preparations of iodine directly into the spinal canal by means of lumbar puncture or of Cathelin's method (Corning, Jacob, Sicard, A. Strauss, Schachmann, etc.). Nothing can be definitely said as to the value or justification of this method, in spite of the results obtained in a few cases.

There is more required, however, than specific treatment alone. Nourishing diet is called for in every case. The avoidance of *chills, injuries, over-strain, and sexual excesses* cannot be too stringently advised. Relapses have been observed after the application of cold douches to the back, after hot baths, sexual excesses, hill-climbing, etc. Even although the disease is taking a favourable course, marriage should always be prohibited, or those undertaking it should be made fully aware of the danger and importance of the step.

In many cases mercurial treatment is only efficacious when accompanied or followed by a mild course of cold-water baths or of luke-warm baths and indifferent waters. The sulphur baths of Aachen, Nenndorf, Baden, and Weilbach are particularly favoured. The bathing treatment always requires special care and constant supervision by the physician. Diaphoretic measures may be of good service, especially when combined with specific treatment. In a few cases a prolonged residence in the south had the desired effect ; it was only during or after it that the anti-syphilitic treatment was crowned with success.

In the spastic forms a moderate amount of walking may be permitted, or even recommended, after the more active stage is past, but it should never lead to over-fatigue ; the patient should rest after walking a short distance, etc.

In cases of this kind, in which the specific treatment has ceased to do good, the application of *derivatives to the back* (points de feu) may induce a certain improvement. I have seen a case, for instance, in which this proceeding, repeated every second week, was always followed by marked improvement. *Electricity*, especially galvanic treatment of the spinal cord, is often very beneficial, and mild *massage*, passive movements in warm baths, may be recommended for the spastic condition.

Surgical treatment—removal of the gummatous accumulation and deposits—has only been employed in a very few cases (Waterman). It can only be suitable in very exceptional cases and with little promise of success. But if specific treatment is unavailing, the uncertainty of the diagnosis may warrant an exploratory laminectomy.

C.—Primary Diffuse Diseases of the Spinal Cord.

Myelitis

Literature: Leyden, "Klinik der Rückenmarkskrankheiten," ii.; *ibid.*, *Z. f. kl. M.*, i.; Oppenheim, *B. k. W.*, 1891; Leyden, *D. m. W.*, 1892; Bruns, article on Myelitis in Eulenburg's "Real-enzyklopädie"; Pfeiffer, *Z. f. N.*, vii.; Hochhaus, *Z. f. N.*, xv.; Redlich, Review in *C. f. allg. Path.*, 1898, and *Verhandl. d. xix Kongr. f. inn. Med.*, 1901; Mager, "Obersteiner," vii.; Marinesco, *Nouv. Icon.*, 1900; A. Pick, *Handbuch der path. Anat. d. Nerv.*

If this designation coincided exactly with the conception of inflammation of the spinal cord, we should have to include under this title a great number of clinically different forms of disease. There has, however, been a gradual abandonment of this wide conception of myelitis. With the deepening and widening of our knowledge of the pathology of the spinal cord in the last twenty years, the conception of myelitis has become more and more restricted as, from time to time, various diseases have been detached from it, these having been recognised as separate morbid entities on account of their special pathological anatomy and symptomatology. An exact delimitation is not at present possible. We shall, however, be approximately correct if we regard as myelitis *the diffuse and disseminated processes of inflammation and softening in the spinal cord.*

Although processes of softening, which are to be considered as necrobiosis from vascular occlusion, sometimes occur in the spinal cord and have been observed in embolic occlusion of the abdominal aorta, in embolism, and especially in thrombosis of the arteries of the spinal cord (Marchand-Tietzen, Homén, Malbranc, Gowers, Weiss, Heiligenthal, Nauwerck, Petrén, Brissaud, D. Singer, B. Sachs, Stanilowsky, Schlapp, Mariani, Dinkler, experimental observations by Brieger-Ehrlich, Singer, Lamy, Rothmann, Katzenstein,[1] and others), yet these are practically unimportant and cannot in general be clinically distinguished from myelitis, or only occasionally as in Heiligenthal's case, when specially characterised by the sudden onset of paralytic symptoms. The boundary between disseminated sclerosis and myelitis is indeed an indefinite one, but yet the two diseases may be easily separated, if we leave out of account the transition type (see below). There is a form of Landry's paralysis, which is due to myelitic processes, but this disease occupies a special place both on account of its clinical character and its pathological anatomy.

With regard to the so-called "funicular myelitis" (Henneberg[2]) see p. 188.

Myelitis is a somewhat common disease. I myself had been led to underestimate its frequency when I drew my conclusions solely from observations at hospital and took my material from a clinique for nervous diseases, where only a small number of recent, acute cases were received. More extended experience has convinced me that the disease is not a rare one. It is associated with no particular *age*. Persons in middle life are most frequently affected. In childhood true myelitis is less common than poliomyelitis. The senile paralyses of the spinal cord require special consideration.

Etiology.—Older writers name as the causes of myelitis, physical over-strain, emotional excitement, sexual excesses, trauma, and very specially *chill*. The significance of these factors becomes, as I showed a number of years ago, more and more open to question in the light of recent observations, although they are on the one hand recognised as accessory and exciting causes, and although recent literature does

[1] *A. f. kl. Chir.*, Bd. lxxvi. [2] *A. f. P.*, Bd. xl.

contain isolated observations in which chill (as in a case of Dreschfeld's) and trauma (Schmaus, Westphal, Grandmaison, Spiller, Hartmann, Nonne, and others) are given as the direct causes of myelitis or of a disease which corresponds to it clinically.

But these factors are quite insignificant in comparison with *infection* and *intoxication*, the etiological importance of which is definitely established beyond any doubt by clinical and experimental observations. It is certain that myelitis may develop after an *acute infective illness* :[1] small-pox, scarlatina, influenza, measles, erysipelas, pneumonia, whooping-cough, dysentery, typhoid, cholera, diphtheria, tonsillitis, etc. The observations of Gubler, Imbert, Westphal, and Ebstein first showed this connection, and then followed those of Leyden-Renvers, Lenhartz, Putnam, Henschen, Schiff, Hochhaus, Pontoppidan, Friedmann, Eliot, Spiller, Lépine, Luzzatto, and others. The disease was traced by Bruns to chicken-pox, by Specker to a septic infection, by Kowalewski to inoculation against rabies, and by Strümpell to a whitlow. In one case under my observation the myelitis was to all appearance due to a suppuration of the antrum of Highmore. Thiroloix and Rosenthal have seen a spinal cord affection, which corresponded to a myelitis, make its appearance in endocarditis ; I have had a similar case (see Fig. 167). The affection should perhaps be regarded as a primary infective disease in the cases of Küssner and Brosin, Achard and Guinon, and others. I myself have seen several cases which might be so interpreted. The form of myelitis which occurs in pregnancy (Kast) and in the puerperium also appears to be of an infective nature. Hösslin[2] has quite recently studied the question. Gonorrhœa may result in a myelitis or a meningo-myelitis (Gull, Barrié, Leyden, Dufour, Kalindéro, Labré, Koelichen). In one case I have seen it develop immediately after vaccination. Dinkler[3] found a transverse myelitis along with appendicitis. Holst saw it occur after septic peritonitis. It also not infrequently follows syphilis, and sometimes accompanies tuberculosis. These forms are to be distinguished from the true syphilitic or tubercular diseases of the spinal cord, in which the specific new growths (solitary tubercle and nodular tuberculosis of Raymond) appear in the cord itself either as primary foci or as extensions from growths in the neighbourhood. We observe in syphilitic and tubercular cases another form of myelitis, which, neither clinically nor anatomically, is distinguished as specific. The cases of this kind which I have reported agree with those observed by Clément.[4] Malaria may also lead to myelitis. The myelitis or meningomyelitis which occasionally follows lumbar anæsthesia (F. König, Walther, and others) is due to a direct infection or the action of a chemical poison.

Isolated observations which I myself have made and have mentioned in my treatise on myelitis, point to a relation between this disease and cachexia due to tumour. Lubarsch and Nonne have also confirmed this. A case by Ballet and Laignel-Lavastine seems to belong to this group. Of the spinal cord affections which arise from *pernicious anæmia, leuk-*

[1] It is worthy of note also that Voinot (*Thèse de Nancy*, 1897), who examined the spinal cord of individuals who had died from various infectious diseases without presenting any spinal symptoms, was able to recognise numerous changes.

[2] "Die Schwangerschaftslähmungen der Mütter," *A. f. P.*, xxxviii. L. Brauer speaks of a pregnancy toxonosis, in reference to a case which he regards as cervical myelitis, but in which there was no pathological examination. See also Rosenberger and Schmincke (*V. A.*, Bd. clxxxiv.), and E. Taube (*Inaug. Diss.*, Berlin, 1905).

[3] *Z. f. N.*, xxvi.

[4] *Lyon méd.*, 1905.

æmia, etc., we have already spoken (see pp. 188 *et seq.*). Their connection with diseases of the urinary apparatus (paraplegiæ urinariæ) is still unexplained. In so far as the myelitis is due to infective processes—which it probably always is—it may be either the direct manifestation of an acute infective disease or the result of the invasion of the cord by a continuous spread of the infective agent through the cellular tissue and the lymph channels.

The origin of myelitis in an ascending neuritis is also not yet certainly proved. I have not been able in any single case to convince myself of the fact that a simple neuritis ascending along a nerve tract to the spinal cord has given rise to a condition of myelitis. Other writers, such as Charcot and Leyden, however, have recently supported this theory of its causation, on the evidence of observations by Bompard, Shimamura, and themselves.

There remain not a few cases in which no cause could be assigned for the myelitis (Hochhaus and others). According to Strümpell, there is always some *exogenous* factor.

Bacteriological examination has led to positive results in only a few cases of myelitis, the number increasing, however, of late. Streptococci and staphylococci were found by Eisenlohr, Barrié, Marinesco, Babes, Thiroloix-Rosenthal, Tooth-Russell, and others. Fürstner found evidence of pneumococci in the myelitic foci, confirmed by Buzzard, Russell, and Marinesco. In one case in which the myelitis had followed a whitlow—undoubtedly by means of or along with an external purulent pachymeningitis—Strümpell [1] found a staphylococcus in the cerebro-spinal fluid obtained by puncture. In another case, however, the fluid was free from micro-organisms, so that Strümpell assumed that the disseminated form of myelitis might have a hæmatogenous toxic origin. Finkelnburg found evidence of a diplococcus in the fluid in one case, and Magnus reports a similar discovery. The rarity of such findings is partly explained by the fact that micro-organisms rapidly disappear from the spinal cord, as has been shown by the experimental examinations of Homén, Hoche, and Marinesco. On the whole a few *specific* exciting causes of infection, such as mixed or secondary infections (Grasset), the so-called ordinary infections, and especially the toxins of micro-organisms, appear to be the originating causes of myelitis.

Experimental investigations of this kind have been made, especially by French writers—I will name only Bourges, Roger, Vincent, Besançon and Widal, Thoinot and Moselli, Crocq, Babinski and Charrin, Gilbert and Lion, Enriquez and Hallion, Phisalix and Claude—and also by Marinesco, Ritter, Moltchanoff, and others, and they have succeeded in producing inflammatory affections of the spinal cord, by introducing cultures of bacilli (bacillus pyocyaneus, staphylococcus pyogenes, Löffler's typhoid bacillus, erysipelas streptococci, diphtheria baccilli, bac. coli, etc.), or their toxic products into the bodies of animals. Salle (*Z. f. N.*, xxxi.) attempted to demonstrate experimentally the paths by which the infective myelitis became extended through the spinal cord.

Hoche [2] and Marinesco [3] have lately studied this question thoroughly and have demonstrated the important fact that embolic foci produced experimentally form a locus minoris resistentiæ in the cord, where micro-organisms circulating in the blood may settle and give rise to foci of myelitis.

Morbid conditions have been seen to follow simple intoxications—

[1] *N. C.*, 1901. [2] *A. f. P.*, xxxii. [3] *R. n.*, 1900.

with CO, ordinary gas, bisulphide of carbon, chloroform, nitrobenzine —which apparently belong to this class.

Symptomatology.—The typical form of myelitis is *transverse myelitis.* We shall start our consideration from a fully developed case, and shall assume that the disease has its site, as is usual, in the dorsal region (dorsal myelitis). This segment of the cord is most frequently affected for the reason that it forms the greater part of the organ. It is also less favourably situated than other regions of the spinal cord as regards its vascular nutrition (Kadyi). The symptoms are approximately such as would be produced by section of the spinal cord at a corresponding level, namely :

1. *Paraplegia.*—The legs are paralysed, the paralysis being, as a rule, accompanied by *rigidity*, as well as by *exaggeration of the tendon reflexes.* At the commencement they are usually in a position of extension, whilst in later stages there is sometimes a flexion contracture, the knees being flexed and the thighs firmly drawn up against the trunk. More frequently the position is a varying one, *i.e.* there appear spontaneous movements and spasmodic contractions in the paralysed limbs, which are thus at one time extended, at another time flexed. The contracture may, moreover, be so severe that the tendon reflexes can no longer be elicited, or it may be exaggerated to this degree even by a mere touch.

2. *Anæsthesia.*—The sensibility may be lost (or diminished) in the legs and at various levels of the trunk, according to the site of the disease, and usually in all its qualities. The upper margin of the anæsthetic area is formed by a zone in which the patient sometimes feels girdle-pain. Slight hyperæsthesia may also be objectively evident.

3. The *reflex excitability* is retained in the legs, and is generally even exaggerated ; a slight touch on the sole of the foot or even a breath of air is sufficient to call forth severe reflex contractions. The modification of the plantar and leg reflexes which is characteristic of spastic paralysis, is usually also present (Babinski, Oppenheim). In a disease which is equivalent to complete transverse destruction of the cord, or other conditions which were described on pp. 115-116, the reflexes and tendon phenomena may entirely disappear.

4. *Paralysis of the Bladder and Rectum.*—The patient has no longer the power to influence these functions by his will, nor has he the sensation of the condition of fulness in the bladder and rectum. This leads to retention of urine or incontinence of urine and fæces (compare pp. 118 *et seq.*). The patient becomes impotent. Priapism sometimes occurs, or erections take place during catheterisation.

5. *Bed Sores* (decubitus).—Pressure, loss of sensation, and soiling from excreta are the factors which combine to produce simple or gangrenous ulcers, especially over the sacrum, the trochanters, or the heels, etc. These are sometimes superficial in nature, but sometimes penetrate so deeply that the bones are laid bare, and the pus or sanious matter makes its way even into the spinal canal. *Trophic disturbances* are possibly also a factor in the production of these ulcers. Pemphigus and cutaneous affections of other kinds are also occasionally observed.

It remains to be added that the muscles of the paralysed limbs usually retain their normal size and their normal electrical excitability, even after the paralysis has existed for a considerable time.

Vasomotor and *secretory* disturbances may supervene ; thus œdema is not uncommon in the paralysed limbs, etc. The sweat secretion is

often abolished there also, but hyperidrosis has been observed. Effusion into the joints and other articular affections (arthropathies) are exceptional in myelitis, and they never attain to such a degree of development as in tabes dorsalis.

Lumbar or Lumbosacral Myelitis.—It is easy to state the modifications which the clinical picture undergoes when the myelitis affects the lumbar region. The paralysis of the legs is then flaccid, degenerative. *The tendon reflexes are absent, the cutaneous reflexes abolished,* the anæsthesia extends only slightly into the inguinal region; there is no girdle pain, but eventually there is radiating pain in the nerve tracts of the extremities. The paralysis of the bladder and rectum is still more complete.

If the myelitis is situated in the upper half of the lumbar cord, there may be ankle clonus with absence of the knee jerks, etc.

It is obvious that when the inflammation is situated still lower down, *i.e.* when the upper portion of the lumbar region is unaffected, the areas of the ileo-inguinal, crural, and obturator nerves will be more or less completely spared, whereby the knee jerks will also be conserved and may even be exaggerated, as I have seen in several cases. A primary myelitis, which is confined to the conus terminalis, would show the following symptoms: paralysis of the bladder and rectum, impotence, anæsthesia in the region of the anus, perineum, on the scrotum, penis, and the internal surface of the uppermost portion of the thigh, and eventually degenerative paralysis in the region of the sciatic nerve (see corresponding chapter).

Cervical Myelitis.—If the myelitis affects the cervical enlargement it is manifested by: (1) *atrophic paralysis of the arms,* (2) *spastic paralysis of the legs,* (3) *anæsthesia* in both legs and arms and on the trunk, (4) finally *oculo-pupillary symptoms.* The remaining symptoms are the same as in dorsal myelitis. When the disease is situated at so high a level, involvement of the abdominal and intercostal muscles may cause *dyspnœa.* A special danger may arise from the expiratory weakness when a bronchial or lung affection supervenes. If the myelitis affects the *upper cervical region,* there is no atrophy in the arms; they as well as the legs are in a condition of spastic paresis or paralysis. A fresh symptom appears in the form of *paralysis of the diaphragm,* and if the disease extends far upwards, signs of affection of the medulla oblongata become evident. A primary myelitis of this region is, however, extremely rare. Atrophy of the muscles innervated by the upper cervical nerves is occasionally observed.

Incomplete Transverse Myelitis.—We have proceeded on the assumption that the focus of myelitis is a completely transverse one. The disease is frequently, however, not so complete; it spares many fibres, or it is practically limited to a part of the cord. This condition finds its clinical expression in the incompleteness of the individual symptoms (paresis instead of paralysis, hypæsthesia instead of anæsthesia), or in the absence of some of them. Partial sensory paralysis may sometimes occur, and even less frequently the type of the Brown-Séquard unilateral lesion. We may, however, take it as a rule that all the signs of transverse interruption of conduction are present, even although some of them may be very slight. Thus the sensory disturbances are almost always insignificant

in comparison with the motor paralysis, and the functions of the bladder may be but slightly affected.

The myelitic process is by no means always confined to a small segment of the cord; it may affect almost the whole of the thoracic portion or part of the thoracic and lumbar cord. The modifica-

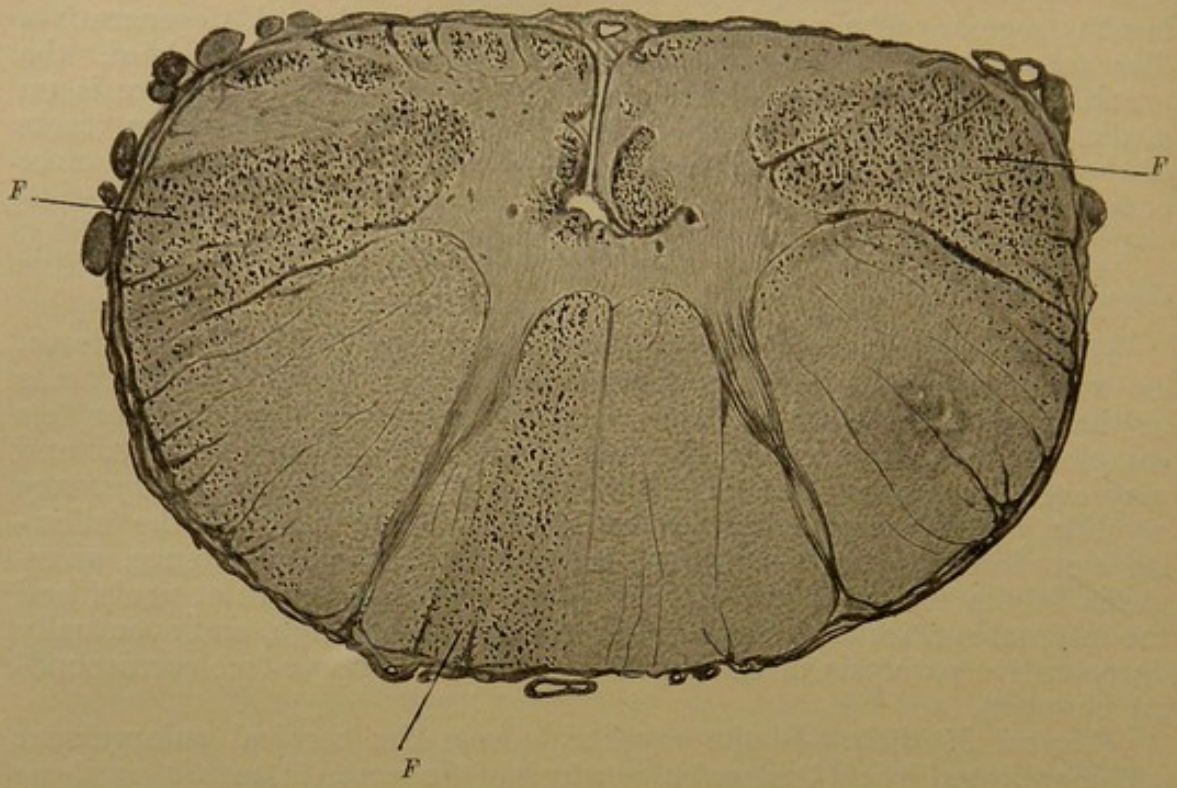

FIG. 160.—Acute myelitis. *F*—Myelitic foci by Marchi method.

tions of the morbid picture which thus arise do not require a special description. The myelitis may also gradually spread through the cord in an ascending or descending direction, or by the formation of new foci, whereby corresponding changes are caused in the symptoms. An ascending tendency is specially observed in the myelitis of pregnancy; this may be so advanced as to produce bulbar symptoms (Hösslin, Rosenberger-Schminke). This mode of extension is, on the whole, however, less common than has hitherto been believed.

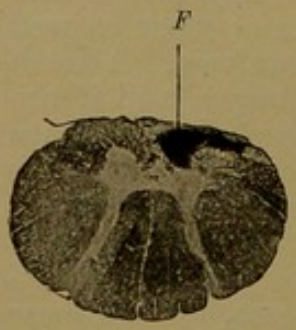

FIG. 161.—Acute circumscribed myelitis. *F*, myelitic focus. Marchi method.

Disseminated Myelitis (Encephalomyelitis disseminata).—There are very frequently present, in addition to the large myelitic foci, several smaller foci in their neighbourhood, or others more widely scattered, which may cause merely slight, if any, symptoms according to their site, extension, and number. There is a form which is distinguished precisely by the appearance of *numerous inflammatory foci* at the most varied sites in the spinal cord. This disease is, it is true, by no means confined to the spinal cord, but may involve the pons, medulla oblongata, and other parts of the brain (see Figs. 162 and 163). In a few cases, however, the disseminated process has been found to be limited to the posterior

columns (Strümpell). The infective (and toxic) origin of this form of myelitis is established beyond any doubt. It follows smallpox, measles, pneumonia, influenza, whooping-cough, dysentery, and chicken-pox. In one case I have seen it follow diphtheria. Cases published by Henschen and Ritter also show this mode of origin. It has to be admitted that syphilis also may give rise to an acute disseminated focal disease of the central nervous system (Bechterew, Dana, Marie, Catola). A number of satisfactory observations (Bruns, Pánsky, and others) are contributed in support of the toxic etiology. Finally, Nonne's clinical observations show that sunstroke, or exposure to great heat, may produce this disease.

The symptoms vary, and depend essentially upon the site and extent

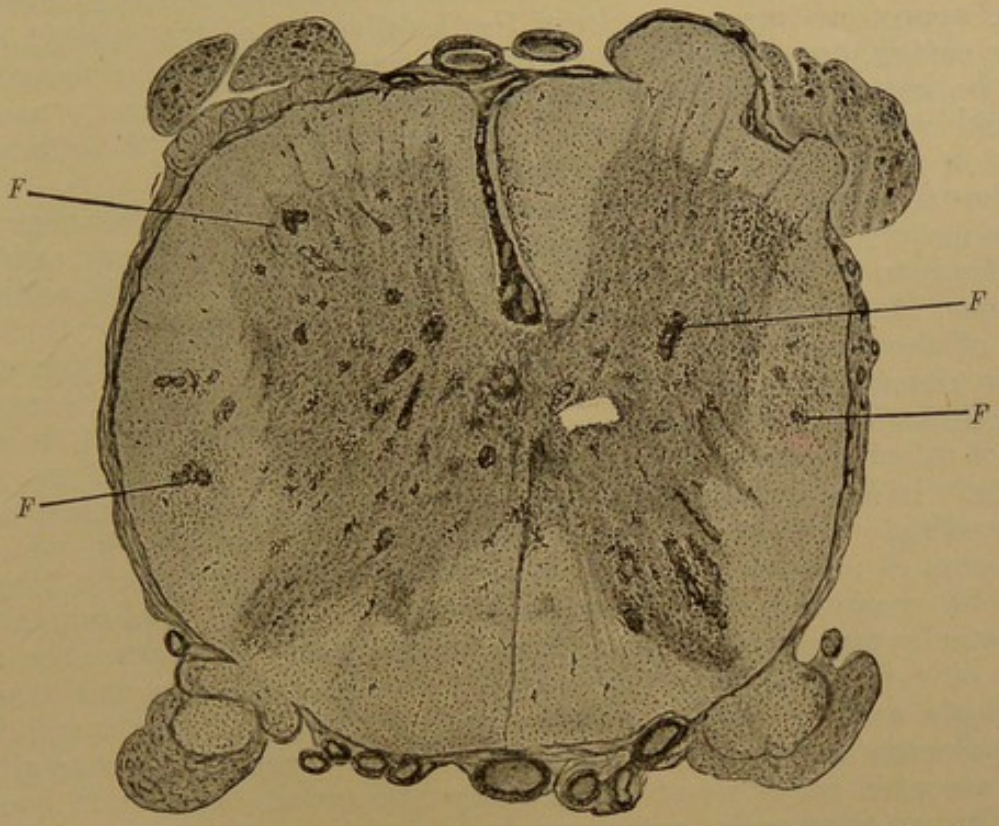

FIG. 162.—Acute disseminated myelitis (or acute disseminated myeloencephalitis). *F* = small foci of round cells, mostly round the vessels. Stain: Van Gieson and alum-hæmatoxylin.

of the foci. If they are confined to the spinal cord, the picture may resemble that of a diffuse myelitis. The tendency of the process to become disseminated may be manifested, for instance, by a simultaneous optic neuritis (Erb, Albutt, Dreschfeld, Dalén, Mayer, Hochhaus, Bielschowsky,[1] Taylor-Collier, Weil-Gallavardin,[2] Kerschensteiner [3]), or by a retrobulbar neuritis, according to Schanz and others. This usually precedes even the process in the spinal cord, and does not of itself prove that the spinal cord affection is a disseminated one. French writers (Devic, Brissaud-Brecy) speak of a *neuro-myélite optique aigue*.

In the cases observed by C. Westphal, the symptoms closely resembled those of disseminated sclerosis, but with this difference, that all the symtoms had developed acutely. Tremor, ataxia, scanning speech, and mental weakness were among the prominent symptoms. The tendon reflexes

[1] "Myelitis und Sehnervenentzündung," Berlin, 1901, Karger.
[2] *Lyon méd.*, 1903.
[3] *M. m. W.*, 1906.

were usually exaggerated, but they might also be absent, as in a case of Fürstner's. In later observations (Leyden, Lenhartz, Oppenheim, Nonne, Lüthje, Dana, etc.) the ataxia, or a mixed form of ataxia and intention tremor, were so prominent that the disease has been described as *acute ataxia*. It should not, however, be forgotten that the so-called acute ataxia may be due to a peripheral neuritis. It is probably the predominant localisation of the foci in the pons and oblongata (possibly also in the cerebellum) which is responsible for the association of symptoms described. The dysarthria and dysphagia observed are also compatible with this. Lastly, cerebral symptoms, disturbances of consciousness, delirium, aphasia, etc., may be conspicuous, especially at the commencement of the illness. Choreic symptoms are also described. These symptoms may arise from the localisation of the morbid process in the cortex, but may also be merely the result of the rise of temperature and the general infection of the organism. Lüthje, who saw a case of typhoid with a similar group of symptoms, attributes the ataxia and affection of the speech to the cortical process.

Thus the clinical picture may be widely removed from that of myelitis. In the majority of the clinical histories, the absence of sensory or bladder troubles is to be noted. Numerous cases of my own as well as those of Fürstner, Mager, Henschen, and others, have shown, however, that affections of the functions of the bladder and rectum are not uncommon. A case described by Süsswein occupies a position apart on account of the pathological anatomical lesion. We must not assume that the symptoms of this disseminated encephalomyelitis can be fitted in to any special scheme. It is apparent from our observations that the clinical picture is exceedingly variable. I have, for instance, seen a case in which the symptoms of myelitis were accompanied at the height of the illness by headache, vomiting, diplopia, and convulsions of the Jacksonian type, the latter symptoms persisting for a considerable time after the spinal group of symptoms has disappeared. In another there was transient hemiplegia and aphasia, and in a third, cerebellar ataxia was present. I have already mentioned, on page 211, that the spinal lesion may give rise to an atrophic paralysis of the poliomyelitic order. I have, in one case, found Brown-Séquard's type of paralysis, and in another there were pains and fever leading to an atrophic paralysis of the arm and a spastic paralysis of the leg on the same side. There are also abortive forms of this kind, in which retention or incontinence of urine are the only symptoms of the process (Oppenheim). Signs of meningeal irritation are also occasionally present. The course of the disease may be a short one. A few cases (Leyden, Oppenheim, Goldscheider, Mager, Henschen, Finkelnburg,[1] Schupfer, and others) point to the fact that it may pass over into disseminated sclerosis (*q.v.*). In any case it cannot usually be definitely distinguished from the acute forms of disseminated sclerosis (*q.v.*).

In the foregoing study of myelitis, we have given our consideration to a certain stage, namely that of the fully developed disease. With regard to the *development* and *course*, the following has now to be said: The onset is as a rule acute; acute myelitis is the most frequent and the best-known form. The above-described symptoms of inhibition of

[1] *Z. f. N.*, xx.

conduction may reach their acme in a day (see, for example, the case quoted on p. 241); indeed, in a few cases the development was almost apoplectiform (Strull, Hochhaus, Oppenheim and G. Flatau, A. Schiff, Muratoff, and others). As a rule days and even weeks go past before the disease attains its full development. With this acute onset the temperature is generally raised, not infrequently considerably so; the fever may persist for days and weeks. The patient shivers, complains of tingling in one or both legs, which may become loss of sensation; then there come weakness, which develops into paralysis, affection of the bladder, etc.; or the illness may be first revealed by motor weakness or affections of the bladder. Pain is but slight as a rule, is seldom very severe, is situated in the region of the back or abdomen, and may extend in the form of a girdle.

Less frequently the disease has a *subacute* development. Days, months, or it may be half a year pass before all the symptoms of transverse myelitis are complete. A course which is chronic from the first is the least common of all. Cases which at first appear to be chronic myelitis, are usually in our experience found on close investigation to be disseminated sclerosis in which the spinal symptoms predominate, or to be tumours of the spinal cord. There is a chronic form of myelitis, however. It is often the result of acute myelitis. But in rare cases it happens that the myelitis has from the very beginning an insidious course. Weakness is felt in one or in both legs, almost always accompanied by rigidity. This very gradually increases. Then either from the first or at a later stage there come sensory disturbances, usually paræsthesia at the commencement and then hypæsthesia, which may or may not very slowly become anæsthesia. Urinary troubles, etc. etc. appear during the first months or after the lapse of a year

With regard to the *differential diagnosis* the chapters on multiple sclerosis, tumour of the spinal cord, and compression of the spinal cord should be consulted. Tumours arising in the spinal cord itself (glioma, etc.) may for a considerable time simulate myelitis, and so also may malignant and metastatic tumours of the spinal column, as Nonne has specially pointed out.

Herpes zoster in the region of the sacral nerves may be associated with paralysis of the bladder and rectum (Davidsohn,[1] Oppenheim [2]), and may thus simulate a picture resembling myelitis.

It has already been remarked that embolism of the abdominal aorta and embolic and thrombotic occlusion of the arteries of the spinal cord and the conditions to which they give rise may produce symptoms similar to or identical with those of myelitis. In embolism of the aorta the diagnosis will be based on the evidence as to the primary vascular disease, especially the absence of the pulse at the femoral artery, etc., and also on the suddenness of the onset of the spinal symptoms, which point to a total interruption of conduction in the cord.

The *softening* caused by local occlusion of a large vessel in the spinal cord can hardly be definitely distinguished from acute myelitis. The criteria for differential diagnosis collected by Langdon (*Journ. of Nerv.*, 1905) are for the greater part uncertain.

Other peculiar affections arising from the vascular system of the spinal cord, which have hitherto evaded diagnosis and may be specially difficult to distinguish from myelitis, are described by Petrén, Brasch, Merewkina, and others.

[1] *B. k. W.*, 1890.

[2] *Mitt. aus Grenzgeb.*, xv.

With regard to the diagnosis of myelitis from the functional neuroses and especially from hysteria, the corresponding chapters should be consulted.

The *prognosis* of myelitis is a doubtful one. Every termination is possible—recovery,[1] improvement, arrest, or an eventually fatal issue. These have all been observed, the latter most frequently. It is of the greatest importance for the physician to be able to give a prognosis as correct as possible in each case. In this respect the following facts should be taken into consideration: the prospect of recovery is on the

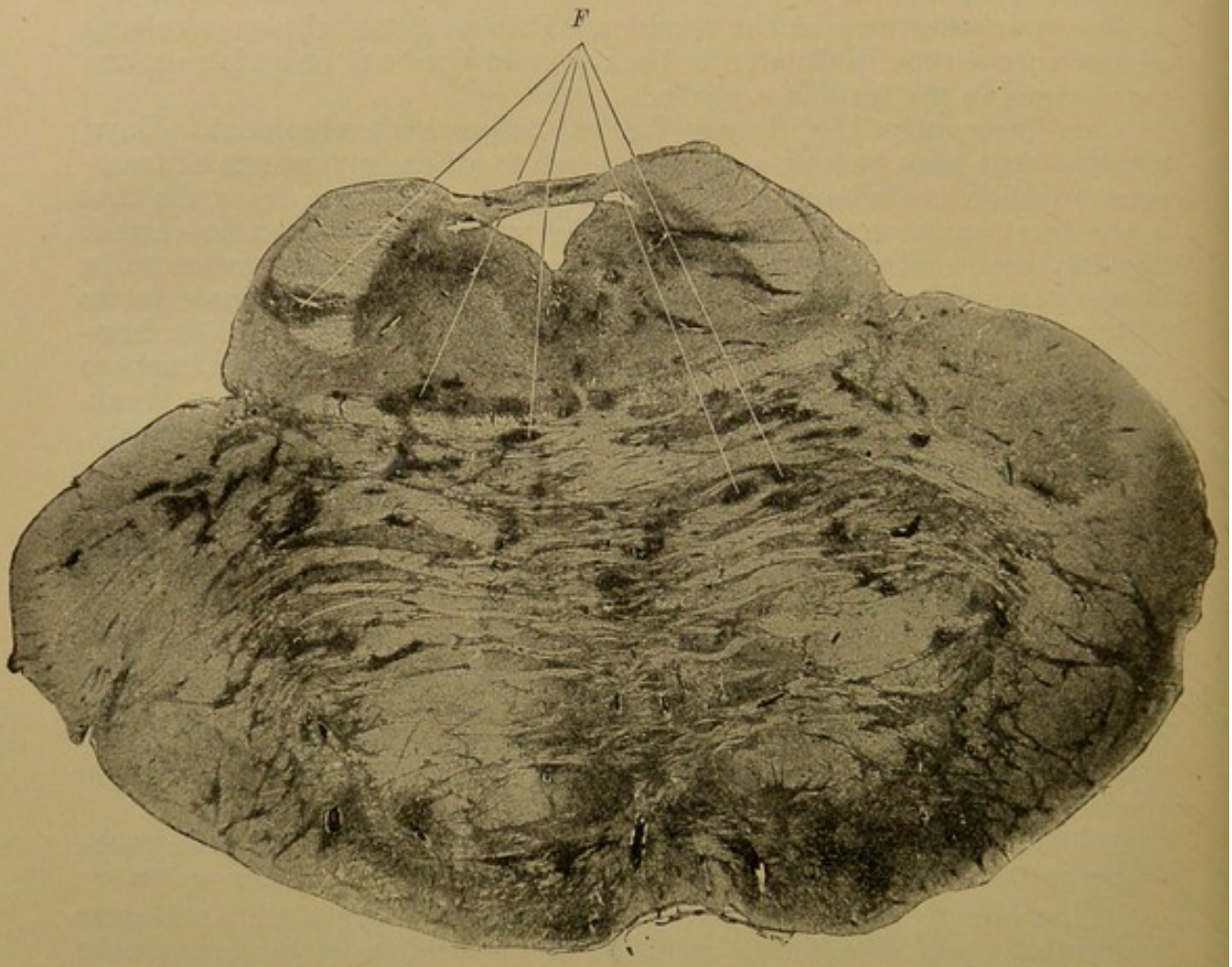

FIG. 163.—Disseminated myeloencephalitis. Numerous foci (*F*) in the pons. Figs. 162 and 163 belong to the same case.

whole better when the myelitis follows an acute infective illness. Among these the most benign form is that which is due to gonorrhœa. This, however, is usually the least definitely characterised. I have seen a case in which a spastic paraplegia or a spastic-ataxic paraparesis with paralysis of the bladder and sensory disturbances had supervened upon an attack of gonorrhœa, in which recovery took place within three to four weeks.

[1] I have seen recovery in a fairly large number of cases of this kind, most frequently in the disseminated, but also in the apparently diffuse form. Friedländer has cited a few of my cases in his thesis (Berlin, 1891). The curability of acute ataxia has already been frequently pointed out. In recent years cases illustrative of the recovery of myelitis have been communicated by Pontoppidan, Stanowski, Eliot, Apostoli-Planet, Semerad, and Krewer, who does not call his cases myelitis, however.

The cases of Hayem, Parmentier, Spillmann, Haushalter, and Bloch are also to be remembered. In the cases of myelitis—apparently mostly of disseminated form—which are due to smallpox, typhoid, erysipelas, influenza, etc., and usually take the form of acute ataxia, termination in recovery is not uncommon. On the other hand an acute hæmorrhagic myelitis of fulminating, fatal course, may develop in typhoid (A. Schiff). Further, an ataxia beginning acutely may become a persistent condition (Nonne). There are, moreover, forms of spinal paralysis which show an *intermittent* character, are connected with malaria, and terminate in

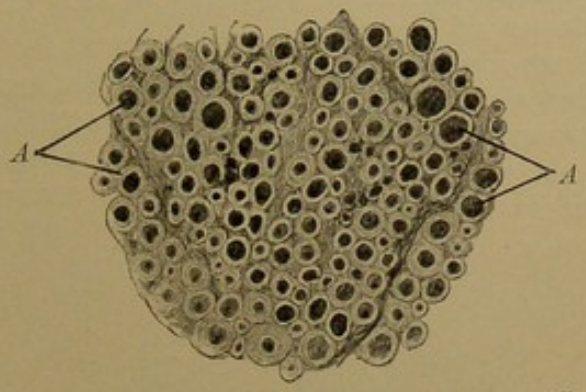

Fig. 164.—Swelling of the axis-cylinders (*A*) in myelitis. Carmin stain.

Fig. 165.—One of the foci (*F*) from Fig. 162 under high power.

recovery. The prognosis of *syphilitic* myelitis is not in general unfavourable. There may be, it is true, a form which occurs early, often at the commencement of the secondary stage, with a rapidly fatal course. A myelitis which develops during the puerperium, from tuberculosis or sepsis, has usually a grave prognosis, although one case of puerperal myelitis with a favourable course is reported (Morell). The myelitis of pregnancy may be cured by the natural or artificial termination of the pregnancy, but may reappear in another (Hösslin, Rosenberger-Schmincke). But it seems to me more probable that such cases are examples of disseminated sclerosis. A further standpoint for prognosis is afforded by the mode of onset: the more acute and at the same time incomplete the development of the symptoms, the more favourable in general is the

prognosis; whilst the cases in which the disease has an insidious, progressive course, and those in which the symptoms after either an acute or a subacute course indicate that the lesion has become a completely transverse one, afford little prospect of recovery. The chances are also the less favourable the longer the symptoms of the spinal disease have lasted.

I have observed cases in which symptoms of *meningitis* and *neuritis* complicated the myelitic process, and which terminated in recovery. In doubtful cases, therefore, the occurrence of severe pain in the back, radiating pain in the extremities, rigidity of the back, as well as the presence of symptoms of neuritis may be regarded as comparatively favourable signs, if we are justified in drawing this conclusion from a few cases.[1] This applies also to the degenerative neuritis which appears at a late stage in the course of a myelitis. The early appearance of a rapidly spreading bedsore, the so-called *acute decubitus*, is regarded as an ominous sign. Complete paralysis of the bladder and rectum is also a symptom of evil omen. Finally, the general condition must be considered in the

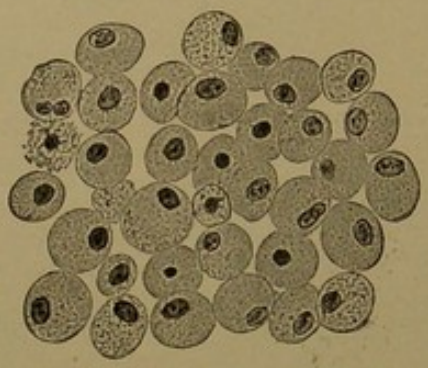

Fig. 166.—Cells from a myelitic focus, probably altered granule-cells. Carmin-alum-hæmatoxylin.

prognosis. Fragile, enfeebled individuals or old people succumb to the disease more easily than robust subjects. Death is usually caused by sepsis due to cystitis or of bedsores.

Pathological Anatomy.—When the spinal cord is removed, the handling of it is generally sufficient to reveal the site of the disease; it is usually of a softer consistency. If transverse sections are made, the affected parts are clearly indicated by the blurring of their details; the grey matter is no longer distinct from the white, and the cord is of a reddish-yellow, yellow-white, or grey-yellow colour. In chronic cases, however, the affected tissue may still be of a firm consistency. If a small portion is placed in the fresh condition and without any preparation under the cover glass, and examined under a microscope, numerous granular cells [2]

[1] Recent observers (Hochhaus, Marinesco, Redlich, and others) have proved that the meninges are usually involved along with the cord in myelitis, and Hochhaus has been able to demonstrate the combination with neuritis and myositis by pathological investigation in one case. A case of Brissaud's also seems to belong to this class. A combination with meningitis was found by lumbar puncture during life in an exceedingly unusual case described by Strümpell. From these observations it is clear that we must be very careful in estimating the significance of meningitic symptoms with regard to the prognosis.

[2] Even now the views of different writers as to the origin of the granular cells disagree. I regard them, with the majority, as being leucocytes, which have absorbed the products of nervous disintegration (Marinesco calls them neuronophages). Hoche thinks that both the leucocytes and the cells of the glial and connective tissue, the latter especially, may become granular cells, and Buchholz thinks they arise from glial cells.

will be found. It also shows either a myelitic focus of very variable extent or several scattered foci confined to small areas (Figs. 160, 161, 162),

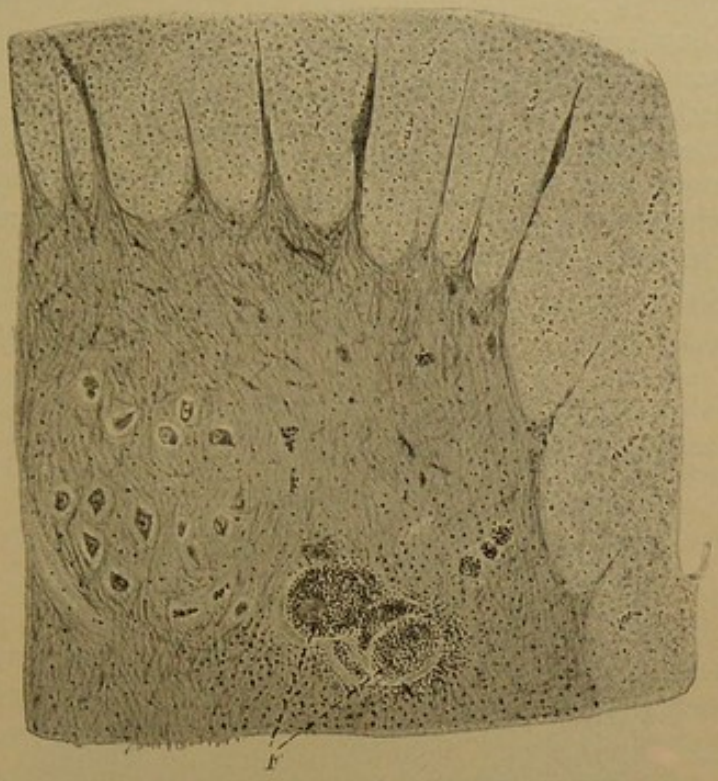

Fig. 167.—Focus of cell infiltration (*F*) in the spinal cord in ulcerative endocarditis. (From a section by Cassirer in Oppenheim's collection.)

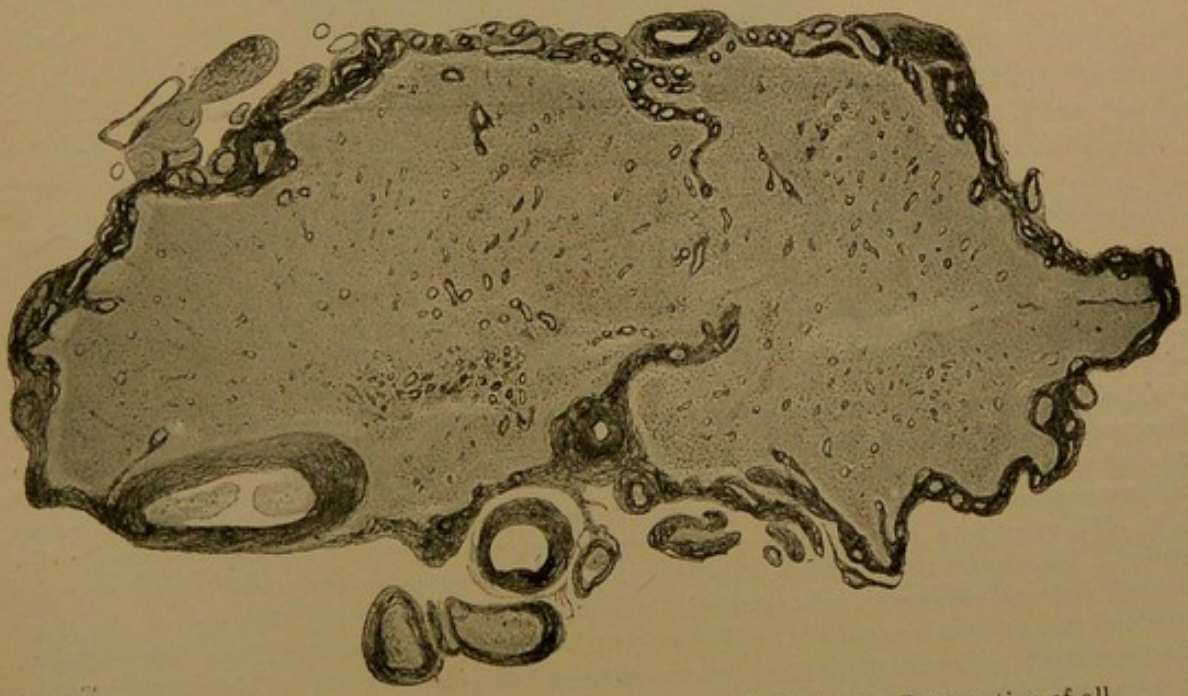

Fig. 168.—Complete transverse myelitis in the terminal stage. Destruction of all the nervous elements, etc. Thickening of the meninges. Carmin stain.

or more or less diffuse foci involving the whole thickness of the cord (Fig. 168). The secondary degeneration can often also be recognised by the naked eye. All this becomes more distinct when the spinal cord

has been hardened for some time in chrom-salt solution, and the affected parts stand out then by their intensely yellow shade of colour.

Precise knowledge as to the nature of the myelitic process is gained only by *microscopic examination* of transverse and longitudinal sections made after hardening. The essential facts, already established by Leyden, Charcot, Westphal, Schultze, and others, have in recent years been confirmed and completed by numerous investigations, amongst which I may mention those of Strümpell, Fürstner, Hochhaus, Schmaus, Marinesco, Mager and Redlich, as well as those by Rhein and the most recent by Harbitz-Scheel.[1]

Although in the description of the histological conditions I have taken the data of these writers into consideration, I have practically

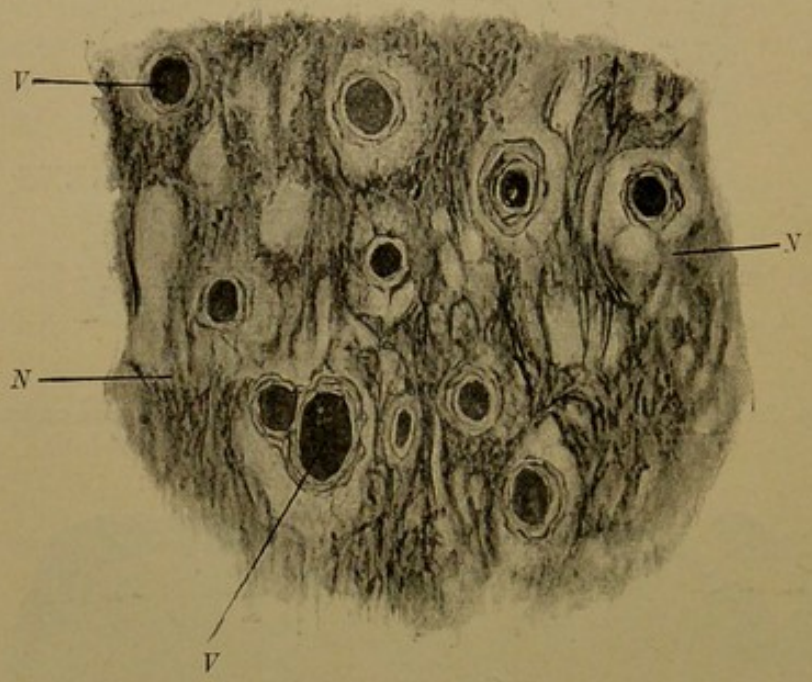

FIG. 169.—Part of transverse section of a spinal cord in myelitis, at the stage of sclerosis. *V* = vessels, *N*, thickened neuroglia.

based it upon my own observations and the evidence of the preparations made by myself and my pupils (Cassirer, Finkelnburg).

The changes which appear in the spinal cord in myelitis are exceedingly numerous and variable. Frequently, perhaps most often (*i.e.* in the majority of the cases upon which there is an autopsy), we find a picture of disintegration, *degeneration*, *necrosis*, *softening* being most prominent. The nervous elements thus appear to be primarily affected. The axis cylinders first become thickened (Fig. 164), often to a comsiderable degree, and the myelin sheaths also swell and appear distended. Disintegration then takes place; blocks of myelin and fatty products of degeneration are formed, and are absorbed by the granular cells which collect into foci. According to the stage at which this process is examined, and also to the acuteness and intensity of the disintegration, the process will appear in the form of the *vesicular condition* (Leyden), of *cavity-areas* (Mager), *i.e.* as wide-meshed tissue, the cavities in which are the result of disappearance of the nerve-fibres and the dissolution of the granular corpuscles, or in the form of *foci of softening*, in which myelin, detritus, and granular cells predominate—the latter may be represented also in stained preparations as so-called epithelioid cells (Fig. 166)—or finally, in a third stage in the form of *cicatricial*

[1] "Pathologisch-anatomische Untersuchungen über akute Myelitis und verwandte Krankheiten," etc., Christiania, 1907.

foci, in which the most prominent feature is the glial proliferation (Fig. 169). In other cases, which indeed comprise the majority of those which show the type of acute *disseminated* myelitis, by far the most prominent appearances are the affection of the *vessels* and the *cell infiltration*, *i.e.* the accumulation into foci of the round cells (Figs. 162, 163, 165, and 167), rarely of epithelioid cells (Fig. 166). With regard to these cell forms and their interpretation, the works of Leyden, Redlich ("xix. Kongress für innere Med."), Weill-Gallavardin (*R. n.*, 1903), and others may be referred to. Hæmorrhages also appear. I assume that in this form the nervous elements are often but slightly altered, at least in the first stage and in mild cases, whilst later, the signs of inflammatory irritation and subsequently those of degeneration become evident. In this form complete restitution, or termination in cicatrisation, sclerosis, and perhaps also in softening is possible. The two main types just described may, however, co-exist, and the involvement of the vascular system may also be very marked in the necrotic form. A *suppurative* myelitis is mentioned occasionally (as by Collins, König), but I have not met with it as a primary disease. It is caused rather by the direct penetration of decomposing, septic matter into the spinal canal, *e.g.* in bedsore, in infected wounds, after injuries, operations, in lumbar anæsthesia, or metastatically (see the section on abscess of the spinal cord). According to Hochhaus, the vascular apparatus of the whole spinal cord, and even beyond it, may be subject to an inflammatory affection, even where the myelitic disease is a circumscribed one. Finally, we have to remember that the optic nerve and meninges may be involved, as is well established by histological observation.

With regard to the general questions and conceptions of inflammatory processes in the nervous system, the articles by Storch (*C. f. N.*, 1900) and Schmaus (*Z. f. N.*, xxvi.) should be consulted.

Treatment.—The treatment of myelitis is not a grateful task, as we are still waiting for the serum-therapy prophesied by Marinesco. Nevertheless medical supervision and medical treatment are necessary; they can do much to protect the patient from dangers and can render him effective help.

Causal treatment plays a part only in exceptional cases, *e.g.* in the syphilitic forms. In the myelitis of pregnancy, the induction of premature labour may be efficacious (Hösslin).

In cases of *acute* myelitis, *absolute* rest is the first requisite. Prolonged confinement to bed should immediately be prescribed. The excreta should even be passed in bed, and with the utmost possible care as to cleansing of the patient. If the attack follows an infective illness, *diaphoretic measures* are, in my opinion, specially indicated. Sweating may be induced by hot packs and woollen blankets and by the administration of hot drinks. Hot steam may also be introduced underneath the bed-clothes by means of suitable apparatus, or an apparatus for local treatment by hot air may be employed. This method has more rapid results than the ice-bag or local derivatives. Local painting with tincture of iodine at a site on the back corresponding to that of the myelitis may, however, be used, or counter-irritation by blisters, or the button cautery. Care should be taken not to apply them to any anæsthetic area which is at the same time exposed to pressure.

As to drugs, preparations of salicylates may perhaps be ordered. We do not as yet possess any specific for myelitis. If the disease is connected with syphilis, or if any factor suggests such a connection, then we must turn to iodide of potassium or to preparations of iodide and mercury. The opinion sometimes expressed that Hg has a beneficial effect, even in non-specific inflammations of the spinal cord, is not confirmed by experience. I have never seen any good result from the use of ergot. If intermittent fever has preceded the illness, then quinine and arsenic are indicated. Injections of strychnine are recommended for the paralytic conditions. Hot baths should be avoided. Wet packs, on the other

hand, may be used after the first stage is over. I have seen remarkable results of this treatment in a few cases.

Finkelnburg found transient improvement after repeated lumbar puncture.

If the disease has reached its height, and if further progress is not taking place, then warm baths of 90° to 95° F. are suitable, and to these may be added sea-salt, Kreuznach lye, or Stassfurt salts. In the chronic cases, so long as patient can move about, a course of baths at one of the mineral spa (Teplitz, Wildbad, Gastein, etc.) may be beneficial, but baths of high temperature should be avoided. The use of the warm carbonic-acid brine baths of Oeynhausen, Nauheim, etc., are well worth recommending. If, however, the spastic symptoms and the exaggeration of reflex excitability are very marked, the latter should not be prescribed.

If the patient is permanently confined to bed, attention should be above all directed to the prevention of bedsores. Care should be devoted to the position, the change of position, smoothness of the bed beneath the patient, air-cushions, etc. The best method of all, however, is a water-bed. Powdering the parts exposed to pressure with starch and zinc oxide or some other dusting-powder, and washing with spirits of camphor, lemon-juice, etc., is useful. Ichthyol collodion may also have a prophylactic effect. The patient should be bathed after each evacuation of the bladder and bowel. Where this is impossible, local washing must take its place. Numerous modes of lifting and transporting are recommended to facilitate all these manipulations of the paralysed patient. Great care and cleanliness is necessary in using the catheter. It must be employed not only in retention, but also in incontinence of urine. Incontinence in men makes it necessary to use a urinal or a suitable vessel if the patient is confined to bed. If the skin of the penis or scrotum is thus irritated, it is advisable to use cotton-wool, which is changed as soon as it is damp, or an absorbent pad of wood-wool or peat-gauze. Raising of the patient in bed facilitates passing of the urine. The bed may be so arranged as to make an immediate flow of the urine possible. If constipation is present, complete evacuation should be aimed at by a mild aperient, or better still by an enema every second day.

Treatment of bedsores necessitates local application of drugs in the form of wet compresses, ointments, etc., in addition to the above-mentioned measures (lukewarm baths, permanent bath). If the ulcer is small, an ointment dressing (boracic vaseline, white precipitate, iodoform, zinc ointment, xeroform, etc.), kept in place by plasters, is sufficient. When it is extensive, wet dressing with spirits of camphor and tincture of myrrh or peroxide of hydrogen is advisable. For indolent granulations tannate of lead or nitrate of silver in ointment or as a wet dressing has often a beneficial effect. Peroxide of hydrogen may also be used in this way. If there is much secretion, dry powders (iodoform, xeroform, bismuth, etc.), with gauze bandages should be employed. Fissured, gangrenous parts of burrowing edges and fistulæ must be removed with the scissors.

If *cystitis* has developed, suitable drugs, of which urotropin deserves the first place, should be administered internally. If washing out the bladder is found to be necessary, a catheter with a double current is used, with luke-warm solutions of 2 per cent. boracic acid, of salicylic acid, or 1 per 1000 solution of nitrate of silver, etc.

We have unfortunately no remedy for the severe *spontaneous spasmodic contractions*. Even morphia may fail in its action. The use of subarachnoid injections of cocaine or lumbar infusions with tropococain, stovain, etc., after Bier, is too dangerous a method to be brought into

general practice. Goldscheider and Leyden-Lazarus found these had a merely transient effect. Cathelin's method should be contemplated in exceptional cases only. Warmth has the most beneficial effect. In no case should the extremities be uncovered or manipulated unnecessarily. In a few cases in which these tremors were the most distressing symptom, I prescribed the *permanent bath* with good results. Massage and careful gymnastics in warm baths may also be helpful. One of my patients, who was particularly troubled by severe contracture of the legs, had himself discovered that he could by a reflex method give another position to his legs. He pinched the skin on the inner surface of the thigh, and the leg, from being greatly extended and excessively adducted, assumed another position out of which he could again move it by the same means. The will power, which could not act directly upon the muscles, called in the help of the reflex in order to attain a certain movement. Dejerine has described a somewhat similar observation and has mentioned a case in which the patient by means of such a device could reflexly pass his urine. In another of our cases the physician in charge overcame the contracture for a time by hyoscin. Nitro-glycerine is also recommended.

I cannot accept Goldscheider's advice to make use of extension in every case of spastic paraplegia, as a compression myelitis can never be with certainty excluded.

In the acute stage nothing can be expected from *electricity*. If the paralysed muscles are in a condition of rigidity with a tendency to reflex spasms, they may be easily injured by faradic and galvanic irritation. A mere touch of the skin is sufficient to elicit spasms, how much more then the electric current. Even the stabile application of the galvanic current to the insensitive limbs is not without danger (see page 287). It is well, therefore, to confine electrical treatment in general to the later stages, and to chronic and convalescent cases. Direct galvanic treatment of the spinal cord is always harmless. Faradic or galvanic stimulation of the muscles is particularly suitable in flaccid atrophic paralysis, and is well adapted to hasten recovery, if the disease has this tendency. If the paralytic symptoms have more or less disappeared and a sensory disorder remains, the faradic brush may sometimes be efficacious. Although electric treatment for the paralysis of the bladder is not suitable while the paraplegia lasts, it may be useful in cases where the bladder paralysis is the only remaining symptom of the myelitis. If a spastic paresis has remained for a considerable time, whilst the other symptoms have disappeared, massage and gymnastics (passive movements, especially in warm baths) may have a good effect.

Appendix

Senile Paraplegia. Senile Paralysis

In old age, conditions of weakness in the lower extremities may occur, which, according to the investigations of Démange-Leyden,—Eisenlohr, Oppenheim-Siemerling, and Sander, reporting similar results—are to be attributed to a process allied to arterio-sclerosis or a corresponding affection of the vascular system of the spinal cord, and to sclerotic changes in the vicinity of the vessels, especially in the white matter. As a rule

there is spastic paraparesis, which may amount to contracture; less frequently there are also sensory disorders and sphincter paralysis (Oppenheim). The arms are sometimes affected. Along with the spinal symptoms, there may be others caused by involvement of the vascular system of the brain and the resulting conditions (dementia, dysarthria, etc.).

We must remember, however, that senile spastic paraparesis may also be caused by small foci in the brain which involve the motor tracts of both sides. This occurs especially when the foci are situated in the pons. There may be no other sign of a brain affection.

I have also seen a few cases of this kind in which symptoms were associated with spastic paraparesis which suggested those of paralysis agitans (*q.v.*); the resemblance was incomplete, however, the tremor in particular not being quite typical. There may of course also be a combination of the two affections.

Other forms of senile paresis with diminution of the tendon reflexes, etc., are also attributed to spinal arteriosclerosis, by Hirsch, for instance (*Journ. Nerv.* and *Ment. Dis.*, 1903), but this view still requires histological confirmation. Further contributions to this question have been recently made by Pic-Bonnamour (*Rev. de Méd.*, 1904), Crouzon, Collins-Zabriske (*Med. Rec.*, 1904), B. Sachs, Dupré-Lemaire (*R. n.*, 1905). See also discussion in *R. n.*, 1906. Lejonne-Lhermitte (*Nouv. Icon.*, 1906) have on the other hand shown that "senile paraplegia," may have a muscular origin (atrophy and consequent contracture of the muscles). See also Lhermitte, *Thèse de Paris*, 1907.

Disseminated Sclerosis, Multiple Sclerosis, Multiple Cerebro-Spinal Sclerosis.[1]

This is a disease of *youth*. It most frequently commences at the end of the second and beginning of the third decade. It not infrequently shows its first signs between thirty and forty-five, but seldom makes its appearance later.

We very seldom meet the fully developed disease in early childhood, but a few cases may be traced back to the fourteenth year, and isolated cases even to the first childhood. Amongst the cases which I confirmed post-mortem there were three in which the illness had begun in the thirteenth to the fifteenth year; I have described one of these fully. Frequently there are isolated symptoms which have lasted so long that they must be considered congenital or early acquired (Oppenheim [2]).

In two of my cases paralysis of the abducens was traced to earliest childhood, in one speech disorders. Several times I have found symptoms which could only be interpreted as stigmata of degeneration, *e.g.* medullated nerve fibres in the retina, etc.

Although the occurrence of disseminated sclerosis in childhood is verified only by the results of a very small number of autopsies, we must remember that the disease runs as a rule a very long course, extending over decades. Eichhorst has demonstrated the disease by microscopical examination in a child of eight months whose mother also suffered from disseminated sclerosis.

Schupfer states that only a few of the clinical cases of infantile focal sclerosis are above reproach, and that only two or three of the cases examined post-mortem belong to this class; the case which he communicates, however, resembles disseminated myeloencephalitis rather than multiple sclerosis.

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[1] Of the most recent work on this disease the monograph of E. Müller, "Die multiple Sklerose des Gehirns und Rückenmarks," Jena, 1904, deserves special praise. But I may be permitted to add that my treatises, particularly the description in the earlier editions of this text-book, contain all that is essential, at least from the symptomatological point of view. Müller's recent contributions will be mentioned in the text.

[2] *B. k. W.*, 1887.

The frequency of the disease has hitherto been decidedly underestimated. In our experience, which agrees with that of E. Müller, Morawitz, Hobhouse, Mercuvita, and others, this affection is the most common of all the chronic organic diseases of the central nervous system.

As to the *causes*, we have but little positive knowledge. We think with Marie that there is no doubt that *acute infective diseases*, such as typhoid, smallpox, scarlatina, measles, etc., may result in this disease. It has also developed after influenza (Nolda, Massalongo and Silvestri, Rendu, Maixner, Bramwell). I have observed five or six undoubted cases of this. In others it was a consequence of cholera, whooping-cough, and acute articular rheumatism. I have found it develop several times after the puerperium, and Hösslin reports similar cases. An observation of Spiller's shows its relation to malaria. It followed diphtheria in a case of Henschen's, but this case should be regarded rather as disseminated myelitis. Krafft-Ebing laid great weight on "chill." I have also pointed out that intoxication with metallic poisons may be the basis of the disease. It is doubtful whether the cases observed by Schlockow in zinc-workers belong to this class. I have seen a case, however, in which a chronic tin poisoning produced simultaneously a nephritis and the typical symptoms of disseminated sclerosis. The tin was found in the dye of the coloured stockings which the patient had worn for years, and in the urine. It is doubtful whether the symptoms observed in manganese poisoning belong to disseminated sclerosis (Jaksch, Embden, Wagner). Jaksch is inclined to refer them to "pseudosclerosis."

Ceni and Besta have experimentally produced the affection by poisoning with aspergillus fumigatus. The disease has in a few cases followed carbonic oxide poisoning. Gerhardt traced it in one case to mercury poisoning. It has been also attributed to alcoholism. Disseminated sclerosis has no connection with syphilis, but in the form of disseminated foci it has been described in cerebro-spinal syphilis (Bechterew and others), and Catola has recently shown the probable existence of a disease due to syphilis and allied to disseminated sclerosis.

The assumption of an infective and toxic origin has been disputed by Strümpell, Hoffmann, Klausner, and others. In not a few cases it followed an *injury* (fall on the back), and in a few it was the consequence of *violent emotion*. I have found that when this was so, other factors, such as overstrain and chill, were associated with the mental shock. Thus two persons were affected in the same way by having fled from a fire naked or insufficiently clad. Negro saw it develop after an earthquake in an individual who had to camp out in the open under snow. A stroke of lightning has also been held responsible. Great emphasis has recently been laid upon the traumatic origin (Jutzler, Mendel, Jacoby, Hoffmann, Schlagenhaufer, Grossmann, and others).

Frequently, perhaps in the majority of cases (in fifty per cent. according to Hoffmann), no causal factor can be ascertained. Strümpell ascribes no importance to exogenous factors, and regards the disease rather as an endogenous and constitutional one, and with Ziegler he regards as its cause a congenital tendency to proliferation of the glial tissue. E. Müller agrees with him. I have already drawn attention to the established fact that congenital anomalies of development or symptoms dating from earliest childhood may be present, but I

regard these merely as laying the foundation of the constitutional tendency which renders the individual more susceptible to the injurious influences regarded as "causes."

Symptomatology. (*Typical Picture of Charcot's Disease*).—The description applies to the disease at its height.

The patient complains of weakness and rigidity in the legs, of tremor and fits of vertigo. Less frequently other troubles are prominent: disturbances of vision, headache, pains in the legs, troubles of speech, etc.

The *objective* symptoms are much more marked. There is in the legs more or less marked *motor weakness*, usually associated with *muscular rigidity*; the tendon reflexes are correspondingly exaggerated, passive movements made difficult, and other signs of the *spastic* condition, described on p. 6, are present.

The gait is markedly that of spastic paraplegia, but may be modified in numerous ways by factors to be mentioned later (affections of the equilibrium, tremor, ataxia, etc.), or, if the motor weakness is far advanced, walking may be quite impossible. Frequently, and often at an early period, the *impairment of the cerebellar co-ordination* renders the gait uncertain, so that along with the spastic paresis there is *staggering* and danger of falling, especially in rapid stopping and turning.

Active movements of the limbs, sometimes also of the head and trunk, are accompanied by tremor, which is so definitely characteristic that it may almost be called pathognomonic. It is absent during rest. This is not contradicted by the fact that the head sometimes trembles whilst the patient is sitting, as the neck muscles are thrown into action to keep the head erect. It accompanies the movements, always the voluntary, and sometimes also the reflex automatic and accessory movements. The tremor consists of ample, irregular oscillations, which may be suitably termed *waggling*. It appears specially in the muscles of the trunk and the large muscles of the extremities; the whole extremity oscillates (not the hand and fingers only), and the range of the oscillations is therefore very considerable. The single oscillations succeed each other comparatively slowly, about four to six to the second. It is as a rule most marked in the arms, and the head is frequently involved; it waggles and makes nodding movements, as in affirmation. As long as the patient is lying down with his head well supported, there is no sign of the tremor, but as soon as he rises, this waggling commences and becomes specially noticeable when he walks. The tremor usually affects the lower extremities also. In rare cases it extends to the muscles of the face and jaw, as in that described by Bruns, in which dislocation of the lower jaw was caused by the tremor. The designation of *intention-tremor* applied to this form (Charcot, Schultze) is inaccurate; Schultze seems to admit this and to prefer the name *motor tremor*.

I have sometimes succeeded in inducing the tremor, which seemed absent on simple examination, by asking the patient to rapidly follow with his hand an object, the direction of the movement of which was continually changed. At other times it appeared after I had fatigued the extremities by muscular exercises.

In the rare cases in which the tremor does not cease during rest, there is perhaps a combination of disseminated with diffuse sclerosis (*q.v.*).

The *tremor* is not always associated with motor weakness; even when it is severe the strength of the arms may be well conserved. In

advanced stages of the disease, however, paresis and rigidity are often present, but the latter is not usually so marked in the arms as in the legs.

Another remarkable symptom is *nystagmus*. When the patient looks straight forward, or still more when he looks to the side, we note rhythmic tremors of the eyeballs, which are constantly being turned back from the extreme lateral position to that of rest. The slightest degree of nystagmus may sometimes appear even in healthy people in forced lateral turning of the eyes, so that only high degrees of the condition are helpful in diagnosis. The nystagmus is seldom rotatory. Kunn describes a constant tremor of the eyes during fixation on an object.

Affections of Vision.—In the majority, or at least in a very large percentage of cases, the optic nerve is affected. This is usually only discoverable by the opthalmoscope. A *partial optic atrophy* pallor of parts, especially of the temporal halves of the optic discs, is almost always present (Uhthoff [1]). Sometimes there is pallor of the whole optic disc. *This condition hardly ever becomes complete atrophy of the optic nerve.* The process is usually limited to, or most marked in one side. Neuritic changes may precede the atrophy. In one case I noted that this neuritis developed during the illness, while the patient was under treatment; the onset was acute and accompanied by pain in the eyes and their neighbourhood; in a week there was hardly a trace left. In the great majority of cases affection of the optic nerve is one of the early symptoms, and it may indeed precede the other symptoms by several years, ten or more (Oppenheim-Frank, Bruns-Stölting). Bruns (and also Nonne and Rosenfeld) has observed marked choked disc, but this is a very unusual occurrence in our experience. The apparent optical affection is usually a *functional disturbance*, which may vary greatly according to its intensity and form. There may be diminution of the central acuity of vision (hardly ever amounting to blindness), an irregular narrowing of the field of vision, a central scotoma for white and colours, or merely an achromatopsia.[2] The affection of sight may disappear, remain constant, or improve. I have only once found a complete blindness prove transient. The sight may be affected without any discoverable ophthalmoscopic changes.

Scanning Speech.—In advanced stages of the disease, the speech is usually so very slow that the words are cut up, the syllables being separated from each other by pauses. The patient speaks almost like a child learning to read. This symptom is preceded for a considerable time by simple slowing of the speech (bradylalia), but is by no means constant.

Apoplectic Attacks.—In a very small number of cases, there are, during the course of the disease, attacks of loss of consciousness which leave behind a hemiplegia. This, however, usually disappears rapidly, in a few hours or days. The apoplectic symptoms may, however, improve slowly, or some of them may persist. The attack, which may resemble epilepsy, is accompanied by a rise of temperature. Attacks corresponding to those of *petit mal* also occasionally occur, but in our experience these are very rare.

[1] "Untersuchungen über die bei der multiplen Herdsklerose vorkommenden Augenstörungen," Berlin, 1889, A. Hirschwald. Also *A. f. P.*, xxi., *B. k. W.*, 1885 and 1889; "The Opthalmoscope," 1905, etc., and Graefe-Saemisch, "Handbuch," 2nd edition, Bd. xi., 1904.

[2] The similarity of the visual disturbances with the toxic amblyopia seems to point to the fact that poisons play a part in the etiology of this disease.

These are the most important symptoms of disseminated sclerosis. As a rule, however, other disorders are also found. There is frequently *headache*, and in most cases *vertigo*. This usually comes on in attacks, which may be so severe that the patient falls to the ground. It is specially apt to occur on looking upwards and in walking.

The *intelligence* is often diminished. The patient is uninterested and forgetful. High degrees of weakmindedness, sensory hallucinations and delirium are, however, quite unusual. This circumstance is particularly important because multiple sclerosis is not infrequently confused with paralytic dementia. Some observations prove (Tigges, Claus, Schultze, Fürstner, Zacher, Bechterew, Petroff, Hunt) that these two conditions may be combined, but this is exceedingly rare (see differential diagnosis).

Dannenberger, Dupré and Lannois, Seiffer (*A. f. P.*, Bd. xl.), Georg and Räcke (*A. f. P.*, Bd. xli.), have recently made contributions to the question of mental disorders in multiple sclerosis. According to Seiffer, moderate affections of the intelligence are present in the majority of cases. He endeavours to show that there is a special form of dementia due to multiple sclerosis, "polysclerotic dementia."

Another symptom is, as I[1] was the first to show (Marie, Lannois, and others came later to the same opinion), erroneously brought into relation with the decline of mental power, namely—*uncontrollable laughter*. This is a symptom which frequently appears even in the early stages; the patient has to laugh against his will, although his mood is not gay. This distresses him greatly. The laughter may come in spasmodic bursts.

In one case I found that the laughter occurred specially when certain movements were attempted, *e.g.* in turning the eyes sideways.

Paralysis of the ocular muscles is a not uncommon symptom, and the external muscles are almost exclusively involved. Myosis, and inequality of the pupils, occasionally appear; reflex immobility of the pupils is very rare.

This has been observed only in three cases, so far as we know (Uhthoff, Probst, Pini), whilst sluggishness of the pupils is more frequently mentioned. Aniscoria (inequality of pupils) is not unusual, according to Kuhn and E. Müller. In the great majority of cases the reaction of the pupils is normal throughout. Unilateral paralysis of accommodation was once found by Hoffmann. Ptosis, usually of a transient character, is mentioned by Wilbrand-Saenger, Williamson, Marburg, and others.

The paresis usually affects only one or several ocular muscles, and has mostly the character of associated paralysis or paresis (Parinaud). *Ophthalmoplegia* is very rare, but I have seen three cases of this kind, in two of which there was an autopsy. In one case I saw the ophthalmoplegia disappear.

In another of my cases there was bilateral paralysis of conjugate deviation, the movements of convergence being conserved. Similar cases are described by Raymond-Cestan and Ballet. Bouchaud mentions isolated paralysis of convergence. I have observed in one case remarkable slowness in the movements of the eyes.

The vocal apparatus is occasionally involved. The voice is monotonous, easily fatigued, and may also be hoarse. In some cases there is also

[1] *Charité-Annalen*, xiv., 1889.

paresis of the tensors of the vocal cords which seldom develops into complete paralysis (Leube, Lori, Lähr, and others). A tremor of the vocal cords is sometimes noted in phonation; if an "E" is intoned and prolonged for a time, a striking tremolo is noticeable in the voice. Rethi has recently written a monograph on the question ("Die laryngealen Erscheinungen bei multipler Sclerose," etc., Vienna, 1907). The tremor may also affect the respiratory muscles and produce a kind of jerky breathing (Oppenheim).

Affections of *sensibility*, while rarely entirely absent throughout the disease, are seldom permanent or severe. According to my own and Freund's (*A. f. P.*, xxii.) experience, there are as a rule *slight*, *temporary* affections of the sensibility, which, if the patient is seen but seldom, may escape observation. He complains at times of paræsthesiæ, of a feeling of numbness, of tingling in the ends of the extremities or at other parts, and objective examination reveals diminution of the sense of touch, pain, and temperature, etc., and disturbance of the sense of position. There is almost always merely a *hypæsthesia*, sometimes a partial sensory paralysis. Isolated thermal hypæsthesia may even occur (Reichel). A delayed conduction of thermal stimuli has been noted (Gothard). The sensory symptoms may disappear in a few weeks, or even in a few days, to return later, but they may also be of a permanent nature. They rarely take the form of hemianæsthesia, but transient hemianæsthesia may be one of the signs of the disease.

We have found that Brown-Séquard's syndrome may occasionally be present for a time. I have once observed a "bilateral Brown-Séquard syndrome."

There are frequent complaints of pains of a stabbing, boring, dragging character in the extremities, joints, and intercostal region; but they are seldom prominent and are hardly ever of the lightning character. In exceptional cases they become so intense in the region of a certain nerve that we may speak of them as neuralgia. I have seen a case in which trigeminal neuralgia was one of the first and most permanent symptoms of sclerosis, and in which a sclerotic focus was found post-mortem at the point of emergence of the trigeminus.

The function of the bladder is in my experience frequently affected. What has been already said with regard to the sensibility applies here also: a complete and permanent paralysis is exceptional; there is much more often slight and transient impairment of the function, a difficulty in passing the urine, retention or incontinence, which, however, lasts some days or weeks only, then disappears to return at a later period. Incontinence of fæces is a rare symptom.

Strümpell and his pupil E. Müller have done us a service in showing that *disappearance of the abdominal reflex* is a frequent and early symptom of the disease. Although I disagree with these writers in regarding the abdominal reflex as inconstant in healthy persons, I must admit that my recent experience has shown that it is absent specially often and usually at an early stage in disseminated sclerosis. This seems to be true also of the cremaster reflex (E. Müller). Finkelnburg thinks that weakness of the abdominal muscles has a diagnostic value in this disease.

If we turn now to the *unusual* symptoms, present only in a small number of cases, we find: (1) *muscular atrophy*. The muscles as a rule

retain their normal size and electrical excitability. A moderate degree of atrophy of one muscle group or of a whole extremity is not altogether rare; but degenerative atrophy revealed by a qualitative change of the electrical excitability is unusual. Complete reaction of degeneration has never indeed been noted. Muscular atrophy was very marked in cases described by Brauer, Probst, Glorieux, and Wegelin.

In one of our cases the development of the typical symptoms was preceded for three years by an atrophic degenerative paralysis of the right arm of radicular distribution, which had so favourable a course that it had been misinterpreted by celebrated neurologists, and the patient had been permitted to marry. Lejonne exhaustively studies the muscular atrophies of disseminated sclerosis in his thesis (Paris, 1903), and endeavours to distinguish a special form, the amyotrophic.

(2) *Ataxia.*—In not a few cases the spastic paresis of the lower extremities is associated with ataxia. This is recognised by the gait; the legs are pushed forwards with difficulty, the toes drag on the ground, but the leg is excessively lifted and is brought to the ground again with a stamp of the heel. The ataxia can also be distinctly recognised in the movements of the patient while on his back, but it may be difficult to distinguish slight degrees of it from the tremor which may also be present. If the disorder increases when the eyes are closed, ataxia is certainly present.[1] Acute ataxia of the upper extremities or of one arm is a not unusual symptom of disseminated sclerosis; the onset may take place in this way (see below). The ataxia is associated with atony of the muscles and even with Westphal's sign, in exceptional cases only. The diagnosis can then be made from the accessory symptoms only, or eventually from the Babinski's or Oppenheim's sign, which can be demonstrated in spite of the hypotonia. In my experience this absence of reflexes is not a permanent, but merely a temporary symptom. In later stages I have occasionally seen the hypotonic alternate with the hypertonic condition.

A marked fatigability is sometimes found in the area of a certain muscle. I was the first to draw attention to this symptom (*B. k. W.*, 1887), and my experience has been recently confirmed and supplemented by E. Müller (*Med. Klinik*, 1905) and Claude-Egger (*R. n.*, 1906).

Early Stage.—The diagnosis of a disease is always most difficult in its first stage. This is particularly the case as regards sclerosis. It commences with *spinal or cerebral*, rarely with *bulbar* symptoms. Motor weakness in one or both lower extremities is usually the first symptom, and the *spastic spinal paralysis* may for months or even years be the only sign of the disease. By repeated and careful examination the diagnosis can be sooner or later established from the evidence of the cerebral symptoms, particularly of affection of the optic nerve, nystagmus, slowness of speech, attacks of vertigo, forced laughter, etc. etc. The cerebral symptoms may even usher in the illness. Headache, vertigo, visual disorders, and in exceptional cases epileptiform attacks may be the initial symptoms. I have particularly often found that disturbances of sight (optic neuritis or atrophy) have preceded the onset of the other symptoms for a considerable time, so long, indeed, that they are not connected with the nervous disease by either the patient or his physician. D. Frank [2] has collected my observations, which were partly published

[1] I regard the attempt of some writers to identify the intention tremor with the ataxia as wholly unjustifiable.

[2] *Z. f. N.*, xiv.

in earlier papers, and we have shown that there is a special type of this disease which is characterised by this early isolated affection of the optic nerve. Bruns and Stoelting [1] were able to confirm and supplement our observations. They found that about 30 per cent. of their cases commenced with optic atrophy. In one of my own cases unilateral optic affection was the only cerebral symptom during twenty years. In a few others the first sign was a severe attack of vertigo with vomiting. Tremor, commencing usually in one arm, may also usher in the disease. According to Mackintosh this onset with tremor or ataxia is comparatively frequent.

Course.—The disease almost always runs a *chronic* course, either simply *progressive* or with *remissions*. In the first case the symptoms become gradually aggravated, fresh ones appearing in slow succession, until death ensues after five to twenty years. There are isolated cases, however, in which the disease has a much more rapid course, proving fatal within a year (Fürstner, Gudden). In the really acute cases, such as the one described by Henschen, we are dealing not with sclerosis, but with acute disseminated myeloencephalitis (as is shown by the combination with peripheral neuritis in this case). Flatau-Kölichen, Wegelin, and others agree with him. Marburg [2] has recently given a detailed description of these acute forms; he states that the acute onset is often merely an apparent one, the preceding symptoms having remained latent or unnoticed. The acute process may also be superadded to a chronic one already in existence. Schlagenhaufer describes a case which terminated fatally within two months, and which from the pathological appearances he considered to be a focal sclerosis. Such cases show the close relation of disseminated myelitis to sclerosis. More frequently we find repeated remissions, improvement, or an arrest of progress, which, to the uninitiated, may even simulate recovery. These remissions may last for months, or even for a year and longer; then comes the relapse, and after a number of such attacks the condition becomes one of permanent illness. This *relapsing course*, to the frequency of which I directed attention in my earliest work, is particularly remarkable. In some of these cases we might speak of a progressive disease with regressive symptoms.

The relapses may be spontaneous or may be the consequence of some noxious agent (chill, over-strain, trauma, confinement, infective disease). Thus Uhthoff has shown that over fatiguing of the legs in walking may increase or cause relapses in the visual troubles. In one of my cases the disease progressed in stages each of which followed a confinement.

Atypical Forms.—Disseminated sclerosis may simulate spastic spinal paralysis, showing all the symptoms of this disease not merely in its first stage, but throughout its whole course. Still more frequently multiple sclerosis appears as a combination of *spastic spinal paralysis* with partial *optic atrophy* (Oppenheim). A few cases are known (Charcot, Bouicli, Edwards, Bikeles, Glorieux, etc.) in which the paralytic symptoms corresponded to the type of a *spinal* (or *cerebral*) *hemiparesis* of slow or

[1] *Z. f. Augenheilk.*, iii. See also the monograph by E. Müller, and his treatise in the *Z. f. N.*, Bd. xxix., and *N. C.*, 1905, in which he supplements our experiences with a large number of cases. Similar contributions are afforded by the work of Kampherstein (*A. f. Aug.*, Bd. xlix.) and Mackintosh (*R. of N.*, 1906), etc.

[2] "Die sog. akute multiple sklerose" (Encephalomyelitis periaxilis scleroticans), Leipzig-Wien, 1906.

relapsing onset (*the hemiparetic form of disseminated sclerosis*). The tremor in such cases tends to be limited to one side of the body. I have seen and described a case of this kind (with the post-mortem report).

In a small number of cases bulbar symptoms are prominent: troubles in deglutition and mastication, disorders of articulation and phonation (without real muscular atrophy). Marburg emphasises their frequency in the "acute" form. The diagnosis can then be made only from the accessory symptoms and the marked remissions. Glycosuria has occasionally been found (Weichselbaum, Richardière, U. Rose [1]). I have seen attacks of *acceleration of the pulse* and *conditions of asphyxia*. Nervous deafness occasionally occurs. Hemiatrophy of the tongue was noted in some cases. Although foci in the pons or medulla oblongata are as a rule the cause of the bulbar symptoms, they may also arise from bilateral foci in the cerebrum (Jolly, Claude). Gastric crises are mentioned, but are quite exceptional.

The literature contains some cases (Pitres, Dejerine) which show that disseminated sclerosis may simulate the picture of amyotrophic lateral sclerosis. Probst has described an interesting case of this kind, which, however, does not appear to be pure on account of the degeneration of the columns. Disseminated sclerosis is more frequently confounded with transverse; but the cerebral symptoms as a rule reveal the true nature of the disease.

Further, I [2] have observed in a number of cases in young adults a disease which I was at first forced to interpret as an acute inflammatory affection of the upper cervical cord, or of the cervical cord and the oblongata, because of the *acute* development of an *ataxia of the upper extremities with or without bulbar symptoms*, *e.g.* a crossed or alternate hemiparesis with corresponding alternating distribution of the sensory disturbances in one side of the face and the opposite side of the body. Many of these symptoms disappeared rapidly. My assumption that these represented the first stage of disseminated sclerosis was verified in several cases of this kind by the subsequent course.

I have also observed two cases which are very instructive in this respect. A woman of fifty-four, who consulted me for paraparesis, stated the following facts as to the development of her illness. She had had typhoid fever thirty-two years previously. This was followed by paralysis of the legs, weakness of the arms, and speech troubles (dysarthria). After three months she regained the power of walking, although with tremors and flail-like movements of the limbs. This condition gradually improved in the course of years. The patient, however, had always to use a stick. A year ago, the trouble became severely aggravated, and a sudden attack of vertigo was accompanied by disturbance of speech and a feeling of numbness in the left half of the tongue and left hand. Present condition: spastic paraparesis of the legs, intention tremor in the legs and left arm, hemihypæsthesia in the left side of the face and left arm, scanning speech, dysarthria, waggling of the head in walking. The second case is that of a woman of thirty-five, who at the time of my examination showed all the typical symptoms of disseminated sclerosis. She stated that at the age of eighteen she became acutely affected after typhoid by left hemiplegia, dysarthria, dysphagia, and bladder weakness. While the majority of these symptoms soon disappeared, a moderate degree of dysuria and rigidity in the right leg remained permanent; this condition persisted without a change for over ten years, and then, a few years ago, new symptoms developed in consequence of over-strain in nursing her husband.

These and other cases clearly show that disseminated sclerosis may develop from a post-infective disseminated myelo-encephalitis.

[1] "Multiple Sklerose und Diabetes mellitus," *Z. f. kl. Med.*, Bd. lv.

[2] On my suggestion, Cassirer has devoted a special discussion to this form (*M. f. P.*, xvii.).

Some further details may now be added as to the apoplectiform course of the disease. It has already been mentioned that apoplectiform attacks with subsequent transient hemiplegia may appear in the course of disseminated sclerosis. It occasionally happens that other symptoms and groups of symptoms may also develop in this apoplectiform way; for instance, an individual suddenly falls down unconscious or overcome with sudden vertigo. After this attack there is paraplegia or even paralysis of all four extremities, which slowly disappears. Such attacks may recur. Hemianæsthesia has been observed to return in this way in several cases. In one case I have seen, during the course of the sclerosis, an onset with severe vertigo of paralysis of the facial, auditory, and trigeminal nerves of one side, which entirely disappeared in a few weeks. Some months later a hemiataxia suddenly appeared, which also subsequently vanished.

In another case the first attack only produced the well-known affection of the optic nerve; after an interval of two years came the sudden onset of paralysis of the larynx and palate with glycosuria. After a rapid recovery there followed a year later symptoms of ataxic paraplegia in the legs and paralysis of the bladder and rectum, etc., with recovery again within a few months.

Further, a woman of twenty-seven, had at the age of fifteen hemiplegia with diplopia and dysarthria; recovery in two months followed by normal condition; at the age of eighteen, right optic neuritis with recovery: a similar attack in her twenty-third year also followed by recovery; patient then quite well. Three months after confinement onset of spastic paraparesis, slight bladder symptoms, paræsthesiæ in the hands; the picture is now one of a fully developed multiple sclerosis.

I have collected over a hundred cases of this kind, in which the disease showed acute exacerbations with intervals of varying duration.

In a course of this kind it is sometimes possible to localise the underlying focal lesion during the various exacerbations. Thus in one of my cases the symptoms at a certain stage pointed to a focal disease which had involved the posterior columns and anterior horns in the lower cervical region. My prediction that the ataxia of the arms and the atrophic paresis of the small hand muscles with partial reaction of degeneration would rapidly disappear was entirely fulfilled. In another case I found the knee-jerk markedly diminished in one leg during an exacerbation of the disease. The presence of thermal hypæsthesia in the area of the third and fourth lumbar roots pointed to the existence of a focus at a corresponding level of the cord, etc.

Complications.—Multiple sclerosis is somewhat frequently associated with hysteria. Its combination with tabes dorsalis has been observed by Westphal (apparently also by Mills), with paralysis agitans by myself and Jolly, with infantile myxœdema by Raymond-Guillain, and with syringomyelia by A. Schüller.

Differential Diagnosis.—The most important facts only will be mentioned. The distinction from paralysis agitans no longer affords any difficulty. Disseminated sclerosis has some symptoms in common with paralytic dementia, such as tremor, affection of speech, apoplectiform attacks, spastic paresis (which is present in a considerable proportion of cases of paralysis of the insane). These symptoms themselves, however, have a quite different character in the two diseases. The tremor in paralysis is inconstant, is not closely connected with voluntary movements, sometimes appears during rest, and the single oscillations are very unequal. The speech is not scanning, but is characterised by syllable-stumbling. The trembling and quivering of the lip muscles also gives rise to a peculiar tremulousness of speech which is not observed in sclerosis. In addition there are mental symptoms which in paralysis tend to appear

at the very commencement, whilst in sclerosis they are comparatively slight, even in the later stages. The other symptoms also differ in this respect. Mixed forms may, however, appear, as already noted. Siemerling's pathological researches seem to prove this fact. I myself have only twice seen this combination of disseminated sclerosis and paralytic dementia. The remarks upon pseudo-sclerosis and diffuse sclerosis may further be consulted on this point.

One must be specially careful in diagnosing disseminated sclerosis in childhood, although it undoubtedly does occur. The hereditary family forms of spastic paraplegia and diplegia, and the congenital or hereditary nervous diseases most closely allied to them, are specially apt to give rise to confusion (Pelizaeus, Freud, Sutherland, Cestan-Guillain, Jendrássik, Pásker, Bäumlin, etc.). I have twice recently had an opportunity of observing typical disseminated sclerosis in two members of one family. Reynolds reports similar cases (*Br.*, 1904).

In one of my cases the *essential hereditary tremor* had been transmitted through three generations. As the tremor had quite the character of intention tremor and was associated with an inherited scanning speech, the resemblance to sclerosis was striking.

Congenital nystagmus is also sometimes combined with other nervous symptoms, and may simulate the picture of disseminated sclerosis (Lenoble). As a rule, however, it is distinguished by being constant and of a rotatory character.

If the illness commences with cerebral symptoms, it may possibly be mistaken for cerebral tumour, encephalitis, or simple apoplexy. To distinguish it from tumour, weight should be laid specially on the absence of symptoms of brain pressure. Although optic neuritis may occur, it is very rare, only exceptionally reaches the degree of choked disc, is very transient, rapidly disappears, or produces merely a partial atrophy, and is often confined to one eye. The constant headache is also absent, and usually also the vomiting, slowing of the pulse, and the stupor. A few remarkable cases of disseminated sclerosis have, nevertheless, been described by Bruns and Nonne in particular, in which the symptoms of brain pressure, especially the choked disc, were so marked that the diagnosis of brain tumour was at first the only possible one. Rosenfeld has also seen choked disc in disseminated sclerosis. It may be that the combination of the sclerotic process with internal hydrocephalus modified the morbid picture in this quite unusual way, although this was not found by Rosenfeld. The evidence is opposed to tumour if the visual affection has for a long time preceded the other symptoms and has entirely or partly disappeared. The tremor, which may appear in tumour, is rapid and not of the character of intention tremor. It may resemble the tremor of sclerosis only in tumours of the cerebellum and its peduncle. In tumour the symptoms as a rule increase gradually, in sclerosis they show remissions and exacerbations. That diagnosis may be difficult is shown by a case of Westphal's, where he diagnosed disseminated sclerosis in a boy while the sectio revealed tumour of the optic thalamus.

Hereditary cerebellar ataxia (compare page 197) may occasionally give rise to perplexity with regard to the diagnosis, as may also a rare, diffuse cerebellar disease in adults, such as Schultze, Arndt (Oppenheim), Dejerine, Ladame, and Babinski describe. (For the symptoms of these, see the chapter on diseases of the cerebellum.) Touche saw symptoms in softening of the cerebellum, which reminded him of those of disseminated sclerosis.

The differential diagnosis between disseminated sclerosis and Friedreich's disease has been exhaustively discussed by Mingazzini.

There is a form of disseminated sclerosis which appears under the guise of acute encephalitis (especially of the pons and medulla) with focal symptoms. The diagnosis can only be established from the later course. As I have already mentioned, symptoms of alternating hemiplegia (Wizel) and of crossed hemiplegia (Oppenheim) may also arise. An interesting case which we observed and at first believed to be encephalitis, has been worked up and published by O. Maass. In a few cases (Strümpell, Bikeles) headache, vertigo, and apoplectiform or epileptiform attacks with subsequent hemiplegia were the only symptoms of sclerosis.

If an apoplectiform attack is the first symptom, the diagnosis cannot be definitely established at the commencement. It is, however, always very suspicious if young persons who suffer neither from heart disease, syphilis, nor alcoholism, have an apoplectiform fit with rapidly disappearing paralytic symptoms. The probability is great that the case is one of encephalitis, commencing sclerosis, or paralytic dementia, but it is only the further course which can definitely decide this. The disease may on account of its spinal symptoms be confused with the combined affection of the postero-lateral columns, and still more easily with spinal or cerebro-spinal syphilis. The determining criteria may be found in the corresponding chapters (compare also my memoirs, those of my pupils Cassirer [1] and Pini [2] and the monograph of E. Müller).

There is a general vascular disease of the central nervous system—probably of toxic origin—which may give rise to a picture very similar to that of disseminated sclerosis, the basis of which is not, however, sclerotic foci, but multiple foci of cerebro-spinal softening with secondary degeneration of the tracts in the brain and cord (Oppenheim). *Multiple encephalo-malacia* arising from arterio-sclerosis may also be closely related in its clinical symptoms to disseminated sclerosis. But the age, which is usually advanced, the evidence of atheromatosis in the heart and vascular system, the marked accentuation of the mental weakness and other cerebral symptoms, etc., usually make the distinction certain (compare chapter on pseudo-bulbar paralysis). Lannois and Lemoine have confirmed the diagnosis of disseminated sclerosis in a case in which diffuse processes were associated with degeneration of the tracts in the cerebro-spinal nervous system.

I remember one case only in which the diagnosis of polyneuritis had been given by other physicians on account of the pain, paræsthesiæ, hypotonia, and ataxia, whilst I, in view of the relapsing affection of the sight and the transformation of the hypotonia into hypertonia, was compelled to diagnose disseminated sclerosis. On the other hand, I saw a typical intention tremor follow incomplete recovery from a severe toxic (non-mercurial) polyneuritis.

Westphal [3] has shown that there is a general neurosis which cannot be distinguished either in its symptoms or course from the most common type of disseminated sclerosis. He calls it *pseudo-sclerosis*. In these cases the early appearance of severe mental disorders (apathy, delirium, etc.) was remarkable, and also the slowing of the movements of the eyes and face—nystagmus being absent—and the paradoxical phenomena, whilst ankle clonus (see below) was not present. Westphal himself insists that in doubtful cases the condition of the optic nerve may suggest the diagnosis, as naturally we should not expect it to be affected in this

[1] *D. m. W.*, 1896. [2] *Z. f. N.*, xxiii. [3] *A. f. P.*, xiv.

neurosis. Charcot's school has without sufficient reason classified Westphal's pseudo-sclerosis along with hysteria.

Our knowledge of this remarkable disease has lately been extended by Strümpell (*Z. f. N.*, xii.), who, after seeing a case of this kind, made the diagnosis in a second case during life. Frankl-Hochwart ("Obersteiner," x.) was also able to recognise the affection during life and to confirm his diagnosis by an autopsy. Strümpell also considers the early onset and the marked degree of dementia of great diagnostic importance; he thinks, further, that there is something characteristic in the kind of tremor which was distinguished from intention tremor in his patients by the range and slowness of the oscillations (2 to 3 per second), and also by the fact that it occasionally occurs even in rest, a circumstance which has been mentioned by other observers. The slow languid character of the voluntary movements has likewise been noted by Strümpell. He also lays stress on the fact that the affection of the motility seldom if ever amounts to complete paralysis, and then only for a short time. On the other hand hemiparesis or paraparesis are more frequently found. Cases published hitherto show that apoplectiform and epileptiform attacks are almost constant symptoms, that during the course of the disease contractures may develop—though not the true spastic conditions—and that the speech is not only scanning, but is markedly dysarthritic. Later observers have confirmed Westphal's view that nystagmus and affections of the optic nerve are absent in this disease. Atrophy of the optic nerve was present only in one exceedingly doubtful case of Mingazzini's, and nystagmus in Bäumlin's case. The cases of these writers and of Fickler (*D. m. W.*, 1904) seem to show that there is a hereditary, family form of this disease. In Frankl-Hochwart's case, the intermissions were very pronounced, and the long duration of the illness, extending over fifty-seven years, was particularly remarkable. He also mentions the symptom of propulsion.

Although according to these facts the diagnosis of pseudo-sclerosis from disseminated sclerosis may, occasionally at least, be made with some certainty, yet great and insuperable difficulties may arise from the fact that the clinical signs of pseudo-sclerosis may be superposed on those of so-called *diffuse cerebral sclerosis*. This term is, of course, applied to absolutely different conditions, such, for instance, as a form of atrophy and induration of one cerebral lobe of one hemisphere, which is found in many cases of infantile spastic hemiplegia. We may pass over these localised forms, as well as Bourneville's tuberose sclerosis.

Diffuse cerebral sclerosis has been shown to occur in young and adult subjects, and to present the picture of paralytic dementia or one closely allied to it. This may be the only change, or it may be accompanied by those of paralytic dementia, as in the cases of Greiff, Zacher, Fürstner, Strümpell, etc. There is another type of this disease, occuring in adults but more commonly in children, the clinical features of which show close relations to disseminated sclerosis, to pseudo-sclerosis and paralytic dementia. Cases are described by Berger, Bullard, Erler, Schmaus, Ganghofner, Heubner, Strümpell, D. Gerhardt, H. Weiss, Mingazzini, Rebizzi, Potts-Spiller, and others. Heubner has endeavoured to show that the disease runs a definite course, characterised by paralytic and mental symptoms, by a spastic paresis of the lower limbs which soon spreads to the arms, and by apathy and dementia, passing into stupor. Finally, there is generalised paralysis. Dysarthria and dysphagia also occur. Optic neuritis was only once noted, by Heubner himself. Hereditary syphilis and injuries to the head seem to play a part in the etiology. Weiss thinks that it may be the consequence of an acute, or even of a fœtal meningitis. The description given from their own observations by Strümpell, Weiss, and others does not entirely tally with that of Heubner, but it is almost identical with the account of the symptoms of pseudo-sclerosis as above described. Weiss lays special stress on the general tremor which takes place when there is any attempt at movement. Frankl-Hochwart mentions that dementia is a very constant symptom and is distinctly progressive. On the other hand, there are no long intermissions. Mingazzini states that along with a spastic hemiplegia of one side there is incomplete hemiplegia of the other. The boundaries between diffuse and pseudo-sclerosis are all the more indefinite in that Westphal and Strümpell have shown that a certain increase in the consistency of the tissue may also be present here and there in pseudo-sclerosis.[1] Although this

[1] Among other lesions we may mention the unusual extension of the Pacchioni granulations noted by Frankl-Hochwart, and the chronic leptomeningitis of Bäumlin. Other remarks which deserve consideration may be found in Campbell's paper ("Cerebral Sclerosis," *Br.*, 1905), but his description applies mainly to other forms.

attains a much higher degree in diffuse sclerosis, so that the white matter of the hemispheres, the corpus callosum, etc., may appear as firm as leather, the pons and medulla are shrivelled, diminished in size, and indurated, yet the histological changes are slight and not specially characteristic. There is therefore no sharp pathological distinction between pseudo-sclerosis and diffuse sclerosis. At present we can only say that there is a morbid form which clinically is closely allied to multiple sclerosis, and which is distinguished from it by its development in childhood (though it may appear later), in the early onset of dementia, by certain peculiarities in the tremor and speech disorder, by muscular contractions or tremors occurring during rest, and by the absence of nystagmus, of affection of the optic nerve, and of true spastic phenomena. To this

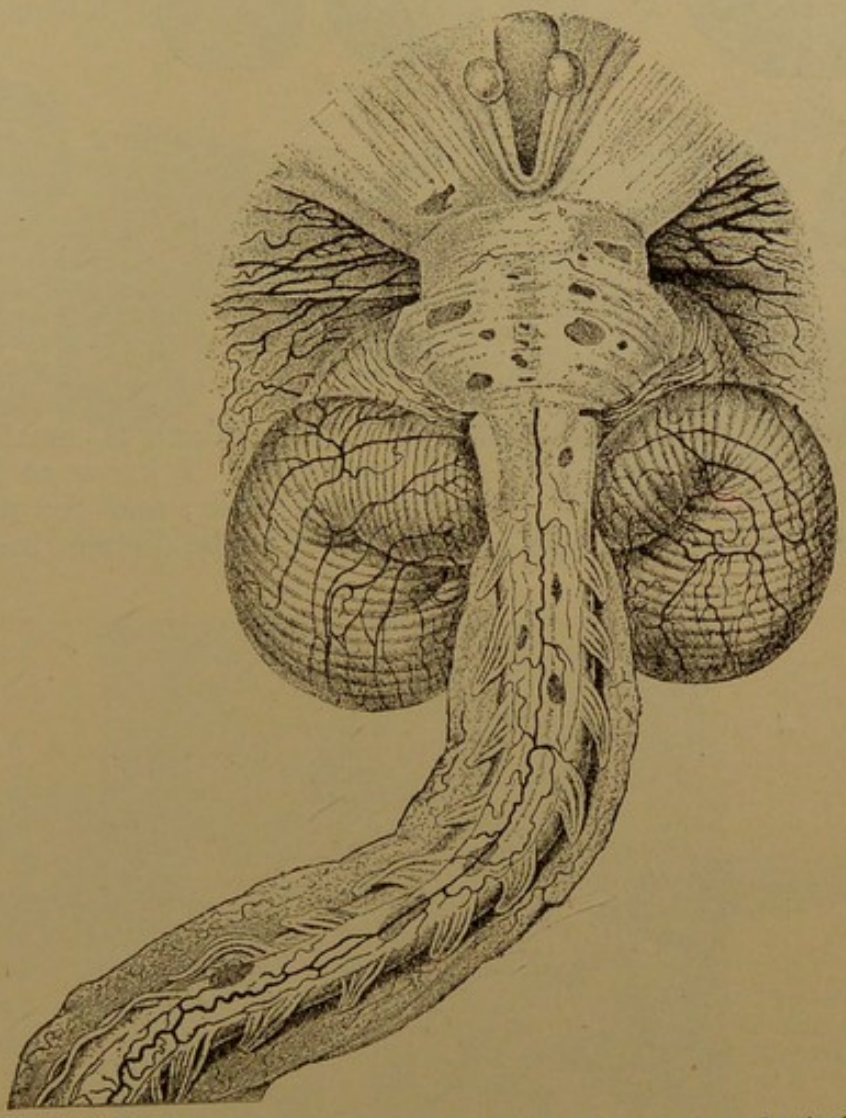

FIG. 170.—(After Leyden.) Disseminated sclerosis of the brain and spinal cord.

so-called pseudo-sclerosis there corresponds a negative pathological condition, or a slight degree of diffuse cerebral sclerosis. The same clinical picture, however, or one more resembling paralytic dementia, may also correspond to a condition of true diffuse cerebral sclerosis with degeneration of the pyramidal tract (Strümpell). We are not yet in a position to diagnose these affections definitely during life. The combination of progressive spastic paralysis with progressive dementia in childhood will suggest the possible presence of such diffuse cerebral sclerosis. In spite of the rarity of disseminated sclerosis in early childhood, we may in very young subjects sometimes give this as a probable diagnosis, but the question whether the disease is a diffuse or a pseudo-sclerosis must then generally be left open.

As regards the diagnosis between disseminated sclerosis and hysteria, the chapter on hysteria and p. 180 should be consulted. I should like to

remark here that in my experience disseminated sclerosis is extremely often mistaken for hysteria. This fatal error is made by eminent clinicians as well as by general practitioners. The fact that young girls and women are often affected by hysteria gives rise to this mistake;

FIG. 171.—Sclerotic foci at various levels of a spinal cord. *h* = foci. (By Weigert or Pal methods.)

FIG. 172. — Sclerotic foci in spinal cord. (Weigert's method.)

and yet the mere fact of the spastic paresis, which is usually present, should be sufficient to prevent this confusion. I know at least forty or fifty cases in which this error has been made, and the unfortunate sufferers have been, even in advanced stages of the disease, incited to be

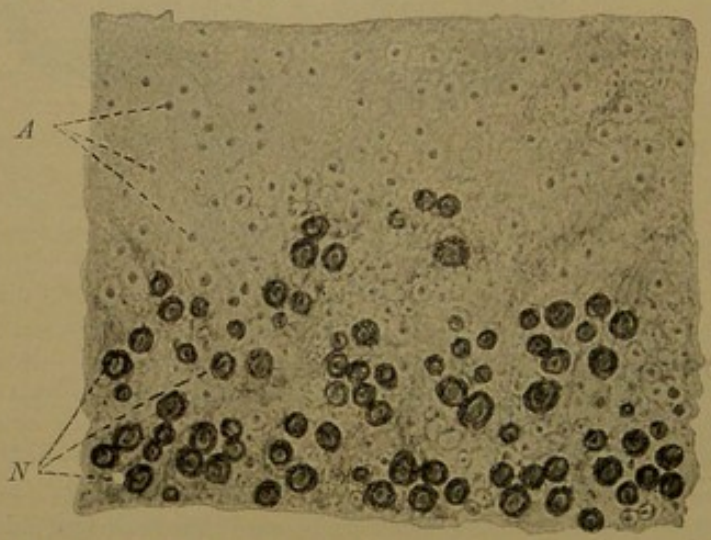

FIG. 173.—Naked axis-cylinders (*A*) in sclerotic foci. *N* = normal nerve-fibres. From a section stained with chloride of gold.

energetic, and distressed with courses of strenuous gymnastic treatment.

The tremor of mercury poisoning greatly resembles that of sclerosis (Charcot), but it is not associated exclusively with active movement, appearing sometimes also during rest and being increased on movement.

Nystagmus occurs in so many morbid conditions (brain tumour, affections of the ears, the nystagmus of hill-climbers, congenital nystagmus) that the diagnosis should not be determined solely by it.

A kind of pseudo-sclerosis—which yields to quinine—may develop in consequence of malaria.

Pathological Anatomy.—The symptoms just described are due to foci scattered over the whole central nervous system, and also some of the cranial nerves. They may for the most part be recognised by the naked eye (Figs. 170, 171, and 174 to 178). In the spinal cord their grey-blue colour shines through the pia mater. They are specially prominent on transverse section (Figs. 171, 172). Whilst the smallest foci can only be seen by the microscope, the larger foci in the cord reach the dimensions at a pea or a bean, and permeate the whole thickness of the cord, leaving only a few portions of it free. They are even more extensive in the pons (Figs.

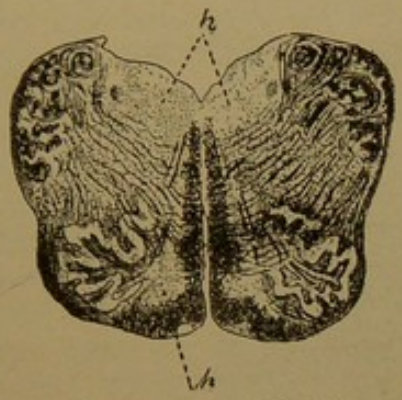

Fig. 174.—Sclerotic foci in the nuclei and roots of the hypoglossal nerve. *h* = foci.

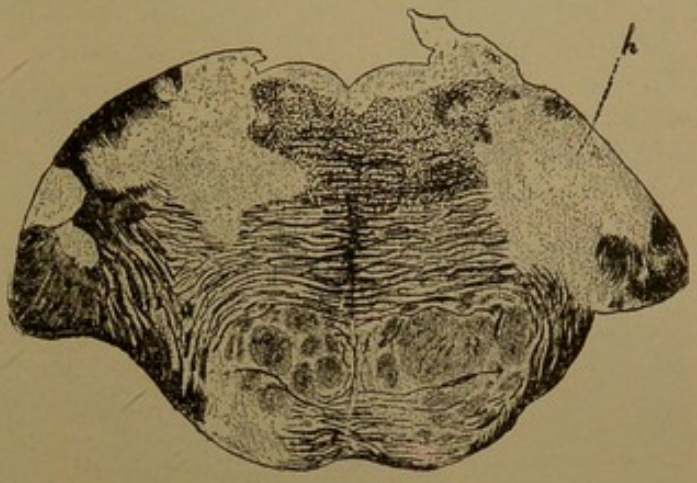

Fig. 175.—Sclerotic foci in pons.

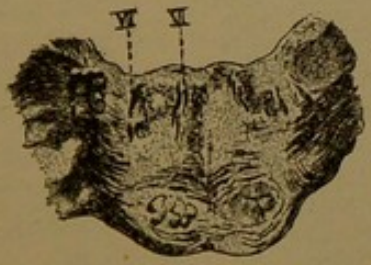

Fig. 176.—Numerous sclerotic foci in the pons at the level of the root of the abducens and facial.

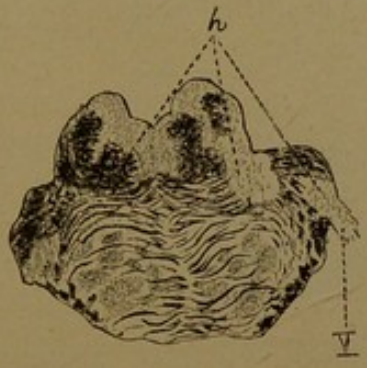

Fig. 177.—Sclerotic foci in the pons; one at the point of origin of the trigeminus.

Fig. 178.—Sclerotic focus in the optic chiasma. Frontal section.

Figs. 174 to 178 from sections stained by the Weigert method.

175, 176, 177) and medulla oblongata (Fig. 174); here, they may occupy one-quarter to one-half of the transverse area, or even the whole of it. The largest foci are found in the cerebrum, especially in its white matter. The cortex itself is frequently involved, as shown by observations of my own and of Taylor, Sander, Philippe-Jones, and Dinkler. These foci are as a rule multiple; we generally find them everywhere in large numbers and varying greatly in size. When they are very numerous in the spinal cord, however, there may be only isolated foci in the brain; the reverse is less often the case. They occur very frequently also in the optic nerve tract or chiasma (Figs. 178 and 180), and much more rarely in other cranial nerves. We require further proof before we can say whether the roots of the spinal

nerves and their peripheral portions may be affected. The sclerotic foci are found both in the white and in the grey matter. They stand out from the former more distinctly on account of their colour. They are usually sharply defined, roundish, angular, but may assume any form.

They are generally of a firmer consistency than normal tissue. There is often some induration of cerebro-spinal tissue outside the foci of sclerosis ; there is thus both a diffuse and a disseminated sclerosis. Consequently some parts, such as the pons and oblongata, are as a whole shrunken and diminished in size. Histological examination of the foci shows disintegration of the myelin sheaths of the nerves, persistence of

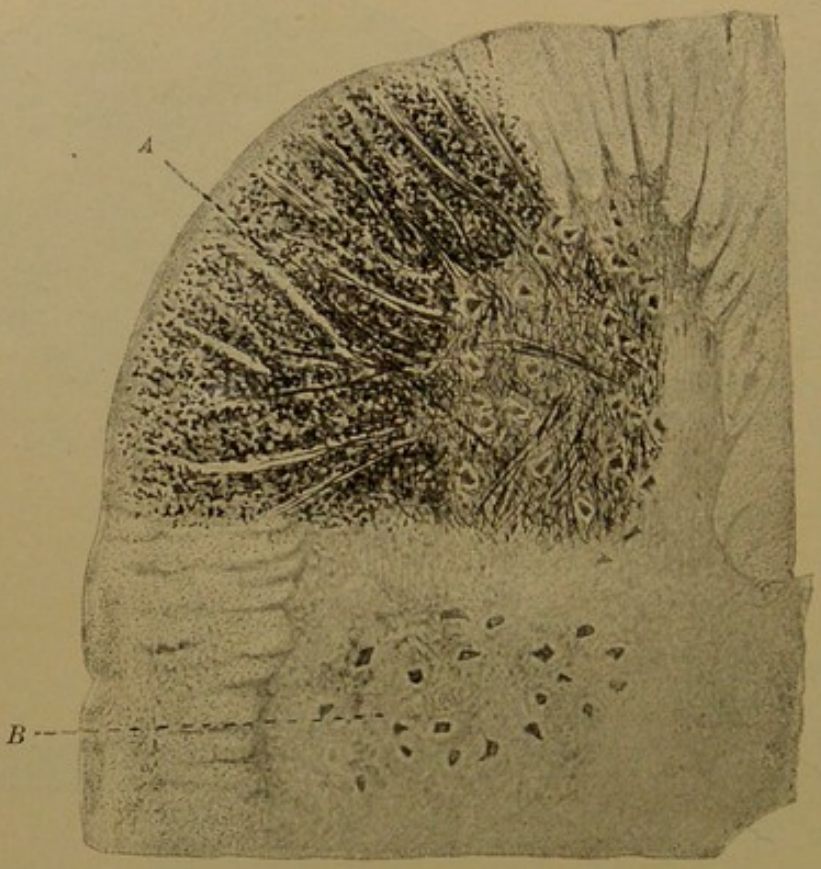

FIG. 179.—Sclerotic focus in the grey matter of the anterior horn ; atrophy of the nerve fibres. Ganglion cells intact. *A* = normal parts. *B* = sclerotic focus. Pal's carmin method.

many of the axis-cylinders (Charcot, Schultze, see Fig. 173), increase of the interstitial tissue, which may assume a fibrous condition, and frequently formation of new blood-vessels. Thrombosed vessels have also been found in the foci (Ribbert). Uhthoff-Lübben and Elschnig found intact axis-cylinders in the optic nerve. The foci in the grey matter affect the nerve cells to a much slighter extent than the nerve fibres (Fig. 179).

There is much difference of opinion as to the nature and starting-point of the process. Some writers (Charcot, Leyden, Erb, Goldscheider, etc.) think it an inflammatory, others (Köppen, Herz, Huber, Sander, Redlich, Thomas), a primary disease of the nervous parenchyma ; and Ziegler, Strümpell, Schüle, Probst, Thoma, Rossolimo regard the neuroglial proliferation as the primary process. This last view is specially defended by E. Müller, who makes a sharp distinction between secondary sclerosis after multiple encephalomyelitis and primary, true disseminated sclerosis. He agrees with Strümpell in calling the condition a multiple gliosis. According to

Schmaus congenital structural defect in the form of a hypoplasia of the nerve tissue is the essential underlying condition; in addition to this he admits that there is a lesion of the perivascular lymphatic system. Rindfleisch, Ribbert, Taylor, and Williamson consider that the affection arises from the vascular system, but Taylor himself has recently questioned this view. Rosenfeld also finds end-arteritis.

Marburg regards the "discontinuous" degeneration of the myelin sheath with the comparative persistence of the axis-cylinders as the essential part of the process, and traces an analogy between the process in the acute forms and Gombault's periaxillary neuritis. Bornstein and I have pointed out the great differences of the histological appearances in different cases, so that there can hardly be any idea of a uniform pathological process.

It should be remembered that we find the foci at every stage of development, and further that some are the result of acute myelitis and circumscribed encephalitis (Oppenheim, Leyden-Goldscheider), and that others have arisen insidiously. Secondary degeneration is almost always absent in sclerosis. The histological characteristics of multiple sclerosis, viz., the persistence of axis cylinders and absence of secondary degeneration, seem *a priori* to negative its origin in a myelitis. It should be remembered, however, that there is probably only one form of acute disseminated encephalomyelitis, viz., that accompanied by cell infiltration, which may give rise to sclerosis, and we have noted in the preceding chapter that in it the tendency to disintegration of the nervous elements, to softening and necrosis, is not present at least at the commencement. The close relation of this affection to disseminated sclerosis has been specially demonstrated by Finkelnburg in a case examined in my laboratory.

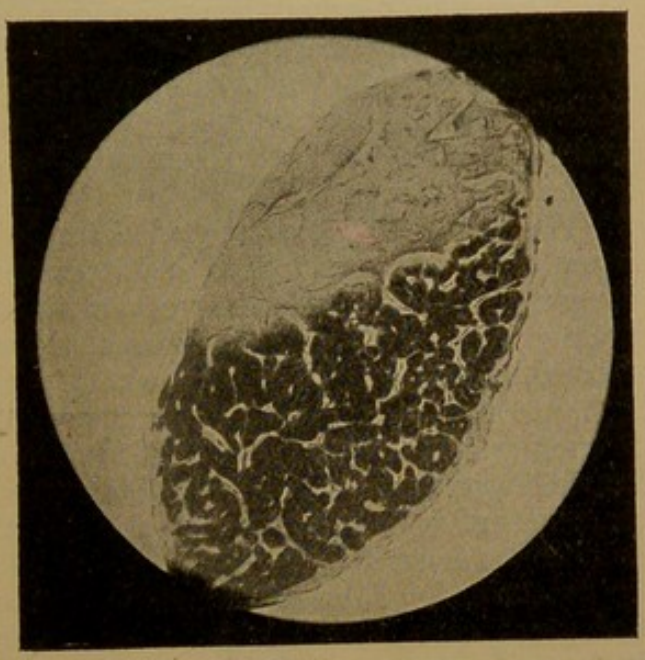

Fig. 180.—Sharply defined sclerotic focus in the optic nerve. (Pal's method.)

It is doubtful whether regeneration of the axis-cylinders takes place (Popoff, Erben, Lapinsky, Strähuber). Bartels and Bielschowsky protest against such an interpretation of the results.

When we see in the histological examination of a case of multiple sclerosis how severe is the destruction that the process has caused in all the parts of the nervous system, we are at first astonished that the patient has been able to exist at all, and that most of the functions were not abolished but merely impaired. The histological peculiarities just described, viz., the integrity of the axis-cylinders and nerve cells, explain this condition. The sclerosis creates, as I am wont to say in my lectures, multa, but not multum.

Prognosis.—The prognosis as to life is not absolutely unfavourable, as the disease has a very long duration, extending over decades. The so-called acute form is a very rare disease. When the symptoms indicate that the medulla oblongata is involved, death may occur at an early stage. On the other hand, I have seen a case of this kind, with repeated attacks

of asphyxia, loss of voice, and signs of vagus paralysis, in which life was retained for years.

The prognosis as to recovery is unfavourable, although the possibility of arrest, improvement, or even recovery is strongly insisted on by Charcot and even more so by Marie. Remissions which may simulate recovery are not uncommon. I have treated a case in which all the symptoms disappeared under the influence of erysipelas of the face, but I have been unable to examine the patient at a later date. Among a large number of cases, which I have been able to follow up, I have seen five or six in which there was an apparent recovery, no symptom being detected five to ten years later. We owe a valuable observation of this kind to O. Maass.[1] I have seen the disease progress with unusual rapidity in a vegetarian, who had undergone a Kneipp cure during the stage of spastic paraparesis. The form which appears in early childhood is apparently not always progressive.

Treatment.—Physical over-strain must be entirely avoided. Rest may cause a marked improvement, even in advanced stages. I have very frequently seen this in hospital cases. Hot baths are harmful. Electrical stimulation of the muscles which are in a condition of spastic paresis should be avoided. The confusion of disseminated sclerosis with hysteria is still unfortunately too often responsible for serious errors in the treatment.

We have no specific remedies for this disease. Nitrate of silver in the usual doses and also iodide of potassium are generally prescribed. I have recently used Crédé's silver ointment method in a number of cases, and believe it has done good in some of them. I have given mercury, which is recommended by others (Mühsam), without any benefit, and in one case I have seen a transient optic neuritis develop during treatment by inunction. Veronal may be recommended for the tremor (Combemale). Marie hopes that the future will bring us an effective anti-toxic method (?). A mild galvanic current applied to the back or head appears to be sometimes helpful. A course of baths at Oeynhausen or Nauheim may be prescribed. Mild hydropathic treatment may often be recommended, but great care must be taken in carrying it out. I have once seen, for instance, a sensory trouble appear directly after the use of a wet pack, but it very quickly vanished. If the disease takes the form of an acute relapsing myelitis and encephalitis, antiphlogistic-diaphoretic treatment should be adopted during the attacks. I have seen local blood-letting (by leeches) produce remarkable results under such circumstances. In some rather advanced cases we have succeeded, by using massage and active and passive movements in warm baths, in restoring the lost power of walking to a certain degree. It must always be remembered, of course, that such spontaneous remissions may occur even in the later stages of the disease.

Abscess of the Spinal Cord

is a very rare disease. In the cases reported (Ollivier, Jaccoud, Feinberg, Demme, Nothnagel, Ullmann, Eisenlohr, Homén, Schlesinger, Skala, Chiari, Cassirer, Silfvast) the disease has had either a traumatic or a

[1] *B. k. W.*, 1907. See also B. Bramwell, "The Prognosis of Diss. Scler.," *R. of N.*, 1905. He found recovery in four cases.

metastatic origin—following putrid bronchitis, gonorrhœa, purulent cystitis, suppuration of the prostate.

A case described by Pribytkoff-Maloljetkoff seems to us uncertain, as no primary focus of suppuration was found, and the suppuration was attributable to actinomycosis. The cases of Turner and Collier (*Br.*, 1904) are interesting because the exciting cause of infection, arising probably from the decomposed contents of the bladder, had settled in a part of the cord injured by compression from disease of the spine, and had led to an abscess which had extended from this site.

In the majority of cases the abscess was situated mainly in the grey

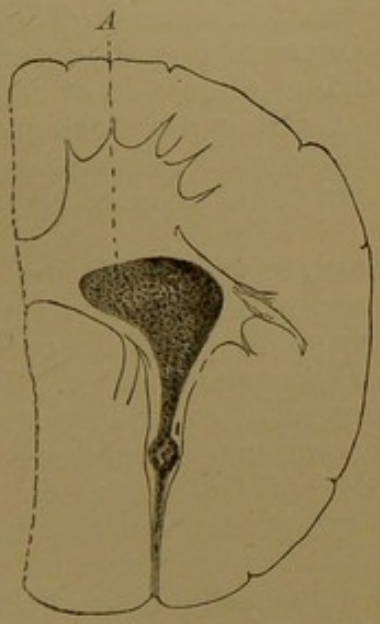

Fig. 181.—Abscess in the grey matter of the spinal cord (*A*).

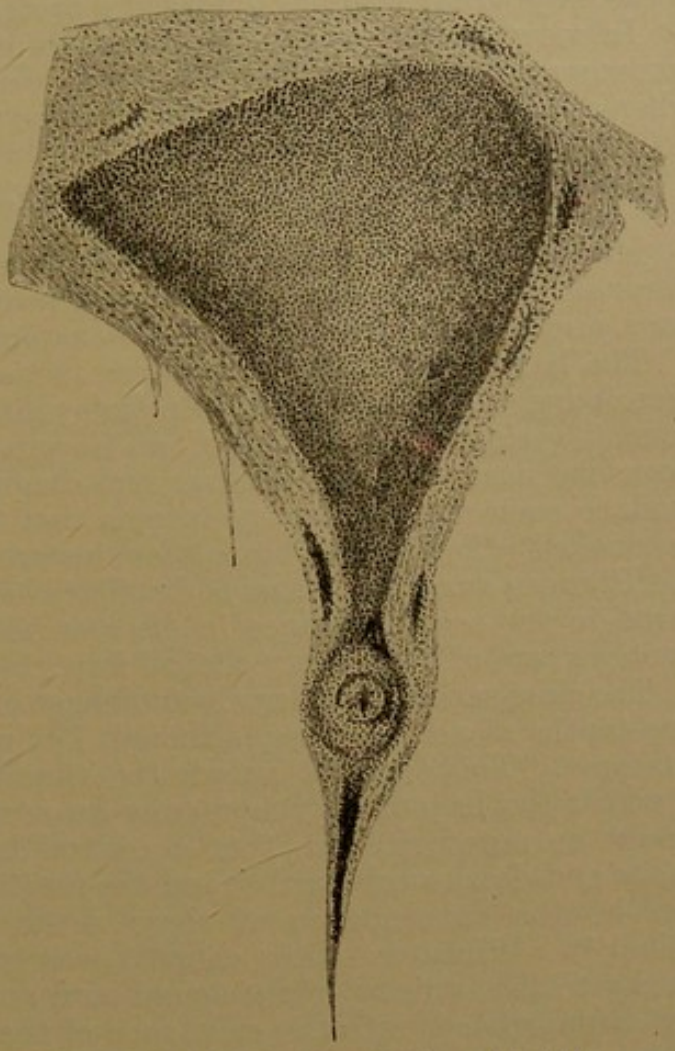

Fig. 182.—Abscess (*A*) from Fig. 181 under higher power.

From a section prepared by Schlesinger in Oppenheim's collection.

matter (Figs. 181 and 182), and was associated with suppurative meningitis. Occasionally foci of suppuration develop simultaneously in the brain. The upper sements of the cord are usually most affected, but suppuration of the lower segments, especially of the conus, has been observed (Schlesinger[1]). In one case the focus extended from the conus to the second thoracic segment. The clinical picture is seldom pure and shows little that is characteristic. Development of the paralytic symptoms is usually preceded some hours or days by signs of meingitic irritation—

[1] *Z. f. N.*, x.

particularly pain in the back and radiating pains. Then there appear either rapidly, in a moment, or more gradually in the course of some hours or days, symptoms of diffuse disease of the spinal cord, paraplegia, anæsthesia, paralysis of the sphincters, etc. The process may thus simulate the picture of an acute ascending myelitis. Retrobulbar optic neuritis was observed by Silfvast. In one of Homén's cases the paralysis of the lower limbs was accompanied by weakness, tremor, and incoordination in the arms. The general condition also shows the effects of the septic infection (fever, subnormal temperature, loss of strength, rigor, etc.). The disease runs a fatal course within a few days. Chiari is the only writer who has observed a protracted course.

In view of the sudden onset of symptoms of a diffuse and rapidly spreading affection of the spinal cord, we can only make the diagnosis when a focus of suppuration is found to be the source of the abscesses.

Hæmatomyelia. Apoplexy of the Spinal Cord

Hæmatomyelia is as rare as hæmorrhages into the brain substance are common. Its occurrence is, however, proved by good clinical observations, and by not a few post-mortem examinations.[1]

The hæmorrhage may take place in tissue already diseased. It has been found with myelitis, acute poliomyelitis, tumours and cavity formations ("syringal hæmorrhage" of Gowers; see next chapter). These *secondary* hæmorrhages have no real diagnostic interest, but they may produce such outstanding symptoms that the original disease is at first overlooked (Taniguchi's case, for instance, of carcinoma-metastasis; and Pfungen's analogous case). Capillary hæmorrhages, such as have been found in the cord substance in diseases with severe tonic or clonic contractions, are of hardly any clinical importance.

The chief cause of primary hæmorrhage of the cord, to which I attribute nine-tenths of all the cases, although Lépine gives a smaller percentage, is *trauma*. This does not include the injuries of the spine and of the cord in which the hæmorrhage is merely an accidental, accessory factor. It should be noted, indeed, that a central hæmatomyelia, extending upwards and downwards far beyond the limits of the traumatic focus, may also be the consequence of these severe injuries of the spinal cord (Minor[2]). Whilst in these cases, however, the symptoms will be due mainly to the bruising of the spinal cord and its roots, there is no doubt that hæmorrhages into the substance of the cord may be the only effect of injuries which have left the vertebræ themselves intact. Thus, a fall on the back, or from a height on to the buttocks or the feet, or a blow against the back may cause hæmatomyelia. The observations of Thorburn, Kocher, Stolper, Bailey, and Oppenheim show that *forced bending of the head forwards*, as in passing below a gateway, diving head foremost into water, etc., is very specially apt to produce hæmatomyelia (rupture-hæmorrhage according to Stolper).

Violent muscular strain must also be regarded as the cause in a considerable number of cases. It is shown to have occurred in *lifting a*

[1] The existing material has recently been collected and reviewed by Pfeiffer (*C. f. allg. Pathol.*, vii.) and Lépine ("Etude sur les hématomyelies," *Thèse de Lyon*, 1900). An article of special value has been written by Browning on Spinal Hæmorrhage, *Med. News*, 1905. See also the literature in Minor, "Handbuch der pathol. Anat. d. Nerv.," ii.).

[2] A. *f. P.*, xxiv. and xxviii.

weight, in military drill, even in coitus. One of my patients while lifting a box was suddenly struck down with paraplegia, which was of a flaccid, atrophic type, and accompanied by partial sensory paralysis. In another a Brown-Séquard paralysis came on after bowling, and disappeared in a few weeks. I have seen signs of a spinal hæmorrhage in a few cases in children after a fall on level ground. Whether in such cases there must be some predisposing condition, such as an abnormal fragility of the vessels, or some obstruction in the vascular system, or whether healthy vessels may also rupture under these conditions, is a question difficult to answer.

The onset of hæmatomyelia is certainly favoured by a *hæmorrhagic diathesis*. Thus I have observed in the case of a "bleeder" that the mere attempt to break with the foot a piece of wood at some height from the ground brought on symptoms of spinal hæmorrhage. In another case, where such symptoms appeared in a soldier after simple manual exercises during drill, there was no suggestion of a general tendency to bleeding. Steffen describes hæmatomyelia in *purpura*, Teichmüller in *pernicious anæmia*. It has also been observed in *typhoid*, as in the cases of Curschmann and Schiff, and also in the *puerperium* or after a *severe confinement*. Finally, *suppression of the menses*, *hæmorrhoidal bleeding*, etc., may give rise to hæmatomyelia.

In a remarkable case described by Bruce, where a tubular hæmorrhage occurred during the vomiting of pregnancy, there was a tumour and a cavity which had been latent till then, the hæmorrhage being thus secondary.

Difficult labour (dystocia) may also give rise to hæmatomyelia in the new-born child (Litzmann, Schultze, Pfeiffer, Couvelaire). O. Schäffer has found hæmorrhages in the spinal canal in 10 per cent. of all the post-mortem examinations of new-born children. In some cases Schultze's method of inducing respiration is said to be the cause (Knapp), but in such cases other conditions are usually also present. I have seen a case in which the paralysis of the whole body of an infant could be explained by a hæmorrhage into the cord. Raymond has given the same explanation in another case. The association of traumatic rupture and laceration of the spinal roots with hæmatomyelia is pointed out by Déjerine.

The case of Guizetti-Cordero,[1] in which a hæmatomyelia was due to a flattened aneurysm of the central spinal artery, is unique.

Alcoholism seems to increase the predisposition to hæmatomyelia (Jestkoff). In isolated cases (Boinet) no cause of any kind can be assigned.

Spinal hæmorrhages almost always occur in the grey matter (Fig. 183); the apex of the posterior horns is sometimes affected. The grey matter is richer in vessels, looser, more yielding, and is supplied by specially large arterial branches. This is the reason why it is more frequently affected than the white matter. According to Minor's investigations, the lateral columns are almost always spared. The experimental observations of Goldscheider-Flatau [2] have given important evidence as to the mode of extension of hæmorrhages in the cord.

With regard to their site, any segment may be affected, the enlargements, in particular the cervical, being more frequently involved than the other parts of the cord. The hæmorrhage extends over the whole central grey matter, or is limited to one side, even to one horn or to

[1] *Rif. med.*, 1903.

[2] *Z. f. kl. M.*, xxxi.

the anterior and posterior horns of one side (Fig. 183). It is seldom so extensive as to give rise to swelling of the cord. The extravasation of blood appears blue through the attenuated substance of the cord, even before it is opened. It tends specially to spread in the longitudinal direction of the cord, in the form of an elongated tube (tubular hæmatomyelia, a term which Minor would replace by that of "hæmatomyelia centralis longitudinalis"), as in a case of Levier's, in which the column of blood extended eleven cm. through the whole lumbar region and the conus medullaris, and in one of Leyden's, in which a fissure of the cord filled with blood stretched through almost its whole length. Fickler gives another explanation of the onset of tubular hæmorrhage. Multiple hæmorrhagic foci or a disseminated extension are seldom seen (Bailey). Minor speaks of a hæmatomyelia annularis. The tissue into which the bleeding takes place is crumbled and softened, and a myelitis may develop after the hæmorrhage.

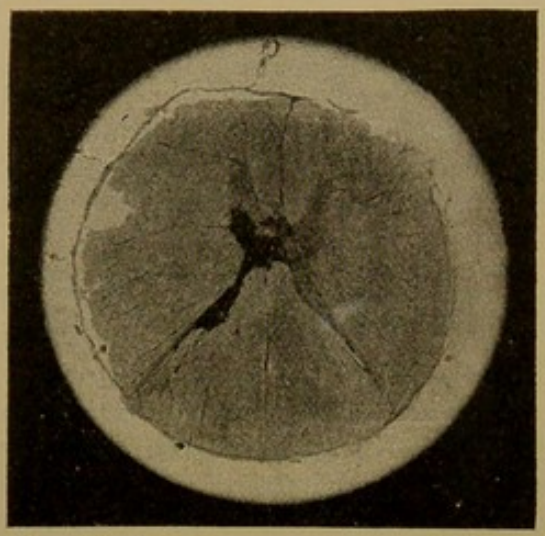

Fig. 183.—Transverse section of spinal cord in hæmatomyelia. Hæmorrhage in the grey matter of the left anterior and posterior horn. (From a section by Minor in Oppenheim's collection.)

Symptomatology.—The symptoms of hæmorrhage are those of a sudden and unexpected interruption of conduction in the spinal cord. The patient falls to the ground without warning, becomes paraplegic, cannot move the legs or any limb, and is affected with anæsthesia, paralysis of the sphincters, etc. Vasomotor and trophic troubles may soon occur. Sometimes the paralytic symptoms increase and extend somewhat during the first hours, but this seldom happens after a longer interval. Fischer has described, it is true, a remarkable case in which the extension of the spinal symptoms was slower and continued for several days, but the case is in other respects so unusual that we can draw no practical conclusions from it. The necrobiotic and inflammatory processes developing in the region of the hæmorrhage may, of course, give rise to symptoms which appear only after some days and which gradually increase. Fickler[1] speaks of a "late spinal apoplexy."

In other cases the symptoms vary according to the site of the hæmorrhage, and in any case they point to a lesion affecting the grey matter, mainly or exclusively (*central hæmatomyelia*). The lumbar enlargement is not infrequently the site. There is flaccid paralysis of the legs with absence of the tendon reflexes, anæsthesia either complete or partial, etc., and later, signs of muscular degeneration. The cervical enlargement is more frequently the site of the hæmorrhage. We find atrophic paralysis of the upper, spastic paralysis of the lower extremities, etc.; the former is usually partial, and various nerve areas are involved in the paralysis, according to the segment of the cord affected. If the hæmorrhage is in the lower part of the cervical enlargement, oculo-motor symptoms are present. Taylor and Collier state that optic neuritis occurs

[1] *Z. f. N.*, xxix.

in hæmorrhage of the upper cervical region. Schott describes bulbar symptoms in a case of this kind, which was, however, only clinically observed. The hæmorrhage is frequently limited more or less completely to the grey matter of one side, and involves the white either directly or by compression. The symptoms which result in such cases are those of an acute *Brown-Séquard paralysis*. The picture derives its characteristic features from the preponderance of the affection in the grey matter. Thus in some cases (Minor, Oppenheim, Raymond-Guillain) the following symptoms were present : *Partial atrophic paralysis of one arm, spastic paralysis of the corresponding leg, partial sensory paralysis, and specially analgesia and thermo-anæsthesia* of the leg of the opposite side. This group of symptoms is evidence of a hæmorrhage in the anterior and posterior horn of one side of the cervical enlargement, which compresses the neighbouring antero-lateral column. The localisation and character of the sensory symptoms may also correspond to the type of syringomyelia. Minor has noted that in severe lesions of the cord, there may be a zone of partial sensory paralysis forming the upper margin of the total anæsthesia : this he attributes to the central hæmatomyelia.

In dorsal hæmatomyelia, as I myself have found, the symptoms due to lesion or compression of the white matter are naturally the most prominent, so that the picture does not essentially differ from that of an acute myelitis.

Egger found that the sensory conduction was retarded.

Hæmorrhage in the conus terminalis is rare (Oppenheim, Raymond, Schiff, Higier, Bregman, Laignol). Schlesinger saw it occur in straightening of a congenital dislocation of the hip by Lorenz's method. The symptoms are paralysis of bladder and intestine and anæsthesia in the region of the third and fourth sacral nerves (usually partial).

If death does not rapidly ensue—and this is not usually to be expected in uncomplicated cases—there is as a rule *improvement* within a few days or weeks. Some of the paralytic symptoms, which are merely the result of compression, gradually disappear. The urine, which has at first to be drawn off by a catheter, can often be passed spontaneously on the third or fourth day. If there is at first total paraplegia, some of the muscles will gradually regain power of movement, etc. This improvement, however, only reaches a certain degree ; symptoms caused by destruction of the substance of the cord do not disappear, and as the grey matter is most affected, when the hæmatomyelia is in the cervical, lumbar, or lumbosacral region, symptoms of degenerative paralysis and sensory disorders remain permanent. ("The nuclear symptoms remain, the distant symptoms vanish.")

At the commencement, there is frequently pain in the back, not however severe or persistent. There may be rigidity in the back, but only when the spinal meninges are involved. Within the first days and weeks, the temperature may be slightly raised. In one case where the hæmorrhage gradually extended along the cord from below upwards, there was on the second or third day an aggravation or an extension of the paralytic symptoms from the lower to the upper extremities.

In small hæmorrhages the symptoms may be so slight that they are easily overlooked. In a few cases of workpeople who had been blamed for malingering, I was able to diagnose a traumatic hæmatomyelia. In one of these the atrophic paralysis (with reaction of degeneration) was

confined to the triceps, in another to the gluteal muscles, and in a third along with the partial sensory paralysis there was merely atony limited to certain groups of muscles (unilateral absence of the supinator and triceps jerks) and fibrillary tremors.

The *prognosis* as to life is not unfavourable. In extensive hæmorrhages death may occur during the first days, or later from decubitus, cystitis, etc. This, however, is not the usual termination. Improvement is the rule, complete recovery the exception. The symptoms which persist in unchanged intensity after the lapse of a few months—the muscular atrophy with reaction of degeneration—may be expected to be permanent. The prognosis for the future is somewhat influenced by the fact that hæmorrhage—especially, it would seem, if occurring in the grey matter of the cervical region—may give rise to the development of a gliosis (Minor, A. Westphal).

Minor has shown that traumatic hæmorrhage of the cord may be associated with the formation of cavities. This is termed hæmatomyeloporosis by van Gieson, in contradistinction to syringomyelia. Kienböck also would strictly separate "traumatic central myelodelesis" from syringomyelia as a non-progresive process. Kölpin (*A. f. P.*, Bd. xl.) on the other hand maintains that there is a relation between syringomyelia and hæmatomyelia. Lloyd, Pitres, Lax, and Müller have also contributed to this subject.

The forensic questions connected with hæmatomyelia and meningeal apoplexy have been discussed by Deetz (*Vierteljahrschr. f. ger. med.*, xxvii.).

Differential Diagnosis.—Hæmorrhages into the spinal meninges give rise as a rule to marked *symptoms of irritation* : severe pain in the back, radiating pains in the nerve tracts, stiffness in the back, tenderness of the muscles to pressure ; the other symptoms are those mainly of affection of the roots. It is unusual for symptoms of compression of the roots and cord to develop gradually and only to attain their full development after many weeks, as in the fatal case described by Bull. Browning states that intense pain is not usually present in epidural hæmorrhages. On the whole these spinal hæmorrhages are not of great clinical importance, as they are almost always accompanied by other conditions due to the injury (Stolper). Lumbar puncture may decide the diagnosis (Kilian, Jacoby, Braun, Gaussel), should not the possibility of hæmatomyelia contraindicate its use. Hennneberg's observations have shown that hæmorrhages in the spinal meninges may be caused by lumbar puncture itself.

In a case of meningeal hæmorrhage under my observation, the muscular tension was so great that the muscles felt as hard as boards.

Epidural hæmorrhages, which are rare, may give rise to similar symptoms from compression of the cord (Miles, Gaussel). With regard to the results of lumbar puncture under these conditions, see Gaussel, *R. n.*, 1905.

Browning, who discussed the differential diagnosis, thinks that dissociated sensory symptoms do not appear in these epidural hæmorrhages.

Myelitis has almost never so sudden an onset, although a few cases (Williamson, Strull) are reported in which it was completely developed within a few hours. As a rule there are premonitory symptoms, and in cases of the most acute course there is usually a high rise of temperature. A rapid regression of the symptoms is also unusual in myelitis. In the myelomalacia due to occlusion of the vessels, the paralytic symptoms may develop in an almost apoplectiform way (Langdon, Dinkler, Oppenheim, Mariani). In doubtful cases of spontaneous onset, the diagnosis

of myelitis or myelomalacia is warranted rather than that of hæmatomyelia, although in a case of Fischer's in which a spinal hæmorrhage was found post-mortem, the diagnosis had been given as that of acute myelitis. On the other hand it must not be forgotten that trauma may also give rise to myelitis and myelomalacia.

Acute poliomyelitis is usually ushered in by a feverish stage; the paralytic symptoms otherwise show a pure affection of the anterior horns. The possibility of a confusion cannot, however, be excluded, as an anterior hæmatomyelia, a hæmorrhage limited, according to its symptoms, to the anterior grey matter of the cord, does occur (Raymond, Giwayo).

Treatment.—Absolute rest for the first two or three weeks is necessary, more so in this than in any other disease of the spinal cord. The patient must be carefully carried to bed and should lie not on his back, but upon his side or abdomen. In strong subjects, blood should be withdrawn by venesection or by leeches applied to the back. Injections of ergotin are also recommended. The patient must avoid coughing, sneezing, and straining at stool as far as possible. The well-known precautions should be taken to avoid bedsores and cystitis. If the first stage is passed, warm baths may be used. I have seen good results from baths in Oeynhausen in two cases. Electrical treatment should be employed for the atrophic paralysis. (For further details see chapter on Myelitis, etc.)

In one case of meningeal hæmorrhage (Bugge) recovery was induced by lumbar puncture, 160 c.c. of blood being withdrawn. Albertin also notes good results from this measure, whilst in a case of Kilian's, death followed in spite of puncture. Epidural hæmorrhages are most accessible to surgical treatment (Browning).

Diseases of the Spinal Cord from Considerable Alternations of the Atmospheric Pressure. (Diver's Disease, Caisson Disease.)

In divers, and bridge or harbour-workers who are compelled to work under the water in caissons, *i.e.* diving-cages, under an atmospheric pressure of 1-4 or 5 atmospheres, paralytic symptoms of cerebral and spinal origin may develop when they emerge from the cage and are subjected to the sudden great lowering of the air pressure. Spinal symptoms are the more frequent. Immediately on leaving the caisson, the patient complains of pressure on the head, vertigo, pain and buzzing in the ears, nausea, and weakness in the legs. These may increase to paraplegia within a few moments. The symptoms usually correspond to a diffuse affection of the thoracic cord; there is spastic paraplegia, anæsthesia, weakness of the bladder, etc. Severe pain is also usually present. Symptoms indicating a special affection of the posterior columns are less common. In a severe case which was under my observation for a considerable time, the paralysis was associated with very great rigidity of the muscles of the legs and abdomen, and every movement, every stimulation of the skin gave rise to severe clonic contractions in the latter, to involuntary loss of urine, and to erection of the penis.

If the paralytic symptoms are slight, complete recovery is possible, and it takes place within a few weeks or months. In grave cases the disease is incurable. Death may occur immediately.

The results of post-mortem examinations have been recorded in a small number of cases only (Leyden, Schultze, Renssellaer, Catsaras, Hoche, Schrötter, Lie). Leyden found small fissures and tears in the thoracic cord, which he attributed to foci of softening. Small foci of local necrobiosis have been found in other cases. It is thought (P. Bert, Leyden, Hoche, Parkin, White-Bainbridge, Macnaughton, experimental investigations by Lépine) that in consequence of the sudden decrease in the pressure of air, gas-bubbles escape from the blood and thus cause an embolism of the small arteries in the cord (specially in the lateral and posterior columns of the thoracic region) with secondary softening. The condition and its causes are exhaustively discussed by Boinet and

Audibert (*Arch. gén. de Méd.*, 1905). We cannot here enter into the other reports and theories put forward, especially by American physicians.

Friedrich and Tausk describe changes in the internal organs. With regard to prophylaxis a stern warning should be given against the rapid passing from the increased atmospheric pressure into the normal one. The transition must be gradual, a rule which, so far as my experience goes, is already carried out, as since the introduction of medical supervision the troubles arising from work under compressed air are becoming more and more rare. Schrötter recommends that the patient should immediately on the onset of the illness be brought again under increased pressure of air (recompression). The hygiene of work in compressed air is thoroughly discussed by Silberstern. It is also treated by Parkin (*R. of N.*, 1905) and Zografidi. The treatment of the disease after its development is otherwise practically that of myelitis.

Tumours of the Spinal Cord

Of the comprehensive and important papers of recent times on this subject the following should be mentioned: Horsley and Gowers, "A Case of Recovery after Removal of the Spinal Tumour," *Brit. Med. Journ.*, 1888; Bruns, "Geschwülste des Nervensystems," 2. Aufl., Berlin, 1908; H. Schlesinger, "Beiträge zur Klinik der Rückenmarks- und Wirbeltumoren," Jena, 1898; Henschen and Lennander, *Mitt. aus d. Grenzgeb.*, x.; H. Oppenheim, *B. k. W.*, 1902, No. 2 u. 39; F. Schultze, *Mitt. aus. d. Grenzgeb.*, xii.; H. Oppenheim, *Mitt. aus d. Grenzgeb.*, xv.; Cushing, "Annals of Surgery," 1904; Walton and Paul, *Boston Med. and Surg. Journ.*, 1905-6; Auerbach und Brodnitz, *Mitt. aus d. Grebzgeb.*, xv.; Stertz, "Klinische und anatomische Beiträge zur Kasuistik der Rückenmarks- und Wirbeltumoren," *M. f. P.*, xx.; H. Oppenheim, "Beiträge zur Diagnostik und Therapie der Geschwülste im Bereich des zentralen Nervensystems," Berlin, 1907.

If we except gliosis, which will be considered in a special chapter, we may say of the other tumours of the spinal cord that they arise for the most part from the meninges. Whilst, according to H. Schlesinger's statistics, tumours of the vertebræ with secondary involvement of the spinal cord are considerably more frequent than all the meningeal and spinal neoplasms taken together, the proportion of those arising from the meninges to those coming from the cord itself is given as 7 : 3 or 6 : 4. Intraspinal tumours are most frequent in the region of the enlargements, whilst in the thoracic cord extraspinal tumours are considerably more common. Tumours developing outside the dura are rare; these are lipomata and hydatid cysts. According to our personal experience, for every fifteen cases of extra-medullary tumour, hardly one originates epidurally, tumours of the vertebræ being, of course, excepted. Within the dura, and arising from it or from the leptomeninges, we have sarcoma, endothelioma, psammoma, syphiloma, tubercle, and especially fibroma and its mixed forms, also myxoma, angiolipoma, and other mixed growths. Glioma seldom forms a solitary tumour nodule in the cord. As a rule it is diffuse and spreads over long extents of the spinal cord, and its distribution may also be a disseminated one. Microscopically, there may be no evidence of tumour, so that the nature of the disease is only explained by histological examination (Stertz, Oppenheim), as we find in certain brain tumours also.

There are gliomata in which the cell elements are less prominent than the fibrous elements (Stertz).

Sarcomata appear as solitary tumours in the meninges or nerve roots, or they may form multiple nodules. These develop in the nervous substance itself and on the membranes of the nervous central organs (Schultze, Hippel, A. Westphal, Schlagenhaufer, Nonne, Spiller-Hendrickson), are confined to the membranes in which they form multiple nodules, or spread

diffusely over the surface (Richter, Ollivier, Schulz, Orlowski, Nonne, Stanley Barnes). Grund observed the extension of an intraspinal growth on the meninges, as did Seiffert. The tumour character may be so little marked that confusion with chronic meningitis is possible (Rindfleisch). Cases are described in which the spinal cord itself was to a great extent destroyed by these tumours (Forster, Malacaster, Ross, Holmsen). In multiple and diffuse sarcomata the structures of the posterior cranial fossa, especially the cerebellum, are usually affected (H. Schlesinger). On the whole the sarcomata arising in the meninges have little tendency to extend into the substance of the spinal cord itself (A. Westphal). Of

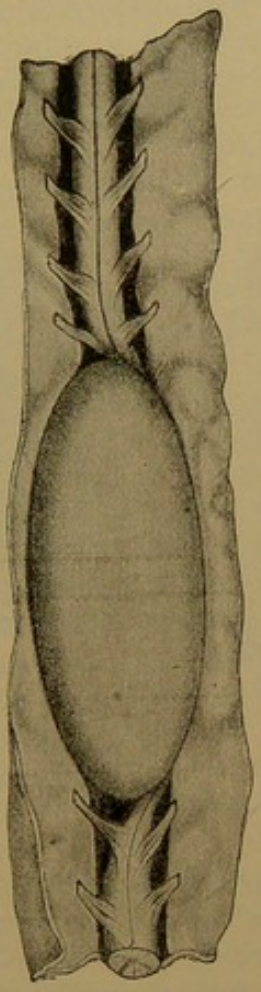

Fig. 184.—Tumour of the spinal cord. (After Braubach.)

Fig. 185.—Neuroma of the cauda equina. (After Lanceraux.)

the rare forms of tumour we may mention cylindroma, myolipoma, neuroepithelioma gliomatosum microcysticum, melanoma or chromatophoroma (Ribbert), and a form once found in the spinal cord by Pick and Esser. Aneurysms of the arteries of the spinal cord are apparently exceedingly rare (cases of Guizetti-Cordera, Raymond-Cestan).

Carcinoma-metastases in the membranes of the cord have been found only in exceptional cases (Bruns, Ballet, and Laignel-Lavastine).

Multiple neuromata and sarcomata may appear on the nerve roots and simultaneously in the spinal cord, brain, and cranial nerves. The combination of general neurofibromatosis with glioma of the spinal cord has occasionally been observed. Devic and Toldt describe multiple

angio-sarcomatosis with involvement of the spinal cord. Hydatid cysts sometimes occur; cysticerci are much rarer in the spinal canal or cord. Pichler described one such case some time ago. I myself observed a cysticercosis of the meninges of the brain and spinal cord (see chapter on Cerebral Cysticercus). Hydatids usually lie between the dura and the bones; they may project externally after erosion of the bone. In one case (Friedeberg) there were numerous cysts in the sacral canal as well as in the spinal canal, as high as the upper thoracic region; the sacrum itself was destroyed by them. Cysticerci on the other hand tend to appear within the dura. Cysts of other kinds and of unknown genesis are found in exceptional cases in the meninges (Schlesinger, Schmidt, Spiller); their connection with so-called serous spinal meningitis (see below) specially requires explanation. Syphiloma, glioma, sarcoma, and

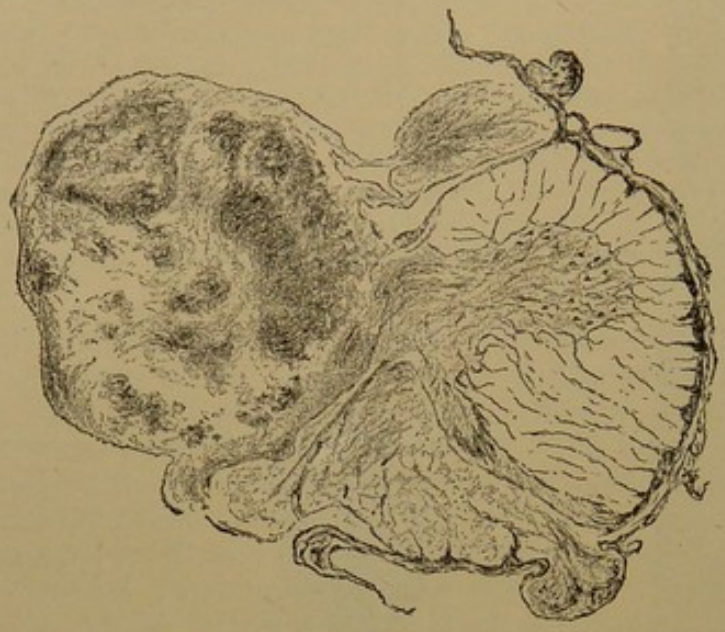

Fig. 186.—Tumour of the spinal cord. (Transverse section.)

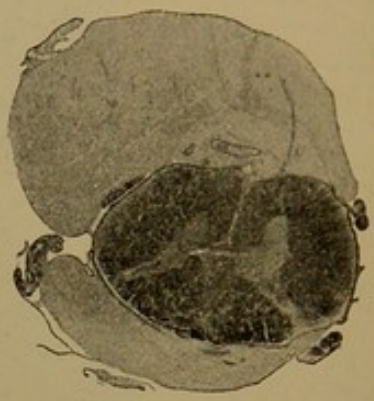

Fig. 187.—Sarcoma of the spinal cord, which compressed it from before and behind, without extending into it. Pal-carmin section. (Photograph.)

tubercle are found in the cord. Glioma and tubercle frequently arise in the grey matter, and sarcoma may have the same origin (Schiff).

Lipomata are congenital. In other kinds of tumour also congenital disposition apparently plays a part. Heredity constitutes an essential factor in the etiology of *multiple* tumours. There is nothing definitely known otherwise as to the etiology, if we except infectious and parasitic tumours. The fact is worthy of note, however, that symptoms of the disease have repeatedly become manifest for the first time after injuries.

The growths arising from the spinal meninges are usually small, the size of a pea, almond, cherry, or hazel-nut, or on an average that of a date or olive (Figs. 190, 192, etc.), but if they grow mainly in the vertical direction they may attain a great size and may extend over two inches or more of the cord (Figs. 184, 191). They would undoubtedly become even larger, did not the compression of the cord cause early death or make operative interference necessary. It seems probable from clinical observation that extradural tumours grow as a rule from above downwards. They may also spread right round the cord and enclose it as if in a tube or case (Orlowski), (see Fig. 187). In the sacral canal they

may often grow to a considerable size, and they have repeatedly been accidentally discovered after death on the filum terminale (Spiller and others).

The majority of these tumours (syphiloma excepted) are of *slow growth*. They compress the adjacent roots, and bearing in upon the cord from in front and behind, more frequently from the side, they gradually compress it until it is reduced to a quarter of its normal size or to the thickness of a lead-pencil, whilst the tumour forms a kind of nest in it and lies embedded in a niche of the cord (Fig. 188). In my own cases the changes in the form and size of the cord were often very slight, and they appeared to disappear immediately on removal of the growth. In other cases there was considerable displacement and contraction of the cord, which was flattened like a ligament. The histological changes are sometimes very slight. More frequently there are processes of inflammation, degeneration, and softening in the neighbourhood of the tumour, and also secondary degenerations. The central parts of the spinal cord often suffer more from the compression than the peripheral. The side opposite the tumour may also be more severely affected than that lying directly beside it (Giese, Auerbach-Brodnitz). It is not unusual for the tumour to destroy the substance of the cord (Fig. 186). In exceptional cases the growth may protrude outwards through the intravertebral foraminæ or after erosion of the vertebral bodies, as in those described by Cladek, Orlowski, Schede, and Schultze. Tumours situated outside the dura frequently exist for a considerable time before they compress the cord, but this is not always the case.

New growths may appear at any level of the spinal cord. It is natural that the thoracic region should be most affected. In six of my own cases, I found tumours in the cervical cord, usually corresponding to the lower part of the cervical enlargement. They are comparatively frequent also in the cauda equina (Fig. 185); fibroma, glioma, sarcoma, fibrosarcoma are found, and in one case there was an extradural lymphangioma of considerable vertical extent.

Symptomatology.—The symptoms are in many cases so slightly marked that the diagnosis, if made at all, can be only a provisional one. In the majority of earlier cases the disease is only recognised post-mortem. The diagnosis of myelitis is frequently made, particularly in tumours arising from the cord itself; sometimes, however, as in Strube's case, even in growths coming from the dura. With the evidence obtained from the celebrated case of Gowers and Horsley, that these tumours are accessible to operative treatment, the interest attached to the clinical symptoms has been so much increased and the symptomatology so far advanced that a definite or probable diagnosis can now be made in the majority of cases.

Root symptoms are usually the first sign of disease, and as only one or two roots are as a rule at first compressed by the tumour, *neuralgic pains* in the area of a certain nerve, especially unilateral or bilateral *intercostal neuralgia*, are usually the earliest symptom. These pains are at first slight, increase in time, and are then described as pricking, boring, burning, or stabbing. They are at first paroxysmal, and may even disappear for a considerable time, whilst later there is a dull persistent pain with exacerbations from time to time. There is very often an increase of the pain on coughing, sneezing, etc. Recent cases show that such pains may

be entirely absent (Clarke, Bailey, Sibelius, Schultze, Oppenheim). In one of our cases they appeared first shortly before death ; in another, on the contrary, intense pains were present only during the first stage of the illness. Schultze has specially emphasised the fact that pain may be absent, or very slight and transient, and he endeavours to explain this as being due to a paralysis caused by compression of the pain-conducting tracts. The pain usually at first, and for a certain length of time, affects one side and may then gradually pass to the other. It is sometimes at the commencement accompanied by hyperæsthesiæ, but I have very rarely found this so. This *neuralgic stage* may persist for a period of several months, a year, or even several years before fresh symptoms appear. In one of my cases the intercostal neuralgia lasted for two and a half years before it was joined by other slight symptoms. If the tumour is situated on an anterior root of the cervical enlargement or of the lumbo-sacral cord (or the cauda equina), symptoms of motor excitement in the area of a certain nerve may be the first sign : tremor, muscular tension, spasms,[1] may appear, but these are generally absent or are soon masked and suppressed by the development of a *degenerative paralysis* of the corresponding muscles. In one of my cases in which the tumour was localised so exactly that it was directly found on the part laid free by operation, compression of the anterior roots (eighth and ninth thoracic) had caused atrophy of the abdominal muscles, whilst compression of the corresponding posterior root had been revealed first by pain and then by absence of the abdominal reflexes and anæsthesia. I have confirmed this by another case and by two which were cured by operation. From the cases reported hitherto we find that symptoms arising from irritation of the posterior roots are usually the most prominent, even where, from the position of the tumour, anterior and posterior roots are equally endangered. There are exceptions, however. In compression of the anterior roots the symptoms of paresis may exist long before there are any changes of electrical excitability (Bruns and Oppenheim). Œdema and herpes may be signs of compression of the roots, but the latter is much more frequent in vertebral tumours.

During the later stages the first sign of the increasing root compression may be an anæsthesia in the region affected by the neuralgia, or much more rarely, there is as the sign of further growth of the tumour in a vertical direction, an *upward or downward extension of the neuralgic or anæsthetic area*, or finally, there are usually immediate signs of compression of the cord. It should be noted that symptoms of meningeal irritation, viz., pain and rigidity in the back, may also occur. The latter is, however, not usually severe, and the movements of the spinal column as a whole are neither limited, nor do they as a rule definitely increase the existing pains. In some of my cases, at least, the rigidity of the back was very marked. In a few cases there was also a scoliosis, but I attributed this to the fact that the patient sought to maintain an attitude in which his pain was as slight as possible. In a few cases (Böttiger-Krause, Oppenheim, Schultze, Stertz) there were no root symptoms whatever ; this might be specially expected if the growth develops in the inter-radicular regions of the cord.

Compression of the spinal cord at any site above the lumbar region causes *spastic paralysis*, and the *muscular rigidity* and *exaggeration of*

[1] General tonic spasms with opisthotonus, etc., have occasionally been observed.

reflex excitability is more marked in tumours which compress the cord than in any other disease. As one side is as a rule first affected, the paralysis usually commences in the leg of the same side, whilst the exaggeration of the reflexes and tendon phenomena are generally bilateral, although they are most marked on the same side. The *spinal hemiparesis* usually becomes slowly or rapidly transformed into paraparesis or paraplegia, and the muscular rigidity not infrequently increases, gradually or rapidly, to such a degree that it eventually causes a marked flexion contracture in the hip- and knee-joints.

Sensibility may remain unaffected in the lower limbs for a considerable time ; but there is more frequently, even at the stage in which the motor weakness is limited to or more marked in one leg, a hypæsthesia or an anæsthesia of the opposite limb, thus constituting a Brown-Séquard *hemiplegia*, provided always that the tumour compresses the cord above the lumbar region. In several of our cases the thermo-anæsthesia of the opposite leg preceded the development of the homolateral paralysis. Henschen and Lennander have also found this.

The symptoms of a unilateral lesion may long remain clearly defined, but in the nature of things a bilateral extension of the paralysis and sensory affections must follow sooner or later, often in a short time. Even then, however, the Brown-Séquard character is often distinctly marked for a considerable period. In one of our cases where the tumour was in the lumbar region, at the level of the third and fourth roots, the homolateral spastic paralysis was associated with hypotonia of the quadriceps and diminution of the knee jerk, and with bilateral anæsthesia on the foot, but there was thermo-anæsthesia only on the gluteal region of the opposite side.

There may be early impairment of the *functions of the bladder and rectum* ; if paraplegia has developed, corresponding troubles are hardly ever absent. At first there is usually increased, distressing desire to micturate, to which the patient must immediately give attention (imperative micturition) ; subsequently there is incontinence of urine or paradoxical retention. Occasionally, under these conditions, the incontinence of the bowel is more marked than that of the bladder (Oppenheim).

In this stage of complete or partial spinal compression the signs of root irritation, *i.e.* the neuralgic pains, frequently persist ; but pains, which may be severe, are also felt in the lower half of the body, in the limbs which are affected with paralysis and anæsthesia. As has already been mentioned, however, they may be absent or may disappear as the paralysis progresses.

The modifications which the clinical picture undergoes when the new growth has a cervical localisation do not require special description. Compression of the cord in this case generally gives rise, first to spinal hemiplegia, later to paraplegia of all four extremities. The paralysis of the upper limbs may be of spastic or atrophic nature, or may combine these characteristics. The radicular type of the degenerative paralysis is here very clearly marked. The symptoms produced by involvement of the oculo-pupillary centres and tracts and of the phrenic have already been described. Bulbar symptoms have occasionally been among the signs of tumour of the cervical cord. Schlesinger attributes them to an œdema of the medulla oblongata, and Nonne to toxic influences.

In tumours which compress the lumbar cord, the root symptoms

play a prominent part, as the nerve roots are here more thickly grouped, and even a small tumour may press upon a large number of roots. The atrophic paralysis is produced in this way, as well as by compression of the lumbar cord, and the spinal symptoms are essentially those of lumbar myelitis, whilst severe pain in the nerve tract of the lumbo-sacral plexus forms an important differentiating sign. I have found, however, that the cord sometimes suffers under those conditions, much more and earlier than the roots, so that the clinical picture is entirely controlled by the spinal symptoms.

Tumours of the cauda equina at first give rise to intense pains in the region of the sacrum, radiating into the region of the anus, bladder, and perineum, and also into the sciatic nerves. Then come symptoms of paralysis, which in one case may be limited to the bladder, whilst in others there is degenerative paralysis in the region of the sciatic plexus; and when the tumour extends higher up, functional disorders may appear in the region of the upper lumbar nerves (Laquer). In a case which I diagnosed there was an onset with pains in the anus, sacrum, and legs, followed by incontinence of fæces and later retention of urine. I found an anæsthesia in the ano-genital region and over the Achilles tendons, paralysis and loss of the anal reflex, absence of the Achilles jerk, and reaction of degeneration in the levator ani. I diagnosed the existence of a tumour at the level of the third or fourth sacral nerves, and it was found on operation by Sonnenburg exactly at this site.

In another of our cases in which tumour of the cauda appeared to be much more probable than one at the corresponding level of the conus, it was not discovered on operation to be so. See also the reference by Cassirer, *Z. f. N.*, xxxiii.

Trophic disturbances, perforating ulcer for instance, also occur in tumours of this region (Dublay).

Tumours which compress the posterior columns may give rise to *ataxia*; if they are situated in the upper lumbar region there may be an early disappearance of the knee jerks. In one of our cases in which the tumour compressed the cord from behind, the clinical picture had a great resemblance to that of combined system degeneration.

Extradural growths frequently produce bilateral root symptoms, and these are frequently extensive owing to their pronounced tendency to extend in a longitudinal direction (Böttiger).

In cases of *multiple and diffuse tumours*, the symptoms usually point to several foci. Thus the picture is often mainly that of endocranial growths (A. Westphal, Schröder, Raymond-Cestan, Henneberg-Koch), but as a rule the type corresponds to one of cerebro-spinal disease, and may show great resemblance, for instance, to cerebro-spinal syphilis. In a case of multiple tumours of the spinal cord, roots, brain, and its nerves, observed in Erb's clinic, the clinical picture greatly resembled that of disseminated sclerosis, the cranial symptoms, however, corresponding more nearly with those of tumour. There were also, as in a case of Sieveking's, *small cutaneous tumours*, which made the diagnosis possible. In one of our cases the picture corresponded to one of multiple cerebro-spinal root neuritis, and the multiple fibromata of the skin confirmed the diagnosis. The appearance of tumours (sarcoma, neuroma, fibroma, echinococcus, etc.) at other sites is a material support in the diagnosis of tumours of the spinal cord. But it should be remembered, that

metastatic tumours occur much less frequently in the cord and its membranes than in the spinal column. Stertz also, for example, found in a case of carcinoma of the rectum a tumour in the spinal cord of quite another character.

The course of the disease is a chronic one, and it may extend over several years, ten it may be. On the other hand, in benign tumours the course may be run in a comparatively short time—in four months in a case of Schultze's. In one of my cases also, in spite of the mild nature of the intradural growth, the development was rapid ; this is, however, an unusual occurrence. In exceptional cases there may be spontaneous remissions, which may give rise to errors in diagnosis.

As a rule we can distinguish three stages : the first, which is usually of longest duration, is the stage of root symptoms (generally unilateral), the second that of Brown-Séquard paralysis, which sooner or later passes into the third, that of complete compression of the cord or bilateral paralysis.

I have only once (along with Bielschowsky) seen in an extramedullary tumour the stage of spastic paraparesis precede that of the hemiplegia.

The *diagnosis* of tumours of the spinal cord and its adnexa is at present a matter of grave responsibility, which requires a great deal of careful consideration and exceedingly thorough, repeated examinations. The general and special diagnostic signs have already been mentioned under the symptomatology. We shall here discuss only a few special points.

In the differential diagnosis from other diseases of the spinal cord, *spinal syphilis* has to be specially considered. Gummatous tumours may act upon the roots and cord like any other growth ; the clinical picture, however, does not show the steady development and course, but a very rapid onset and an intermittent course with a tendency to sudden exacerbations and remissions. Further, isolated gummata are rare, while multiple foci and diffuse changes are common. The symptoms therefore usually indicate, not a single focus, but multiple lesions at various sites of the spinal cord or cerebro-spinal nervous system. This rule certainly fails in cases of multiple new growths (which generally reveal themselves by their simultaneous extension over the areas of the body which can be directly examined—general neurofibromatosis, etc.). The history of the case, the physical examination and the serum reaction, are all of course important aids to the diagnosis of spinal syphilis. Finally, in every case where there is a possibility of a specific process, antisyphilitic treatment should be first instituted and the diagnosis made from the results obtained.

The diagnosis from *disseminated sclerosis* is a matter of less difficulty. Doubts may arise, however, when in tumour the root symptoms, especially the intense localised pains, are for a long time absent. In a case of this kind I gave the probable diagnosis of disseminated sclerosis on the first examination, but the further course showed that the case was one of multiple tumours.

In another case where I had on the first consultation given a diagnosis of disseminated sclerosis, the subsequent course led me to suspect an extramedullary tumour, and this was found on operation at the expected site.

Careful general examination may furnish valuable indications for the diagnosis of multiple tumours and their distinction from disseminated sclerosis in the shape of neuromata, angiomata, etc., in other parts of the body, but it also exceptionally happens that the tumour in the spinal cord is not identical in character with those found in other parts of the body (Stertz).

It should be remembered that the diagnosis of disseminated sclerosis from certain (gliomatous) forms of tumour may be very difficult, even on histological investigation (Stertz, Oppenheim).

According to a case of Rindfleisch and a communication by Dufour, cytodiagnosis may lead to the recognition of meningeal sarcomatosis from the presence of tumour cells. Schönborn and Grund have given valuable data as to the diagnostic value of lumbar puncture in tumours of the spinal meninges.

It is specially shown by Schultze's communications that extramedullary tumour may be mistaken for spinal pachymeningitis. Joachim [1] describes a case in which an extramedullary growth was simulated by a chronic meningomyelitis.

It is of special practical importance to decide whether we are dealing with a *vertebral*, a *meningeal*, or a *medullary* tumour.

Localised deformities of the spinal column point to a spinal tumour, if caries can be excluded; an endovertebral tumour, especially a hydatid cyst, may of course erode, deform, and break through the spinal column. In one remarkable case (Fischer) a growth of the spinal cord had made its way through the vertebra towards the abdominal cavity. This, however, happens so rarely and under such unusual circumstances that the rule above stated retains its diagnostic value. Even before the outward changes in form become visible, X-ray examination may reveal a vertebral tumour, but radiography often fails us (as we see from an observation by Stertz), and on the other hand a tumour situated in the spinal canal may make room for itself by bulging the canal outwards (as Jolly demonstrated in one of my cases by radiography). This of course cannot occur when the growth is malignant. If the observations specially reported by Leyden and Grunmach, that various diseases of the cord substance itself may give rise to trophic changes of the vertebral bones (halisteresis, etc.), are confirmed, radiography would become of even less value in diagnosis. In any case the appearances must be interpreted with the greatest care.

If the localised deformity of the spinal column corresponds to tumour (and caries of the vertebræ), former experience would have obliged us in doubtful cases to attribute diffuse scoliosis and kyphoscoliosis to medullary new growths, *i.e.* to gliosis and syringomyelia. I have found, however, that even extramedullary tumours of the spinal canal may be combined with scoliosis. Considerable deformities of this character are of course more frequent in gliosis, perhaps also in neurofibromatosis (Haushalter).

Difficulty in moving the trunk and painfulness of this movement occurs specially in tumours of the spinal column, and is as a rule very marked in these. There is also considerable local sensitiveness of the vertebral column to pressure and percussion. This may be entirely absent, so far as our experience goes, in meningeal tumours, even in advanced

[1] *A. f. kl. M.*, Bd., lxxxvi.

stages, but it is very rarely missing in vertebral growths. It may also be very marked in extradural growths of the spinal canal.

Further, extension to the cord and roots follows rapidly in vertebral tumours. Pain, limited to one or several neighbouring root areas, may for a considerable time be the only symptom ; but the further progress of the disease ceases to be slow, and the symptoms of a diffuse involvement of the spinal cord, usually from the first bilateral, appear either all at once or in rapid succession. It must be remembered, finally, that vertebral tumours are usually malignant and metastatic, and that on the other hand the metastases of malignant growths hardly ever directly attack the spinal cord or meninges, but first affect the vertebræ themselves. Advanced age of the patient, cachexia, and especially evidence of a malignant tumour at other sites are signs that the spinal symptoms are due to a vertebral tumour. A case of Senator's shows, however, that even these deductions should be made with some reserve.

What we have said as regards the differential diagnosis of vertebral tumours applies practically also to *caries*. With regard to the details, the chapter on this subject should be consulted. The evidence of the tubercular diathesis, the vertebral symptoms (to which may be eventually added those of a burrowing abscess), the effect upon the general condition, which is often apparent, the fever sometimes present, the bilateral appearance of symptoms of compression, which is at least the rule—well-marked Brown-Séquard symptoms being present in my experience in less than 5 per cent. of cases of caries—are factors which afford an almost certain basis for differential diagnosis. The diagnostic value of tuberculin injection in such cases is doubtful ; we should therefore advise that it should not be employed, although the recent experiments and methods of Pirquet and others may be taken into consideration. Lumbar puncture may assist the diagnosis, but it frequently fails to do so.

Some time after I had found an accumulation of cerebro-spinal fluid in the spinal canal, due to other conditions (see p. 279), and giving rise to symptoms of compression, Krause and I found this serous spinal meningitis in a few cases in which we had diagnosed a new growth. We debated this question in our Report to the Naturforscherversammlung d. J., 1906, and in the discussion which followed. I doubt all the same whether this is an independent disease, and cannot exclude the possibility that there was at least in some of these cases a combination of intramedullary lesion with the serous meningitis.

On the other hand I suspect from observations of my own that transient processes of this kind, *i.e.* local serous exudations, may occur in the spinal meninges, and may be the cause of temporary symptoms of irritation in the area of certain nerve roots.

Böttiger speaks of a pseudo-spinal tumour, as he saw in a case corresponding to tumour of this region recovery which was spontaneous or due to treatment with arsenic. Such cases do not in the meantime justify the conclusions drawn from them.

The greatest difficulty is that of determining whether a new growth arises in the cord or in its immediate neighbourhood, *i.e.* the *meninges and roots*, or in the vertebral column itself. This can often not be decided with absolute certainty, as we learn from Nonne's statistics (Stertz) as well as my own cases.[1] The important fact should be remembered, that the growths developing within the canal, if we except gliosis, mostly arise from the meninges. This specially applies to the benign tumours, which grow slowly.

[1] On this question, which has already been thoroughly discussed in the previous edition of this work, consult Malaisé, *A. f. kl. Med.*, 1904, Stertz (*loc. cit.*), and my papers mentioned above.

In tumours of the cord substance itself, the root symptoms are as a rule by no means prominent, and the clinical picture is more like that of a chronic transverse myelitis or a gliosis. Root symptoms may indeed be entirely absent, even in extramedullary tumour. Growths arising in the cord frequently have a course like that of ascending, subacute—in exceptional cases even of acute (Nonne, Stertz, Spillmann-Hoche)—or chronic myelitis, corresponding to their rapid or gradual extension from the lower segments of the spinal cord to the upper (Saenger, Wyss, Orlowski, and others). The course may be very protracted, extending over many years. In one of my cases the disease had existed for eight years, when death took place after an explorative laminectomy. Stertz describes a case of intramedullary glioma with a duration of over ten years.

Remissions and fluctuations in the course occur specially in intramedullary tumours, but are not altogether wanting in extramedullary growths.

From a diagnostic point of view it should be specially noted that in extramedullary growths the symptoms of irritation or paralysis do not, as a rule, extend upwards, and that the upper limit of the level-symptoms thus remains unchanged or shows only a comparatively slight upward extension. (It may be thus expressed: the symptoms seem to show that the tumour becomes thicker, but not longer.) I have, however, seen exceptions to this rule.

Tubercle of the spinal cord arises usually in the central grey matter, and its clinical features are therefore very similar to those of syringomyelia, but are characterised by their rapid progress (H. Schlesinger).

Oberndörfer (*M. m. W.*, 1904) has lately discussed its differential diagnosis.

Gliosis has specially to be diagnosed from extramedullary tumour. Confusion between the two has indeed led to unfortunate operations (Fürbringer-Hahn). The following points should be specially considered in this respect:—

1. Root symptoms are absent in gliosis. This is specially the rule as regards the posterior roots, whilst the atrophic paralysis does not in itself show whether it arises from an affection of the anterior horn or of the anterior roots. The diffuse extension in gliosis usually points to an intramedullary site of the disease.

2. In gliosis the symptoms of irritation, *i.e.* the pain in the back and the radiating pain, are not usually prominent. This is not an absolute rule, however. A combination of gliosis with pachymeningitis may make the disease a painful one. On the other hand, pain is by no means rare in extramedullary tumour. Symptoms of motor irritation, especially spontaneous contractions in the legs, such as arise when a tumour compresses the cord, are not likely to be met with in gliosis, as in it the spastic phenomena are absent or but slightly developed (see next chapter with regard to a rare type accompanied by severe spasms).

3. Vasomotor and trophic symptoms in the skin, the soft parts, the bones and joints, decide in favour of syringomyelia. Œdema may, of course, occasionally appear in extramedullary tumours.

4. The distribution and character of the anæsthesia in gliosis show that the lesion is in the *posterior cornu*; in extramedullary tumours they indicate *interruption of conduction* in the cord. In the first case there is a "*segmental*" *anæsthesia to temperature and pain on the same side*; in the

latter, besides the root anæsthesia there is anæsthesia of the lower half of the *opposite side* of the body, corresponding to Brown-Séquard's paralysis, the anæsthesia to temperature and pain being of course most marked. This description applies naturally to the main types only, and does not include every particular case and every possibility.

5. The symptoms in gliosis point to an extension of the process mainly in the vertical direction, in extramedullary growths, chiefly to a transverse extension. If therefore the paralysis remains unusually long or permanently in the Brown-Séquard stage, whilst the symptomatology reveals a gradual extension in the vertical direction, the evidence is decisively for syringomyelia, as is shown in a case recorded by Stertz. But there are exceptions to this also.

6. The course in gliosis is usually more insidious than in extramedullary tumour. If, after the medullary symptoms have developed, the process goes on very slowly and intermittently, the evidence is in favour of gliosis.

7. The more marked degrees of scoliosis and kyphoscoliosis are found specially in gliosis.

Some of the above criteria may become of reduced value because a gliosis is sometimes associated with a true glioma, and the symptoms are modified accordingly.

The knowledge that the tumour is extramedullary does not satisfy the requirements of the diagnosis. We have still to determine its precise *site on the cord*, to establish the *level-diagnosis*. For this we must refer to what has been said in the chapter on Localisation in the Spinal Cord, p. 125. For this purpose a very exact and frequently repeated examination is required, and the results should be immediately registered on one of the well-known schemata.

The most important points to be considered are the following :—

1. *Site and Extent of the Pain.*—The localisation of the pain in the back is of a certain importance, but that of the root pain is still greater. Pains and paræsthesiæ may of course be caused by irritation of the long intraspinal sensory tracts—and therefore girdle pains and girdle sensations at the abdomen may be produced by tumour of the cervical cord (Henschen-Lennander), but these are neither so constant and persistent nor so intense as the root pains. It follows that for purposes of localisation only the best localised and most constant pains should be taken into consideration.

2. The *root paræsthesiæ* and the *anæsthesiæ* as a rule extend through the same root areas as the pains. For purposes of localisation great caution is required with regard to the latter. The theory of the descending course of the root fibres, of the multi-radicular innervation of cutaneous areas, which causes total anæsthesia in a certain area of innervation to appear only after extirpation of two or more neighbouring posterior roots —the existence, according to Sherrington's experiments, of individual differences in the relation of the roots to certain cutaneous areas— all these points have to be taken into consideration. If, for instance, the anæsthesia extends into the area of the seventh dorsal nerve, then the sixth and probably also the fifth dorsal root must be involved, and the upper margin of the tumour therefore reaches to the fifth dorsal segment.[1]

[1] Böttiger thinks that, even where there is only hypalgesia as the highest root symptom, the tumour should be looked for at a segment higher than would be assumed from the distribution of

It is due to neglect of this and other facts that tumours of the spinal cord are often sought at too low a level and are therefore either not found at all, or only after removal of another vertebral arch. Horsley therefore advises that laminectomy should be practised 8 to 10 cm. above the upper margin of the anæsthetic zone. It is not wise, however, to stop here, and the question ought to be decided after consideration of all the facts in each individual case. In two of our cases the tumour was found somewhat lower than we had anticipated. In one of them there was a circumscribed arachnitis above the tumour, which had accordingly modified the symptoms.

Fugitive pains in the upper root areas should not, however, lead us astray, as disorders of the circulation (œdema), occlusion of the cerebro-spinal fluid, which I found several times above, and Cushing and Stertz below the tumour, and perhaps toxic influences, may affect segments of the spinal cord above the tumour and give rise to corresponding symptoms.

Some writers maintain that local hyperæsthesia above the anæsthetic zone is of special importance in determining the site, but I have hardly ever found this symptom.

3. *Loss of the Root or Spinal Reflexès.*—The reflexes are abolished in the corresponding area, both by compression of the roots and of the cord itself. If the tumour is situated, for example, on the eighth and ninth dorsal roots, the supra-umbilical (epigastric) reflexes are usually absent; if it compresses the tenth and eleventh dorsal roots or the corresponding segment of origin, the infra-umbilical (abdominal) reflexes tend to disappear (see p. 134). Great care is required, however, in judging of this factor, because the abdominal reflex is by no means constantly present even in healthy persons, and it may also be affected by growths situated above its segments.

4. *Degenerative paralysis* as a root symptom, whether it be due to compression of the anterior roots or of their segment of origin. This is a symptom of great importance as regards tumours in the region of the cervical, the lowest dorsal, and the lumbo-sacral cord.

5. Extension of the symptoms of *affection of the long conduction tracts*, due to the lesion of the spinal cord itself, *i.e.* an extension of the spastic paralysis, of the conduction anæsthesia, *e.g.* of the upper margin of the anæsthesia of the opposite side in Brown-Séquard paralysis, etc. etc.

These symptoms are in some cases associated with

6. *Certain External Signs.*—These include tenderness on pressure of one or more vertebræ, dulness of the percussion sound over the vertebra corresponding to the site of the tumour, noted in several of our cases, and finally the conditions revealed by radiography (see above), in the interpretation of which great experience and care are required.

The order of the development of the various root and cord symptoms sometimes indicates whether the tumour is situated in the vicinity of the posterior or of the anterior roots, and whether it compresses the cord from behind, from in front, or from the side. Errors of interpretation may, however, creep in, as the various roots do not suffer to the same extent under similar pressure, and the side of the spinal cord adjacent to the tumour is sometimes not so severely compressed

this sensory disturbance. From my own experience, I must say that this view is not always correct, and that the tumour is frequently found at the level of the cord from which the symptoms show the highest affected root to originate.

as the opposite side (Giese, Auerbach-Brodnitz. A case of E. Meyer's may possibly be explained in this way). Bilateral root symptoms may, of course, be present from the outset, and the spinal symptoms may be the first evidence of the disease.

Previous to the era of surgical treatment, the *prognosis* was very grave. Recovery, either spontaneous or as the result of medical treatment, cannot be looked for, except in the case of syphiloma. An arrest due to regressive processes (calcification, etc.) is only possible in the case of parasitic growths. Henschen has seen a case which he could only attribute to the regression of a neuroma. I have also known one case in which, after an operation had been contemplated, the condition improved to a considerable extent under the influence of some febrile illness.

It is possible that neuroma of a spinal root may, on account of a temporary swelling, such as sometimes occurs in peripheral neuroma, exert transient pressure upon the cord. I think I have seen one such a case, in which the advisability of operation had already been considered.

A course of this kind is quite unusual. Remissions may indeed occur, due to the benign character of most of these growths, but as a rule the course is steadily progressive and fatal. The prognosis has been considerably more favourable since the introduction of surgical treatment (see below).

Treatment.—If there is any suspicion of syphilis, a thorough anti-syphilitic course should at once be instituted. In other cases we were formerly limited to symptomatic treatment for alleviation of the pain, although Erb had in 1878 raised the question of operation, and tumours growing outwards or those pressing from outside towards the spinal canal had been surgically treated (Secat, Gerster, Abbé, etc.). Then Gowers and Horsley showed that even occult tumours of the spinal cord could be surgically removed, and that complete recovery might be thus induced. The case which they reported was that of a man of 42, who in 1884 became affected with left-sided intercostal pain of great severity, which until 1887 was the only symptom. The diagnosis of intercostal neuralgia seemed moreover to be justified by the fact that for a time the pain was successfully controlled. Then there appeared paralysis of the left leg, followed by paraplegia of both lower extremities and accompanied by exaggeration of reflexes, spastic symptoms, and sphincter paralysis. The diagnosis of tumour of the spinal cord was made, and it was decided to remove it by operation. After the spinal column was opened, an almond-shaped tumour was found at the level of the upper thoracic cord on the left side in the subdural space. With its excision the paralytic symptoms gradually disappeared, and the man was shown to a Medical Society in London as completely cured. He died only a short time ago (according to a verbal communication from Gowers). Cases with similar recoveries have since been described by Lichtheim-Mikulicz, F. Schultze-Schede, Böttiger-Krause, Sachs-Gerster, Laquer - Rehn, Oppenheim - Sonnenburg, Oppenheim-Borchardt (4), Putnam - Warren, Williamson, Eskridge - Freeman, Henschen-Lennander, Hahn, Putnam-Krauss-Park, Abbé, Spiller, Bailey, Muskens, Odiorne-Warren, Wolsey, Auerbach-Brodnitz, Baldwin, Stursberg, Schultze-Bier, H. Köster, and others

Gowers and Horsley are of opinion that intradural tumours are almost always operable, as they are benign tumours of slow growth, and are but

loosely connected with the cord. H. Schlesinger, from the results of his post-mortem investigations, came to a much less favourable conclusion. The surgical experiences collected since then, however, appear to practically confirm the view of Gowers and Horsley (Figs. 189-193).

Statistical accounts of the cases hitherto treated by operation have been given by Sachs-Collins, Starr, Krause, Böttiger, Williamson, Lloyd, Köhlisch (*Inaug. Diss.*, Berlin, 1905), but from different points of view and under different conceptions of what constitutes a spinal-cord tumour and a recovery from it. Williamson, who includes vertebral tumours, has collected twenty-four cases with complete recovery or considerable improvement. Schultze (*Deutsche Klinik*, 1905)

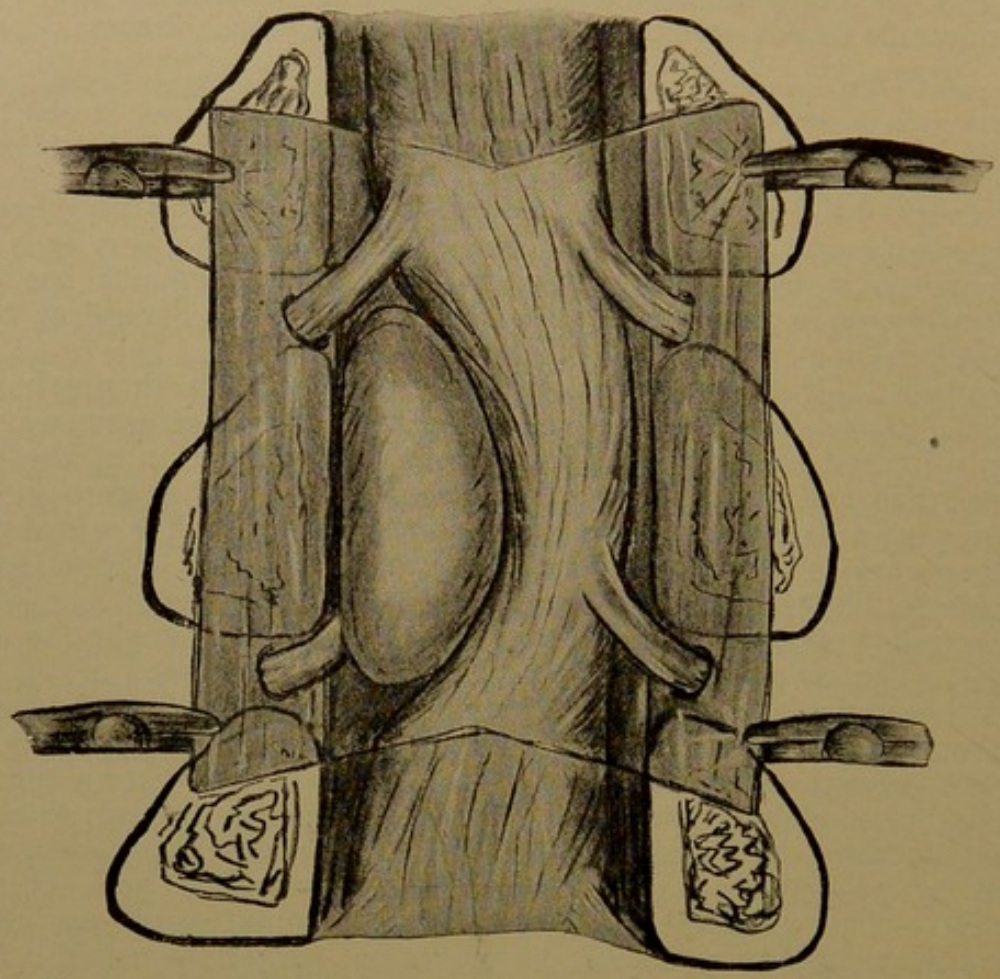

Fig. 188.—Representation of the relative positions of the tumour and the spinal cord in a case of endo-vertebral new-growth, exactly localised by Oppenheim and removed by Sonnenburg. (Somewhat schematic.)

calculated that out of sixty-two cases, there were twenty-four recoveries. In the literature to which I have access, I find sixty-five cases in which there was operation for tumour in the spinal canal. In thirty-three of these, in 50 per cent. therefore, recovery, or improvement which nearly approached it, took place—a very wonderful result. But we must remember that only some of the cases with unsuccessful termination are recorded, and that the report sometimes follows too soon upon the operation. Köhlisch's statistics include vertebral tumours, caries, and abscesses, and thus lose their value. Illustrative cases of complete recovery obtained by surgical treatment are furnished by the detailed clinical histories given by Gowers-Horsley, Oppenheim-Sonnenburg, Oppenheim-Borchardt, Henschen-Lennander, and by F. Schultze. My own experiences include, apart from vertebral tumours, twelve cases of extramedullary growths. In ten the tumour was found exactly at the expected place, once a segment lower, and in another case, complicated with arachnitis, two segments lower. The operation was always successful, and in six cases led to

more or less complete recovery. The lady operated on six years ago is now so well and agile that she is known as an excellent dancer. Cases of intramedullary tumour or "serous meningitis" have sometimes been erroneously diagnosed as extramedullary growths, but in these cases the laminectomy performed has been intentionally of an exploratory nature. F. Schultze has also been very successful, as, according to his recently published statistics (*M. m. W.*, 1907), his experiences agree both in number and result with my own. The latest digest is given by Stursberg in his résumé, *C. f. Gr.*, 1908.

The conditions are most favourable when the tumour is not too large, when it is solid, sharply limited, and benign, arising from the meninges or in the subarachnoidal space, pressing into the arachnoid tissue, displacing the spinal cord to one side, but with a pressure which is neither severe

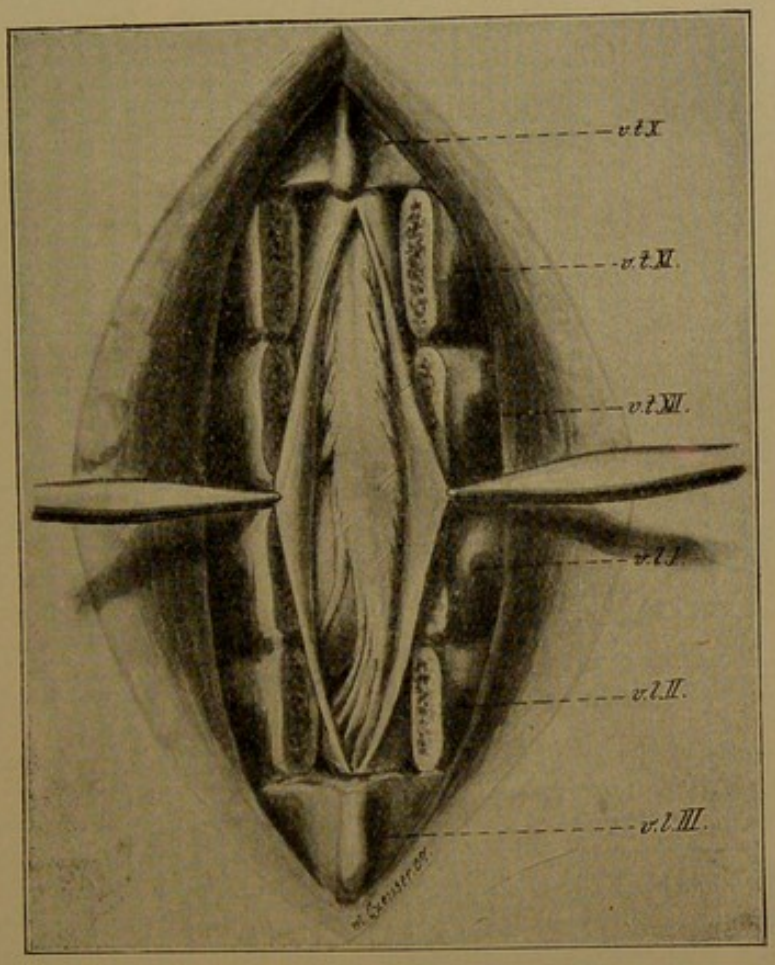

FIG. 189.—Tumour on the anterior surface of the lumbar cord, afterwards successfully removed. (Oppenheim and Borchardt.)

nor prolonged, and when it is easily loosened from the surrounding tissue, as shown for example by Figs. 188 and 189.

See also the illustrations of my own cases in Figs. 190-193.

The cases reported by myself, Böttiger-Krause, and Henschen-Lennander give details as to the progress of the improvement which followed successful operation. In two of these there was partial recovery from the symptoms of irritation and paralysis within the first few days. The gradual restitution of the functions of the spinal cord could then be followed almost from day to day. In other cases the improvement was much slower, and it was interrupted and retarded by complications (cystitis, etc.). It is by no means unusual for an increase of the paralytic symptoms to follow immediately upon a successful operation. This is caused by the manipulation of the cord, by section of the roots, etc. The prognosis is particularly favourable if the spastic paralysis does not develop into complete flaccid paralysis, and if the reflexes are not entirely abolished.

If the compression has lasted for a considerable time, or the pressure been particularly severe, the cord may of course be so much affected that even although the operation for removal of the

tumour has been a complete success, there is recovery merely in the surgical sense of the word, the paralytic symptoms still continuing to exist (cases of Tyther-Williamson, Collins-Lloyd, Oppenheim-Hirschlaff-Borchardt, etc.). In other cases, such as Erb has reported and I myself have

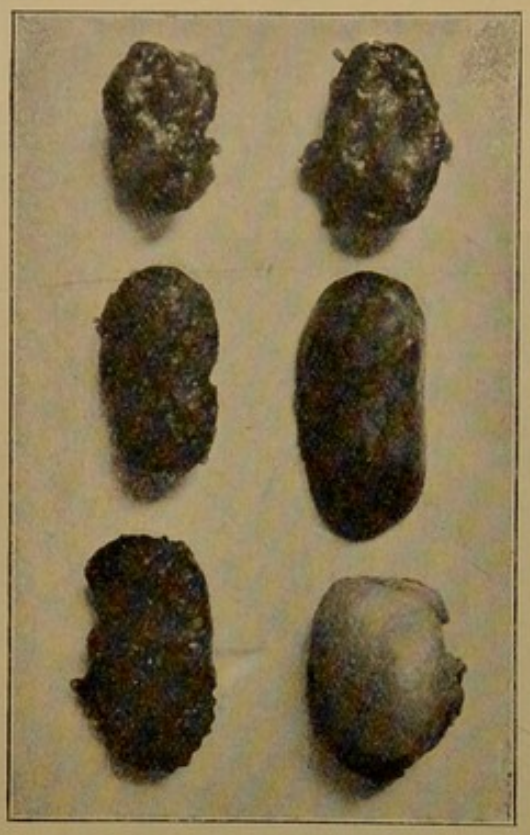

FIG. 190.—Tumours of the spinal meninges removed by operation. (Oppenheim.)

FIG. 191.—Tumour of spinal meninges removed during life. Natural size. (Oppenheim-Krause.)

seen, the spinal cord is injured during the operation, in the chiselling of the bone, the result being that the paralysis increases in intensity and extent after the operation.

Apart from these unusual cases, the unfavourable results of operation are due to the following factors, viz., operation under an erroneous general diagnosis (Schultze-Pfeiffer, Raymond, Joachim, Starr), or under an erroneous level-diagnosis (Starr, Hirtz-Delamare, and others); death within a few days or weeks after operation in cases which have been correctly diagnosed, from shock, exhaustion, meningitis, septic bed-sore, excessive loss of cerebro-spinal fluid, etc. (Schultze-Schede, F. Krause, Starr, Erb, Oppenheim-Sonnenburg, Putnam-Elliot, Walton-Paul, Ward, Sick, Raymond, Oppenheim-Krause, Quante, Quensel, etc.); the intramedullary site of the tumour (Hahn, Edinger, Oppenheim-Borchardt, Goldscheider-Schlesinger, Putnam-Warren; in the latter case, however, there was some improvement); the great size, malignant character, or multiple nature of the tumour (Schede, Remak-Krause, Kron, Mitchell, Clarke, Powell, Muskens, Bregmann); undue delay in undertaking the operation (Starr).

FIG. 192.—Tumour of spinal meninges removed during life. Natural size. (Oppenheim.)

Tumours may be successfully removed from any level of the spinal cord, but the operation is naturally most often performed upon tumours in the thoracic region. Surgical methods have also been adopted in many cases of tumour in the region of the lowest segments of the cord and cauda equina (Laquer-Rehm, Sachs-Gerster, Oppenheim-Sonnenburg, Remak-Krause, Dejerine-Chipault, J. Frenkel, Schultze-Schede, Warrington, Davis, Ferrier-Horsley, Hildebrand, etc.), and there has been recovery in some of these. A successful operation has even been performed in a case of tumour of the highest cervical region (*Putnam-Warren*), but the tumour was apparently a vertebral one, and the ultimate result is not known.

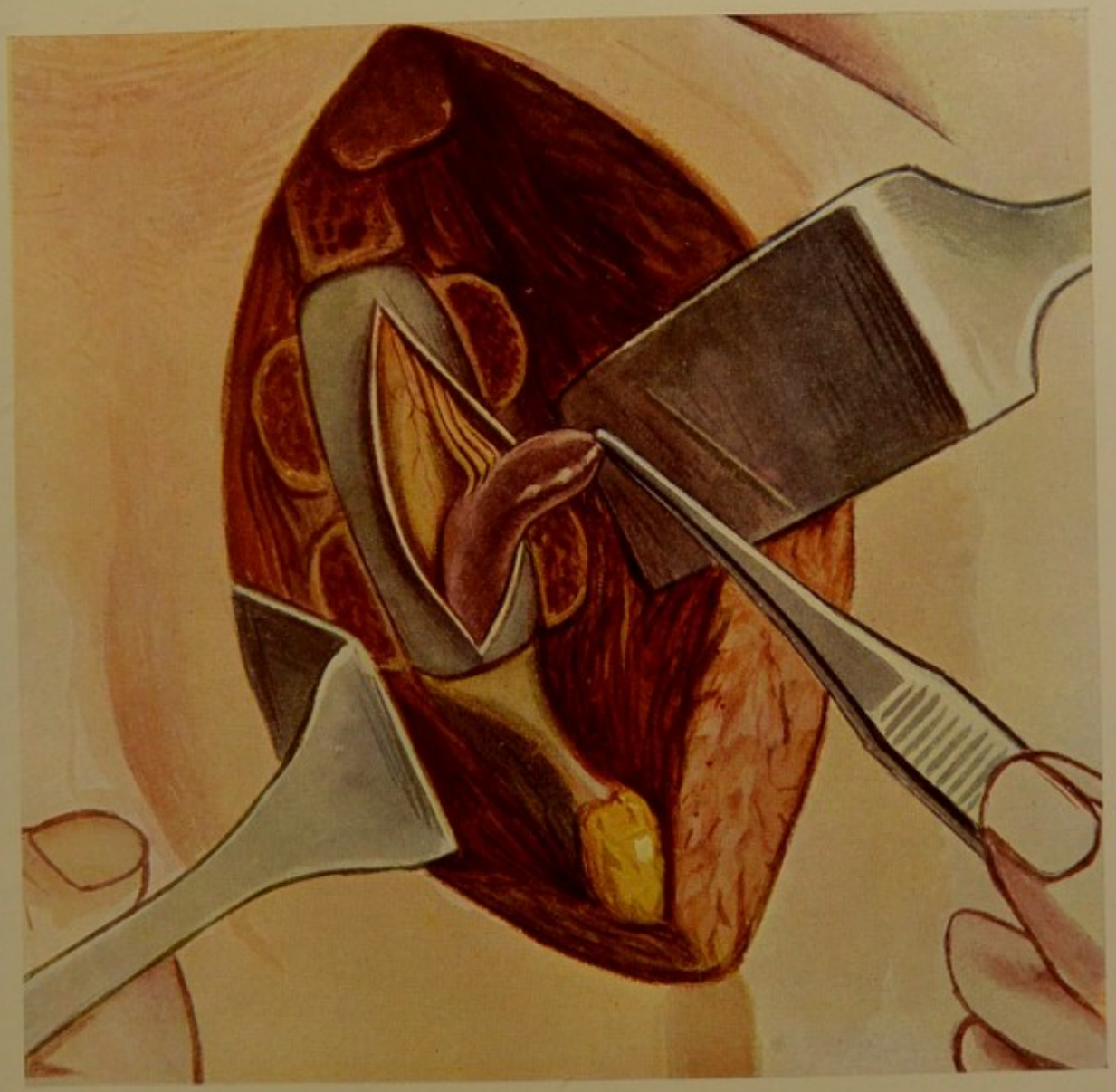

Fig. 193.

Otto Schulze & Co., Edinburgh.

In the cases cured by operation the tumour has most frequently been a fibroma and its varieties (fibrosarcoma, fibromyxoma, etc.), or hydatid, but these cases also include sarcoma, psammoma, endothelioma, lymphangioma, etc. Schmidt records a successful operation for cyst (*Z. f. N.*, xxvi.). Complete recovery takes place chiefly when the tumour is small and fibromatous in character, but in Abbé's case an endothelioma of great longitudinal extent was successfully treated, and in another favourable case operated upon by Auerbach-Brodnitz, the fibrosarcoma extended through the whole cervical cord and made it necessary to remove the third to the seventh vertebræ.

It can thus no longer be doubted that extramedullary growths developing within the spinal canal, if not amenable to specific treatment, give the indication for radical surgical treatment. This is justified only, however, if there are sufficient data for the level-diagnosis, and if this can be made with at least some certainty. Multiple tumours and those arising from the spinal cord itself are not accessible to treatment. The difficulties of differential diagnosis may justify explorative laminectomy (Oppenheim). This step is the less to be feared as Putnam-Warren have found that it has a favourable effect even when the tumour is an intramedullary one, and we (Oppenheim-Borchardt) have at least found no bad effect. Metastatic tumours are better left alone. It is of course most desirable that operation should take place as early as possible, but even a duration of several years does not exclude success if the paraplegia has not been of too long standing. The chances are best when the disease has not yet progressed beyond the second stage—that of Brown-Séquard paralysis.

This is not the place to describe the methods of the operation itself. I would merely make a few remarks on the ground of what I myself have seen. I do not regard osteoplastic methods as advisable, and would recommend that the portions of the vertebræ which have been chiselled out should not be replaced. Krause and Borchardt also follow this plan, and I would agree with them in advocating that the operation be completed at one time. Great care and patience are necessary in the operation of chiselling, and it may occupy a considerable time, as the bones are often unexpectedly firm and thick. I have, however, had an opportunity of seeing, along with Horsley, Krause, and Borchardt, how very complete the technique has been made in this respect. Gentle and careful manipulation is essential to preserve the spinal cord from severe and irremediable injury. When the posterior segment of the vertebra has been removed, the dura mater bulges up, and it is usually evident at the first glance that it is stretched from severe pressure. The new growth is often apparent to the eye, and can be easily felt by the finger. After the dura has been opened up, the tumour can be distinctly seen and felt, if it has a posterior or posterolateral position, but it can, as a rule, be seen in its whole circumference only after laminectomy of several vertebræ. In some of our cases two were sufficient. The spinal cord is generally pushed to one side, sometimes to such an extent that it can only be recognised lying deeply after the tumour has been removed. In the majority of my cases the tumour stood out very distinctly from the paler spinal cord by the blue-red colouring of its capsule.

There may be a gush of cerebro-spinal fluid, either immediately or after the removal of the tumour. Some surgeons (Sick and others) advise that an excessive flow of the fluid should be prevented by tying a ligament round the dura before it is opened, but cases of stabbing injury have repeatedly shown (Giss, Demoulin, Mathieu) that a severe and prolonged escape of the fluid does not necessarily endanger life. None of the surgeons with whom I have been associated in treatment have adopted Sick's method. A rise of temperature may be caused by obstruction of the fluid and serous meningitis (Oppenheim, Auerbach-Brodnitz).

Treatment by extension can hardly be altogether avoided, in view of the difficulty of diagnosis. I would point out, however, that my own experience shows that it may have a very unfavourable effect upon the course of intravertebral growths, as it is usually followed by a rapid increase in the symptoms of compression.

It may be difficult to locate bullets in the spinal canal, as they are often situated at a point other than that of the primary injury of the cord, and they may sink downwards, passing through a considerable part of the cord (Oppenheim, Loison, Raymond-Rose). In such cases radiography may prove to be a very valuable and trustworthy aid. It has shown the position of the shot in many cases, such as those described by Konrád, Sailer, Langdon-Wolfstein, Loison, Demoulin, Visdin, Eskridge-Rogers, Wilde, Engelmann, and others, and has enabled it to be successfully removed in some of them. Engelmann (*M. m. W.*, 1904), Raymond-Rose (*R. n.*, 1906), and Faure have reported results of this kind. The bullet is of course responsible not only for the compression and the lesion which it directly causes, but also sometimes for other secondary conditions, such as fracture of the vertebræ, hæmorrhage of the cord, myelitis, abscess, etc., as is shown by an interesting case reported by Federmann (*D. m. W.*, 1905), and one which I treated along with Bergmann. See also Rosenstein, "Revolverschussverletzungen der Wirbelsäule," etc., *Inaug. Diss.*, Berlin, 1906.

Spinal Gliosis and Syringomyelia[1]

These two pathological conditions will be discussed together, as they are usually combined and cannot be distinguished as regards their symptoms.

By spinal gliosis we mean a process of new growth which takes place in the centre of the spinal cord, in the grey matter, and which does not usually lead to a material increase in the size of the organ. Cavities (syringomyelia) are produced by the disintegration of this new growth, especially in its central parts, and these may appear throughout the whole extent of the spinal cord.

The form of cavity-formation in the spinal cord which, although giving rise to no important symptoms, was recognised long ago (Ollivier, Lancereaux), was congenital dilatation of the central canal, a condition analogous to hydromyelia-hydrocephalus. Some writers (Hallopeau, Joffroy) subsequently thought that these cavities had their origin in a central myelitis of the grey matter. Still later it was recognised that disintegration of tumours might be another cause of cavity formation (Simon, Westphal). This process was at first interesting merely from the anatomo-pathological point of view, and we therefore find syringomyelia discussed in the earlier text-books under the heading "Rara et Curiosa." Careful clinical investigation of this disease began with the cases of Kahler, Schultze, and others, and it has advanced so greatly in a short time that we no longer have any difficulty in diagnosing the disease during life. Recently Hoffmann and H. Schlesinger in particular have advanced our knowledge of the subject.

Pathological Anatomy.—To all outward appearance the spinal cord is either unaltered, or merely swollen in parts (very frequently in the region of the cervical enlargement). We can feel the fluctuation in this region and can often tell by palpation that the organ has been transformed into a *tube*. On section of the cord the cavity, which varies greatly in size and may be large enough to admit the tip of the little finger, usually at once meets the eye. As a rule it is small and resembles a central canal which is more or less dilated, especially posteriorly. In many cases the obvious change is not so much the cavity formation, as the tumour, round, oval, or very irregular in form, which lies in the centre of the cord or in some part of its grey matter (Figs. 194-197). This tumour has grown mainly in the longitudinal direction, and it may

[1] The best and most exhaustive monograph is that by H. Schlesinger, "Die Syringomyelie," 2nd ed., Vienna, 1902. See the literature quoted in it.

extend through the cervical, or even the cervical and thoracic portions of the cord, and upwards into the medulla oblongata.

The medulla oblongata may be involved in various ways. The tumour-process and the formation of cavities (syringobulbia) may both extend into it. Fissure-like cavities are specially common in the medulla oblongata. These fissures frequently run in the direction of the spinal trigeminal root, and they may often involve the nuclei of the vagus-accessory, the solitary bundle, or the lemniscus (Schlesinger, A. Westphal, Philippe-Oberthür, Maixner, K. Wilson). The formation of fissures does not usually extend beyond the facial nucleus (Schlesinger). In a remarkable case described by Spiller (*Brit. Med. Journ.*, 1906), the process had passed beyond the brain stem into the cerebrum.

As a rule there is a fissure or cavity in at least one segment of the spinal cord. The new growth may be limited to the grey matter of one side, or even to one posterior horn, which may for a great part of its course be transformed into tumour tissue.

Microscopical examination shows the tumour to consist of glial cells and fibres, which take part in very varying proportions in its structure, and may undergo many transformations. The spaces of the fissures

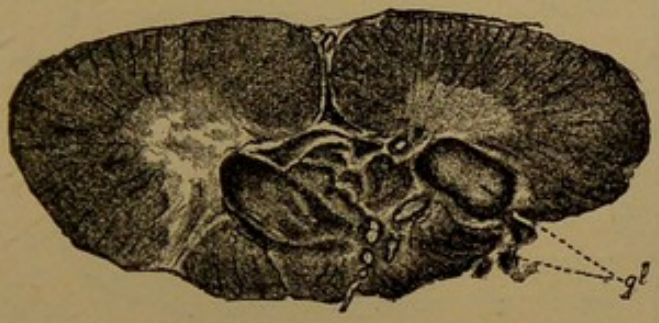

FIG. 194—Spinal gliosis. (From a section stained with carmin by Westphal in Oppenheim's collection.)

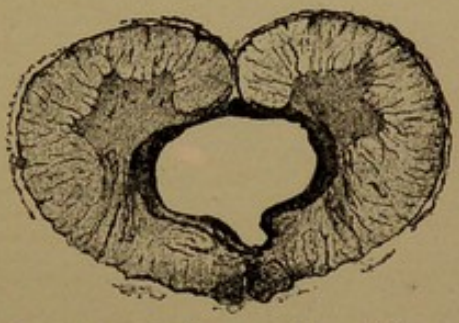

FIG. 195.—Syringomyelia. (From a section by Westphal in Oppenheim's collection.)

and cavities are lined by a firm, compact membrane, in which a layer of epithelium can sometimes be recognised. The tumour arises from the central parts of the spinal cord, from the posterior commissure, the posterior horns, etc., and extends beyond the grey matter and towards the posterior columns. The cavity exceedingly often follows the course of the posterior median septum, and separates the posterior columns from each other, but it does not reach the pia mater.

Thomas and Hauser emphasise the *vascular* origin of the disease (*Nouv. Icon.*, 1904).

The formation of neuroma has also been found in syringomyelia (Raymond, Schlesinger, Bischofwerder, Saxer, Hauser, etc.), but it is not improbable that these are mostly the formations which have been described on p. 323 (Hellich). Fickler regards the symptom as a process of regeneration (*Z. f. N.*, xxix). A combination of general neuro-fibromatosis with syringomylia does, however, occur.

The foundation of the process is usually some *congenital anomaly of development*, which affects either the central canal itself—by arresting it at a fœtal stage of development, during which it sends out a posterior process, by constricting this diverticulum, etc. (Leyden [1])—or nests of glia cells are left behind from the fœtal period, in the neighbourhood of the central canal, in its posterior line of closure, or in the region of

[1] Various anomalies of development in the central canal of the spinal cord of the infant and in its neighbourhood have been recently described (Zappert, Rolly, Ivanoff, Utchida).

the posterior septum. These nests tend either spontaneously or under the influence of some irritant (*e.g. trauma*) to proliferate, and by their increase to form these longitudinal tumours (Hoffmann). If this process has originated from the cells around the central canal, the latter will be found in the centre of the tumour. A second cavity may also be present, due to disintegration of the growth or to a previously existing doubling of the central canal. The cavity may or may not be lined with epithelium, according as it is of primary or of secondary origin.

Cavities may be produced by processes of many other kinds. Circumscribed affections of the meninges, which cause the membranes to adhere to each other and to the spinal cord, seem specially to have this effect. Traumatic necrosis and softening, as well as venous congestion in the spinal cord, may lead to the development of cavities within it. They may occur in caries (Thomas-Hauser, Alquier-Lhermitte, etc.). It is doubtful whether we can from this point of view classify them into various forms (Marinesco, Philippe).

Changes in the peripheral nerves have been found in a few cases

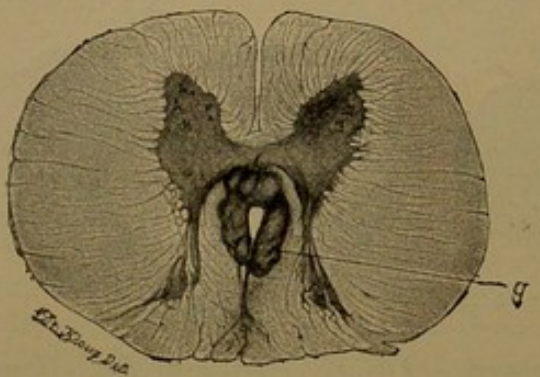

Fig. 196.—Gliosis and syringomyelia. (From a section stained with nigrosin.)

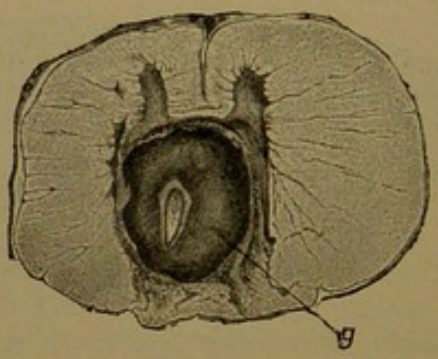

Fig. 197.—Spinal gliosis with commencing syringomyelia.

(Oppenheim and others), but these do not seem to be of any practical clinical importance.

Causes.—It has been frequently observed that the disease developed after a trauma—a fall on, or a blow against the back. It is conceivable that when there is a congenital predisposition (the anomalies of development mentioned above) the trauma gives the impulse to the proliferation of the cells and the formation of the tumour. Experimental investigations (Schmaus, Fickler) point to this mode of production. In any case we must maintain the possibility of a traumatic origin, and we must remember that injuries are specially calculated to bring such a latent disease to full development. According to Minor, central hæmatomyelia due to trauma is not infrequently the starting-point of the process. Hæmorrhages of the spinal cord from injuries during birth may possibly also have this effect (Schultze). Kienböck has, from a critical review of all the material, expressed his disbelief in the traumatic-hæmorrhagic origin of true gliosis and syringomyelia, whilst A. Westphal (and also Kölpin and Steinhausen) have with good reason supported it. My experience also is in favour of a traumatic etiology in gliosis. The theory has been put forward that, given the existing disposition, a trauma or a whitlow affecting an extremity, *e.g.* the hand, may by means of an ascending neuritis reach the spinal cord and so engender gliosis

(Guillain), but this view has little to support it. Sicard has recently contradicted it and expressed the view that such a traumatic neuritis may be capable of rousing the disease from a latent state. In one case a splinter of bone driven into the spinal cord is said to have produced syringomyelia. The formation of cavities is frequently found in syphilitics and in combination with other affections of the spinal cord.

Symptomatology.—In typical cases in which the gliosis has involved mainly the cervical enlargement of the spinal cord, we find the following triad of symptoms: 1. *Progressive muscular atrophy* in the upper extremities (including the shoulder girdle); 2. *partial sensory paralysis* in the upper extremities, the cervical and trunk region; 3. *vasomotor symptoms* and *trophic changes* in the skin, subcutaneous tissue, bones, and joints.

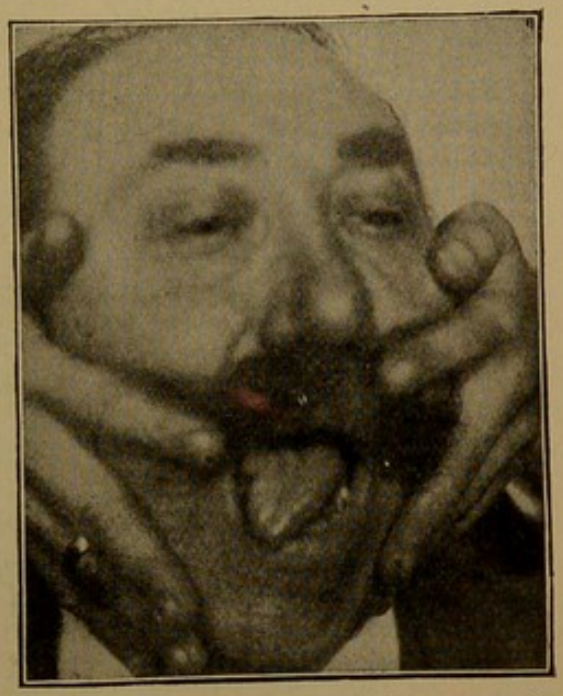

Fig. 198.—Hemiatrophy of the tongue in syringomyelia. (Oppenheim.)

Round these symptoms, which show the nature of the disease, a number of others, less characteristic, may be grouped.

The muscular atrophy usually begins in the hands, less frequently in another segment of the upper limbs and muscles of the shoulder. The small hand muscles are the first to become atrophied, so that the interosseous spaces become depressed, the balls of the thumbs and little fingers atrophy, and the *claw-hand position* develops. All the symptoms remind one of the spinal form of progressive muscular atrophy. Indeed gliosis was formerly usually mistaken for this condition. The atrophied muscles show as a rule fibrillary tremors. Electrical examination reveals reaction of degeneration—generally, however, only in a few muscles or parts of muscles, and in not a few cases simple quantitative diminution of excitability. In a few particularly insidious cases I have found no marked change of electrical excitability, although there was distinct atrophy.

The atrophy is not usually symmetrical in its development, one hand being chiefly or solely affected. The muscular function is altered correspondingly. As the atrophic paralysis usually affects mainly the region of the ulnar and median nerves, or the eighth cervical and first dorsal roots, the hand may, from the preponderance of the muscles innervated by the radialis (sixth and seventh cervical), assume the position of "preacher's hand."

This atrophic paralysis, which progresses very slowly, is combined with a form of sensory disorder peculiar in its character and manner of extension. Whilst touch and pressure are as a rule well perceived, and whilst the sense of position and attitude of the limbs is unaffected, *the sensibility to pain* (for the prick of a pin, the faradic brush, etc.) is more or less completely abolished and the *sensibility to heat and cold* is also diminished or absent.

The sense of heat may alone be absent, that of cold being conserved, or *vice versa*. It may also happen that extreme temperatures alone will be mistaken, whilst moderate heat and moderate cold are correctly perceived (Dejerine-Tuilaut). This differentiation may, of course, be quite uncertain, even in normal conditions, when the person examined is inattentive or unintelligent.

This partial sensory paralysis is not confined to the area of the distribution of a single nerve, but extends over the whole arm, or the arm and segments of the trunk, over both arms, trunk, and neck, and sometimes over half the body. The distribution corresponds to the area of innervation of the posterior spinal roots, or of the single segments of the spinal cord (Lähr, Hahn ; see pp. 126 *et seq.*). In the lower limbs it may correspond to the Brown-Séquard type. The sensory disturbances may also extend to the trigeminal region (see below).

Brissaud specially favours the view of a segmentary ("metameric") distribution, whilst Dejerine, with whom Huet-Guillain, Hauser, Lortat-Jacob, Veraguth, Lewandowsky-Catola (*Med. Klinik*, 1906), and others agree, believes in the root distribution of the anæsthesia. Lewandowsky and Catola distinguish between a radicular and a funicular type of anæsthesia, the latter being due to partial lesion of the sensory tracts in the white matter.

Paræsthesia are usually present, especially a feeling of cold, heat, or a mixed sensation, "a cold burning." It may extend to the mucous membranes. Pain is a not unusual symptom. French writers (such as Raymond-Lhermitte) speak of a type of the disease which is characterised by great pain. The patient has sometimes no idea of the diminution in his sensibility.

There are very numerous *trophic* and *vasomotor* symptoms. Vesicles frequently form on the skin (hands), leaving behind cracks and badly healing ulcers. The wounds and scars, so often found on the hands of these patients, are partly due to the fact that they are insensitive to painful and thermal stimuli, and are easily burnt without being aware of it. The hand, or the hand and forearm, are often of a livid, or simply a red colour, and œdema of these parts is sometimes noted (Remak, Schlesinger, Gnesda). Urticaria facticia, pemphigus, and many other exanthemata occur. Phlegmonous processes, whitlows, keloids, bone necrosis, mutilation of the phalanges (Fig. 199), thickening of the terminal phalanges, ankylosis of finger-joints, are all occasionally found. I have found a Dupuytren's contracture in two cases of gliosis. Neutra and others have also observed this. A condition of the hands resembling acromegaly (Schlesinger, Lunz, Chauffard, Oppenheim, Raymond-Guillain), and a myxœdematous condition of the skin (Sainton-Ferrand) have been described. A combination of gliosis with acromegaly has also been noted. Marinesco has described a peculiar condition of the hand as "main succulente," and he regards this symptom as pathognomonic in opposition to Dejerine, who thinks the hanging position of the arm is the cause. Its main characteristic is diffuse swelling, especially on the back of the hand, by which the deeper parts are completely hidden ; this is due, according to Marinesco, to a hyperplasia of the subcutaneous connective tissue, but vasomotor disorders also play a part in it. The hand is diminished in size from the muscular atrophy of the hypothenar eminence ; it feels cold and dry, etc.

There may be abnormal fragility of the bones, so that *spontaneous fracture* may occur, usually without pain, in the bones of the forearm,

and sometimes in the humerus, and this may especially be one of the first signs of the disease (Schultze). Kofend reports spontaneous fracture of the heads of both humeri. Trophic affections in the joints are more common, especially those allied to the *arthropathies* of tabes (Bernhardt, Klemm, Graf, Sokoloff, Londe, Gnesda, H. Schlesinger, Brissaud-Brouardel, Deycke-Pasche, etc.[1]), but the joints of the upper extremities are almost always affected. The arthropathies may lead to spontaneous dislocations, as I have seen at the wrist-joint. A recurrent dislocation of the shoulder has been repeatedly observed (Schrader).[2] Suppuration occasionally takes place in the affected joint. A more exact idea of the changes in the bones and joints may be obtained during life by means of radiography, (Kienböck, Nalbandoff, Hahn, Hudovernig, etc.; see Fürnrohr, *l.c.*). Tedesco reports a chronic diffuse bone-atrophy. We cannot definitely say how the scolioses or kyphoscolioses of the vertebral column,

Fig. 199.—Mutilation of the fingers in spinal gliosis. (Oppenheim.)

which are present in the majority of cases, are produced. The investigations of Nalbandoff point to a trophic origin, which is accepted by most writers. In a few cases, as in one which I have described, they have existed along with other malformations (Fig. 200) from earliest childhood, and must be regarded as congenital defects of development. Peculiar formations are also occasionally found in the thorax (boat-shaped protrusion of the anterior parts of the thorax, thorax en bâteau (*Marie*)).

Among the *anomalies of secretion*, we should specially mention *unilateral hyperidrosis*, which is complained of in many cases.

The relation of renal calculi, which has been noted in a few cases (Schlesinger), to this disease is still doubtful. It is more frequently found in traumatic affections of the spinal cord (Bramann-Müller, Wagner-Stolper), and is attributed to a pyelitis secondary to a cystitis.

[1] See also Borchardt, "Die Knochen-und Gelenkerkrankungen bei Syringomyelie," *Z. f. Chir.*, Bd. lxxii.

[2] Zesas (*Z. f. Chir.*, Bd. lxxx.) has pointed out the frequency of spontaneous dislocations of the shoulder-joint in this condition.

Changes in the pupils and palpebral fissures must be included among the symptoms of typical spinal gliosis. The pupils and palpebral fissures are frequently contracted on one or on both sides. The pupils retain their reaction to light, but they dilate incompletely in the dark. If one side only is affected, the pupil is as a rule contracted on the side in which the other symptoms are most marked.

Unusual symptoms and *atypical forms* of spinal gliosis.—As the medulla oblongata becomes involved during the later course of the disease, *bulbar symptoms* may be present in addition to those already described. The most common of these is anæsthesia in the region of the fifth nerve, caused by the extension of the process to its spinal root. It has been

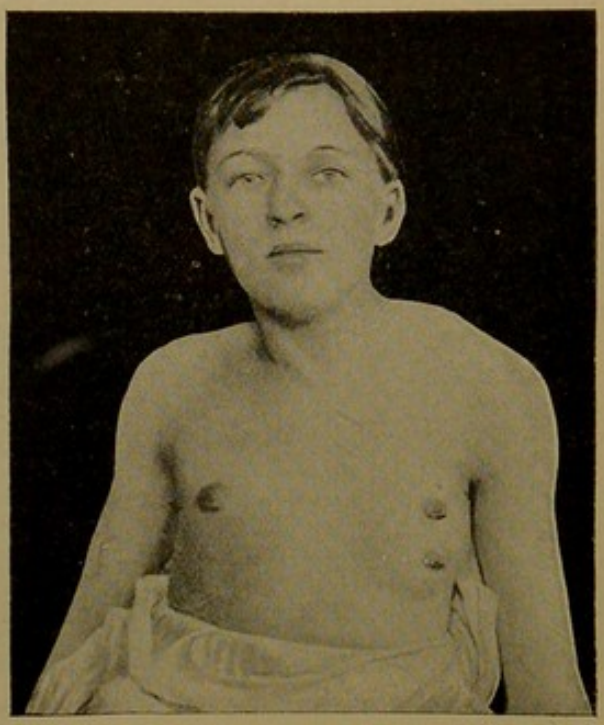

Fig. 200.—Kyphoscoliosis and polymastia in a case of gliosis. (Oppenheim.)

pointed out by Schlesinger, and specially by Sölder,[1] that the distribution of the sensory symptoms does not correspond to that of the peripheral nerves, but follows other laws (segmental type of distribution).

He finds the anæsthesia distributed in concentric rings round the mouth and nose, and limited by the line of the vertex, ear, and chin. Kutner and Kramer (*A. f. P.*, Bd. xlii.) have recently subjected the question to thorough investigation. From a case observed by Egger, syringobulbia also seems to produce symptoms of alternating hemianæsthesia. See further as regards this question, Gerber, *W. m. W.*, 1907.

Paralysis of the soft palate and vocal cords, and even complete recurrent paralysis, have been noted in some cases and are attributed to involvement of the vagus accessory.[2] The paralysis of the larynx is

[1] *Jahrb. f. P.*, Bd. xviii.

[2] I gave permission for my earlier cases of this kind to be reported in 1890, in Brunzlow's "Berliner Dissertation": "Ueber einige seltene, wahrscheinlich in die Kategorie der Gliosis spinalis gehörende Krankheitsfälle," and have since then greatly extended their number. Among the recent cases see Raymond-Guillain (*R. n.*, 1906), Kutner-Kramer (*A. f. P.*, Bd. xlii.).

usually unilateral (Schlesinger), but it has been found to be bilateral also. The spinal accessory is seldom affected (Schmidt, Hoffmann, Weintraud). Taste disorders are described by Ivanoff and attributed to the glosso-pharyngeal. I have found *nystagmus* to be by no means uncommon. Glycosuria has been seldom observed, and vomiting in a few cases only. There is occasionally hemiatrophy of the tongue (Fig. 198). Chabanne, Schlesinger, and Dejerine describe facial hemiatrophy. Schlesinger also once mentions atrophy of the temporal muscles.

Atypical forms are caused by an unusual localisation of the process. Thus the gliosis may be limited to one-half of the spinal cord (Rossolimo, Dejerine, Oppenheim, Dercum-Spiller, Sano, etc.). The clinical symptoms of this *unilateral gliosis* are generally confined to one-half of the body *i.e.* to the arm and trunk of one side. Cases have indeed been described in which only one posterior horn was affected, and therefore partial sensory paralysis combined with vasomotor troubles in the corresponding side of the body were the only symptoms of the condition. The process may also be limited to the anterior cornua, muscular atrophy being then the only symptom. I have found in one case muscular atrophy in the right, and vasomotor, secretory, and sensory symptoms in the left arm. In another case under my care the typical symptoms of gliosis appeared in the right arm and left leg (crossed gliosis). If the disease occurs in the thoracic and lumbar cord, the clinical condition is modified only as regards the topical distribution of the symptoms. I have seen three cases in which the symptoms were at first limited to one lower extremity.

If the affection arises in the medulla oblongata, *bulbar symptoms* appear at the onset, and these are always asymmetrical and usually unilateral (Oppenheim-Brunzlow, H. Müller, Lamaq, Schlesinger, Raymond, A. Westphal, Ivanoff, etc.).

Optic neuritis and choked disc are occasionally found. Weisenburg and Thorington[1] think that these are due to a combination with hydrocephalus. It is not clear whether atrophy of the optic nerve and immobility of the pupils may occur in pure gliosis, or whether they always indicate a complication with tabes or general paralysis. The cases reported by Rose-Lemaître (*R. n.*, 1907) are purely clinical. Paralysis of eye muscles is a not uncommon symptom. Redlich describes a combination with psychoses. Mental symptoms are frequent in the later stages of the disease (Marie-Guillain).

Although partial sensory paralysis is the most characteristic sign of the disease, the tactile sense, or less often the sense of position, are affected in not a few cases; but analgesia and thermo-anæsthesia are usually more marked. I have hardly ever found the vibratory sense to be affected. Polyæsthesia and a retardation of sensory conduction have occasionally been noted.

Morvan has described a group of cases in which analgesia and thermo-anæsthesia are associated with very characteristic symptoms in the shape of tactile anæsthesia and whitlows on the fingers. He regards this as a special form of disease and terms it "parésie analgésique à panaris des extrémités supérieurs." More recent observations show that it undoubtedly depends upon gliosis, but leprosy may give rise to a condition closely allied to it.

[1] *Amer. Journ. Med. Sc.*, 1905.

A more extensive involvement of the white matter, as already stated, will naturally give rise to spastic symptoms, which, in exceptional cases, may be the outstanding feature of the case. French writers (Marie-Guillain, Alquier-Guillain) regard these cases as a special type. Guillain (*Thèse de Paris*, 1902) and Raymond-François (*R. n.*, 1906) describe as characteristic of this form, a special deformity of the hands—main en pince—in which the three last fingers are in a condition of flexion contracture, whilst the thumb and finger can be freely moved.

The symptoms of gliosis may undergo a material change should the tumour affect specially the posterior columns or be accompanied by a degeneration of the posterior columns, which, as I [1] have shown, is a condition so closely related to tabes dorsalis, not only from the anatomical but also from the clinical point of view, that the ordinary symptoms of gliosis may be more or less completely masked by those of the tabes dorsalis. I have named this gliomatous pseudo-tabes. Finally, syphilis may produce allied clinical conditions, as I have found chiefly from therapeutic experience. Thus I lately made the diagnosis of syringobulbia in a case which showed astonishing improvement under energetic iodide treatment. The patient was the son of a syphilitic father.

Congenital anomalies of development in the vertebral column, especially spina bifida, are repeatedly noted, as by Minor, Dufour, and others, and in the other organs also, such as anomalies in the formation of the skull and jaw, hydrocephalus, web fingers or toes, hypoplasia of the genitals, etc. The combination with hydrocephalus may give a particular stamp to the condition. I made the diagnosis of cervical gliosis in an interesting case in which the presence of cervical ribs at first suggested that the paralytic symptoms were due to them. Soon afterwards I saw an exactly similar case, and other writers, such as Marburg, have also recently pointed out their occurrence.

Development and Course.—The disease has such a gradual onset that its first commencement can seldom be ascertained with any precision. The majority of patients in whom the disease could be diagnosed were between 25 and 45 years of age, but it has been proved that it may commence in childhood. In one case I have been able to trace the onset back to the age of 14, in another to the third or fourth year. It is usually the trophic troubles (in the muscles, skin, or joints, etc.) which direct the patient's attention to his condition, and if he then comes under medical examination the sensory disturbances may usually be detected. The further course is very insidious and may extend over ten to twenty years or much longer. I have treated a lady of 68, in whom the disease had commenced at the age of 18. In another case I found no marked progress of the disease during ten years. Remissions occasionally occur. These were specially remarkable in a case reported by Müller and Meder, in which a paralysis of all four extremities which had been present at the commencement of the illness completely disappeared. The acute paralytic conditions which sometimes occur in the course of the illness may be attributed to hæmorrhage, as Bruce found in one case. Gowers describes this as syringal hæmorrhage. Œdema or serous exudations, which take place in the cavities, may be permanent or may be absorbed.

It is only in the later stages that the symptoms in the lower extremities, due to the diffuse morbid process in the upper part of the

[1] *Charité-Annalen*, Jahrgang xi., 1886; also "Über atypische Formen der Gliosis spinalis," *A. f. P.*, xxv.

spinal cord, are associated with signs of interruption of conduction: spastic paraparesis of the legs, bladder symptoms, etc., etc.

Death is due to cystitis, bed-sore, septicæmia, an involvement of the medulla oblongata, or some intercurrent disease.

Differential Diagnosis.—The distinction between progressive muscular atrophy of spinal origin and amyotrophic lateral sclerosis is discussed elsewhere, pp. 229 to 235. So long as there are no sensory symptoms, a definite differentiation is not possible. With regard to amyotrophic lateral sclerosis, it is to be noted that the atrophic paralysis of the arms in gliosis is usually flaccid in nature and is not associated with increase of the tendon reflexes. The diagnosis may be difficult in the rare cases of chronic anterior poliomyelitis in which slight sensory symptoms are caused by the spreading of the affection to the posterior columns (see corresponding chapter). Gliosis may also simulate spastic spinal paralysis, but the partial sensory paralyses are hardly ever permanently absent in such cases.

The diagnosis from caries and peripheral neuritis or plexus affections is a particularly practical question. Caries of the cervical spine may produce a similar clinical picture, viz. progressive muscular atrophy of the upper extremities, especially of the hands, sensory disturbances, and eventually changes in the pupils, such as described above. Here, however, the symptoms of vertebral disease and tuberculosis are always present, at least in an illness of long duration, and the sensory symptoms have not as a rule the characteristics of dissociated sensory paralysis. Further, the signs of interruption in the conduction of the cord usually appear at an *early* stage.

In affections of the brachial plexus and the nerves of the arm, whether the neuritis be of traumatic, toxic, or infective origin, the symptoms of irritation (pain) are conspicuous. It is only in very rare cases that the anæsthesia is limited to the sensibilities for pain and temperature, and the distribution is not of the root type but corresponds to the area of distribution of the affected nerves. This of course is not so if the affection occurs in the roots themselves.

In the vasomotor neuroses (acroparæsthesiæ) objective signs are entirely absent or are limited to a slight dulness of sensation, especially of the tactile sense in the tips of the fingers.

Atrophy of the small muscles of the hand may develop as the result of vasomotor troubles of long duration, as Luzzatto has described from my polyclinic. The anamnesis, as a rule, reveals this origin, and the atrophy is usually very circumscribed and non-degenerative.

Hysteria may easily give rise to confusion, the more so that it may be associated with gliosis (both originating mainly on the basis of congenital anomalies of development). Hysterical patients sometimes complain chiefly of paræsthesia in the upper extremities and a feeling of weakness in one or both arms. Amongst the objective symptoms, hemianæsthesia may give rise to doubts in the diagnosis, but here the special senses are almost always involved. There is also no atrophy, or it is non-degenerative and limited to single muscles. Concentric narrowing of the field of vision is also occasionally found in a gliosis which is not complicated by any other sign of hysteria.

H. Curschmann has lately given us an exhaustive discussion of the differential diagnosis between hysteria and syringomyelia (*Z. f. N.*, Bd. xxix.).

Some cases of hæmatomyelia have a symptomatology closely allied to that of gliosis. But hæmatomyelia is almost always, in opposition to gliosis, a regressive disease. In doubtful cases the further course must determine whether a gliosis has developed from the hæmatomyelia.

Hoffmann thinks gliosis can be diagnosed from glioma of the spinal cord (myxoglioma, angioma, gliosarcoma), (see preceding chapter). The latter is distinguished by the acute or subacute onset of the symptoms and a much more rapid course. The symptoms rather resemble those of a transverse myelitis, extending gradually upwards and downwards, and eventually reaching the medulla oblongata. The symptoms of irritation predominate. We have, however, seen cases of glioma of the spinal cord, with a very insidious course. Cases reported by Nonne-Stertz and others also show that the glioma may be associated with gliosis and formation of cavities at other parts of the spinal cord.

There may be very great difficulty in distinguishing syringomyelia, especially of Morvan's type, from leprosy, from its macular form in particular. The clinical conditions may be so much alike that the attempt has been made to represent syringomyelia simply as a form of leprosy, a product of the lepra bacillus (Zambaco, Marestang). This has, however, been rejected, because, though in post-mortem examinations of leprosy other affections of the spinal cord, such as degeneration of the posterior columns (probably merely the result of the peripheral process, Lie, Nonne), or fine cell changes (Lie, Shuzo Kure), have been occasionally found, on the other hand no pathological evidence of gliosis has been discovered; and conversely, in spinal cords with gliosis the lepra-bacillus has only been found in a few instances (Babes, Uhlenhuth-Westphal).

According to Jeanselme and Nonne, the lepra bacillus has not been found in the spinal cord of a single typical case of syringomyelia. There is, however, one case of true leprosy (Gerber-Matzenauer [1]) in which the spinal cord showed the syringomyelic process, but no evidence whatever of the bacillus.

With regard to the *differential diagnosis* the following points are of importance: Leprosy until recently did not occur in Germany. It first appeared endemically a few years ago in East Prussia, and has occurred sporadically only in isolated cases in other parts, in Hungary for instance.[2] The cutaneous affections present in leprosy tend to take the form of multiple circumscribed plaques, which occasionally extend to the lower extremities and the skin of the face, whilst syringomyelia usually commences with local circumscribed symptoms in the upper extremities. The many trophic disorders in the face to which leprosy gives rise, are generally absent in gliosis. This is the case also as regards the peripheral paralysis of the facial nerve sometimes noted. The pigmented or non-pigmented spots of diminished sensibility are found only in leprosy. The sensory disturbances have usually an insular distribution or one corresponding to the peripheral nerves, whilst in syringomyelia they follow other laws (see above). The muscular atrophy in leprosy practically always begins at the distal ends of the limbs, whilst in gliosis it may begin in the shoulder. Paralytic symptoms of spastic type in the legs, paralysis of the bladder and bowel, typical bulbar symptoms, nystagmus, etc., are in favour of gliosis. Febrile attacks which occur in leprosy are absent in gliosis (Laehr). Fusiform thickenings on the peripheral nerves are due to leprosy. They are found particularly often on the great auricular nerve (Baelz). The hypertrophy of the nerves may be considerable; Jeanselme and Huet, for instance, found the musculo-

[1] "Obersteiner," ix.

[2] During the last few years I have seen a number of atypical cases—some of them very mild or abortive—of nervous leprosy from certain parts of South America, which had been unrecognised and wrongly diagnosed. Similar observations seem to have been made in Brittany (see the Thesis of Plateau, Paris, 1904, and Jeanselme, "La Lèpre en France et dans ses Colonies," *Presse méd.*, 1904).

spiral and ulnar nerves to be swollen to the thickness of the little finger. The most certain sign of leprosy is the presence of the lepra bacillus; Pitres and Sabrazès succeeded in finding it in a case otherwise difficult to interpret, but it is by no means always detectable. The existence of lepra nodules is of course specially characteristic.

The pathological processes have lately been studied specially by Lie (*Norsk Magaz. f. Laeger*, 1904, and *X. Dermatol. Kongress*, Berlin, 1904) and Nonne (*Lepra in Bibliothec. internat.*, 1904). The bacteriological-therapeutic problems, which have been raised by Deycke and Reschad (*D. m. W.*, 1905) cannot be discussed here.

The differentiation of syringomyelia from Raynaud's disease (see below) and allied tropho-neuroses may be an exceedingly difficult matter. Analysis of the trophic and sensory symptoms also gives rise to perplexity when the patient has had in his work to handle corroding, or excessively hot substances.

In the cases which are attributed to some injury of the limbs, the disease has usually been already in existence, but it may have been materially aggravated by the trauma. Its pre-existence may be proved by the facts that the injury caused no pain, that a comparatively slight trauma produced a fracture, etc.

In three adult cases I have found a disease which the patients regarded as congenital, the symptoms of which were shortening, atrophy, and paresis of one leg, with partial sensory paralysis and congenital anomalies of development (kypho-scoliosis, webbed toes, etc.). In two of these there were also affections of the functions of the bladder. As there were no other signs of spina bifida, it seemed as if there might be some other developmental anomalies of the spinal cord, corresponding perhaps to the type of syringomyelia.

The *prognosis* as to recovery from gliosis is absolutely unfavourable. But although the disease is usually progressive, an arrest may occur in rare cases. Remissions are not infrequent. In three of our cases in which there had been syphilis and symptoms which made the diagnosis of spinal gliosis imperative, the use of iodide of potassium led to improvement bordering on recovery. Patients suffering from gliosis may be able for a considerable time to carry on their occupation.

Treatment.—The patient must be urgently advised to avoid the risk of injury, and especially to be careful not to come into contact with anything hot. Over-strain of the arms should also be forbidden.

We have at present no specific treatment for this disease, but I have found that the administration of iodides is advisable in every case. Galvanic treatment of the spinal cord and mild faradisation of the atrophied muscles may be tried. Lumbar puncture has lately been recommended as a palliative measure, but I cannot think that it is a rational procedure.

Some writers have recently reported good results from the use of X-rays (Raymond, Pescarolo, Gramegna), (see Colombo, *Z. f. diät. Therapie*, 1906), Beaujard-Lhermitte (*Sémaine méd.*, 1907), who give details as to its administration, and Bougouı-Lhermitte (*R. n.*, 1907). It is very difficult, however, to determine the results on account of the gradual course of the disease and the tendency to remissions which is present in some cases.

Appendix

Diseases of the Cauda Equina (and of the Conus Medullaris)

Diseases of the cauda equina cannot be sharply distinguished from those of the spinal cord, and only occupy a special place from the fact that the cauda equina consists of a number of closely compacted nerve

roots. Morbid processes may extend to it from the lumbar vertebræ (the second to the fifth), the sacrum or the meninges, or may arise in the cauda itself (see Figs. 45 and 46). It is most frequently affected by injuries, fractures, or dislocations, and their consequences, traumatic hæmorrhages, stabbing (and bleeding), *e.g.* in lumbar puncture (Henneberg, Decroly), or lesions and compression from bullets, etc. Tubercular disease of the sacrum and of the sacro-iliac synchondrosis not infrequently spreads to the cauda (Naz, Wolff, Hahn, Cestan-Babonneix, M. Bartels,[1] Conta, Rossi,[2] Pick). In one case of this kind I was able to confirm the diagnosis by an autopsy. Tumours—neuroma, sarcoma, glioma, lymphangioma, etc.—may also occur in the cauda equina or extend to it from the surrounding membranes or bones. An aneurism of the hypogastric artery was diagnosed by Bálint as the cause of the compression. Syphilitic growths and syphilitic meningitis may also involve the nerves of the cauda (Westphal, Eisenlohr, Oppenheim, Delteil [3]). L. R. Müller's case of a primary, purulent inflammation of the cauda equina does not seem to me to be beyond question. Nor has Raymond sufficiently proved his diagnosis of a primary neuritis of these nerves.

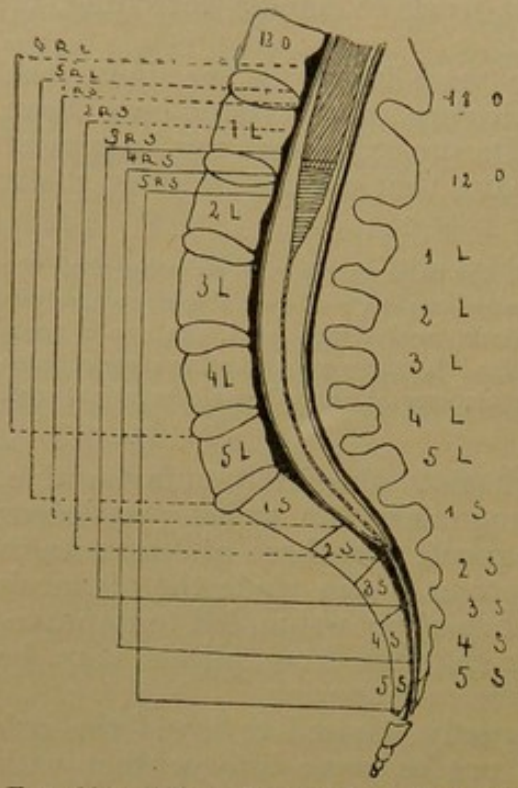

Fig. 201.—(After Raymond.) Diagram of the relations between the conus terminalis, cauda equina, and spinal cord. The vertical lines illustrate the areas through which the lower lumbar and the sacral nerves pass within the spinal canal and the sacrum.

The cauda equina can only be affected as a whole when the disease is situated high up—at the level of the second lumbar vertebra. The lower down the seat of the lesion is, the smaller is the number of the roots involved. The cauda equina innervates the muscles and the regions of the skin and mucous membranes supplied by the lumbar and sacral nerves. Consequently, affections involving the whole of the cauda give rise to atrophic paralysis of all the muscles of the lower extremities, to anæsthesia in them extending as high as the lumbar region, to paralysis of the bladder and rectum, and to impotence (see, however, Müller's theory, pp. 119 *et seq.*). As a rule, the two upper lumbar nerves are intact; the anæsthesia does not therefore extend so far, but leaves the region of the ilio-hypogastric and ilio-inguinal nerves free, and the sensibility of the testicle unaffected.

If the lesion affects the cauda equina below the third lumbar vertebra (see Fig. 202), the anterior crural and obturator nerves no longer fall within its range, but there is an atrophic paralysis of the sciatic nerve or of the muscles innervated by the sacral plexus (gluteal muscles, flexors of the knee, all the muscles of the foot), anæsthesia in the corresponding cutaneous area, and an ano-vesical and sexual paralysis. On the other

[1] *Mitteilungen a. d. Grenzgebiet.*, xi. [2] *Arch. de Neurol.*, xx. [3] *R. n.*, 1907.

hand the knee jerks, the cremaster reflex, and the sensibility of the testicle are conserved.

If the lesion is situated still lower, as in dislocation of the fifth lumbar vertebra (Kahler), the sciatic nerve is only partly involved. If it is still lower, within the sacral canal and below the emergence of the second sacral root, the muscles of the lower limbs are no longer paralysed, but we have the very characteristic symptoms of an affection of the three lowest sacral nerves and of the coccygeal nerve, *i.e.* paralysis of the bladder and rectum and of the genital functions (paralysis of the muscles of ejaculation, etc.) and the anæsthesia of the "riding-breeches" form, described on p. 134, which involves the urethra, the mucous membrane of the bladder, the anus and its neighbourhood, the perineum, the posterior scrotal region, and a strip of skin on the posterior inner surface of the thigh, innervated by the small sciatic nerve. The reflex closing of the sphincter ani, the anal reflex, and the electrical excitability of the sphincter are also lost. Reaction of degeneration may develop in this muscle (Bartels) and in the levator ani (Oppenheim). In one of my cases there was severe prolapsus ani.

It has been shown experimentally by Gianuzi and Merzbacher that section of the corresponding posterior roots may cause the sphincter ani to become flaccid.

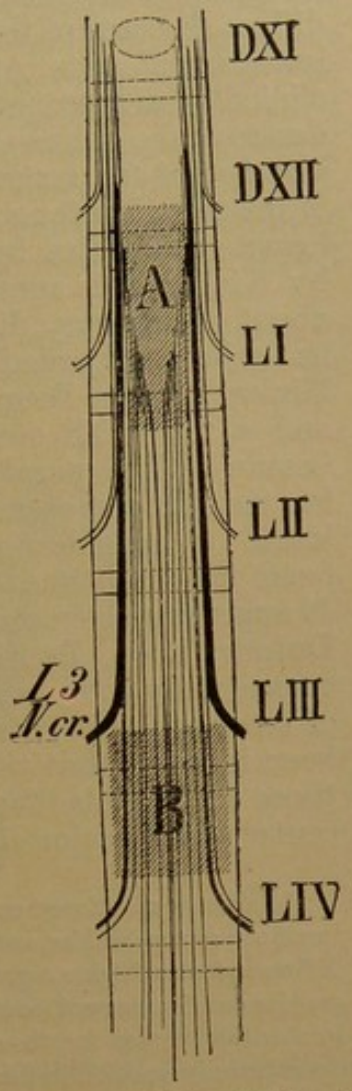

Fig. 202. — (After Schulze.) Diagram of the lower segments of the spinal cord and the cauda equina.

DXI — LIV = XI. dorsal to IV. lumbar root. The three lumbar roots (*L3*) (with their crural and obturator fibres) are shown by the dark shading.

A = longitudinal focus at the level of the lower segment of the cord.

B = longitudinal focus at the level of the third lumbar vertebra, affecting the cauda equina. The figure is to show that the two foci would produce symptoms on the whole similar.

Similar cases of lesion of the roots of the cauda equina, in which the above-mentioned group of symptoms was more or less completely developed, have been reported by Westphal, Lachmann, Thorburn, Mills, Schultze, Oppenheim, Cestan-Babonneix, Pansini, Curcio, Gehuchten, Bálint, Benedict, Weisenburg, Ferrier-Ballance, and others. In the case described by Lachmann there was a glioma in the upper part of the filum terminale, compressing only the nerves for the bladder.

We need not again insist that the lower the site of the lesion is the more limited becomes the area of the paralysis and sensory symptoms, so that the genital apparatus and the functions of the bladder and bowel are less and less involved, until ultimately when the lesion affects only the coccygeal nerve the paralysis is restricted to the levator ani and the anæthesia to the coccygeal region (Hammond).

In one case of affection of the cauda equina the symptoms of erythromelalgia were present (Auerbach).

Disease of the cauda equina therefore produces a group of symptoms —as shown also by Bechterew's experiments [1]—which is practically similar to that caused by diffuse affections of the lumbo-sacral cord. There is an almost entire agreement between the symptoms arising from diffuse diseases of the conus terminalis [2] and those caused by lesions of the lower sacral roots. As I [3] was first able to prove, by a case which I examined clinically and also post-mortem, an affection which more or less completely destroys the conus gives rise to the following symptoms : *paralysis of the bladder, of the rectum ; anæsthesia in the " riding breeches form " ; absence of the sexual reflexes, of the Achilles tendon reflex, etc.*, the motility of the lower extremities being completely conserved. An early case recorded by Kirchhoff is specially incomplete in this respect, nothing being said about the sensory disturbances. But a number of relevant clinical cases have been recorded (Bernhardt, Rosenthal, Raymond, Schiff, Köster, Higier, Ziegler, Bregmann, Billaud, Vitek, Fischler, H. Schlesinger, etc.), and a few with corresponding pathological reports (Sarbo, Raymond-Cestan). The paralysis of the bladder was not always complete, in so far that in some cases emptying of the bladder was automatically regulated, a considerable quantity of urine being spontaneously passed from time to time (L. R. Müller, Raymond-Cestan, Bálint-Benedict).[4] It appears also from the cases described by Rosenthal, Bernhardt, Müller, Dufour, Leon, Pansini, Bálint-Benedict, etc., that generative power is occasionally conserved or that at least there is still sexual desire and power of erection, whilst ejaculation may be impaired or lost. It has therefore been inferred that the corresponding centres and tracts are distinct from those for the bladder and bowel, and lie higher up than these, while the centres for erection lie above those for ejaculation.

As already mentioned on p. 120, L. R. Müller teaches (Fürnrohr agreeing with him) that the functions of the bladder and bowel are affected in the same manner in diseases of the conus as in diffuse lesions of higher segments of the spinal cord, as the lowest centres for the bladder, intestine, and for erection are not located in the conus, but in the sympathetic ganglia of the pelvis. He also locates the centres for the emptying of the seminal vesicles in the sympathetic. I have already described my position in this matter on p. 121, and referred to the views of Gehuchten (*Névraxe*, 1902-1903), and others. Bálint and Benedict have lately, on the ground of their experience, expressed their agreement with Müller's views, according to which when the conus is destroyed emptying of the bladder and bowel takes place in the same way as in the new-born infant. It cannot, however, be denied that the spinal cord is involved to a certain extent in the opening and closing reflexes, the lumbar cord innervating the non-striped sphincter muscle of the bladder, and the conus the anal muscles. They think that when there is no interruption of conduction in the higher segments of the cord, the need to micturate is indicated by a sensation of pressure behind the symphyses communicated by the abdominal nerves. They succeeded in proving that the act of parturition takes place independently of the conus or spinal cord, as had already been demonstrated experimentally (Goltz-Ewald). They interpret the persistence of erection in the absence of orgasm and ejaculation in the majority of cases according to Müller's view. Another symptom of involvement of the conus is absence of the reflex closing of the sphincter

[1] L. R. Müller made experimental investigations as to extirpation of the conus and its results (*N. C.*, 1904).

[2] Raymond suggests that the lower part of the sacral cord below the origin of the third sacral nerve belongs to the conus. Gehuchten accepts this limitation. L. R. Müller includes the three lower sacral segments and the coccygeal segment; he has pointed out, following Bräutigam and others, the special anatomical peculiarities of the conus—the slight development of the anterior horn cells, whilst the intermediate groups are well represented, the absence of the posterior commissures, etc.—and certain modifications in the course of the fibres (see p. 119).

[3] *A. f. P.*, xv.

[4] *Z. f. N.*, xxx.

when a finger is introduced into the anus or when the skin round the anus is rubbed (anal-reflex). L. R. Müller attributes this disappearance to the lesion of the lowest segment of the spinal cord.

The diseases of the conus with which we have to deal are traumatic myelitis and hæmatomyelia [1] (Figs. 203 and 204), (the latter is diagnosed by Schlesinger, for example, as the cause of corresponding symptoms which appear after Lorenz's operation for congenital dislocation of the hip-joint), primary hæmatomyelia of the conus, as Raymond and Schiff believe, and syphilitic processes. I have seen several cases in which the symptoms clearly indicated the presence of specific disease of the conus medullaris or the cauda equina, terminate in improvement or incomplete recovery. Gliosis may also apparently occur in the conus (Oppenheim, Gordon).

We cannot here discuss the congenital malformations which occur at the lowest segment of the spinal cord (see section on Spina Bifida). We

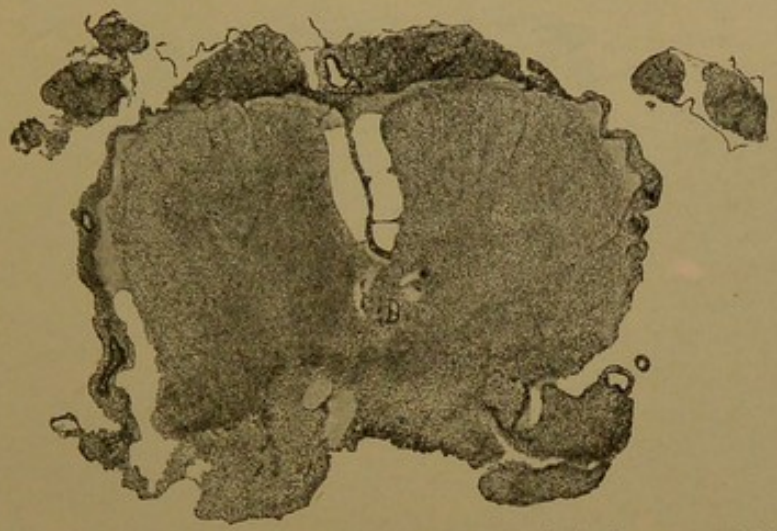

Fig. 203.—Traumatic myelitis and partial destruction of the lower segment of the cord. (From a section stained with carmin and hæmatoxylin.)

may point out, however, that they may be associated with symptoms of conus disease (Schlesinger).

It is obvious that in these affections of the conus, as soon as the lesion extends to the upper sacral segments symptoms of paralysis may appear in the lower extremities, commencing in the thigh and the small muscles of the foot. The paralysis is atrophic in character. Many writers include the region of the centres for these muscles with the conus, and refer the site of origin of the peroneus which lies higher up to the so-called "epiconus" (Minor). It is more correct, however, to include amongst the pure conus affections only those in which the process is localised below the second sacral segment and in which the muscles of the lower extremities are quite intact.

Minor (*Z. f. N.*, xix. and xxx.) gives the name of epiconus to the part above the conus from which the first and second sacral and the fifth lumbar arise. He has lately indicated the fourth lumbar as the highest limit. In lesion of this segment there is atrophic paralysis of the sacral

[1] In addition to direct injuries to the spine falls on the buttocks are very important causes of lesions in the conus. The spinal column is bent forwards, and the spinal cord is so stretched that solution of the continuity takes place at the site of least resistance, viz., in the conus (Fischler). A traumatic lesion of the conus may thus occur without any injury to the spine.

plexus, the peroneal region being specially affected and the tibialis anticus muscle being intact. The glutei may be involved; the sphincters are unaffected if the process is confined to the grey matter; the Achilles jerk is absent, but the knee jerk can be elicited. Acute anterior poliomyelitis may be limited to this region.

In a case of spondylitic compression of this region under my care the atrophic paralysis was confined to the peroneal muscles, whilst in the calf muscles the tonus was so exaggerated that ankle clonus could be elicited. In a similar case I have also found an exaggeration of the anal reflex which was tonic in character, *e.g.* an extension of the area from which the reflex could be elicited to the sole of the foot, and of the reflex movement to the whole of the gluteal muscles. Fürnrohr has made similar observations. Lesions of the epiconus are described also by Weisenburg, Bernhardt, Marinesco, etc.

As the symptoms of lesions of the conus and cauda are so nearly allied, the efforts of many writers (Schultze,[1] Raymond, Pansini, Coenen, Bálint-Benedict, Cestan-Babonneix, etc.) have been directed to the discovery of signs which would assist the diagnosis. It was presumed

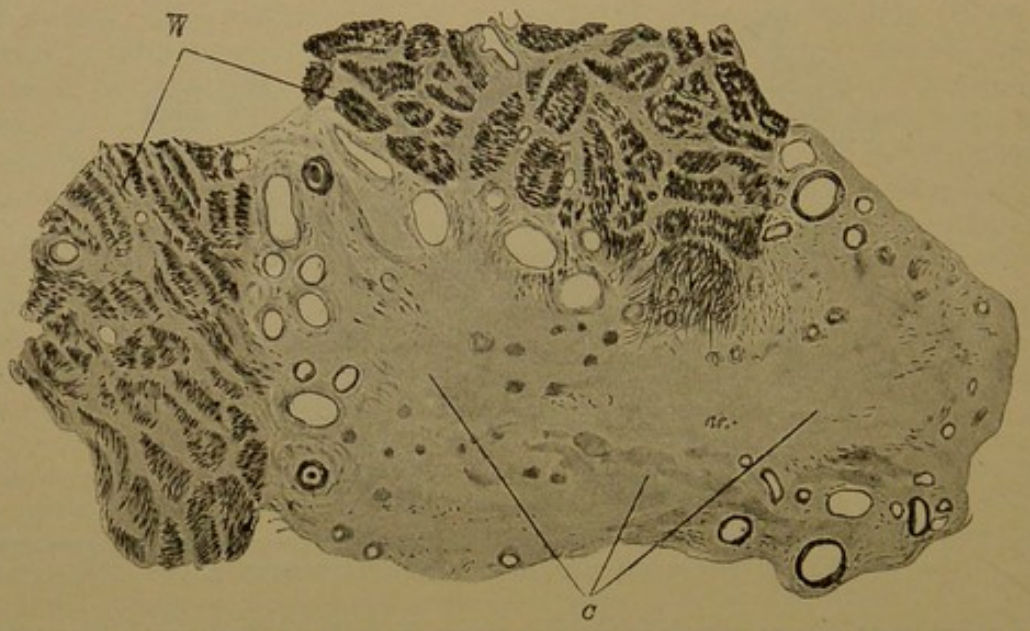

Fig. 204.—Complete destruction of the conus (*C*), with normal condition of the roots (*W*) in a case of fracture of the first lumbar vertrebra. Pal's method. (From a section prepared by Sarbo in Oppenheim's collection.)

that affections of the conus, when due to compression, always involved the roots surrounding it, and therefore the lumbar nerves, which as a rule were unaffected in lesions of the cauda equina. But the cases of Thorburn, Oppenheim, Kocher, and especially that of Erb-Schultze (where, in a fracture of the twelfth thoracic and first lumbar vertebræ, a fragment of bone had injured the cord as it lay between the roots), show that even under these conditions the lateral lumbar roots may remain intact (Fig. 203); indeed, it is certain that a trauma affecting the conus and its surrounding roots may cause severe and permanent injury to the former, whilst the roots remain more or less intact (Oppenheim, Sarbo, Müller, Fischler). I have seen this also in tumours of this region.

It has been stated that, in cases of doubt, fibrillary tremors in the atrophied muscles indicate involvement of the conus, but this is very uncertain. It must be admitted that disease of the conus is almost

[1] *Z. f. N.*, v.; see also literature in Fischler, *Z. f. N.*, Bd. xxx.; Billaud, "Des syndrômes du Cône terminal et de l'épicône," Paris, 1904.

always bilateral, but we do not know very much about strictly unilateral affections of the cauda. I have been obliged by the symptoms to diagnose a traumatic hæmorrhage in the sacral canal in a case in which there was found to be atrophic paralysis of the gluteal muscles (with R.D.) limited to one side. Unilateral symptoms of irritation and paralysis are also described in tubercular affections of the cauda equina (Cestan-Babonneix, Bartels, Oppenheim). But there is hardly ever a symmetrical distribution of the paralytic symptoms in affections of the cauda.

The following criteria may be used, carefully of course, for differential diagnosis: (1) The site of the pain, of the local tenderness to pressure, and particularly the site of any deformities that may be present. If these are on the lower lumbar vertebræ or on the sacrum, a disease of the cauda is indicated; but I have seen exceptions even to this rule. In a few cases a manifest dulness of note on percussion over the bones was an early and valuable sign, but it requires to be interpreted with great caution. (2) Very marked anæsthesia and the presence of *partial sensory paralysis* (analgesia, thermo-anæsthesia) are signs in favour of disease of the conus, but the absence of dissociation symptoms is not a positive proof of affection of the cauda. (3) Diseases of the cauda almost always give rise to *intense pain* in the sacrum, the bladder, perineum, anal region, the area of the sciatic nerve, etc., which is absent as a rule in affections of the conus. This is the most important criterion. In compression of the cauda the pain tends to precede the paralytic symptoms for a considerable time. On the other hand tumour of the conus may compress the neighbouring roots and thus give rise to very severe radiating pain. A sudden onset and above all a rapid extension of the symptoms are to a certain degree characteristic of disease of the conus. (4) Marked asymmetry of the paralytic symptoms hardly ever occurs except in affections of the cauda equina. The condition of the tendon reflexes is not a definite guide to the diagnosis, in spite of the contrary opinion of Zingerle and others. Absence of the Achilles jerk is the rule in affections of the conus and cauda; but the knee jerk is present and often exaggerated in the former, whilst it may be absent in lesion of the higher part of the cauda. In the cases from my polyclinic described by Köster,[1] radiography was used in order to make the diagnosis a definite one. I have also frequently employed this method, but usually without success. In cases of a traumatic hæmorrhage, the paralysis and anæsthesia become completely developed very soon after the conus is affected, but when the hæmorrhage occurs within the sacral canal a longer time elapses before the compression affects the roots—fifteen hours in a case of Mills. Trophic symptoms develop on the whole more rapidly and frequently in lesion of the conus, but bedsores have also been observed in diseases of the cauda.

Where hæmorrhages occur into the sacral canal, the cerebro-spinal fluid is stained red. This may be discovered by lumbar puncture, but when the hæmorrhage takes place in the substance of the conus this indication may fail.

In *tuberculosis of the sacro-iliac synchondrosis* or of the sacrum, other signs are present, such as swelling over the affected area, local pain and tenderness to pressure, and peculiar pain on lateral compression of the iliac crests (Erichsen's sign). In one case, however, I found neither swelling nor Erichsen's sign. There is also a rigid attitude in which the pelvis is usually lowered. The

[1] *Z. f. N.*, ix.

diagnosis may be confirmed by the appearance of burrowing abscesses in the pelvis or gluteal region, and eventually by the results of X-ray examination. According to cases described by Naz, Cestan-Babonneix (*Gaz. des hôp.*, 1901), and especially by Bartels (*Mitt. a. d. Grenz.*, xi.), the nervous symptoms are usually those of an asymmetrical affection of the cauda equina, *i.e.* they are most marked on the side of the morbid focus; pain in the sciatic region (with Lasègue's sign), atrophic paralysis of this nerve or of the external popliteal and of the gluteal muscles, paralysis of bladder and rectum (the latter may be present alone or may be specially marked, but may also in my experience be absent), anæsthesia in the region of the sacral roots, loss of the Achilles jerk and sometimes diminution of the knee jerk, etc. I was able to confirm this in two cases by operation and autopsy. In one, the development of all the objective symptoms was for a long time preceded by vague pains. The former were almost entirely confined to one side.

As regards the differential diagnosis, peripheral injuries of the sacral nerves are also to be considered, but these affections are usually unilateral and often accessible to direct examination, from the rectum or vagina. In compression of the sacral nerves in their extravertebral course, by tumours, etc., the symptoms are either altogether or for a long time unilateral, and are at first confined to the area of one nerve (sciatic, etc.), and there is no marked impairment of the functions of the bladder and rectum. Although this is of no importance as regards injuries affecting the sacral nerves directly after their emergence (new growths on the anterior surface of the sacrum, etc.), yet in such cases also the symptoms are often limited for a considerable time to one side (L. R. Müller). Tabes dorsalis may commence with symptoms in the region of the sacral roots, but these hardly ever simulate a perfectly pure disease of the cauda. Neuralgia may occur, limited to the ano-vesical region (W. Mitchell), but it is not accompanied by the objective symptoms of a lesion of the cauda.

Gluteal herpes may, according to cases of Davidsohn (*B. k. W.*, 1890) and myself, be preceded by transient paralysis of the sphincters, and may thus for a time give rise to difficulties in the differential diagnosis.

There may be a permanent paralysis, either congenital or acquired, and limited to the sphincter vesicæ, more rarely to the sphincter ani—as I have seen in children and adults (see also pp. 203, 204). The pathological explanation of this paralysis has not yet been ascertained. The congenital cases may be due to defective formation or to complete absence of the centres in the cord (infantile nuclear atrophy).

The *prognosis* of diseases of the cauda equina is on the whole more favourable than that of affections of the spinal cord. Although the course may be rapidly fatal (Schultze), arrests and improvements frequently occur, especially in the traumatic and syphilitic diseases of these nerve tracts. It must of course be remembered that the cauda equina is less easily injured than the conus (Fischler). The prognosis of the tubercular process is grave, but not hopeless. It is comparatively favourable when the lesion in the cauda is treated surgically, as shown by the cases of Thorburn, Laquer, Shaw, Busch, Sachs, Gerster, Ferrier-Horsley, and Ferrier-Ballance. In Bolton's case the symptoms were caused by a bullet in the sacral canal, and the condition improved after its removal. We owe interesting reports of this kind to Engelmann, Raymond, and others (see p. 367). I have also seen marked improvement, approaching recovery, in a case of syphilitic disease of the conus, and in another of traumatic conus lesion the improvement was so advanced that when I

saw the patient again eight years later, he could empty the bladder and intestine merely by muscular effort, and had not only regained normal sexual power, but had become the father of two children in the interval. H. Schlesinger reports similar cases.

With regard to *treatment* we can only infer that antisyphilitic measures are suitable if there is any suspicion of syphilis, and that in cases of injury or new growths, surgical interference is advisable as soon as it is seen that expectant treatment is of no avail (see p. 371). Sick for instance successfully performed laminectomy in fracture of the upper lumbar vertebræ with lesion of the conus. Tubercular diseases of the sacrum and the sacro-iliac synchondrosis may be successfully treated by orthopedic (Sayre) and surgical methods (Bardenheuer, Ferrier-Ballance [1]).

Third Group

This comprises diseases of the spinal cord *without organic or hitherto recognised anatomical cause.* These include forms and conditions, such as so-called spinal neurasthenia, spinal irritation and so on, hitherto placed in the category of the functional neuroses, but which we regard as functional diseases of the whole nervous system, and chiefly of the brain, and therefore discuss at another place.

We are, however, justified in considering *concussion of the spinal cord* at this point. A fall on the back, a blow or push against it, concussion of the whole body, *e.g.* in a railway accident, may have many severe results. It is certain that hæmorrhages may occur in the cord and meninges without any external injury or solution of continuity of the spinal column. Severe lesions and even rupture of the cord are said to have been observed under these conditions. The symptomatology of these affections does not require special description. The direct effect of concussion may also be a condition of generalised paralysis which indicates a functional inhibition of the spinal cord (and eventually of the brain) and terminates fatally in a few days, the most minute examination failing to reveal any anatomical change. These symptoms, which are practically identical with the idea of *shock*, were known long ago and were ascribed to molecular concussion of the spinal cord or to reflex inhibition. Kocher indeed refuses to admit that there is such a thing as spinal shock; he maintains that such cases are due to organic lesions of the spinal cord or of the brain. Stolper takes a similar standpoint. Schmaus and Hartmann lay most stress on the demonstrable changes caused by the trauma, and think that, apart from the actual crushing and tearing, the alterations in the pressure of the cerebro-spinal fluid may produce exudations of lymph and direct mechanical injuries of the nervous elements which may lead to their necrosis (as in shown by the investigations of Bikeles and Kirchgässer). But he does not entirely abandon the idea of commotion (molecular concussion).

Fickler (*Z. f. N.*, Bd. xxix.) maintains that a transient paralysis of the spinal cord may be the result of an injury which has neither affected the spinal column nor produced gross lesions in the cord itself. He thinks, however, that the symptoms arise not from molecular concussion, but from contusion due to the abrupt movement of the cord.

Symptoms of Shock.—There is "great prostration; the facial features are sunken; the eyes are dull and hollow and have a fixed, lifeless look;

[1] *Br.*, 1904.

the extremities are cold, the skin is white as marble, the hands and feet are slightly cyanosed, the temperature is 1-1½° C. below normal, the pulse can hardly be felt, and paralysis of the sphincters is present. There is no loss of consciousness; the voluntary movements are all possible, although limited and feeble, and the extremities when passively raised fall lifelessly back again. The sensibility is diminished, and only the strongest sensory stimuli are perceived as painful," etc. It is obvious that the brain plays an essential part in these functional disturbances. On the other hand, paralytic conditions of the spinal type may arise directly from such concussions without any detectable pathological change, although this is extremely rare.

In many cases the symptoms of a nervous disease develop gradually and insidiously after the injuries described; it was formerly thought that they must be due to a chronic meningo-myelitis, and the spinal site of the disease seemed to be so certain that the nervous affections arising after a railway accident were summarised under the name "*railway-spine.*" It cannot be denied that a railway accident which does not cause severe external injury may yet give rise to myelitis, due to a simple blow on the back. Gowers describes one such case in the latest edition of his text-book, and the investigations and cases of Schmaus, Westphal, Spiller, Bikeles, Jolly, Hartmann, Fickler, etc., show that myelitis may arise in this way, but such a result is exceedingly rare, and the affections of the nervous system occurring after such accidents should for the most part be regarded as neuroses (see chapter on Traumatic Neuroses).

Appendix

I. Congenital Malformations of the Spinal Cord and its Membranes

Some of these malformations, such as amyelia (absence of spinal cord), atelomyelia (defect from arrest of development of a certain segment), diastematomyelia (fissure of the cord), diplomyelia (doubling of the cord), make life impossible, or they are associated with other malformations which prevent the continuance of life. These forms have, therefore, no clinical interest, but detailed investigations have recently been made on cases of anencephaly, amyelia, micromyelia, etc. (Monakow, Leonowa, Muralt, Petrén, Brissaud-Briandet, etc.), which, along with the experimental observations of Schaper and others, have proved of great value as contributions to the study of the development and relations of the different segments of the nervous system, and of certain physiological questions. Leonowa has shown that in amyelia the spinal ganglia, posterior roots, and sensory nerves are normally developed. Other malformations, such as *asymmetries* and *heterotopia*, neither prejudice life nor give rise to any symptoms by which they can be diagnosed. It has been proved also by Ira van Gieson that most of the changes in the spinal cord described as heterotopia have been artefacts. The occurrence of a real heteropia cannot, however, be doubted. Anomalies of development of another kind, such as an abnormal localisation of Clarke's column, have been described by A. Pick. Stewart (*Br.*, 1904), Bruce (*R. of N.*, 1906), A. Westphal (*A. f. P.*, Bd. xli.), and Altmann (*Inaug. Diss.*, Breslau, 1906), have reported interesting malformations of the spinal cord.

Rachischisis and *spina bifida* are the only malformations of practical interest.

Congenital fissures in the spinal column are due to an arrest of development, which produces either merely a defective closure of the spinal column and spinal membranes, or the simultaneous formation of a tumour, due to a cystic projection of the meninges through the fissure in the form of a subcutaneous swelling (*meningocele*). The process usually involves the cord, also displacing it outwards (*myelocele or meningomyelocele*). Finally, Recklinghausen describes, by the name of *myelocystocele*, a cystic tumour arising within the cord itself and representing a cystic dilated segment of the embryonic medullary tube, which protrudes through the fissure of the vertebral body. This malformation is frequently associated with the development of other tumours

(lipoma, myolipofibroma, dermoid, teratoma, teleangiectasis) in the lower segment of the spinal canal or in the cysts.

Rachischisis is complete or partial. The latter alone is of clinical interest. It is generally situated in the lumbo-sacral, much less frequently in the cervical, and only exceptionally in the dorsal region. There is no posterior closure of the dural sac and the meninges at the fissure, so that the bottom of the sac is occupied by the ventral portion of the leptomeninges, and above this there usually lies the so-called area medullo-vasculosa of Recklinghausen, a red,

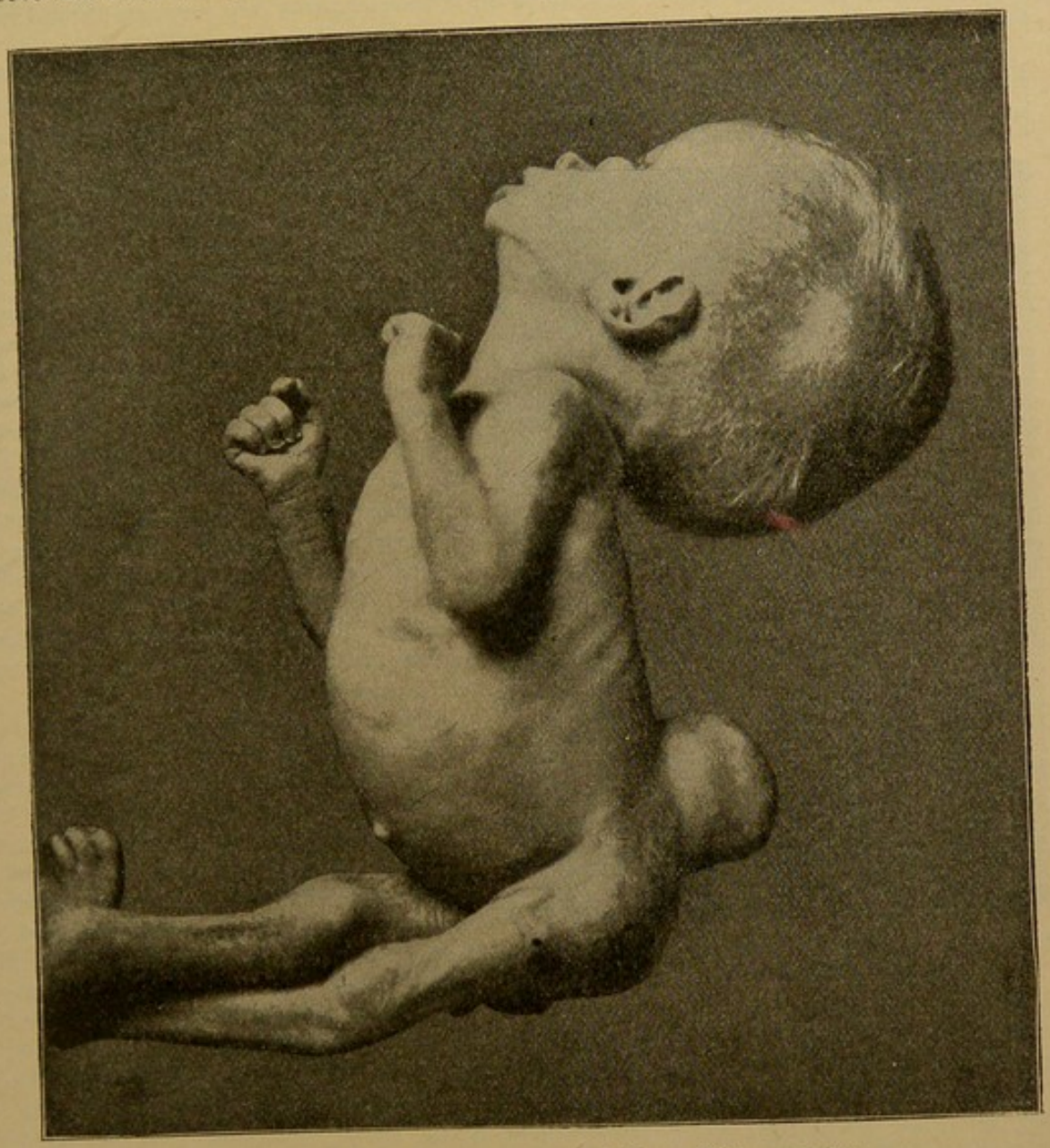

Fig. 205.—(After Zappert.) Spina bifida. Hydrocephalus.

velvetlike membrane composed of vascular plexuses and nervous elements, and which represents the more or less contorted lowest segments of the spinal cord. If the fissure is closed on the outside of the skin (*spina bifida occulta*), fibres and tracts may pass between it and the above-mentioned area, which may injure the nervous structures by exercising pressure and traction upon them. This influence may be specially active during the period of growth (Brunner, Jones, Katzenstein). But even in the forms which commence with the formation of cysts, pressure of the fluid and laceration of the nerve roots may subsequently cause degenerations.

Myelomeningocele is the most common form of spina bifida. In it the apex of the tumour is formed by the area medullo-vasculosa. The spinal cord, therefore, reaches its posterior wall.

whilst the accumulation of the fluid lies ventral to it. The central canal is open, or small grooves lead into it from the upper and lower poles of the tumour. In most cases of spina bifida the development of the lower segment of the spinal cord is more or less impaired, as it is flattened out when it enters the cyst formed from the meninges, or becomes attached to its walls. The lowest nerve roots have their origin from this flattened portion. The nerves of the cauda equina are themselves often degenerated, and are abnormal in their course, as they have to bend upwards in order to reach the point at which they emerge from the spinal canal. Anterior cystic spina bifida, in which the tumour is directed towards the abdominal cavity, is a very rare form of malformation. Neugebauer and Grossmann have recently described such cases.

The tumours are as a rule situated in the lumbo-sacral region of the spinal column, in the middle line or somewhat to one side, and they vary from the size of a nut to that of a child's head (Fig. 205). They are elastic and usually fluctuating, and the defect in the vertebræ may sometimes be felt at the base or the side of the tumour. Radiography has lately been used in diagnosis (Beck, Levy-Dorn, Milner, Oppenheim), and it may even afford information as to the contents of the sac. If the skin does not form part of the tumour (spina bifida occulta), it simply stretches over the tumour or fissure; it may be normal, or thin and drawn in like a funnel, and it is sometimes covered with hair (hypertrichosis), and may sometimes be the seat of an angioma. Spina bifida is not infrequently accompanied by other malformations (hydrocephalus, gliosis, syringomyelia, scoliosis, intestinal fissure, ectopia vesicæ, umbilical hernia, etc.). Schwalbe describes displacement of and overlapping between the cerebellum, medulla oblongata, and spinal cord.

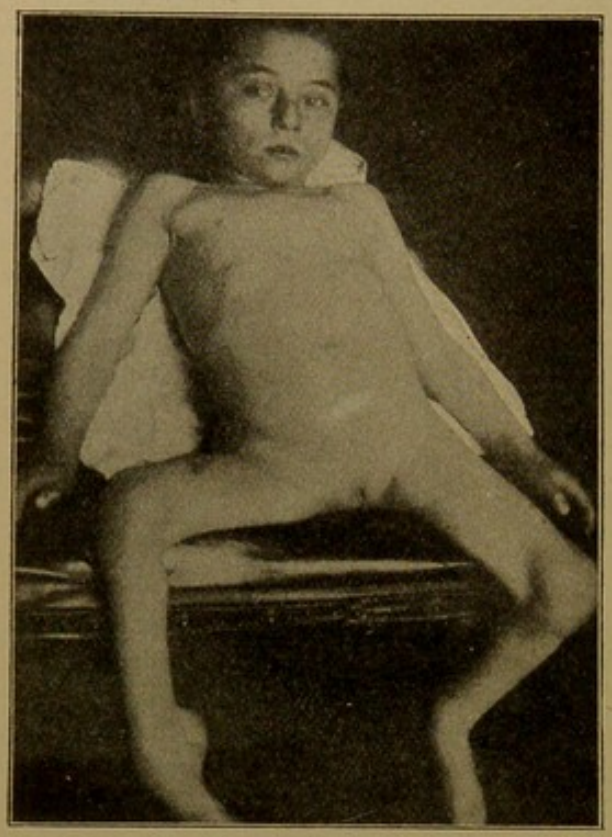

Fig. 206.—Atrophic paralysis of the leg muscles in spina bifida. (Oppenheim.)

Pressure on the tumour may cause derangement of the cerebral functions, symptoms of cerebral compression, or complete loss of consciousness.

We shall not here discuss the question of the mode of origin of these malformations, but we would draw attention, mainly on account of the details and the points still under dispute, to the exhaustive work of Recklinghausen (*V. A.*, Bd. cv.), to the papers by Marchand, Ziegler, Borst, Muscatello, Ruyter (*A. f. klin. Chir.*, Bd. xl.), Hildebrandt (*A. f. kl. Chir.*, Bd. xlvi., and *Z. f. Chir.*, xxxvi.), Bockenheimer, etc. (*Arb. aus Bergmanns Klinik*, 1902), and to the Report of the London Committee. With regard to the various theories see also the work of Katzenstein (*A. f. kl. Chir.*, Bd. lxiv.), the résumé by Borst in the *Centralbl. f. allg. Pathol.*, 1898, and Tillmanns (*D. m. W.*, 1904).

The symptoms arising from the pathological changes in the lumbo-sacral cord and the nerve roots are of special *neurological* interest. They are to a great extent congenital, but may become evident during the first years of life, at the period of puberty and even later. They may in part develop in later life as the result of injuries, of traction on the roots and cord, and of secondary inflammations.

The typical symptoms of spina bifida are as follow: *Club-foot* (pes varus or equino-varus) due to paralysis of the leg muscles (Fig. 206), usually with the exception of the tibialis anticus, is the most common. Ruyter found this symptom in eight out of twenty cases. There may also be a *complete paraplegia* with atrophy of all the muscles of the extremities, and various conditions of the electrical excitability. I have sometimes found reaction of degeneration, sometimes quanti-

tative diminution, and in a few cases no change whatever. There may also be paralysis of the bladder and intestine, anæsthesia and formation of ulcers (especially perforating ulcer), and occasionally prolapsus ani. The knee jerks may be diminished. Spastic paralysis, as in Reiner's case, is rare, and appears only when the spina bifida is situated unusually high, in the dorsal and cervical regions. The paralytic symptoms as a rule show a distribution which is characteristic of an affection of the cauda equina. In a case which I have recently seen the spina bifida gave rise to no definite nervous symptoms, but other signs were present which pointed to a complication with gliosis.

The patient seldom lives to old age, and the more marked the symptoms of paralysis, the greater is the danger to life. Death may be the consequence of ulceration of the skin. It may be due to rupture of the cyst either directly or secondarily as a consequence of an ascending meningitis, as in a case of Haushalter's.

The treatment is *surgical*; compression, ligature, puncture, followed by injection of iodide and removal of the sac—the method which is now most in use (Schede, Kölliker). Operation is indicated in meningocele. It has also been reported as successful in myelomeningocele (Bayer and others), but as a rule this is not to be expected. The condition of the nervous elements can often be determined only after the sac is opened; the operation is then modified accordingly. The part containing the nervous structures should be detached and replaced in the spinal canal. In spina bifida occulta Maass and Katzenstein and others have had good results from the removal of the fibrous plates of the membrana reuniens posterior, or of the adhesions formed by the muscles, connective tissue, and elastic tissue, which compress the cord. Excision of the tumour should be followed by osteoplastic closure of the fissure, as practised by Dollinger, Sklifossowsky, Rochet, and others, but this is not suitable in spina bifida occulta .Hydrocephalus contraindicates operation (Broca, Schede). Bockenheimer gives statistics derived from sixty-three cases in Bergmann's "Clinique." Bergmann operated upon all three kinds of spina bifida, if other marked malformations, hydrocephalus or severe paralysis of the extremities were not also present. Nicoll (*Glasgow Med. Journ.*, 1902) reports remarkable results. Böttcher (*Bruns Beitr.*, Bd. liii.) gives the following figures: of thirty-nine cases with operation, twenty-three =70 per cent. died, and twelve recovered completely. These were mostly cases of pure meningocele.

II.

We must at this point consider certain forms of paralysis of the spinal type, which are exceedingly difficult to explain on account of the lack or insufficiency of knowledge as to their pathological anatomy.

First, we have the so-called spinal *reflex paralyses*. This name is given to the forms of paralysis which resemble a pure paraplegia or a transverse myelitis, and which sometimes develop after diseases of the urinary passages, the intestine and uterus. In the majority of cases the earlier views formed upon insufficient data, that these were functional conditions due to vascular spasm and without any anatomical pathological cause, cannot be maintained. It has indeed been shown experimentally that strong stimulation of the viscera, such as bruising or laceration of the kidneys, may produce a very transient paraplegia, but the cases under consideration almost always showed pathological changes, either neuritic, meningitic, or myelitic. Disease of the mucous membrane of the bladder, of the urethra (blenorrhagia), of the prostate (prostatitis or abscess), etc., may produce conditions of paralysis which are due, as many writers think, either to the fact that the inflammation or suppuration has spread as a neuritis along the nerves to the spinal cord and its membranes, or to the direct action upon the spinal cord of the infective organisms, or the toxins which they have produced-

The theory of reflex paralysis cannot, however, be entirely abandoned, as we shall see later. We might here point out that individuals with phimosis sometimes suffer from paraparesis of the legs and paralysis of the bladder, which disappear when the phimosis is cured by surgical operation. I have seen in a male patient an undoubtedly functional paraparesis follow the introduction of the catheter into the urethra.

The transient paralyses which sometimes follow obstinate diarrhœa, the use of drastic aperients, irritation from worms, etc., are also to be regarded as functional; they have been thought to be *paralyses from exhaustion due to anæmia*. Dejerine has described paraparesis as a transient symptom following severe attacks of lightning pain.

Although a few cases have been explained as the result of an *ascending neuritis*, extending, after injury of the peripheral nerves, to the spinal cord and producing a myelitis which gives rise to paralytic symptoms of spinal type, the great majority of central paralyses developing after such injuries nevertheless belong to the class of the traumatic neuroses. Simple conditions of weakness with difficulty in walking, such as follow acute infective diseases, exhausting illnesses, and severe loss of blood, without any sign of spinal affection, are not caused by pathological organic changes in the spinal cord, but are the consequence of defective nutrition or toxic influences, possibly aided sometimes by anæmia of the spinal cord. The course is always favourable. On the other hand the cases of Lichtheim and others (see p. 189), show that severe anæmia may lay the foundation for radical structural changes in the spinal cord, and it may in many cases be difficult to determine whether the existing paresis is a functional disease or is produced by degenerative changes in the spinal cord.

Periodic Paralysis of the Extremities

This includes a number of obscure cases, to which greater attention has of late been devoted. Some of these had already been brought into relation to *malaria* (Cavaré, Romberg, etc.); they showed intermittent paraplegia, with or without anæsthesia, and sphincter paralysis of several hours' duration, which usually disappeared after an outburst of sweating. The attacks came on after the manner of malaria, of the quotidian or tertian type, and they could be cut short by quinine. In a case of this kind under my own observation, there was during the attack a considerable rise of temperature, and in the intervals slight symptoms of paralysis. The constant use of quinine for a considerable period resulted in recovery.

The cases of periodic paralysis of the extremities in which no connection with malaria could be detected are of special interest. These include a case described by Westphal (*B. k. W.*, 1885-1886), and myself (*Charité-Annalen*, xvi.), which may serve as typical of this group: a boy of thirteen, after scarlatina, developed attacks of paralysis, which occurred at irregular intervals, usually every second week. They almost always commenced during the night, and the paralysis lasted till the following mid-day or evening. At the height of these non-febrile attacks, there was a flaccid paraplegia of all four extremities and of the trunk muscles, whilst the cranial nerves retained their normal function. The tendon reflexes were abolished, the electrical excitability diminished, and in certain muscles entirely lost. Sensibility was normal. In addition to paralysis of movement the patient complained of thirst and perspiration. Micturition was usually difficult. The attack gradually passed off. In the intervals the boy—or, I may now say, the man—is healthy, but there is often slight weakness in some muscles, and abortive attacks occur.

In the later course of the disease, I found that there was during the attacks a *dilatation of the heart with symptoms of mitral insufficiency*, which did not exist in the intervals.

The patient has developed normally, and has married, but is still subject to the same conditions as formerly.

We can merely suggest theories as to the nature and causes of these paralytic attacks. It may be that there is a virus somewhere in the body, which reproduces itself from time to time.

Similar cases have been reported by Hartwig, Fischl, Cousot, Goldflam, Greidenberg, Hirsch, Taylor, Mitchell, Donath, Putnam, Singer, Oddo, Buzzard, Schlesinger, etc. The majority of these relate to a family form of the disease. Oddo and Audibert collected sixty-four cases of this kind. They mention an occasional involvement of the muscles of the throat and neck. Cheinisse (*Semaine méd.*, 1904) gives a full report of the cases recorded. Whilst as a rule the cranial nerves are unaffected, there was ptosis at the height of the attack in a case observed by Singer, and dysphagia has been noted by Mitchell-Flexner-Edsall and by Fuchs. The attacks are specially apt to come on after a long rest, during or after sleep, or after over-fatigue. They last from fifteen minutes to a week; several hours is the average duration. There are cases in which an attack occurs daily or weekly, but the intervals may extend to months and years. In a few cases the attacks were apt to appear on certain days.

The changes in the heart which I have observed have also been reported by Goldflam, Mitchell, and specially by Hirsch. Fuchs has (*W. kl. R.*, 1905) also noted these, and H. Schlesinger (*W. kl. W.*, 1905) has found the pulse to be slow and irregular. Goldflam also observed *qualitative* changes of the electrical excitability. This is in contrast to our experience and to the investigations of Oddo and Darcourt. Oddo points out that the mechanical excitability of the muscles may be diminished or absent during the attack. Goldflam has found toxins and albumen

in the urine after the attacks; and the latter has been also reported by H. Schlesinger. Singer speaks of the increased toxicity of the urine, and Löbl and Schlesinger found excretion of aceton in the urine. The reports of toxic bodies in the urine (and fæces, where Crofts states he has found them) must be accepted with reserve. Westphal and I have already described certain changes in excised portions of muscle, to which, however, we were not inclined to ascribe any practical importance, although they were very remarkable. Goldflam (*Z. f. N.*, vii.) describes hypertrophy and vacuolisation of the primary fibres, with intercalation of a vitreous substance between them, and on this account he regards the disease as a muscular one. Bernhardt describes its combination with progressive muscular dystrophy. Oddo terms the affection a myoplegia in contrast to myotonia, and regards both as developmental diseases of the muscular tissue.

Osteomalacic Paralysis

During the course or even at the onset of osteomalacia, severe pain in the back and lower extremities may be associated with weakness in the muscles, which affects the power of walking. The gait becomes shuffling and waddling. The paresis is specially evident in the flexors of the hip, and in the ileopsoas (Köppen, Stieda, Laufer). The condition may consequently resemble a dystrophy so closely that a differential diagnosis may be difficult to make. Paræsthesiæ and tremor also occur, and exaggeration of the tendon reflexes (Winkel) and contracture of the adductors have been described (Trousseau, Latzko). Symptoms of paralysis are less often noted in the upper extremities and trunk muscles. Degenerative changes were found in the muscles by Friedreich and Weber. Friedreich and Jolly have drawn attention to the combination of osteomalacia and dystrophy. The diagnosis has to be made on the ground of the condition of the skeleton (with the assistance of an X-ray examination), but it should be remembered that the muscular affection may be the earliest symptom (see Hösslin, *A. f. P.*, Bd. xl.).

Schlesinger has called attention to a syphilitic and hysterical "pseudo-osteomalacia" (*D. med. W.*, 1906), which I have also met with.

The combination with symptoms of exophthalmic goitre or the relation of osteomalacia to the thyroid gland has been described by Hoennicke, whilst the results of treatment, *e.g.* the curative effect of castration on the disease (Fehling) point to a connection with the sexual organs. Phosphorus has frequently been recommended as a drug; I have found osteomalacic paralysis pass off under its use.

For literature, see the review by Laufer (*C. f. Gr.*, 1900), Zesas (*C. f. Gr.*, 1907), and the treatise by Völsch (*M. f. P.*, xxi.), which has just appeared.

DISEASES OF THE PERIPHERAL NERVES

On the Anatomy of the Peripheral Nerves

The cerebro-spinal nerves and their branches practically consist of medullated nerve fibres provided with sheaths of Schwann, and contain only a few non-medullated fibres. Even the smallest of the latter, according to Boveri and Kölliker, possess a thin medullary sheath.

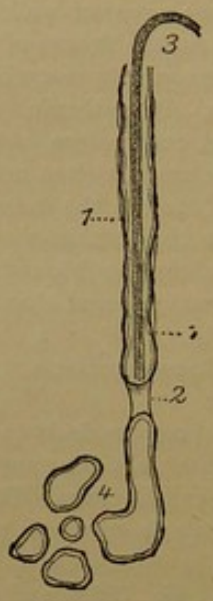

Fig. 207.—Medullated nerve-fibre with the sheath of Schwann (1), nerve-medulla and axio-cylinder (3). The latter is isolated above. Below the medulla (2) has escaped and formed myeline drops (4). (After Schwalbe.)

The medullated fibres of the peripheral nerves are composed of an *axis-cylinder*, a *medullary sheath*, and a *sheath of Schwann*.

The axis cylinder (neuraxon), which lies in the centre or occasionally somewhat eccentrically, is seen in fresh preparations to be covered by myelin, and it only becomes distinctly visible after the latter is removed, or in sections stained with a reagent that imparts to it a certain tint. In sections of medullated nerve fibres stained with carmine, for instance, it stands out as a red point, or in those stained with chloride of gold it assumes a reddish-brown colour. The new methods of Mallory, Bielschowsky, etc., are specially well adapted to stain it and demonstrate its structure. In teased preparations it is sometimes seen projecting from the free ends of the broken nerve fibre, from which the myelin has escaped (Fig. 207). When examined under a high power, a fine, longitudinal striation can be seen. It consists of extremely fine fibrils (axis fibrils), embedded in an interfibrillary substance (Schiefferdecker's axoplasm, Kaplan's axostroma, Apáthy's perifibrillary substance).

The axis cylinder is surrounded in its fresh state by a homogeneous and very delicate *medullary sheath*. After death, or in the various methods for demonstrating the structure of the fibre, this sheath shows a double outline. The medulla or myelin of the nerve escapes at the point where the fibre is torn, in the form of myelin droplets. With osmic acid it stains a deep black, like fat. In the isolated fibre also, the myelin is so changed by the process of preparation or by the effect of fluids used for staining, etc., that it appears split up and notched in various ways.

The *sheath of Schwann* (or neurilemma) is an elastic, structureless membrane, lying in immediate contact with the medullary sheath. It contains at various points on its inner surface, oval nuclei (*n*, Fig. 208), which stand out from the medullary sheath.

These nuclei on the sheaths of Schwann, which are known as neuroblasts, are supposed by some investigators to play an important part in the origin and regeneration of nerve fibres (see below, however).

There are in the fibres, in addition to the numerous notches already mentioned, constrictions of two kinds, viz., (1) Ranvier's nodes. These occur at regular intervals. At these points the myelin is more or less completely absent, so that the sheath of Schwann is drawn in all round the fibre and seems to surround the axis-cylinder directly (Fig. 208). In the segment lying between two such notches, and just about half-way in it, a nucleus is found. It is thought that Ranvier's nodes represent the points at which the nutritive substances pass inwards. On the other hand, it has been suggested that they serve for isolation (Bethe). (2) Lantermann's clefts. These are numerous fine fissures in the myelin, lying obliquely to the longitudinal axis. They are probably antefacts.

The non-medullated nerve fibres (grey or Remak's fibres) are found in the sympathetic and olfactory nerves.

Transverse section of the peripheral nerves (Fig. 209) shows an arrangement of numerous bundles of fibres. Each bundle is surrounded by a layer of connective tissue, which consists of concentric laminæ (perineurium). Within this bundle the closely compacted fibres are held

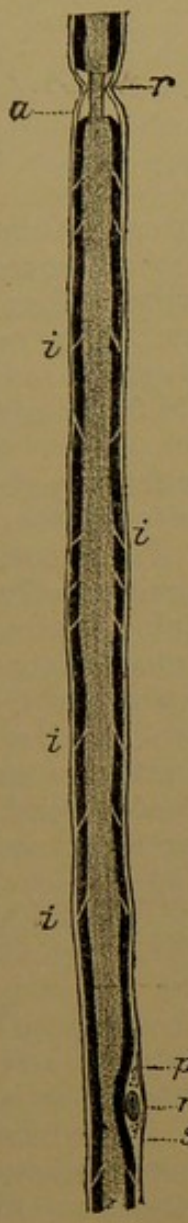

Fig. 208.—Medullated nerve-fibre. *a*, axis-cylinder (in centre). *s*, sheath of Schwann. *n*, nerve-nucleus. *p*, fine granular substance at the poles of the nuclei. *r*, Ranvier's nodes. *i*, *i*, Lantermann's clefts. (After Schwalbe.)

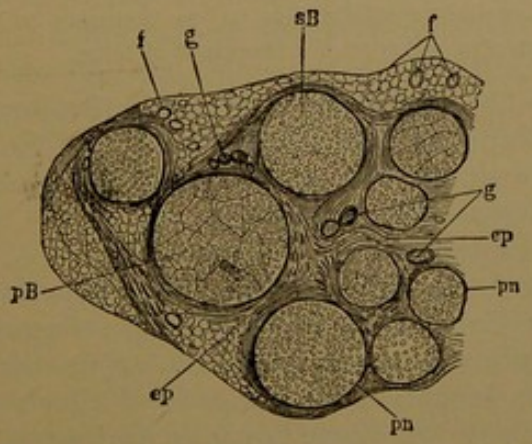

Fig. 209.—Transverse section through a nerve-trunk. pn, perineurium. ep, epineurium. sB, secondary bundle. pB, primary bundle. f, fat. g, vessels. (After Seeligmüller.)

together and gathered into other smaller bundles by the endoneurium. An outer connective tissue investment—the epineurium—unites the bundles enclosed in their perineurium into a nerve trunk. It is usually permeated by fat cells, and contains the bloodvessels which

send branches through the perineurium into the interior of the nerve bundle. The perineurium and endoneurium contain the lymph tracts of the nerves which communicate with those of the central organs (cord and brain).

PERIPHERAL PARALYSIS

Traumatic Paralysis of the Peripheral Nerves

Injuries which affect a peripheral nerve lead to a more or less complete interruption of its conduction, and thereby to disorders of movement, of sensation, of reflex excitability, and of vasomotor and trophic functions.

The effects of pressure, stabbing, bruising, section, etc., differ only in degree. Traction on the nerve may also affect it more or less severely, and may produce transient or permanent disturbances of function (Weir Mitchell).

If slight pressure is exercised on the nerve for a short time, about a quarter-minute, the myelin may be injured at the site of the compression, whilst the axis-cylinder remains unaffected. The function is then as a rule rapidly restored. It has been shown experimentally, and also in a case of compression paralysis of the musculo-spiral nerve, examined clinically and pathologically by Dejerine-Bernhardt, that the organic changes at the point of pressure may be very slight and limited to the myelin sheath If the compression is severe or prolonged, radical changes take place in the nerve, which are not merely limited to the site of the compression, *i.e.* to a localised disintegration of the medulla and axis-cylinder, but which extend to the whole of the distal section of the nerve.

That a nerve is not always severely affected, even in prolonged compression, is shown by the remarkable rapidity with which its function may be restored after operation to free it from the compression of a callus or of a fragment of bone.

These changes are most evident when the nerve has been completely divided and re-union prevented. The divided ends retract, and the direct result of the trauma, viz., *traumatic degeneration*, affects the stump of the peripheral as well as that of the central segment. But whilst the severe changes in the central segment of the nerve are limited practically to the stump, and consist in degeneration or atrophy of the nerve fibres, with the formation of connective tissue, and eventually of a neuroma, the peripheral segment of the nerve undergoes degeneration throughout its whole length, according to Waller's law. Within two to four days *the myelin of the nerve becomes disintegrated* throughout its whole extent into irregular masses and granules, then into droplets and globules. The breaking up of the myelin is followed by disintegration of the axis cylinder, whilst the nuclei of the sheath of Schwann increase in size and in number. According to Ranvier this nuclear proliferation is a primary process, the disintegration of the myelin being a result of it. Other writers, such as Mönckeberg and Bethe, think that the degeneration originates in the primary fibrils of the axis-cylinder. The products of the disintegration are gradually absorbed, the sheaths of Schwann are filled with new-formed nuclei and the remains of the debris of the myelin, whilst changes

take place in the epineurium and perineurium which, in combination with those of the nerve, ultimately produce a real *cirrhosis* of the peripheral segment of the nerve. The intramuscular ramifications of the nerve and the end-plates (Gessler, Ranvier, Weiss) also degenerate.

Batten has found that the first changes after section of a nerve are in the nerve endings in the muscle, whilst others think that the degeneration progresses in a centrifugal direction. The condition of the so-called muscle spindles and other nervous apparatus in the muscles, which apparently have sensory functions, cannot be discussed here, the results of various investigations (Horsley, Batten, Russell, Ruffini, Marinesco, etc.) being so contradictory.

The changes in the central segment of the nerve and even in the nucleus which have been recently discovered are so insignificant, both in intensity and importance, that we may disregard them here. Nor can we discuss the degenerations in the nerves and spinal cord found after amputation. We would refer to what has been already said with regard to the theory of so-called retrograde degeneration (p. 124).

Rumpf (*A. f. kl. M.*, 1903) has investigated the chemical changes in degenerated muscles.

The degenerative processes are not, however, confined to the nerve and its ramifications; they also extend to the muscles. The primary fibres become smaller and lose their transverse striation, their contents dissolve into granules, and there is a wax-like degeneration, with proliferation of the nuclei of the sarcolemma and the internal perimysium. The muscles appear to the naked eye at first pale-red, then yellow; they become markedly atrophied, and finally show a condition of atrophy with formation of connective tissue, if regenerative processes in the nerves have not in the meantime led to restoration of conduction.

Regeneration takes place the more easily the less complete the interruption of the continuity of the nerve. In simple temporary constriction of the nerve by ligature Leegard found restoration of conduction on the thirtieth day. Clinical observation also shows that a paralysis due to compression frequently disappears with astonishing rapidity after removal of the compressing agent (neurolysis, etc.), (Busch, Wölfler). Even after complete section, regeneration may occur if the divided ends are not separated from each other or are directly re-united (nerve-suture), but this usually requires a long time. The investigations of Waller, Ranvier, Vanlair Büngner, and Stroebe have shown that the idea which formerly prevailed, that in these cases restoration of conduction took place by first intention, from simple union of the two ends of the divided nerve, without any previous disintegration or new formation of fibres, must be abandoned.

According to the theory which prevailed until lately and is not yet entirely refuted, the regeneration always arises from the central stump, from which new fibres and new-formed axis-cylinders spring, making their way through the cicatrix and growing into the peripheral stump (neurolisation of Vanlair). This latter, however, merely plays the passive part of a conducting path. This view, which has been frequently opposed in recent years, explains the facts demonstrated by Vanlair, Gluck, Assaky, etc., that in traumatic and operative lesions of the nerves the loss of substance can be made up or bridged over by the interposition of an indifferent material, *e.g.* a decalcined bone tube (tubular suture), a bundle of catgut (suture à distance), etc. The new-formed nerve fibres which are sent out as buds from the central stump, use the substance introduced as a bridge or conducting path in order to reach the peripheral section.

Objections have, however, recently been raised against this theory by various writers, amongst whom we may mention Ziegler, Büngner, Wieting, Kennedy, Ballance-Stewart, and especially Bethe. They maintain that their investigations prove that the new axis-cylinders do not arise from the old, but from a protoplasm formed by proliferation of the nuclei of the sheaths of Schwann. Wieting thinks that they develop in connection with the old axis-cylinders and under the influence of stimulation from the centre. According to Kennedy, Bethe, Modena, and others, young axis-cylinders are formed in the peripheral portion as well as in the central stump, even when there has been no union of the sectioned ends. These young, new-formed fibres only mature and become capable of conduction when the two ends are united by suture. Attention is also drawn to this by Ballance and Stewart. Bethe is most firmly convinced that regeneration may take place independently in the peripheral segment of a sectioned nerve when the impulses from the centre are completely shut off, but his interpretation of his results and his theory are challenged by Langley-Anderson, Mott-Halliburton, Münzer, Medea, Ramón y Cajal, Lugaro, Marinesco, Besta, and others, whilst Lapinsky, Zander, and Modena take up an intermediate position.[1] The question cannot therefore be regarded as solved, although Harrison's [2] valuable experimental investigations show it to be very probable that the old theory of the central origin of regeneration is the correct one. Clinical facts do not always appear to be in full agreement with the old view as regards regeneration founded on animal experiments, since after suture of a nerve its function is in some cases so speedily restored that the regenerating fibres could not have had time to reach the peripheral end of the nerve (Nélaton, Langenbeck-Nicaise, Gluck, Monod, Bardenheuer, Reclus, Chaput, Reynier). In the majority of such cases, it is true, it was the sensory conduction that was restored, and it has been thought possible to give another explanation of this (see below). This return of sensibility was, however, frequently found within a few days after the union of the sectioned ends, even when the interruption of continuity had been in existence for months or even for over a year (Kennedy, etc.). These facts can hardly be explained otherwise than by the assumption that the nerve suture had created the conditions for the development within a few days of power of conduction in the nerve, a process which is most easily explained by the theory of Kennedy and Bethe. An observation of Durant's also supports this view. Other attempts at explanation have been made (assumption of unusual

[1] Of the very abundant recent literature on this point we may mention: Büngner, *Zieglers Beiträge*, x.; Kennedy, *Philos. Trans. Roy. Soc. London*, 1897; Stroebe, *Zieglers Beiträge*, xiii.; Bethe, *A. f. P.*, xxxiv.; *ibid.*, *N. C.*, 1903, 1904; *ibid.*, *Allg. Anat. u. Physiol. d. Nervensyst.*, Leipzig, 1903; Bethe-Mönckeberg, *A. f. mikr. An.*, Bd., liv.; Ballance-Stewart, *The Healing of Nerves*, London, 1901; Gluck, "Nervenplastik" (Greffe nerveuse), *B. k. W.*, 1903; *ibid.*, *Z. f. diät. Ther.*, ix. (refers to his earlier works); Münzer, *N. C.*, 1903, 1904, 1905, etc.; Münzer-Fischer, *N. C.*, 1906; Henriksen, *Nord. Med. Arkiv.*, 1903; Mott-Halliburton-Edmunds, Regeneration of Nerves, *Proc. Physiol. Soc.*, March 1904; Langley-Anderson, *Journ. of Physiol.*, xxx. (1904); Kennedy, *Brit. Med. Journ.*, 1904; Braus, *D. m. W.*, 1904; Wieting, *Zieglers Beiträge*, xxiii.; Durante, *Nouv. Icon.*, 1904; Modena, *Obersteiner*, xii. (1905); Lapinsky, *V. A.*, Bd. clxxxi. (1905); Ludlum, *Rev. of N.*, 1905; Head and Ham, *Journ. of Physiol.*, 1905; Perroncito, *Arch. ital. di Biol.*, 1905; Segale, *Rif. med.*, 1905; Lugaro, *N. C.*, 1905; Marinesco, *R. N.*, 1905, and *Journ. f.-Psych.*, vii. (1906); Zander, *D. med. W.*, 1906; Medea, *Riv. speriment.*, 1906, *R. n.*, 1906; Ramón y Cajal, "Mecanismo de la Regeneracion," *Trab. del. Lab.*, iv., 1905, 1906. See also Bethe's latest communication in *A. f. d. ges. Phys.*, 1907.

[2] Further Experiments in the Development of Peripheral Nerves, *Amer. Journ. Anat.*, 1906; ref. in *N. C.*, 1907.

anastomoses, etc., Gluck and others), and Head (see below) has specially shown that investigations must be very thorough and critical in order to show the real condition of the sensibility and the return of sensory function after section of the nerve. The fact that return of the motor functions is usually so much longer deferred is not surprising from this point of view, as the muscles are atrophied and require a considerable time for their regeneration.

The views of various writers differ greatly on this point. Whilst some report rapid recovery within three to six weeks, especially after the nerves in the distal part of the arms have been sutured, Head has proved that after section and primary suture of the ulnar nerve, for instance, the power of movement only begins to return on an average after 346 days, and recovery is only complete in one to two years. Here also there are many contradictions to be explained. It is quite in harmony with the earlier view that restoration of function is generally slower and more incomplete the nearer the site of the injury lies to the centre. Etzold, for instance, found symptoms of incomplete regeneration appearing years after section of the plexus in the axilla, whilst in several cases of section of the nerves in the upper and fore-arm, and especially above the wrist-joint, there was return of movement and even complete recovery within three to six weeks after suture (Langenbeck, Tillmanns, Bruns, Wölfler, Schüller, Kölliker, Schede, Mader, Bardenheuer, Taylor, Chaput, etc.). Bruns also (*N. C.*, 1902) found that a plexus-paralysis showed much less tendency to recovery than paralysis of the peripheral nerves. This is contradicted by the experience of Thorburn and others.

The younger and stronger, the more " vital " the individual, the more easily do the nerves regenerate (Raymond).

With the degenerative changes in nerves and muscles there are corresponding changes in electrical excitability, as described on p. 41, the *reaction of degeneration*, the course of which has been minutely investigated by Erb, Ziemssen, and Weiss, in experiments on animals. After very transient increase the excitability of the nerves diminishes for both currents from the second day onwards, and is entirely abolished within the second week (at latest on the twelfth day). Whilst the muscle does not respond to the faradic current during this period, there is an evident increase of the excitability for the galvanic current during the second week, with the changes in the mode and in the formula of contraction which are characteristic of reaction of degeneration. This increase may be so great that, according to E. Remak, a current of the twentieth part of the usual intensity is sufficient to elicit a contraction (0,1 M.A. on the affected side against 2 M.A. on the healthy side). If regeneration does not take place, the direct galvanic excitability gradually diminishes, commencing usually after three to nine weeks, whilst the sluggish A.C.C. may be detected by strong currents one or more years later.

In general, the slighter the lesion of the nerve, the less is the affection of the electrical excitability. It may remain normal or be but little diminished when the compression is slight, and even when it is sufficiently severe to cause complete paralysis. A slight increase has even been observed in some cases where the lesion has been very insignificant. But electrical stimuli applied *above the site of the lesion* have as a general rule no effect in such cases. In injuries of medium severity there is frequently an incomplete or partial reaction of degeneration. It may be noted further that an unequal response of the nerves to the two kinds of current has been observed in rare cases, viz., diminished faradic and normal or even increased galvanic excitability.

There are a few cases which do not entirely accord with the above laws; thus Ballance and Stewart found that the electrical excitability was not abolished in the exposed peripheral stump of the facial, even a considerable time after section.

The *symptoms* caused by injuries of peripheral nerves depend not only upon the severity of the lesion, but also upon its site and the nature of the nerve affected. Lesions of *mixed* nerves are of course most common, and they may serve by way of example. The *motor functions* are constantly, or almost constantly affected. The muscles which derive their motor nerves from the portion of the nerve distal to the lesion are more or less completely paralysed. If they are innervated also by branches of another nerve, the paralysis may be absent or incomplete.

In pressure of gradual onset and progress the distal muscles of the corresponding nerve area may show paralysis and atrophy before the proximal muscles (E. Remak).

The paralysis is always *flaccid*, and the *tendon reflexes* of the affected muscles are diminished or absent. The paralysis is rapidly followed by *atrophy*, which is only absent when the paralysis is very slight. Even before this becomes visible, the degeneration may be revealed by electrical examination.

Anæsthesia is by no means so constant as paralysis. It has been experimentally proved (Lüderitz) that when pressure is gradually exerted the interruption of conduction appears in the motor earlier than in the sensory fibres; sensibility may even be retained when all power of movement is absolutely lost. It is a still more remarkable fact that even in section of sensory and mixed nerves the *sensory symptoms may be absent or may rapidly disappear*, or may be limited to a much narrower area than we should expect from the anatomical conditions. This is chiefly due to the numerous anastomoses which connect the peripheral ramifications of the sensory nerves to each other, many cutaneous areas thus receiving their innervation from different nerves. There may also be anastomoses, though rare and inconstant, in the nerve trunks, as in the nerves of the fore-arm (Tessier, Gegenbaur, Létiévant, etc.). The expression "collateral innervation" has been used to imply that after section of a nerve the stimulation from the peripheral branch may pass through an accessory branch into a neighbouring nerve, and may reach the main stem of the injured nerve higher up (*i.e.* above the site of the lesion) through a second lateral branch. The fact that after section of a nerve, the median for instance, its peripheral ends are still sensitive, is due to recurrent fibres which originate in a neighbouring sensory nerve (recurrent sensibility of Arloing and Tripier, Laborde, Vanlair, Létiévant). The following explanations have also been suggested: that nerve fibres grow from the healthy surroundings into the insensitive parts (Schuh); that the sensory stimulation also excites the neighbouring touch-corpuscles of the intact nerves; that the sensory fibres run in the deep parts of the nerves, so that they suffer less in trauma which affects the nerve superficially (Viannay[1]), etc. Finally, it has been supposed that the sensory fibres are more capable of resistance and of regeneration. Leegard disagrees with this view, and maintains that the integrity of a few fibres suffices for sensory conduction.

All these opinions have quite recently undergone an important change owing to the exceedingly valuable communications of Head (Head and

[1] "Les paralysies des nerfs périphériques et la systématisation des ces nerfs." Paris, 1905. Franceschi opposes his views.

Sherren, *Br.*, 1905; Head and Thompson, *Br.*, 1907), who sectioned a nerve in his own body in order that he might closely observe the results, and confirmed them by investigation of many cases of injury. He has shown that when the sensibility was tested minutely with fine methods of examination, anæsthesia is never completely absent after section of a peripheral nerve, nor is there ever a rapid restoration of all the forms of sensibility after the nerve has been sutured. Contradictory results and errors of opinion are due to the fact that after section of a sensory nerve, pressure and any deep stimulus are felt, because the deep sensibility is communicated by fibres which do not run along with the cutaneous nerves but accompany the muscle nerves, within the muscles, tendons, fasciæ, etc. Tactile sensibility must therefore be tested with the finest touch—cotton wool—all pressure being avoided; in the same way extreme temperatures must not be used in testing the temperature sense. Head succeeded in showing after section of a sensory nerve, (1) that the deep sensibility is not affected, (2) that the analgesia and total thermo-anæsthesia are limited to a smaller area than that corresponding to the anatomical distribution of the nerve, (3) that there is a more extensive zone in which (*a*) slight touch is not felt, (*b*) the power of localisation, of differentiating between the points of a compass, is lost, and (*c*) the power of distinguishing temperatures between 20° and 40° C., and of indicating whether they are warm or cool, is absent. Further, whilst the area of complete analgesia and thermo-anæsthesia soon diminishes, and this marked disorder of the *protopathic* sensibility, as he calls it, disappears within a few weeks, the disturbance of the fine (*epicritic*) sensibility persists for a considerable time (on an average about a year, but it may be two or more), even after the nerve has been sutured. He gives exact data as regards these points in the various nerves. Head concludes that the theories of the formation of anastomoses and the co-innervation and compensation thus produced apply only to the fibres which serve to conduct pain (and extreme temperatures), whilst this compensation does not apply to the epicritic sensibility; they must, therefore, be connected with quite different fibre systems.

He has also pointed out that in the zone in which epicritic sensibility alone is abolished, protopathic sensibility is retained or recovered, the prick of a needle and strong thermal stimuli causing a severe, pricking sensation, which, however, is diffuse and not sharply localised.

When the nerve is incompletely divided, recovery takes place as a rule much more rapidly, and without these marked differences in the condition of the protopathic and epicritic sensibility. He gives further interesting data as to the relation of trophic and secretory disorders to analgesia and thermo-anæsthesia. The nearer the point of section to the posterior root, the more extensive is the area of the disturbance of the protopathic sensibility. He finally shows, especially in his most recent work, the great differences in the condition of the disorders of sensibility in diseases of the spinal cord as compared with those in lesions of the peripheral nerves, as in the former a distinction between protopathic and epicritic sensibility is impossible. If, for example, there is thermo-anæsthesia, the sensibility for extreme as well as medium degrees of temperatures is involved (except that the intensity of the disturbance of course varies here also); if there is analgesia, it affects also deep stimuli. The parallelism between tactile sensibility and the condition in testing with the points of a compass is lost, the latter rather corresponding to the disturbance in the sense of position, etc. It is only in spinal diseases that the sensibility for heat is lost and that for cold conserved.

So far, there has hardly been any opposition to Head's theory. Dana alone maintains that it does not apply to the cranial nerves (*Journ. Nerv. and Ment. Dis.*, 1906).

It has recently been shown that there are considerably more anastomoses in the motor

nerves than has hitherto been suspected, so that in lesion of a motor nerve, muscular paralysis sometimes fails to appear or is rapidly recovered from, as the muscles receive impulses of innervation from another nerve by means of anastomoses (Bardeleben, Frohse, Marenghi). A remarkable observation by Goldmann (*N. C.*, 1906) may be interpreted in this way, but the writer has stated facts (*Bruns Beitr.*, Bd. li.), which do not allow of this explanation There are other isolated and hitherto unexplained cases in which section of mixed nerves was not followed by any essential disturbance of function.

Leaving out of account a few cases of injury of a mixed nerve (specially of the median) in which the sensibility was mainly affected, it is a rule that severe, persistent, and widespread anæsthesiæ result only from trauma which make several nerve trunks or a whole plexus incapable of conduction. In lesion of a single nerve there are either no pronounced affections of the sensibility, or if these be present they quickly disappear or are limited to an area smaller than one would have expected from the anatomical distribution of the nerve.

Injuries of sensory or mixed nerves, however, give rise almost constantly to *paræsthesiæ* and often to *pain*. These occur specially when the nerve is incompletely divided. The paræsthesiæ and feeling of numbness are frequently, though not always accompanied by a detectable diminution of sensibility ; less frequently by a total anæsthesia. This hypæsthesia involves some or all of the qualities of sensation. According to Herzen and Goldscheider, in cases of compression of mixed nerves the sense of cold and pressure suffers first, the sense of warmth and pain being subsequently affected.

Vasomotor and *secretory* disorders are often caused by lesion of peripheral nerves. Redness is common, as well as a local rise of temperature, which may subsequently become a fall. The hyperæmia usually gives place later to *cyanosis*. *Œdema* is rare ; there is often local *hyperidrosis* or *anidrosis*. *Trophic* disorders occur in the skin, less often in the bones and joints. The skin is sometimes *smooth*, *glossy*, very thin and tender, so that the slightest injury may cause ulceration. Small vesicles which burst and leave *ulcers* that are difficult to heal are occasionally observed. " Perforating ulcer " may be traced back in many cases to a traumatic neuritis of the sciatic nerve or its roots (Fischer, Sonnenburg, Sattler, etc.). Atrophy of the subcutaneous tissue, inhibition of growth, or even atrophy of the bones are less common. Trophic troubles in the bones have been discovered by the X-rays within the last few years (Sudeck, Oppenheim, Hirsch, Goldscheider, etc., and also Fürnrohr, " Die Röntgenstrahlen im Dienste der Neurologie," Berlin, 1906). In paralysis of the roots and plexuses occurring in childhood the bones may cease to grow (Guillain). The growth of the hair and nails may also be affected, but Head attributes the affection of the growth of the nails to other factors, especially to deficient movement. I have very often found these disorders of nutrition in partial lesions of the peripheral nerves which were associated with severe pain and slight paralytic symptoms, *e.g.* in injury of the median or ulnar nerve by a splinter of glass. In a few cases these formed the only sign of the nervous affection. In one case they developed a year after the injury and in another still later, perhaps under the influence of some traction.

Symptoms of motor irritation do not play an important part in injuries of the peripheral nerves. Fibrillary tremors hardly ever occur. Muscular spasms, which sometimes appear, are probably of reflex origin, for many morbid symptoms, especially in lesions of the peripheral nerves, may

be produced by reflex means (see chapter on Reflex Epilepsy, Traumatic Neuroses, etc.). We must finally mention the secondary changes, viz., secondary contracture, atrophy of the capsular and articular ligaments, etc.

With regard to the *differential diagnosis* we must take into consideration *direct traumatic muscular paralysis*, and *muscular atrophy due to contusion of the joints* (in both of which there is merely quantitative diminution of the electrical excitability, the sensibility being intact), and especially the so-called *ischæmic muscular paralysis and contracture* (Volkmann-Leser).

This is the name given to the muscular atrophy which develops after excessively firm application of bandages, especially to the upper extremities. There is first swelling and severe pain in the hand and fingers beyond the bandage. If it is not soon removed the pain becomes more acute and the hand and fingers show an increasing flexion contracture. Shrinking follows rapidly upon the swelling, and the muscles become stiff and hard as boards. Active movements are completely absent; passive movements are very limited and painful. The hardness and rigidity of the muscles, the absence of reaction of degeneration (there is usually merely quantitative diminution of excitability in direct muscular stimulation, indirect excitability being still present), and of severe sensory symptoms permit of the ischæmic muscular paralysis being easily distinguished from the neuritic. The investigations which Lapinsky[1] carried out in my laboratory have indeed shown that, under the influence of arterial ischæmia, it is mainly the *nerves* which suffer, and that this gives rise to a condition which is not quite that of so-called ischæmic muscular paralysis, sensory disorders and atony being also present. Although these facts are important and deserving of consideration, yet the paralytic conditions produced by pressure of bandages corresponds so closely to that described by Volkmann-Leser that they must have been produced by factors other than those employed in Lapinsky's experiments. In any case Lapinsky shows that ischæmia of the nerves is a factor not to be neglected in the paralysis caused by tight bandaging. Hildebrand [2] has also recently noted this; he draws attention specially to the compression of the nerves by the rigid muscles and considers this process from a therapeutic point of view. Riedinger also blames direct muscular compression. I have several times seen symptoms of traumatic neuritis accompanied by muscular changes which corresponded to those of ischæmic muscular paralysis, due to the fact that the corresponding *artery* was also injured and obliterated.

Hildebrand, H. Schlesinger (*Z. f. N.*, xxix.), and Kleinschmidt (*D. m. W.*, 1907) have also shown the importance of this factor with regard to the sensory nerves. The condition of the electrical excitability of the muscles after embolism is discussed by Delherm (*R. n.*, 1903).

It must not be forgotten, also, that the manifold mechanical conditions resulting from peripheral injuries (stiffness of joints, section and cicatricial adhesion of muscles, tendons, etc.) may very materially add to the difficulties of diagnosing traumatic paralysis of the nerves.

Course and Prognosis—The course depends chiefly on the severity of the injury. A simple pressure paralysis may recover in a few weeks, even in a few days. If the solution of continuity be absolute, then complete restoration cannot be expected in less than a few months or

[1] *Z. f. N.*, xv.

[2] *D. m. W.*, 1905.

one to two years. If the nerve stumps be separated and displaced, we can only expect recovery if they are re-united by artificial means. But even in simple section, spontaneous recovery seems to be a rare occurrence. It can easily be understood that when the nerves are previously affected, slight injuries may give rise to severe peripheral paralysis. It is for this reason that slight trauma is often sufficient to produce severe paralysis in persons suffering from chronic *alcoholism*, chronic *lead intoxication*, *cachexia*, and conditions of weakness following infective diseases (Oppenheim and Siemerling [1]). In these cases the paralysis is not simply a traumatic, but a toxico-traumatic one (Oppenheim [2]), etc. These facts, which we had described and proved many years ago, have lately been referred to by other writers (Guillain, d'Abundo, Edinger,[3] etc.).

Leaving out of account the results of surgical treatment, the prognosis should be determined by the *condition of the electrical excitability*. If reaction of degeneration be present, the prognosis as to recovery is doubtful, the course is always tedious, and even in the most favourable cases recovery cannot be expected in less than three to four months. It may, however, take place even after the lapse of a year. If the excitability is unaffected or but slightly diminished at the end of the second week, a rapid recovery may be anticipated, and it may be complete within three to four weeks. Slight pressure paralysis may even disappear in a few days. Partial reaction of degeneration justifies a comparatively favourable prognosis. Recovery will not be rapid, but it may be expected to take place probably within a few months. These laws are by no means absolute, however. There are isolated cases in which for instance the paralysis remains permanently, in spite of normal or but slightly diminished excitability.

Thus we found in a few longstanding cases of facial paralysis (Placzek has described these cases from my polyclinic) that the paralysis had persisted, although the electrical excitability was conserved or had returned. Placzek endeavoured to explain this condition on Erb's theory of axillary neuritis, *i.e.* destruction chiefly of the axis-cylinder with integrity of the medullary sheath. Bernhardt (*B. k. W.*, 1903) attempts another explanation. I suspect that the so-called habit-paralysis (see below) plays a certain part here.

On the other hand, it should be remembered that when the course is favourable, voluntary movements may be restored even although the nerve still fails to respond to electrical stimulation—a symptom which Erb explains by the assumption that it is only the continuity of the axis-cylinder which is at first restored by the regeneration. Bernhardt (*Z. f. N.*, xxvi.) has also expressed this opinion and drawn attention to Gombault's periaxillary neuritis. Pelnár, on the other hand, claims to have shown experimentally that these differences in the condition of the irritability and power of conduction of the nerve occur when its superficial fibres degenerate and those in the centre are conserved.

If the electrical excitability is quite abolished, the outlook is exceedingly grave.

Even in longstanding cases considerable improvement can sometimes be brought about by treatment. Thus electrical treatment proved efficacious (to a certain extent) in a case of Duchenne's after four years, and in a case of my own after about twenty years. Secondary suture of the nerve (see below) may also be successful, even after many years.

In the peripheral paralyses of childhood the prognosis is to a certain degree graver, on account of the fact that the organic paralysis may be

[1] *A. f. P.*, Bd. xviii. [2] *B. k. W.*, 1891. [3] *D. m. W.*, 1904 and 1905.

associated with a habit-paralysis (Ehret). I am personally convinced of the great part which the latter plays, especially in obstetrical paralysis (see below), and my experience has been confirmed by Huet. I have also seen similar conditions in the traumatic musculo-spinal paralysis of childhood, and I have thought it possible that a child suffering from long-standing paralysis gradually loses the memory pictures for the execution of the corresponding movements, so that the function of the nerve remains in abeyance, although its conduction has been restored.

In one such case I have seen that a movement which could not be carried out voluntarily, could be synergically performed, and kept up for a moment after it was elicited by electrical stimulation. Weakness of mind and indolence may help to produce this habit-paralysis; this at least is my experience.

Treatment.—In injuries of the nerves *great care* must be taken of the extremities, which should be kept at *rest*. The injured nerve should not be irritated by pressure and traction. Firm bandages, which compress it directly, should therefore be avoided, and the circulation should be as free as possible. Care should be taken also that the muscles through which the nerve passes or between which it lies should not be contracted. *Local antiphlogistic treatment* is only suitable when signs of acute neuritis are present.

If there is simple compression or contusion, electrical treatment is the most important measure, although its value has occasionally been disputed. In recent cases the stabile use of the cathode of the galvanic current at the point of pressure, the anode being placed on the plexus, on the muscle, or at some indifferent point, is specially recommended. The intensity of the current should be about 6 to 8 milliampères, the size of the electrode of 20 to 30 square c.m. (Remak), or an intensity at which the patient feels a subjective improvement of his power of movement. If there is reaction of degeneration, this treatment may be fittingly associated with direct stimulation of the paralysed muscles by means of "labile" galvanic currents (A.C.C.). If faradic excitability is not abolished, the faradic current may be indicated, but it is not advisable to stimulate the nerves themselves during the first stages. Duchenne has obtained considerable benefit from the use of faradisation, and has induced recovery even in inveterate peripheral paralyses. Even weak induction currents which do not elicit contractions, may, after prolonged application, increase the excitability of the nerves. It is doubtful whether it is advisable to stimulate the nerves with strong currents above the site of lesion. This method is recommended on the assumption that it may be possible to a certain extent to create a path for voluntary impulses through the affected part of the nerve.

It is always wise to *avoid strong currents* in recent cases, and to take into account the individual sensitiveness, as traumatic neuritis may be aggravated by excessive stimulation. It is only in paralysis of long standing that strong currents are suitable. *Galvano-faradisation* and *franklinisation* in the form of the static discharge may also be employed.

Electrical treatment may be combined with *massage* in the neighbourhood of the site of compression; the affected nerve itself, however, must not be mechanically stimulated. Massage is specially valuable in the later stages, in which kneading and stroking of the muscles, passive movements which counteract the contracture, and stiffness of the joints may consider-

ably help the process of recovery, assuming that it is possible to restore conduction to the nerve. Massage of the muscles should be avoided when reaction of degeneration appears in recent cases. Hydropathic bandaging, peat fomentations, local application of hot air, baths containing carbonic acid, are sometimes recommended in traumatic neuritis. Gymnastic exercises, viz., active movements in the paralysed muscles, which are passively supported, etc., are very suitable.

In cases of an open *incised* wound, the nerve should immediately be *sutured* (the stump exposed, the ends refreshed, and finally stretched and drawn together in order to make up for diastasis, etc.). The extremity must then be fixed in such a position that there is no traction upon the sutured nerve.

The statistics of Tillmanns and of Schmidt, who in 1889 collected from the literature 129 cases of nerve suture, show that operation led to a more or less complete restoration in almost two-thirds of the cases. Schede says that, when the nerve is carefully sutured, success is the rule.

Of the recent communications we may specially mention those of Kramer (*Inaug. Diss.*, Heidelberg, 1900), from Czerny's clinic, of Hector ("Die Erfolge der Nervennaht," etc., *Inaug. Diss.*, Berlin, 1901) from Bergmann's clinic, and those of Head-Sherren, Henriksen (*Norsk Mag. f. Laeger*, 1903), Nurmann (*Inaug. Diss.*, Copenhagen, 1904), Tonarelli (*Il Morgagni*, 1904), Sherren (*Lancet*, 1906), Sherren (*Edin. Med. Journ.*, 1906), Medea-Rossi (*R. n.*, 1906), Auffenberg (*A. f. kl. Chir.*, Bd. lxxxii.), Blencke, Taylor (*Journ. Amer. Assoc.*, 1907), Warrington-Jones (*Lancet*, 1906).

Sensation generally returns sooner than power of movement, but this symptom, as already stated, is not always a sign and a consequence of successful suture. As Head has shown also, it is true only as regards the coarse, protopathic sensibility. In a few cases the trophic symptoms were the first to improve. Wölfler occasionally found diminution of the temperature sense, even after the power of movement had returned. Remy noted that after suture of the median nerve, tactile stimuli were wrongly localised, although the sensory functions had been restored. Recovery is not as a rule to be expected in less than a year, although it has taken place much earlier in a few cases (see above), Nerve suture is naturally most frequently performed on the nerves of the arm, but the operation has also been successfully carried out on the nerves of the lower extremity, *e.g.* the sciatic (Cervera, Reynier), the facial (Neugebauer, Betcke, etc.), the accessory (Neugebauer), hypoglossal (Wölfler), the fifth and sixth cervical nerves (Wölfler, Kennedy, Harris-Low, Taylor, Warrington-Jones), the lower roots of the brachial plexus (Bardenheuer), etc.

If the patient does not come under treatment until a late stage, after the wound has already closed, *secondary suture* is indicated if the nerve is found to be still divided. We do not, however, possess any definite criteria by which to distinguish between a contusion causing complete interruption of conduction and section of the nerve. It is only when the paralysis and degeneration are incomplete that we can with certainty exclude total solution of the continuity (if we are not dealing with a muscle which also receives branches from another nerve). The condition of the sensibility may be considered in this respect, Head's data being taken into account. *Expectant* treatment is, however, advisable in doubtful cases, as it has been shown that nerve suture may produce regeneration

even after a period of several, indeed as many as ten to fourteen years (Tillmanns, Cervera). But since there is no danger involved in careful exposure of the injured nerve, it should not be unduly delayed. Surgical textbooks should be consulted with regard to the methods of suture of the nerves.

If a cicatrix of connective tissue or a neuroma have formed between the stumps of the nerve, they must be excised before suture. Many suggestions have been made as to compensating for the defective substance; Vanlair, Gluck, and Assaky (see above) propose union by means of indifferent material, but Schede regards this merely as a matter of theoretical interest; others (Létiévant, Phillipeau and Vulpian, Faure, Gluck) suggest the "greffe nerveuse," in which the peripheral end of one sectioned nerve is united with the central end of another, the function of which is less important (*central* implantation); or where only a single nerve has been injured, its peripheral end is grafted into the freshened margin of an uninjured nerve (peripheral implantation). This has been successfully carried out in a few cases by Després, Gunn, Horsley, Spitzy, etc., whilst Fursac and others report failures. It has of late years been specially performed between the facial and the accessory, and between the facial and hypoglossal nerves (Ballance, Faure, Kennedy, Gluck-Bernhardt, Körte, Bardenheuer, Cushing, Frazier, Taylor-Clark, Löhlein, etc.). It has undoubtedly in several of these cases led to regeneration, but the functional success is usually qualified by disturbing associated movements. Only Gluck and Bardenheuer have succeeded in obtaining a satisfactory functional as well as cosmetic result. The interesting experiments of Langley, Manasse, Bréavoine, Kennedy, Cunningham, Barrago, Floresco, etc., have created a physiological foundation for this operation. Gluck and others have performed it on the nerves of the lower extremities, as Borchardt did in one of my cases, but without success. The condition of the electrical excitability has convinced me that the nerve upon which the paralysed one is grafted is to some extent injured. Létiévant recommends union by means of nerve flaps (*autoplastie à lambeaux*), which are formed by longitudinal splitting of the ends of the divided nerve. The operation has been successfully performed by Tillmanns, Kölliker, Saenger and Sick, etc. Finally, transplantation from animal nerves has also been advised (Gluck, etc.). Peterson has lately collected cases of this kind, and has related an interesting case of his own.

With regard to the literature of nerve-grafting, etc., see Bréavoine, *Thèse de Paris*, 1901; Faure, *R. n.*, 1903, *Presse méd.* 1905; Gluck, *B. k. W.*, 1903, and *Z. f. diät. Ther.*, ix.; Körte-Bernhardt, *D. m. W.*, 1903; Cushing, *Ann. of Surg.*, 1903; Frazier-Spiller, *Univ. of Penn.*, 1903; Ballance-Stewart, *Brit. Med. Journ.*, 1903, *Br.*, 1904; Munch, *Sem. méd.* 1904; Langley-Anderson, *D. m. W.*, 1904; Bardenheuer, *D. m. W.*, 1904; Zesas, *Fortsch. d. Med.*, 1904; Horsley, *Journ. Amer. Assoc.*, 1906; Sick, *D. m. W.*, 1905; Spitzy, *W. kl. W.*, 1905; *D. m. W.*, 1906; Taylor-Clark, *Med. Rec.*, 1904; Chaput, *R. n.*, 1905; Warrington-Jones, *Lancet*, 1906; Head-Sherren (see above); Bernhardt, *Mitt. aus Grenzg.*, xvi.; Alt, *W. kl. R.*, 1906.

From the foregoing analysis it is obvious that secondary suture of the nerve is a justifiable, and in many cases an efficacious operation. Great defects in the nerve of course materially affect the result. According to Wölfler success is uncertain if the interval between the two ends extends over 4 cm. Some surgeons, such as Trendelenburg and Rotter, have made suture possible in such cases by artificially shortening the extremity

by excision of a piece of bone, from the humerus, for instance. Secondary suture has also been successfully employed in a few cases of neuroma after the tumour has been removed (Monod, Bruns-Kredel, Lambotte-Sano, Péan, Goldmann, etc.).

At other times it may be necessary to remove foreign bodies, especially fragments of bone, which press upon the nerve, to free it from a cicatrix or an exostosis, or to excise a callus (*neurolysis*). In cases of this kind the symptoms of nerve lesion appear only some time after the injury, and commence with pain and paræsthesia, followed by atrophy, paralysis, and finally anæsthesia. A few cases under my observation (see E. Weber[1]) proved that the callus of the nerves may become dangerous many years after a trauma, should some fresh injury occur (traction, overstrain). In a case of Neugebauer's the callus paralysis appeared nine years after the fracture; in one described by Guillemain-Mally the ulnar nerve became paralysed twenty-six years later.

A few cases of this kind have been previously described, by Panas (*Arch. gén.*, 1878), Seeligmüller, etc. Mouchet, Vennat, and others report similar observations. I have also seen cicatrix left in the axilla after extirpation of mammary cancer affect the nerve trunks three years later, a degenerative neuritis being brought on by an abrupt movement. This is an important fact as regards diagnosis.

If the symptoms do not improve under conservative treatment, especially electrical, neurolysis is indicated. I have seen a case in which non-surgical treatment of paralysis of the radius commenced one and a half years after the injury, *e.g.* galvanism, massage, applications of hot air, induced recovery, although the paralysis had been complete and absolute until it was begun. Neurolysis should not therefore be too hastily undertaken. Busch saw a paralysis of sixteen years' duration cured by its means. It is remarkable that in this, as in other cases of the kind (Schede, Wölfler, Neugebauer, André, Bräuninger, Kennedy, etc.), improvement commenced immediately after the operation, which would show that in this compression-paralysis the axis cylinders are not completely destroyed. In a few cases relapse was caused by the nerve again becoming adherent to its surroundings; it is advisable therefore to envelop it with epidermis, fat, and so on. Several months or longer elapse before recovery is complete. The return of electrical excitability may take an even longer time (Neugebauer). Radiography may be useful in locating the site of the lesion, if the callus is not distinctly palpable and visible (Oppenheim).

If a dislocated joint presses on the nerve, the dislocation should be immediately reduced. I have seen a case where this had been delayed until the third day, and an exceedingly severe plexus paralysis was the result.

After suture of the nerve, the process of recovery may be aided by electrical treatment. One must not get discouraged quickly; the electrical applications must be continued until convalescence is quite complete, even should this require a year to accomplish. The functional disorders due to the paralysis may be partly adjusted by supporting apparatus (*e.g.* in musculo-spiral paralysis).

Finally, for the treatment of old-standing paralysis, muscle transplantation (see p. 247) is recommended. This has led to excellent results in several cases of musculo-spiral paralysis (Francke, W. Müller, Gönczy).

[1] *Z. f. N.*, xv.

Neuritis, Inflammation of the Nerves[1]

Inflammation of nerves may arise from the perineurium and be practically limited to it (*perineuritis*); it may be situated mainly in the interstitial tissue (*interstitial neuritis*), or within the nerve fibres (*parenchymatous neuritis*). The first two forms correspond to the type of a *true inflammatory process*, the latter is identical with *degeneration of the nerve* and causes changes similar to those we have learned to recognise as secondary to section of a nerve or secondary degeneration (Waller). A sharp distinction cannot, however, be made between these different forms, as inflammation arising from the perineurium and endoneurium usually involves the nervous parenchyma also, and primary degenerative neuritis is almost always associated with changes in the connective apparatus of the nerves. It is obvious that no definite distinction can be made between the different forms as regards their symptoms.

Anatomy.—Acute perineuritis is characterised by *redness* and *swelling* of the connective tissue surrounding the nerves. These may sometimes show a *fusiform swelling*. The vessels of the nerve sheaths are dilated and distended with blood. Small hæmorrhages are less common. Serous transudation, or emigration of the white blood corpuscles, results from the hyperæmia. The local swelling is at first due to the exudate; later circumscribed proliferation of connective tissue may cause a *nodular swelling* of the nerve at one or more points (*neuritis nodosa disseminata*). These changes are seldom confined to the perineurium; the intervening tissue also undergoes inflammatory infiltration, though in less degree, and, if the perineuritis is severe and prolonged, the nerve fibres are also involved.

Interstitial neuritis is due to similar changes in the connective tissue. It also not infrequently causes local swelling and practically always involves the nerve fibres. It is only the slightest forms of perineuritis and interstitial neuritis which do not markedly affect the nervous tissue itself.

Parenchymatous or *degenerative* neuritis, *i.e.* atrophy of the peripheral nerves, is the most common form. It may follow the processes described above, or may have a primary onset. The histological changes have been already referred to.

Gombault's periaxillary segmental neuritis is an exceedingly slight form of nervous degeneration, in which the myelin of the nerve is disintegrated only in a few segments of the nerve fibre, whilst the axis-cylinder remains intact (Fig. 210). Gudden regards it as a process of restitution. It has been found in toxic and infective conditions. Stransky[2] has lately devoted careful attention to these discontinuous processes in the peripheral nerve fibres.

The *causes* of neuritis are very numerous. We have referred to the traumatic origin in the preceding chapter. The exciting cause of neuritis may be not only direct injury of the nerve, but also contusion, traction, or compression in forced muscular movements. Dislocated joints, bone callus, exostoses, tumours, cervical ribs, etc. etc., produce

[1] Amongst the recent literature we would specially mention Bernhardt, "Die Erkrankungen der periph. Nerven in Nothnagels Handbuch der Spez. Path. u. Therp.," 2nd ed., 1904; and E. Remak-Flatau, "Neuritis und Polyneuritis," *ibid.*, Bd. xi., 1900. As regards treatment see Goldscheider in the "Handbuch der physikal. Therapie," Bd. xv.

[2] *J. f. Ps.*, i.

FIG. 210.—(After Stransky.) Periaxillary neuritis, discontinuous process of disintegration, etc. Osmic acid stain.

various conditions of inflammation and degeneration by causing injury to the nerves. It is proved also that less severe compression of the nerve may, when repeated, cause neuritis. Thus we have, for instance, the so-called *crutch paralysis* and *occupation neuritis*, in which constant pressure of the shaft of a hammer or an iron, a gimlet or other instrument, upon the nerves of the hand produces inflammation in them (see chapter on Occupation Paresis).

Over-strain is also a causal factor. Its importance is specially shown by the cases of Auerbach, Edinger, Knapp-Thomas, and Scheffer (*W. kl. R.*, 1903). I have seen a case, for instance, in which no cause for a peroneal paralysis could be discovered except over-strain in working a sewing-machine. Infection, intoxication, and marasmus are of course peculiarly apt to make the nerves sensitive to this influence.

Chemical agents introduced near the nerve may cause inflammation; this has been proved as regards ether (subcutaneous injection), osmic acid, etc. Paralysis following lumbar anæsthesia, which usually affects the abducens, may be explained in the same manner.

The influence of *chill* has hitherto been decidedly over-estimated, but we are not justified in entirely ignoring it in the etiology of neuritis.

Infective diseases are of much greater importance. During and after these, especially in typhoid, small-pox, diphtheria, tuberculosis, syphilis (syphilitic neuritis specially affects the ulnar nerve or corresponding roots), neuritis may develop in its localised as well as its multiple form. *Puerperal* neuritis is usually a polyneuritis.

Chronic intoxication (alcohol, metallic poisons, nicotine) plays a similar part, but it usually produces, except in cases of toxic neuritis of the optic nerve, an inflammation or degeneration involving a number of nerves. Poisoning by *carbonic oxide*, meat-poisoning,

etc., intoxication from bisulphide of carbon (possibly also benzin, phosphoric acid salts, sulphonal, etc.) may also produce neuritis.

Infection and intoxication produce a condition within the peripheral nerves so that slight injuries, which would not affect healthy nerves, may give rise to a neuritis. This sensitiveness is due to slight and to some extent latent disorders of nutrition in the nerves, which give rise to symptoms only when a fresh agent increases the slight inflammation or degeneration into a severe one (Oppenheim and Siemerling [1]).

Among the causes we must also include *rheumatism*, *gout*, *diabetes*, and *leucæmia*. It appears from an interesting observation by Cassirer and Bamberger,[2] that peripheral neuritis may also develop in pentosuria. It may in some cases be attributed to vascular diseases (obliterating arteritis, arteriosclerosis), (Oppenheim, Gombault, Joffroy, Schlesinger, etc; Lapinsky reports corresponding pathological data). It has also been noted in diseases of the veins (Quenu). On the other hand, diseases of the vascular system have been attributed to neuritis (Lapinsky, Moltschanoff). Jores questions the value of Lapinsky's investigations. We have also to remember that it may develop in the course of carcinomatous cachexia (Oppenheim-Siemerling, Gombault, Oberthür, Klippel, etc.), and in old age (Oppenheim-Siemerling, Elsholz, Sternberg, etc.).

Inflammation may extend to the nerves from adjacent organs, *e.g.* to the facial in caries of the petrous bone, to the intercostal nerves in caries of the vertebræ and ribs, etc. Inflammation of the joints, acute and chronic, may also spread to the adjacent nerves. This happens but rarely, however. The *neuritis ascendens* or *migrans*, which plays a great part in earlier lierature, is also rare. Experimental investigations have shown that an artificially produced suppurative inflammation of the nerves may ascend along the nerve and may advance intermittently in a centripetal direction (Kast and Rosenbach). Homén and Laitinen [3] have also proved experimentally that streptococci and their toxins introduced into the nerve sheaths may make their way along the nerves and spinal roots to the spinal cord. Marinesco has explained the condition found in a spinal cord in a case of gangrene in this way. As regards simple, non-suppurative inflammation, however, this mode of spread, has not yet been sufficiently proved, although isolated clinical observations make it probable that neuritis may penetrate in the central direction from the site of its origin continuously or in the form of disseminated foci. Thus it is that inflammation caused by a wound on the finger may extend to the nerves of the arm, in which nodular swellings can then be felt. This is, however, much less common than the earlier neurologists believed [4] (especially R. Remak). Kausch, Krehl, Gerhardt, Chipault, E. Remak,

[1] *A. f. P.*, xviii. [2] *D. m. W.*, 1907.

[3] See Homén: "Die Wirkung der Streptokokken und ihrer Toxine auf verschiedene Organe," Jena, 1898.

[4] In the great majority of cases shown to me as ascending neuritis, I have found that there was not a neuritis, but a traumatic neurosis or hysteria; there were only two or three in which a true ascending neuritis seemed to exist, as in one case in which a musculo-spiral paralysis followed a slight wound of the finger, which the patient had treated with urine, and where influenza had also previously been present. Brodmann has described an interesting case of this kind; the case of Pürckhauer to which he draws attention may, however, be otherwise interpreted. Dejerine still firmly believes in the infective form of ascending neuritis, and Raymond specially maintains that the neuritis may ascend along the nerves and reach the spinal cord, whilst Sicard (*R. n.*, 1905) agrees with our view on this point.

Redlich, etc., have lately contributed a number of such cases and they conclude that the process originates specially from *infective* injuries.

The observations of Raymond-Guillain (*Semaine méd.*, 1905) with regard to neuritis in appendicitis, which may also be regarded as an ascending neuritis, are of interest. Marcou holds this view with regard to "névrite appendiculaire" (*Arch. gén. de Méd.*, 1905). See also Solirène: "Les Complications nerveuses des Appendicites," *Thèse de Paris*, 1906.

Symptoms.—Acute interstitial neuritis and perineuritis may commence with fever and rigor, but this is not the rule when one nerve only is affected. I have found in several cases of facial paralysis of childhood that its development was preceded by a febrile stage, and an inflammatory affection of the nucleus of the nerve could not be definitely excluded. The cardinal symptom of acute neuritis is *pain*, felt at a definite site or usually through the whole length of the nerve. This pain is *intense*, boring, tearing or burning, and is continuous, although it may vary at times in intensity. It is aggravated by movement, by pressure, and by every muscular contraction which causes stretching or compression of the nerve. The skin over the affected part is sometimes red, rarely œdematous. Its temperature may be raised.

The inflamed nerve is very *tender to pressure*, and this sensitiveness affects either the whole trunk or circumscribed parts, especially those at which it emerges from a bony canal or a fascia, or bends round a bone. Pressure produces as a rule not only local pain, but pain which radiates upwards and downwards.

As the sensitiveness of the nerves may vary greatly in normal individuals, and very markedly so, according to our investigations (*J. f. P.*, i.), in neuropathics, this point must be very carefully weighed.

In many cases palpation reveals swelling, and even *fusiform bulging of the nerve* (R. Remak, Henschen, Ross, Dreschfeld, etc.). I have found in the case of an alcoholic who complained of severe pain in the region of the peroneal nerve, that the nerve was so greatly swollen on the inner side of the biceps tendon that it was more than double the normal circumference. In another case of tuberculosis, the ulnar nerve showed a similar condition. Here the swelling was confined to a definite point of the nerve, but in another of my cases the median nerve had throughout almost its whole extent become a thick hard cord. This case was, no doubt, one of relapsing neuritis, and the symptom was apparently due to a generalised neuro-fibromatosis (*q.v.*). Propper found considerable swelling of the long saphenous nerve in rheumatic neuritis. Guttenberg has frequently found this neuritis nodosa on the sacral nerves in women.

The symptoms described so far are those of the inflammatory process; they hardly point in any particular to the nature and physiological significance of the affected structure, as pain, redness, swelling and rise of temperature, are characteristic of every form of inflammation.

The first indications that the nerves are affected are *paræsthesia* and *hyperæsthesia* in the area of distribution of the affected nerve. Symptoms of *motor irritability*, *e.g.* fibrillary tremors, tonic contractions of the muscles, etc., are by no means so constant. The *tendon reflexes* may, undoubtedly be increased in the affected muscles in recent *slight* neuritis (Strümpell, Möbius, Werner, Brissaud), but as a rule they are diminished

and *as the neuritis advances, they may disappear completely*. The *electrical excitability* is not infrequently increased at first, whilst later—as soon as the nerve conduction is affected—there is diminution of the excitability or reaction of degeneration.

Trophic disorders in the skin are not infrequent, herpes zoster, glossy skin, etc., being frequently associated with the neuritis. It should also be remembered that bed-sores, gangrene, pemphigus, and especially perforating ulcers may have a neuritic origin. Trophic disorders of the joints are less common, *e.g.* swelling and inflammation, which sometimes lead to ankylosis. I have also in rare cases seen formation of new bone producing considerable hyperostosis. Some writers ascribe Dupuytren's contracture to neuritis (Eulenburg).

From my own experience I must maintain the view that it is related to affections of the nervous system. I have seen one case, for instance, in which it developed in a syphilitic alcoholic directly after a severe neuritis of the ulnar nerve. Frequently indeed this symptom develops in the course of central processes, especially of gliosis (Oppenheim, Bieganski, Neutra, Testi, Perrero, etc.). We cannot here discuss the traumatic, rheumatic, gouty, diabetic (Noorden, Teschemacher), and hereditary etiology.

During the further course symptoms almost always appear which point to *inhibition of conduction in the nerve : anæsthesia* or *hypæsthesia*, sometimes also retarded pain conduction (Kraussold, Erb, Westphal), *paralysis*, and *atrophy*. These are absent only in pure perineuritis and when the process of recovery sets in before radical structural changes have taken place in the nervous parenchyma.

Chronic perineuritis or *interstitial neuritis* is usually not associated with rise of temperature. The symptoms develop gradually, the pain is less severe, and the tenderness to pressure less marked. Otherwise the symptoms are those of acute neuritis.

The symptoms of *parenchymatous* or *degenerative neuritis* give very little definite indication of the inflammatory nature of the process. There are from the commencement signs of disturbance of conduction in the nerve, which are absent only in the slightest, chronic forms. *Hypæsthesia* and *flaccid, degenerative paralysis* in the area of a certain nerve are the characteristic symptoms, but *pain* and *paræsthesia* are usually also present. The nerve is tender to pressure, but not to such a degree as in the forms described, and there is no swelling of the nerves. The tenderness to pressure is also evident in the muscles involved in the degeneration. It has already been noted that the interstitial tissue is as a rule involved in the disease, and that the symptoms of the various forms pass into each other.

It should be noted that neuritis may be confined to the motor or to the sensory fibres of a nerve, and may therefore to a certain extent be a systemic disease.

If the neuritis affects a purely sensory nerve, such as the cutaneus femoris externus or the long saphenous, the symptoms are of course purely sensory. This neuritis most frequently occurs in the cutaneus femoris externus (see section on meralgia paræsthetica), but it has also been observed by Ballet in the long saphenous nerve and by Remak in the cutaneus femoris internus. A neuritis, limited to the superficial branches of the external popliteal nerve, was recently described by Kutner (*M. f. P.*, xvii.). I myself have seen isolated cases of this kind, and have occasionally met with an affection which I could only interpret as neuritis of the main branch of the trigeminus.

With regard to the *differential diagnosis*, we must remember that some of the injuries named—trauma, poisons, such as carbonic oxide, and infective diseases—may also produce myositic paralysis. The chapters on Neuralgia, Hysteria, etc., should be consulted.

The nature and cause of *paralysie douloureuse* (Chassaignac) are not yet sufficiently explained. Of the recent memoirs on this condition those of Halipré (*Rev. mens. des malad. de l'enf.*, 1904), and Lövegren (*Z. f. k. M.*, Bd. xlix.) may be mentioned.

Course and Prognosis.—Neuritis may have an acute, subacute, or chronic course. An almost "apoplectiform" onset has been mentioned in very exceptional cases (Dubois, Dejerine-Klumpke, A. Westphal[1]). Acute neuritis may end in recovery within a few weeks, and so also may the severe forms of traumatic origin. More usually the condition lasts for a considerable time, or becomes chronic. The cases which are chronic from the very beginning run a lingering course, and may persist for one or more years, after which recovery may take place. The *prognosis* is generally more favourable in cases of acute onset and of rheumatic, infective, or toxic origin. It depends in each case upon the severity of the process, and in secondary neuritis upon the character of the underlying disease. The danger arising from the wandering character of neuritis and from its assumed tendency to make its way along a nerve to the spinal cord has hitherto been greatly overrated.

Treatment.—In acute, recent cases, apart from the necessary skilled treatment of wounds, etc., the first requirement is *rest* for the affected part, as every movement and muscular contraction is apt to excite the inflammatory process. The affected limb should be rendered immobile in a way that will obviate all pressure and traction upon the affected nerve.

General diaphoresis is of special value in rheumatic and infective cases. The local application of cold (ice, ice-water compresses), or of one or more leeches to the site of the greatest pain, is recommended when it can be borne. Mercurial ointment may also be rubbed in. If the pain is less intense, wet packs, hot compresses, hot sand-bags, or the local use of hot air (methods of Tallerman, Hilzinger-Reiner, etc.) should be tried. These are well adapted for the treatment of neuritis of subacute and chronic development, and so are vesicants, which of course must not be applied to anæsthetic areas of the skin. I have obtained the best results in an extremely severe and obstinate case of neuritis and perineuritis of the median nerve, from a blister, with collod. cantharid., extending in a line over the whole course of the nerve in the upper arm. The diet must be light and non-stimulating. Care must be taken to keep the bowels open. A laxative has often a very beneficial effect.

As regards drugs, salicylate of soda, salophen, salol, and aspirin may be recommended, and antipyrin may also be tried. In cases of syphilis, iodide of potassium and mercury should be prescribed. Gowers recommends the latter, given internally, even in non-specific neuritis. Schleich's infiltration anæsthesia has sometimes a palliative effect in neuritis. If the pain be very severe, it may be necessary to give subcutaneous injections of morphia. Cathelin's method[2] has sometimes

[1] *A. f. P.*, Bd. xl.

[2] Epidural injections, etc., translated into German by Strauss, Stuttgart, 1903. See also Pelz, "Die epiduralen Injekt.," clinical report, *C. f. G.*, 1904.

been used with success in neuritis of the sacral nerve, especially by French physicians.

In the acute stage it is not advisable to use the faradic current. On the other hand careful galvanic treatment may be tried, if rapid changes in the strength of the current, especially closing and opening, are avoided. The stabile application of the anode to the chief point of the pain, the cathode resting on a central point of the nerve, has been particularly efficacious in many cases; the current of about 2-4 M.A. with electrodes of about 20 square cm. Some writers recommend strong currents, which I would also advise in subacute and chronic neuritis, but they should be avoided in the acute stage.

If the irritative symptoms have disappeared, or have been absent from the first, and if the disease has a protracted course, then electrical treatment and massage may be strikingly helpful. In addition to the stabile application of the cathode to the painful points or swellings on the nerves, labile galvanic stimulation of the muscles is indicated, and where faradic excitability is not abolished, the faradic current may be used. Massage may help in chronic perineuritis nodosa to dissipate the neuroma, but it must be avoided or used very carefully as long as acute symptoms are present. After the neuritis has disappeared, the muscular atrophy, contractures, and shrinking of fasciæ, which sometimes remain, are most efficaciously treated by massage combined with *active and passive movements*.

Operative treatment may be of advantage in inveterate cases of interstitial neuritis or perineuritis, viz., *nerve-stretching*, in order to free the nerve from adherent tissue and to divide the thickened nerve sheath. This operation is said to have given successful results in some cases of this kind reported by Seeligmüller, Bardenheuer, König, and Barger.

We might also mention other surgical methods, and the various modes of injecting fluid into the nerve sheath, which will be described in detail in the chapter on neuralgia, sciatica, etc.

Finally, chronic neuritis may be cured by the use of thermal baths (Wiesbaden, Teplitz, Wildbad, Gastein), and by brine and peat baths. Local peat fomentations are also to be recommended.

Peripheral Paralysis of the Spinal Nerves

Paralysis of the Phrenic Nerve

Paralysis of this nerve is not common. It may be due to diseases of the cervical cord which affect its nuclear origin, and also, comparatively frequently, to some affection of the spinal column and meninges which injures the third and fourth cervical roots, such as spondylitis, fracture and dislocation, spinal tumour, pachymeningitis, spinal hæmorrhage, and syphilitic disease of the meninges.

The nerve itself, on account of its protected position, is on the whole but seldom affected by *injuries* of the neck. It is occasionally involved along with the fifth and sixth cervical nerves (Naunyn, Moritz), paralysis of the diaphragm being thus associated with Erb's paralysis (*q.v.*). It is seldom compressed by *tumours* in the neck or in the thorax. Holzknecht has recently described one such case. *Tubercular processes* may involve the phrenic nerve (Reynaud). I have lately seen a *narcosis paralysis* of the right phrenic nerve, caused by the head of the patient

being rotated too forcibly to one side during an operation. Possibly the nerve was also directly subjected to pressure from the arm of the operator. The paralysis entirely disappeared within a few hours.

Neuritic paralysis of the phrenic nerve, which may be of *rheumatic* (?), *toxic*, and *infective* origin, deserves special attention. A bilateral phrenic paralysis may appear in the course of alcoholic neuritis. Diphtheritic paralysis often involves this nerve, as Laslett's anatomical investigations show. Ohm [1] has seen the paralysis follow acute articular rheumatism. It has also been observed in lead-poisoning (Duchenne).

Myositic paralysis of the diaphragm, which is due as a rule to the spread of the inflammation from the pleura or peritoneum to the diaphragm, need not be discussed here. Sihle [2] has attributed it to inhalation of ammonia.

Whether the diaphragmatic paralysis observed in tabes (Gerhardt,[3] Levy-Dorn) is of central or peripheral origin cannot be definitely determined.

The *symptoms* of paralysis of the phrenic nerve are those of *paralysis of the diaphragm.* [4] The diaphragm does not contract in inspiration, consequently there is no protrusion of the epigastrium, and on palpation the inspiratory depression of the diaphragm and liver cannot be felt; on the contrary, the diaphragm and the lower margin of the liver recede. In expiration, on the other hand, the hypochondriac and abdominal regions are protruded and the liver descends. The diaphragm can be pressed upwards, and the lower margin of the lung may also be pushed upwards, as may be found on percussion. If the paralysis be incomplete, the slightest counter-pressure of the hand is sufficient to push back the diaphragm which has descended during inspiration.

When the patient is lying quietly on his back *dyspnœa* is not necessarily present, but it occurs on any movement and may be very severe. Life is specially endangered if bronchitis or pneumonia supervene and increase the strain upon the breathing. It is not only inspiration which is affected, but also the power of expectoration, as the quantity of air inhaled is not sufficient and the abdominal muscles cannot act forcibly when the peritoneal cavity is enlarged by the relaxation of the diaphragm. The abdominal pressure is less efficacious for the same reason, as the abdominal viscera may lie higher up and the protrusion of the abdomen on pressure is absent (Grawitz [5]).

Unilateral phrenic paralysis is difficult to recognise, as the functional disorder is very slight. It can usually be detected by careful examination. Absence of the so-called "diaphragmatic sign" (Litten), *i.e.* the visible movements of the diaphragm accompanying inspiration and expiration, may be of assistance in diagnosing a unilateral or bilateral phrenic paralysis. The respiratory sounds are heard faintly, especially at the base of the lung (Suckling). Radiography has during recent years widened our knowledge of this paralysis and its symptoms (Kienböck, Levy-Dorn, Naunyn-Rose, Holzknecht,[6] Moritz [7]).

It has been clinically shown by Lesser (*V. A.*, Bd. cxiii.) and Moritz that unilateral diaphragmatic paralysis may produce scoliosis.

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[1] *Z. f. k. M.*, Bd. lix. [2] *C. f. Phys.*, 1902. [3] *B. k. W.*, 1893.

[4] The fibres which the diaphragm receives from the intercostal nerves seem in man to be too few in number to influence its function.

[5] *B. k. W.*, 1906. [6] Ref. *W. kl. W.*, 1902. [7] *D. m. W.*, 1906.

Electrical examination may also be helpful in diagnosis. The electrical excitability may be abolished for the faradic and galvanic currents and may re-appear with the commencement of recovery, as I have found in a few cases of alcoholic neuritis. In neuritis of the phrenic nerve, points which are *painful on pressure* are sometimes found in the neck over the scaleni, immediately external to the sterno-mastoid or between its two heads.

It is not certain whether there is such an affection as a true *hysterical paralysis of the diaphragm* (Briquet, Duchenne). It is not infrequently simulated, as hysterics allow the muscles of the thorax, especially the superior costal muscles, to act excessively in breathing, whilst the diaphragm is kept inactive. But prolonged observation will show an occasional protrusion of the epigastrium, which will reveal the nature of the case. Then also, although there is great rapidity of breathing in such cases, the other criteria of dyspnœa are wanting, and above all, mental factors have the greatest influence upon the trouble. Even healthy persons sometimes breathe in this artificial way, if requested to make deep inspirations. Wernicke speaks of insufficiency of the phrenic nerves in hysteria and the allied neuroses, which is associated with anxiety and inspiratory dyspnœa.

The *prognosis* is good in the rheumatic and hysterical forms of true or apparent paralysis of the diaphragm. In the cases following diphtheria also, recovery has repeatedly been reported. Involvement of the phrenic nerve in multiple neuritis is an ominous sign; but recovery may take place even in complete degenerative phrenic paralysis, as I have found in several cases. Otherwise the prognosis in the majority of cases is unfavourable.

Treatment.—This comprises treatment of the general disease, removal of tumours which maintain the paralysis, and electrotherapy. In neuritis, counter-irritation on the neck, such as blisters along the course of the nerve (Suckling), may be recommended. Injections of strychnine may do good in diphtheritic paralysis of the diaphragm. General treatment of multiple neuritis, etc.

Paralysis of the Brachial Plexus. Combined Peripheral Paralysis of the Nerves of the Shoulder and Arm

As it is difficult and at the same time of great practical importance to keep before the mind a clear conception of the *anatomical relations* of the plexus, I have thought it wise to preface this section by a description of these, and have followed that given by Schwalbe in his "Textbook of Anatomy."

The anterior branches of the fifth to the eighth *cervical nerves* along with that of the *first dorsal nerve* constitute the brachial plexus. Its roots, which gradually increase in size from the fifth to the eighth cervical nerve, emerge from the intervertebral foramina behind the origin of the scalenus anticus and in front of the scalenus medius. As the three upper roots descend slightly, whilst that of the eighth cervical runs horizontally, and that arising from the first dorsal takes at once an ascending direction in front of the neck of the first rib, all these roots soon unite at an acute angle, and thus form the plexus. This lies in the space between the scalenus anticus and medius, with its three upper roots above the subclavian artery and its two lower behind it. From the outer border of the scalenus it descends obliquely through the supraclavicular fossa, then passes below the clavicle and the subclavius muscle, then behind the pectoralis major and minor to the axilla, after which, while lying between the subscapularis and serratus magnus, it rapidly

breaks up into its long branches to the arm. Below the clavicle the axillary artery lies in front of the median part of the plexus, and it turns gradually on its anterior surface in order to pass backwards into the axilla through the opening formed by the two roots of the median nerve, and to reach the posterior side of this nerve.

There are *many varieties* in the distribution of these nerves in the brachial plexus, and also in the point of origin of the various branches from the main trunks, but a certain *type of ramification* can usually be recognised, which is represented in the scheme I have drawn up (Fig. 211). First—indeed, between the scaleni—the parts of the plexus from the first dorsal and the eighth cervical nerves unite into a common trunk (III.), which is known as the *lower primary cord.* Then the fifth and sixth become united to the upper trunk (I.), *upper primary cord.* The seventh cervical nerve forms for itself a middle trunk (II.), *middle primary cord.* Each of these primary cords is divided into an *anterior* (a^1, a^2, a^3), and a *posterior* (p^1, p^2, p^3) branch, which again enter into new connections and thus form three new trunks, from which the long nerves of the arm are derived: 1. An outer cord (secondary superior and lateral cord), formed of the anterior branches (a^1 and a^2) of the first and second primary cords, gives origin to the musculo-cutaneous nerve, and a root of the median arises from it. 2. An inner cord (secondary inferior and median cord). It usually con-

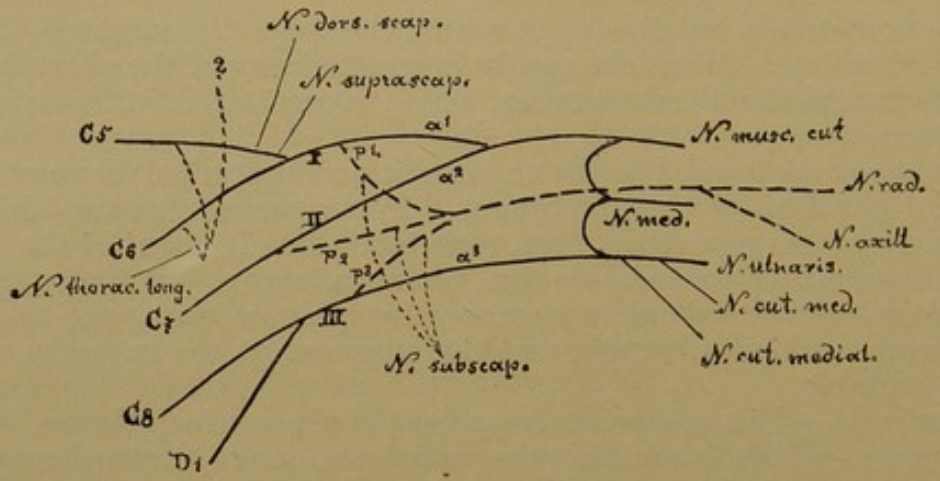

FIG. 211.—Purely schematic representation of the brachial plexus and its branches.

N. rad. = N. musculo-spiral. N. axill. = N. circumflex. N. cut. med. = N. int. cut. N. cut. medial. = N. lesser int. cut.

sists only of the anterior branch (a^3) of the third primary cord, and gives rise to the internal and lesser internal cutaneous nerves, the ulnar, and the inner root of the median nerve. 3. A posterior cord (secondary posterior cord), from the posterior branches of the three primary stems, gives rise to the circumflex and musculo-spiral nerves.

The short nerves of the plexus which go to the shoulder are the posterior thoracic nerves, which originate from the posterior surface of the fifth and sixth (perhaps also the fourth) cervical nerves, before these unite to the primary cord, viz.: 1. the dorsalis scapulæ nerve to the major and minor rhomboid muscle; 2. the long thoracic nerve, which usually arises with two roots from the fifth and sixth cervical nerves, according to some anatomists also largely from the fourth. It runs below the clavicle and behind the brachial plexus to the serratus magnus. From the posterior branches of the three primary trunks, or from the posterior secondary cord, come the subscapular nerve (to the subscapular, teres major, and latissimus dorsi muscles) and circumflex nerves.

The short nerves also include the suprascapular, which has its origin just after the union of the fifth and sixth cervical nerves to form the outer primary cord or even before this. It runs along the superior margin of the brachial plexus, in a lateral and posterior direction with the transverse colli artery, and along the omohyoid muscle, under the trapezius muscle, to the supra-scapular notch, and thence to the supra- and infraspinatus muscles.

The anterior thoracic nerves arise from the anterior side of the brachial plexus and supply the subclavius, and pectoralis major and minor. In the innervation of the pectoralis major

several roots take part—the fifth, especially the sixth, and possibly also the seventh; the clavicular and sterno-costal portions are probably innervated by different roots.

With regard to the morphology and distribution of the fibres of the plexus, and its relation to the nerves and muscles, the works of Harris (*Journ. of Anat. and Physiol.*, 1904), Viannay (*Lyon méd.*, 1905), and Porot (*R. n.*, 1905), may be mentioned among the recent literature.

Isolated diseases of these nerves occur as well as *paralysis of the plexus*, in which the whole plexus or certain segments of it are affected. *We cannot, however, draw a sharp distinction between lesions of the roots and of the plexus*, as it cannot always be determined whether the roots are affected before their union to the plexus or after it (Pagenstecher,[1] Raymond). The distinction is the less important as the causal lesions not infrequently extend to both roots and plexus.

We would further draw attention to the attempts which have been made to differentiate exactly between paralysis of the roots and plexus, especially by Grenet, Warrington-Jones, etc.

Injuries to the shoulder region, a push or blow against the supraclavicular fossa, stabbing and cutting wounds, a fall on the shoulder, etc., may affect the whole plexus or merely some of its roots. This specially applies to *dislocation of the humerus* and *fractures of the joint*, fractures of the clavicle, etc., and also to *forced approximation of the clavicle to the first rib*.

Traction also, especially upon the abducted, extended arm, may produce plexus paralysis. I have several times seen symptoms of neuritis of part of the plexus roots or nerves in persons who have had to rein in an excited horse in riding or driving. Lähr has reported a similar case, and so has Volhard (*D. m. W.*, 1904), and French and English writers have laid special weight on this factor in the production of partial paralysis of the plexus (see below).

Tumours of the supraclavicular fossa by compressing the plexus may give rise to symptoms of paralysis, as I have noted also in cases of aneurism and arterio-sclerotic dilatation of the subclavian artery. There is also a primary neuritis of the brachial plexus (of rheumatic or toxic infective origin).

Obstetrical paralysis and compression paralysis of the plexus due to *dislocation of the shoulder* demand special consideration.

The different nerves of the arm are usually involved one by one after they emerge from the plexus, but they may also be collectively injured by binding the arm (tourniquet, Esmarch's bandage, etc., even by elastic bandages, or handcuffs).

Among the forms of *partial plexus paralysis* [2] the most important is—

(Duchenne) Erb's *combined shoulder-arm paralysis*. This affects constantly the *deltoid*, *biceps*, *brachialis anticus* and *supinator longus* muscles, frequently the *supinator brevis*, sometimes also the *infraspinatus*, less often the *subscapularis*.

This paralysis arises from lesion of the *fifth and sixth cervical roots*, or the upper primary cord of the plexus which originates in the union of these roots (see diagram, Fig. 212). From this trunk arise the *musculo-cutaneous* and some of the fibres which go to the musculo-spiral and *circumflex* nerves, and apparently only the root portion of the musculo-spiral, which supplies the *supinator longus* (sometimes also the *supinator brevis*),

[1] *A. f. P.*, xxiii.

[2] For references to literature see Bernhardt, "Die Erk. d. periph. Nerven," 2nd ed., Wien, 1902. Also Stransky in *C. f. Gr.*, 1902.

and the root of the circumflex, which supplies the *deltoid*. Occasionally the *suprascapular nerve* (for the supra- and infraspinatus), which lies somewhat higher up, and the *subscapular*, are also involved. Erb has demonstrated by his discovery of the supraclavicular point, that these muscles are innervated by a definite root or portion of the plexus, lying superficially. Stimulation of this point, which lies above the clavicle and external to the sternomastoid, elicits contraction of the muscles which are involved in Erb's paralysis.

This paralysis is usually produced by trauma, which either affects the plexus directly at the point mentioned, or presses the clavicle against the plexus by forced adduction of the shoulder to the spinal column, especially by violent backward and outward movements of an arm raised at the shoulder. It was formerly believed (Hoedemaker, Nonne, Schultze) that the fifth and sixth cervical nerves were in this way compressed between the clavicle and the transverse processes of the spinal column ; but recent investigations (Wigand, Büdinger, Kron, Gaupp, Stolper) show that the compression generally takes place between the clavicle and the first ribs,

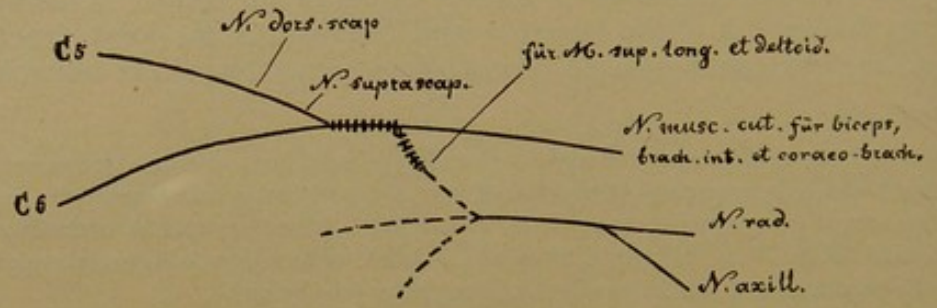

FIG. 212.—Purely schematic representation of the site of Erb's paralysis. N. rad. = N. musculo-spiral. N. axill. = N. circumflex.

or that the spinal roots are subjected to *traction* or even tearing by the violent abduction and elevation of the arm.

According to the investigations and cases of Fieux, Huet, Duval-Guillain (*R. n.*, 1900), Bruns, Clark, Prout, Taylor (*Journ. of Amer. Assoc.*, 1907), etc., the factor of traction is the most important one, and it may be so severe that the mechanical effect extends to the spinal cord, and the roots may even be torn away from it. The fifth and sixth cervical nerves have the longest course, and are most subjected to tension. Philippe and Cestan (*R. n.*, 1901) have shown this anatomically in an interesting case of obstetrical paralysis. This traction is most easily produced, if the head is *bent* towards the opposite side, whilst the arm is elevated (Büdinger, Fieux, Schoemaker). Madlener lays most stress on backward rotation of the arm.

Some cases of infantile obstetrical paralysis (see below) originate in this way. This process also frequently plays a part in the causation of so-called narcosis paralysis (Braun), as during a long operation (usually a laparotomy) the arms of the patient are bent backwards and upwards. Cases of this kind are described by Baum, Büdinger, Krumm, Garrigues, etc. Braun saw a bilateral total plexus-paralysis, and Bernhardt a bilateral Erb's paralysis brought on in this way. Frequently one nerve only, the musculo-spiral for example, is affected.

Most writers take a wider conception of narcosis paralysis. Braun (*D. m. W.*, 1894) has pointed out that the paralysis may be produced by pressure of the head of the humerus against the nerves of the axilla. There is also a tendency to use the term for all the paralyses which arise during narcosis, whether central (cerebral and spinal), or due to chloroform poisoning, etc.

The hemiplegias (Hofmokl, Büdinger, Senger) which in rare cases occur during narcosis from cerebral hæmorrhage and softening have even been included under this designation. Some French writers (Phocas, Mally, Moret, Cabon) go so far as to include all the paralytic conditions which develop during operations under anæsthetics (post-operative and post-anæsthetic paralyses). It has been supposed that acute intoxication from chloroform or ether is responsible, along with the compression and traction on the nerves, for these peripheral paralyses (Casse), and that they therefore belong to the class, which I have called toxico-traumatic, but this is improbable. The anæsthesia, however, by producing total relaxation of the muscles, creates conditions favourable to the onset of this paralysis. Chronic alcoholism is another predisposing factor.

The narcosis paralysis which occurs in the nerves of the lower extremities will be discussed later.

The pressure put upon the nerves in carrying a weight on the shoulder, etc., may also give rise to Erb's paralysis. It has been observed in stone-carriers (Rieger), coal-carriers (Osann), soldiers (pressure of the knapsack (Marsch), and in an attempted suicide from hanging (Pfeiffer). Finally, a primary, toxic, or infective neuritis may be confined to the fifth and sixth cervical nerves, as I have occasionally seen. A woman recently consulted me for this paralysis which had developed after a mono-articular rheumatism of gonorrhœal origin. In another of our cases it followed the ordinary form of acute rheumatism. Krafft-Ebing observed a bilateral Erb's paralysis of neuritic origin, as did Zuelzer; Heyse found it in a phthisical patient who was a stone-carrier. Rendu saw it occur during cerebro-spinal meningitis, and L. Mann found it develop after meat poisoning.

Compression by tumours affecting the fifth and sixth cervical nerves may, of course, produce this paralysis. Thus in one of our cases a metastatic carcinoma, and in another a tubercular abscess had given rise to Erb's paralysis—but under these conditions complete plexus paralysis tends to develop gradually out of the partial form (see below).

The functional disorders caused by paralysis of the muscles supplied by the fifth and sixth cervical nerves have been already described. To put it shortly, the arm cannot be abducted (paralysis of the deltoid). It may sometimes be moved slightly forwards, due probably to the fact that the anterior portion of the deltoid muscle often receives small nerve branches from the anterior thoracic, and also to the action of the supraspinatus. The arm is in the extended position. There is no flexion in the elbow-joint (biceps, brachialis anticus, supinator longus). If the supinator brevis be involved, the forearm and hand are pronated and the hand cannot be sufficiently supinated. If the infraspinatus be affected, the arm is rotated inwards and cannot be properly rotated outwards.

The paralysis is almost always *atrophic*; there is complete or partial reaction of degeneration, less often simple diminution of excitability. The supinator jerk is absent, whilst the triceps jerk is conserved. Pain is sometimes present.

We have not much evidence as regards the condition of the *sensibility*. In many cases it was intact, even in the area of the circumflex nerve, or the anæsthesia had passed off at the time of examination. In others there was a sensory disturbance in the cutaneous area innervated by the *circumflex and musculo-cutaneous* nerves, *i.e.* on the *outer side of the upper arm* over the *middle portion of the deltoid*—not quite extending to the acromion—and on the *external surface of the forearm*. The sensory

fibres of the median for the thumb and forefinger were also sometimes involved.

Interesting data as to the condition of the sensibility are contributed by Warrington and Jones; they agree with Harris that the corresponding sensory fibres run in the sixth, and the motor mainly in the fifth root. Compare Harris (*Journ. of Anat. and Physiol.*, 1904).

In two cases of Erb's paralysis in which sensibility was almost intact, I found trophic disorders in the cutaneous area of the median nerve.

Cases of paralysis of the upper part of the plexus have been reported, which are atypical and which deviate more or less from Erb's type. Thus Rose, in excising a neuroma, had to resect a great part of the fifth and sixth cervical root, and the scalenus, subscapularis, teres minor and major were consequently included in the Erb's paralysis. Heyse found involvement of the long thoracic nerve. Implication of the triceps and pectoralis major has also been reported.

Naunyn and H. Rose (*M. f. P.*, xiv.) saw in one case a combination with paralysis of the phrenic and sympathetic; Frischauer (*W. kl. W.*, 1905) and Moritz (*D. m. W.*, 1906) also noted this combination.

This extension of the paralysis to muscles which are otherwise intact is quite comprehensible if the injury has not been sharply limited to a certain segment of the plexus or to single roots, but passes beyond it or involves the other nerves running through the neck.

The so-called "*hand-ladder paralysis*" (Sehrwald), unilateral or bilateral, which occurs in gymnasts, particularly as the result of "passive hanging," may correspond to an atypical form of Erb's paralysis, as it involves the long thoracic and sometimes also the dorsalis scapulæ and other nerves. It is more often confined to the serratus magnus. According to Sehrwald, the main cause is hyperextension of the arms and rotation on the clavicle, the plexus fibres being crushed between the clavicle and the first ribs, more especially if the head be at the same time bent backwards. But traction on the nerve itself may also be a contributing factor.

Further, there are incomplete, abortive forms of Erb's paralysis, in which single muscles, *e.g.* the supinator longus, are left intact; this only occurs in cases of slight injury.

An attempt has been made, especially by French writers (Grenet, *Arch. gén. de Méd.*, 1900, and *Gaz. des hôp.*, 1904), and by Warrington-Jones (*Lancet*, 1906), etc., to distinguish between *paralysis of the roots* and *of the plexus*. They would also differentiate between intra- and extra-vertebral root paralyses, the levator anguli scapulæ, the rhomboids and the serratus magnus being involved in intravertebral paralysis of the upper root, whilst in the extravertebral form, in opposition to plexus paralysis, it is not these nerves, but the suprascapular, which is affected. As to paralysis of the plexus itself, Grenet would make a definite differentiation according as the proximal or the distal segments are involved. It is not generally possible, in my opinion, to carry these distinctions into practice.

I have seen a few cases in which the distribution of the paralysis corresponded neither to the picture of typical plexus paralysis nor to that of a root affection. Thus a Russian officer, after a gunshot wound in the infraclavicular fossa, developed symptoms of paralysis with complete reaction of degeneration in the muscles supplied by the median and ulnar nerves, with absolute integrity (and normal excitability) of the flexor carpi ulnaris, flexor carpi radialis, the pronators, and the oculo-pupillary fibres. I explained this distribution by the assumption of a lesion of the median fasciculus of the secondary cord of the plexus distal to the point of origin of the branch going to the musculo-spiral, and Bergmann agreed with this interpretation.

The study of lesions of one constituent of single roots is still in its earliest stages. Cases of this kind have been observed by Charcot, Wallenberg, Chipault, F. Buzzard, and E. Bramwell (*R. of N.*, 1903). The disorders of sensibility appear, from Sherrington's experiments, to have a partial (dissociated) character, and, as Buzzard states, they are not accompanied by paræsthesiæ. According to Sherrington, motor symptoms are hardly to be expected, but the few recorded cases are not in accordance with this view, although it is doubtful in how far these have been rightly interpreted, *e.g.* Buzzard's case (*Br.*, 1902). As to the differentiation of peripheral from radicular interruption of conduction, Head's results (*Br.*, 1905, compare this Textbook, p. 408) may be taken into consideration.

The prognosis depends upon the severity of the lesion. The course is usually protracted, and recovery does not always take place. According to Bruns, the prognosis is much less favourable than in peripheral paralysis. In root paralysis also it seems to be more unfavourable than in plexus paralysis, especially in view of the greater difficulty of operative treatment (Warrington-Jones).

From a prophylactic point of view, care should be taken to avoid raising the arms during operations, especially in combination with lateral rotation of the head or bending of the head towards the opposite side.

As regards *treatment*, consult pp. 413 *et seq.* The nerve has been repeatedly sutured in Erb's paralysis, as by Lexer, and recently by Bier, in cases under our obsevration, and by Bardenheuer, Thorburn, Kennedy, Harris-Low, Czerny (Kramer), Taylor, Engelen. Warrington and Jones recommend that surgical measures should be adopted if no improvement has appeared within seven months. Persistent paralysis has led to attempts at transplantation—triceps on biceps, pectoral or trapezius on deltoid—which have been partially successful (Tubby-Steward, Hoffa, etc.).

Paralysis of the inferior plexus (Klumpke's paralysis), which is due to an affection of the eighth cervical and first dorsal roots or the part of the plexus which they form, is less common. Cases of this kind have been described by Flaubert, Seeligmüller, Klumpke,[1] Pfeiffer,[2] Oppenheim, Dejerine, André-Thomas,[3] etc. They may be caused by tumours which compress these roots, by an osteoma-like thickening of the first rib (Müller), by cervical ribs (Seiffer), by surgical operations, such as section of the root (Chipault et Demoulin), by gunshot wound (Brasset[4]), by traction, by syphilitic meningitis (Dejerine, Oppenheim), and by a primary neuritis of these roots (Oppenheim and Feinberg, who saw it follow influenza). It was attributed by Gaussel-Smirnoff,[5] in one case to a meningitic-tubercular focus (?). It occurs as a partial symptom of total plexus paralysis, *e.g.* in dislocation of the shoulder, and the total plexus paralysis may disappear, the inferior paralysis alone persisting.

Cases of paralysis limited to the first dorsal root (Charcot, Buzzard, Bramwell) should not, strictly speaking, be included here.

The small muscles of the hand, some of the muscles of the forearm, especially the *flexors*, are paralysed, whilst the extensors of the hand are only involved if the seventh cervical root is also implicated; in one such case I found paralysis of the triceps, the long thumb muscles, and the extensor carpi ulnaris, whilst the radial extensors of the carpus were spared. *Sensory symptoms* are usually present, both in the area supplied by the *ulnar nerve* and on the *inner surface of the fore- and upper-arm.*

[1] *Rev. de Méd.*, 1885. [2] *Z. f. N.*, i. [3] *R. n.*, 1904.
[4] *N. C.*, 1900. [5] *R. n.*, 1906.

On the hand the anæsthesia sometimes extends into the area of the median nerve, and sometimes it reaches no higher than the elbow-joint (Müller).

In a case described by André-Thomas, the sensory symptoms were manifestly due to a root lesion, which was shown to have affected mainly the roots of the eighth cervical and first dorsal. The distribution of the bathyanæsthesia also corresponded to the root lesion. The author suggests that the inner surface of the forearm is innervated by the eighth cervical, that of the upper arm by the first (and second) dorsal, whilst Bramwell thinks the latter supplies the forearm also. The question requires further investigation.

In the cases under my observation, the distribution of the anæsthesia corresponded to that produced by a root lesion.

Vasomotor troubles may occur. *Oculo-pupillary* symptoms (Horner's syndrome) may develop when the roots are injured above the point where the rami communicantes are given off, and they are therefore not to be expected in true plexus paralysis.

It should be remembered that injuries which affect the shoulder and even the arm, may drag on the nerve-roots so as to produce a root lesion in the immediate neighbourhood of the spinal cord. Cases have been reported in which the roots have been torn away from the spinal cord by forcible attempts to reduce a dislocation of the shoulder.

B. Volhard (*D. m. W.*, 1904) reports a case of this kind; he regards this as a not uncommon occurrence.

Egger and Armand-Delille suppose that the second and third dorsal roots and their rami communicantes were involved in one case.

Apert has made a detailed pathological examination of the affected muscles and nerves, and has found secondary changes in the spinal cord, as Pfeiffer and others had already done. Egger and Armand-Delille (*R. n.*, 1903) have also made a histological examination in a case of total plexus paralysis.

Laehr, by means of the X-rays, saw a dark spot corresponding to the origin of the roots of the inferior plexus, especially to that of the first dorsal, which was perhaps due to a hæmorrhage.

Total Paralysis of the Plexus

This is on the whole a rare affection and is almost always of *traumatic*, rarely of *neuritic* origin. It may be produced during birth or by fracture of the humerus, clavicle, etc. In one of our cases a total and complete plexus paralysis was caused by the fall of a carriage, from a considerable height, upon the shoulder of a man whose head was turned to one side. I have seen a plexus paralysis, not absolutely complete, in a woman who had undergone an operation for mammary carcinoma eleven years previously, caused by dragging of the cicatrix, which extended into the axilla. The paralysis which is caused by *dislocation of the shoulder* is of great practical interest. It occurs specially in cases of dislocation under the coracoid and into the axilla, in which the head of the humerus presses directly upon the nerves, possibly lacerating them. It may also be produced by forcible attempts at reduction (Malgaigne). Duplay, Evesque, Duval-Guillain,[1] and others have recently studied the question of the mechanism of these paralyses. Hæmorrhages within the plexus may be a cause. The mode of production which Kennedy observed in one case is very unusual. Here the patient, by going to sleep with his arm hanging over the back of a chair, had brought on an aneurism of the axillary artery with compression of the adjacent nerve.

[1] "Les paralysies radiculaires du plexus brachial," Paris, 1901.

All the nerves may be involved or only some of them. Or the paralysis which at first is total may gradually become limited to the area of a certain nerve. The *circumflex* and *musculo-spiral* nerves are most often involved, probably by lesion of the posterior cord of the plexus. But as a rule the muscles are affected, not according to their peripheral, but to their root innervation. The paralysis is always *degenerative*. Sensory symptoms are usually present, but they vary greatly in extent. I have seen cases in which there was no agreement whatever between the motor and sensory paralysis; in paralysis of all the brachial nerves, for instance, there was indefinite anæsthesia on the hand or only on the hand and forearm. The sensibility is frequently diminished in the circumflex region also. The fact that sensation is sometimes retained on the inner surface of the upper arm is attributed to vicarious innervation by means of the intercosto-humeral (or the second dorsal root). According to Dejerine-Klumpke, however, this is the only area in the arm that is not anæsthetic in total paralysis of the roots of the brachial plexus.

In a particularly severe case under our observation, in addition to the upper and inner part of the arm, a strip on its dorsal surface had retained or recovered its sensibility.

The *prognosis* of this dislocation paralysis is on the whole unfavourable. As a rule, it is true, it disappears partially or even wholly, but often some muscles remain permanently paralysed and atrophied. Regeneration may require a very long time, even several years. In a patient who had dislocated the humerus by a fall and who was first seen and treated by a doctor on the third day, paralysis and atrophy of the whole arm had developed, which partly disappeared within a few weeks. The muscles supplied by the musculo-spiral and some of those supplied by the ulnar nerve remained longest paralysed, but even after two years they showed a slowly progressive improvement. Alcoholism was a complication in this case.

Laceration of the nerves, or their enclosure in cicatricial tissue, may make operative treatment necessary (Thorburn, Kennedy, etc.).

The paralysis of the plexus in *fracture of the clavicle* is sometimes a direct consequence of the force exerted; it is sometimes due to pressure of a splinter of bone or of an extravasation of blood, or it may be caused by the callus. The whole plexus is usually affected, although the ulnar nerve frequently escapes (Chipault). The pectoralis major is usually involved in the paralysis. The treatment, if the severity of the symptoms requires it, should be surgical, viz., removal of the splinter, resection of the callus, and finally bone suture (Chipault). Complete recovery is thus induced in some cases. Kramer[1] reports the use of neurolysis in these cases in Czerny's clinic.

The combined paralysis of the arm produced by the use of Esmarch's bandage (Langenbeck, Frey, Braun, Bernhardt, Neugebauer) affects sometimes all the brachial nerves in so far as they are compressed in the upper arm, sometimes single nerves, such as the median, etc. It is specially apt to occur in thin persons and in those whose nervous system has been injured by toxic influences (Oppenheim). As a rule the paralysis is slight or of medium severity. Complete recovery took place in two of my cases.

[1] *Beitr. z. klin. Chir.*, Bd. xxviii.

Mally saw paralysis of the median, ulnar, and radial nerves develop after ligature of the brachial artery in the forearm, and attributed it to disturbances of the circulation. See H. Schlesinger (*Z. f. N.*, xxix.) on this question.

Cervical ribs, by compressing the plexus, may also produce symptoms of irritation and paralysis. The first symptoms are pain and paræsthesia in the course of the nerves of the arm and shoulder, and of the long thoracic nerve. Occasionally there are also objective disorders of sensibility, either diminution or complete loss, and signs of atrophic paralysis. The sensory disturbances are rarely bilateral, and their distribution shows that they depend upon a lesion of the roots. Vasomotor and secretory affections occasionally occur. Cases of this kind are described by Bardeleben, Hirsch, Bernhardt, Borchardt, Dejerine, Armand-Delille, Weissenstein, Ranzi, Howell, Russell, etc., and have also been observed by us. The nervous symptoms develop gradually as a rule, but there may be an acute onset due to the effect of trauma, especially to traction (less often to infective diseases). The fact that the disease usually remains latent during the whole life, even in cases of bilateral cervical ribs, and that the nervous symptoms are often present on one side only, is in favour of this mode of origin. An abnormal course and insertion of the scaleni (into the asternal ribs) is apt to produce injury of the nerves by compression and traction. Coote and Fischer were able to satisfy themselves that the nerves had become flattened during life.

The diagnosis of cervical ribs is in the first place founded on the local conditions, viz., visible and palpable bone-tumours in the supraclavicular fossa, the exact nature and course of which may be determined by the use of X-rays. I have seen several cases of an apparently spontaneous neuritis of the brachial nerves in which, in the absence or uncertainty of local conditions, a definite diagnosis could only be made by this means (see Fürnrohr, "Die Röntgenstrahlen," etc., pp. 224 *et seq.*)

As regards the topography it should be noted that the head of the cervical rib is articulated with the body of the seventh cervical vertebra, and the tuberculum with its lateral process, and that, according to Gruber, we can distinguish four forms or degrees of cervical rib: in the first, it does not extend beyond the transverse process, in the fourth it resembles a true rib and has a costal cartilage fused with the cartilage of the first thoracic rib. The frequent association of cervical ribs with a scoliosis situated high up should also be noted.

The symptomatology depends upon the length, and still more upon the arching of the rib, its position, and the consequent relations to the plexus and to the subclavian artery, as compression is specially caused by marked arching or bending.

The symptoms produced by displacement and compression of the subclavian artery are: unusually marked and violent pulsation in the supraclavicular fossa (Fischer), changes in the radial pulse, especially on respiration, movement of the head or raising the arms, pallor of the hand, local syncope or gangrene of the fingers (Hodgson, Coote, Gordon), etc., aneurysmal dilatation of the subclavian artery (Willshire, Adams), or thrombosis of that artery, etc.

Greater attention should in future be paid to hereditary and family conditions. Israel, for instance, noted the disease in two members of one family.

I have already in the third edition pointed out that cervical ribs belong to the so-called stigmata of degeneration, and may be connected with nervous diseases which they have not produced, but which may arise from the common basis of the neuropathic diathesis. Thus I have seen not merely hysteria and hypochondria, but the symptoms of gliosis in two patients who had cervical ribs. In these cases paralysis of the recurrent laryngeal had suggested a direct compression of that nerve.

This factor should always therefore be kept in view when the nervous symptoms are being considered.

It cannot now be maintained that the recurrent laryngeal nerve is directly compressed by the rib (Planet).

I doubt also whether I was right in attributing the unilateral affection of the sympathetic, which I have observed in one such case, to a direct injury of the cervical sympathetic nerve by the cervical rib.

Marburg observed a combination with gliosis, and Levi with disseminated sclerosis.

Cervical ribs have often been removed by surgical operation (Coote, Fischer, Bardeleben, Nasse, Madelung, Gordon, Quervain, Bergmann, Lexer, Borchardt, Israel, Kiderlen, Dejerine, Ranzi, Beck, Tancrast, Winkler, Seiffer, etc.), and in many cases the nervous disorders have subsequently disappeared. I have advised operation in several cases, and have satisfied myself that this is a severe and by no means trifling procedure, which should only be undertaken when the symptoms are marked and intractable In one of our cases upon which Borchardt operated, recovery was at first prevented by the formation of a callus on the resected cervical rib, so that a second operation—with resection of the clavicle—was necessary. Clairmont seems to have observed a somewhat similar case.

Literature: Pilling, "Über die Halsrippen des Menschen," Berlin, 1894; Küster, "Die klinische Bedeutung der Halsrippen," Berlin, 1895; Bernhardt, *B. k. W.*, 1895; Garrè, *Z. f. orth. Chir.*, xi.; Oppenheim, earlier edition of this book, and in Fürnrohr, "Die Röntgenstrahlen im Dienste der Neurologie," Berlin, 1906, pp. 224 *et seq.*; Kammerer, "Annals of Surg.," 1901; Borchardt, *B. k. W.*, 1901; Dejerine-Delille, *Arch. de Neurol.*, 1902; Weissenstein, *W. kl. R.*, 1903; Ranzi, *W. kl. W.*, 1903; Seiffer, *N. C.*, 1904; Bernhardt, *B. k. W.*, 1904; Winkler, *Weekblad f. Genesk.*, 1904; Helbing, *Z. f. orth. Chir.*, xii.; Thorburn, *Brit. Med. Journ.*, 1904; Beck, *Journ. Amer. Assoc.*, 1904 and 1905; Meyerowitz, *Beitr. z. klin. Chir.*, 1905; Seiffer, *M. f. P.*, xvi.; Howell, *Lancet*, 1907; W. Krause, "Fortschr. a. d. Geb. d. Röntgen.," 1907; Russell, *Med. Rec.*, 1907.

Obstetrical Paralysis[1]

Apart from the facial paralysis occasionally caused by application of the forceps, paralysis of the brachial nerves is most apt to occur during parturition. The cases are usually those in which artificial aid is necessary, but more frequently those of head than of breech or foot presentation. In head presentation the introduction of a finger or hook into the axilla, when the shoulder is delayed, may lead to compression, either by direct pressure on the nerve or by the shoulder, and with it the clavicle being pushed backwards and upwards. The use of forceps may cause plexus paralysis if the blades reach the cervical region, but this is on the whole, uncommon. In other cases the plexus may be injured by

[1] Under this heading we include only the infantile form, as paralysis in the mother caused by delivery does not belong to this class. A much wider conception has sometimes been taken of infantile obstetrical paralysis, and some writers speak of cerebral, spinal, and peripheral types (Köster). In the first two groups the condition is the result of hæmorrhage. There is no doubt that hæmorrhage into the spinal cord, and especially into its membranes, frequently occurs in severe confinements (Lietzmann, Ruge, Mauthner, etc.). The child does not usually live, but persistent paralysis has been observed. Laceration of the cord may also occur (Parrot).

A very rare form of peripheral obstetrical paralysis is that of the levat. palp. sup. and rect. sup., due to pressure of the forceps (Nadaud, Berger).

Raymond has noted a combination of plexus paralysis and cerebral changes due to parturition, which has been described in detail by his pupils.

the pressure which is exercised upon the shoulders in order to accelerate the birth of the head. Or it may be due to the *bringing down of the elevated arm*, when the finger or hook is introduced in order to press the arm downwards, or when traction is exerted upon it. We have already stated that the nerves of the brachial plexus may thus be injured by *compression* and *traction*. The chief importance has recently been ascribed to traction, into which the following factors enter, viz., abduction and elevation of the arm, which is at the same time pushed backwards, while the head is bent towards the opposite side (Fieux, Schoemaker, Carter). It is obvious from a number of cases that the roots may also be torn in

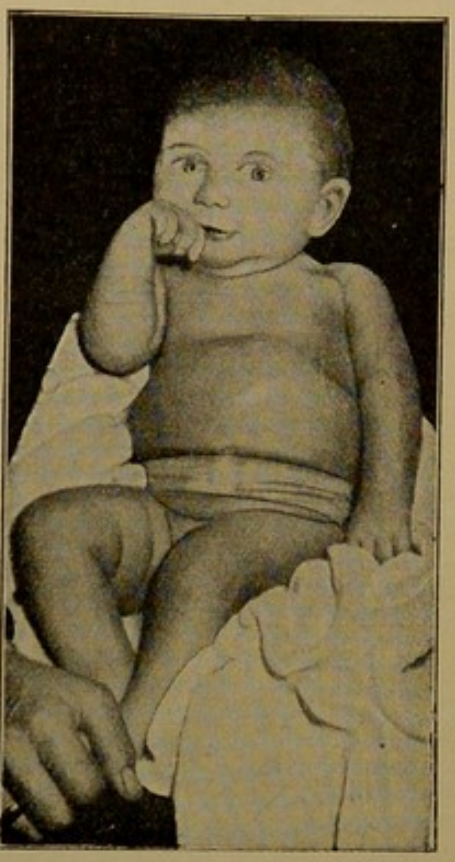

FIG. 213.—Obstetric paralysis of the left arm. Ordinary type (Duchenne-Erb). (Oppenheim.)

FIG. 214.—Paralysis of the right inferior brachial plexus. Obstetric paralysis. (Oppenheim.)

this process. But we must not go so far as to consider traction and tearing to be the regular causes and to leave compression quite out of account. The Prague or Smellie's method have also been held responsible. In very rare cases the plexus has been compressed by the umbilical cord being twisted round the neck.

Obstetrical paralysis very seldom occurs in confinements without artificial help, and then only when the child is very strong, the diameter of the shoulders very great, and their birth very long delayed. Contraction of the pelvis is favourable to the production of paralysis.

Typical obstetrical paralysis has been already described by Duchenne: the deltoid, biceps, brachialis anticus, sup. long., sup. brevis, and infraspinatus are affected. The humerus is rotated inwards, the forearm

extended and the hand pronated. This inward rotation of the arm and pronation of the hand is a very characteristic and constant sign of obstetrical paralysis, by which it can often be recognised at the first glance (Fig. 213). The paralysis is soon followed by atrophy. There are no sensory disorders in this form as a rule, though I have recently found them in one case, in the region supplied by the circumflex nerve. The paralysis sometimes involves the latissimus dorsi, teres major, and other muscles.

The paralysis much less frequently (according to Stransky in twelve out of ninety-four cases) corresponds to the *inferior* plexus paralysis (Fig. 214), with the characteristic oculo-pupillary symptoms, etc., and only in exceptional cases does it extend to the whole plexus or to its roots (cases of Seeligmüller, Jolly, Oppenheim, etc.). The cause is then usually the effect of violent force which leads to fracture of the humerus, acromion, and so on. In such cases the usual sensory symptoms are present, extending over the whole arm; but the medial area of the axilla or even the whole inner surface of the upper arm, innervated by the intercosto-humeral, is generally spared.

Fig. 215. — (After Jolly.) Position of the arms in a case of a typical obstetric paralysis, in which the 7th cervical root was mainly affected, the 5th and 6th being intact.

Atypical cases have been often noted (Huet), in which the roots of the superior and inferior plexus area are involved, but with predominance of this or that type. In isolated cases obstetrical paralysis is limited to one nerve, *e.g.* the circumflex (Oppenheim, Bollenhagen).

Some writers assume that the type of paralysis depends on the position of the child (Peters, Jolly), but constant relations of this kind cannot be demonstrated (Stransky).

Nonne and I have shown by pathological investigation that the lesion in one case of typical obstetrical paralysis affected Erb's point. I found a degeneration of the fifth and sixth cervical roots, which was continued into the circumflex, musculo-cutaneous, and musculo-spiral nerves.

Obstetrical paralysis is seldom bilateral. A remarkable but unusual case of this kind is described by Jolly (*Charité-Annalen*, xxi.); here the muscles of the upper arm, innervated from the fifth and sixth cervical nerves, were spared, whilst those of the forearm and hand, along with the pectoralis major, latissimus dorsi, and triceps, were affected. The contraction of the antagonists (deltoid and biceps) produced a peculiar attitude of the arms (Fig. 215). Jolly assumed a lesion of the spinal roots, particularly of the seventh pair, and suggested that the lordosis of the cervical vertebræ correponding to the face presentation in his case had caused traction of the roots. Philippe and Cestan (*R. n.*, 1900) describe a very severe case complicated by central changes, and on pathological examination they found laceration of the roots.

The complications of obstetrical paralysis include fracture of the humerus and of the clavicle, dislocation of the shoulder, hæmatoma of the sterno-mastoid, and separation of the epiphyses. Küstner thinks that the latter has in many cases simulated the condition of obstetrical

paralysis, especially paralysis of the infraspinatus. This condition, however, produces local pain and soft crepitation. Stransky lays weight on asphyxia, and Schüller on torticollis as predisposing factors.

The *prognosis* is not unfavourable, although the experience of different writers varies widely on this point, and the majority emphasise the slight tendency to complete recovery. There are cases in which the paralysis disappears within a few weeks, others in which it persists for months or even a year and then improves only to a certain extent. I have seen complete recovery in six cases (in one the circumflex was alone affected), whilst Seeligmüller, Bernhardt, D'Astros, Jolly, Warrington-Jones, and others found only partial recovery. This is the rule in the severe forms (see below). The condition may remain unchanged. The prognosis is more favourable in superior than inferior (D'Astros) or total plexus

FIG. 216.—Obstetric paralysis. Duchenne-Erb type, with involvement of the extensors of the carpus. Taken at the age of twelve. (Oppenheim.)

paralysis. Osteo-articular complications make the prognosis graver. Bruns thinks that the effect of traction often extends into the spinal cord itself, and that this is the cause of the unfavourable course.

I had an opportunity of examining a boy of fourteen, in whom the bilateral paralysis had only partially recovered; a remarkable condition had been produced by the secondary changes. The paralysis was typical (Duchenne-Erb), but the deltoids and the biceps had almost completely recovered, while the supinators, especially the longi, had entirely disappeared, the infraspinati had recovered their power of function, though not completely, and the biceps on both sides were in a condition of contracture (due to the total atrophy of the supinator longus). The limitation of supination and external rotation gave rise to peculiar movements on the patient's part. If he wished to put his hand to his nose, for instance, he first abducted the upper arm to the horizontal level, etc. It seemed to me that, on the whole, habit, as well as loss of function in these muscles, had to do with these movements (habit paralysis in Ehret's sense). I found the same condition in another case, and have recently examined a number of such patients. The habit was always the same (Fig. 216), but careful examination showed some differences; thus

in one case I found the shoulders fixed by contracture of the subscapularis, the infraspinatus having lost its power of function although it responded to electrical stimulation.

Huet has since published an almost similar case, without, as he writes me, any knowledge of my communication, and his interpretation of it is similar to mine. He has satisfied himself from a large number of cases that these symptoms occur when the restitution is incomplete. An inhibition of the growth of the bones has also been noted (Guillemot, Huet).

The "congenital limitation of supination of the forearm," described by Drenkhahn (*D. milit. Z.*, 1905) may possibly sometimes be explained in this way.

The outlook is less favourable if there is reaction of degeneration. Yet it may be hardly possible to recognise the alteration of the electrical excitability within the first five to eight weeks (see p. 36). In any case electrical treatment should be commenced as early as possible. Orthopædic treatment is specially suitable at a later stage. If recovery does not take place, Kennedy's neurolysis and nerve suture may be indicated. In one case movement was almost normal nine months after operation. He advises that this operation should be adopted if there is no evidence of any tendency to spontaneous recovery at the end of two months. This period is undoubtedly too short. Taylor also reports satisfactory results. He has found the second year of life to be the most favourable time for operation. The observations of Warrington and Jones are also worthy of attention.

The necessity for suitable and scientifically conducted *gymnastic treatment* is demonstrated by the cases published by Huet and myself.

The advice given by English physicians to fix the unaffected arm in order to compel the child to use the paralysed one should be kept in mind.

See also Payr, *D. m. W.*, 1908.

The most important measures for *prophylaxis* of this paralysis have recently been discussed by Schoemaker.

An interesting communication by Guillemot upon an endemic occurrence of obstetrical paralysis in the district of one midwife shows how important it is that obstetricians should be thoroughly trained and proficient in their work.

For literature on this subject see specially the review of Stransky, "Über Entbindungslähmungen der oberen Extremität beim Kinde," *C. f. Gr.*, 1902; also Huet, *R. n.*, 1902; Stolper, *W. kl. W.*, 1901 (*N. C.*, 1903); Kennedy, *Brit. Med. Journ.*, 1903; Albert-Weil, "Les paralysies radic. obstét.," etc., 1905; Thoyer-Rozat, "L'Obstétrique," 1904; Bauduy, *Thèse de Paris*, 1905; Vigier, "Des paralysies obstét.," *Thèse de Montpellier*, 1904; Taylor, *Journ. Amer. Assoc.*, 1907; Warrington and Jones, *Lancet*, 1906.

Paralysis of Individual Nerves of the Shoulder and Arm

Some of these nerves are rarely paralysed alone, whilst others are frequently so.

The Long Thoracic Nerve

may be directly injured in the supraclavicular fossa, or in the axilla. It may be paralysed after *injuries to the shoulder*, after a fall, blow, or push on the shoulder, or after stabbing in the axilla. *Carrying weights* may act as a trauma. It is also possible that the nerve may undergo compression from strong contraction of the scaleni. The paralysis may also develop as the result of *overstrain*, especially from work which necessitates prolonged or constantly repeated elevation of the arm.

Claude-Descomps attributed one case partly to overstrain in cutting-out cloth and partly to some infective condition. It has also followed gymnastic exercises (climbing a ladder with the hands alone).

It is easily understood why *men* should be more often affected than women, and the right side more frequently than the left.

Isolated paralysis of the long thoracic nerve has been observed after *diphtheria* and *typhoid* (Nothnagel, Bäumler, Souques-Castaigne), and after influenza (Bernhardt, Rad), and is said to be caused by *rheumatism*. I have once seen it occur during the puerperium. This nerve is not usually involved in paralysis of the upper cord of the plexus. I am very doubtful as to the existence of an hysterical serratus paralysis (Verhoogen, Biro), but such a condition may be simulated by contracture of the antagonists (Seeligmüller). In many cases the cause remains obscure.

The symptoms are those of *serratus paralysis* (see p. 15, Figs. 5-7). Sensory disturbances are not present as a rule, but there may be pain along the course of the nerve. When the paralysis is severe, electrical examination shows reaction of degeneration.

The disturbance of function is not so marked in paralysis of the serratus as in that of the deltoid, but the patient cannot lift a weight or use a heavy tool (hammer, hatchet). The power of raising the arm above the horizontal is often retained. Steinhaus says this is the rule and attributes it to the integrity of the upper part of the muscle, the nerve roots to which are less liable to injury; but Struthers, who has made a careful anatomical investigation in a similar case, does not agree with this view. The trapezius, especially in its middle and lower portion, is often paralysed along with the serratus. Souques states that associated paralysis of the serratus and the scapular part of the trapezius is the usual type of this paralysis, and ascribes it to the synergic action of these muscles in certain movements. E. Bramwell and Struthers also note the rarity of pure serratus paralysis.

The *prognosis* depends upon the primary disease. It is favourable in the rheumatic and post-infective forms, whilst in severe traumatic cases the paralysis may be permanent. Recovery often commences only after many months. If the disease is seen to be incurable, the functional disturbances may be removed by grafting the pectoralis major upon the serratus.

Tubby (*Brit. Med. Journ.*, 1904) and Samter (*D. m. W.*, 1906 and 1907) report results of this kind.

Literature: Bernhardt in "Nothnagel's Handbuch"; also Bareiro, *Thèse de Paris*, 1895; Souques-Castaigne, *Nouv. Icon.*, xii., 1899; Steinhausen, *Z. f. N.*, xvi.; Bramwell and Struthers, *R. of N.*, 1903; Struthers, *R. of N.*, 1903; Bernhardt, *B. k. W.*, 1905; Biro, *Z. f. N.*, xxiii.; Claude-Descomps, *R. n.*, 1906.

Paralysis of the Circumflex Nerve[1]

A fall or blow on the shoulder, even a fall on the hand with contusion of the shoulder, compression of the nerve (as in pressure of a crutch), rheumatic and infective influences, may produce paralysis. There is also a form of lead paralysis which is limited to the deltoid. I have seen paralysis of the circumflex nerve with sensory troubles occurring in chronic lead-poisoning after the patient had for a considerable time

[1] The circumflex nerve is termed the *nervus axillaris* by continental authors.—Tr.

carried weights on his shoulder ; in this case therefore the paralysis was a toxico-traumatic one. Diabetic neuritis may also be confined to the circumflex nerve, as may puerperal neuritis (Tuma, Syllaba).

Raymond [1] reports bilateral paralysis of this nerve, which followed prolonged elevation of the arms (in sleep) and was therefore due to *traction*. A case of circumflex paralysis caused by pressure exerted during sleep had already been described by Seeligmüller. Strauss has lately published another similar case. Paralysis from dislocation may be limited to the circumflex nerve, but more frequently other nerves are also involved. The dislocation may even produce laceration of the nerve. In exceptional cases obstetrical paralysis (Oppenheim) and narcosis paralysis (Skutsch) are limited to this nerve. Wallerstein saw it become paralysed as the result of a jerk of the arm in throwing off a weight.

Circumflex paralysis is either of purely motor nature, in which case it is usually limited to the deltoid muscle, the teres minor not being markedly involved, or it is associated with anæsthesia in the area of the skin supplied by the nerve. The symptoms due to this paralysis have been already described on p. 15. It was there pointed out, on the data furnished by Duchenne, Kron, Kennedy, Rothmann, Loewe, Steinhausen, Hoffmann, Hasebrock, and myself, that in spite of complete paralysis of the deltoid, abduction of the arms could sometimes be carried out by means of the compensatory action of other muscles, viz., the serratus magnus, trapezius, infra- and supra-spinatus, triceps, biceps, and pectoralis major. Kron succeeded by methodical strengthening of these muscles in overcoming the restriction of movement due to paralysis of the deltoid. He found that the muscles then became hypertrophied.

I have seen a case in which the onset of the paralysis, which was of a relapsing kind, was immediately followed by an attempt at compensation between the infraspinatus, trapezius; and pectoralis major, the result being a condition which suggested ataxia when the arm was raised. It was interesting to find that in this case, although the electrical excitability was otherwise normal, the weakest electrical stimulation from Erb's point produced merely an external rotation of the arm. The infraspinatus had, it seemed to me, gained greatly in strength on account of the previous paralysis.

During the futher course relaxation or even ankylosis of the shoulder-joint may develop It is doubtful whether these trophic disturbances are of nervous origin. Paralysis of the deltoid will not be confused with a primary (rheumatic, arthritic) ankylosis of the shoulder, as in the latter the arm is fixed in the shoulder-joint and the scapula follows the movement of the arm. The patient can also contract the muscle, although he cannot move the limb at the joint, and there are no symptoms of degeneration or sensory disorders. Paralysis of the circumflex nerve should not be confused with the direct muscular paralysis (and atrophy) produced by a blow or percussion against the muscle, in which there is never any reaction of degeneration or sensory disturbance.

Circumflex paralysis may also be stimulated by contracture of the adductors (pect. major, latiss. dorsi) or by voluntary contraction of these muscles. Such contracture may be recognised by sight and palpation. Freund and Sachs describe an ingenious device for detecting simulation

[1] "Leçons sur les Maladies du Système nerveux," i., 1896.

of deltoid paralysis. They laid the patient upon his abdomen on a table and allowed his arms to hang perpendicularly from its edge. The patient pretended that he could not raise his arm to his hip (because he thought this was a function of his deltoid, whereas it was in reality a function of the adductors), and he brought the arm which had been passively raised to the thorax back to its former position against resistance by a movement of the deltoid.

Paralysis of the Musculo-Cutaneous Nerve

Paralysis limited to the musculo-cutaneous nerve has been observed only in a few cases (Erb, Bernhardt, Windscheid, Strauss, J. Hoffmann, A. Hoffmann, Seiffer, Fischler[1]), in one after extirpation of a tumour in the supraclavicular fossa, in another after dislocation of the humerus, and in a third from pressure of the sharp edge of a slab of marble carried on the shoulder. I have seen it occur after a sword-wound in the axilla, and in fracture of the head of the humerus. In a third case which I have lately seen neuritic paralysis confined to this nerve developed after pneumonia. It was attributed by Bernhardt (*B. k. W.*, 1905), in one case, to gonorrhœa.

The symptoms are *paralysis of the flexors of the elbow*, with the exception of the supinator longus, and diminished sensibility in the area supplied by the *musculo-cutaneous nerve* on the *radial side surface of the forearm*.

The coracobrachialis has been several times found intact, probably because the lesion affected the nerve below the point at which it passed through this muscle (Hoffmann). The brachialis anticus muscle, which receives branches from the musculo-spiral nerve, is not necessarily paralysed, so that the paresis may in one case be confined to the biceps or in another may extend only to the inner portion of the brachialis anticus. If the paralysis affects, as usual, all the flexors with the exception of the supinator longus the forearm can only be flexed by this muscle or with the aid of the flexors of the wrist and fingers (see p. 18). In the position of supination, flexion is considerably restricted or impossible. In Hoffmann's case flexion was pretty strongly carried out by the sup. long. and the outer part of the brachialis anticus ; the coracobrachialis was also strongly contracted. Electrical stimulation from Erb's point only produced contraction of the deltoid and supinator longus (and of the external part of the brachialis anticus). In Hoffmann's case also stimulation of the musculo-cutaneous nerve was followed by contraction limited to the coraco-brachialis muscle. The depression on the outer surface of the upper arm between the insertion of the deltoid and the origin of the supinator longus, due to the muscular atrophy, is characteristic.

In one case I found the supinator jerk absent on the side of the paralysed nerve, and ascribed this symptom to the impairment of the sensibility in the region of the *musculo-cutaneous nerve*, as the muscle itself was intact.

In one of my patients the hypæsthetic zone did not extend quite to the lateral margin of the forearm. On the other hand there were obvious trophic troubles of the skin which extended beyond the area usually innervated by this nerve.

The course corresponds to the severity of the lesion. In one case in which the middle form of reaction of degeneration was present, recovery took place at the end of three months under galvanic treatment.

[1] *N. C.*, 1906. For earlier literature see Bernhardt in Nothnagel's " Handbuch," etc.

Paralysis of the Supra-Scapular Nerve

Isolated paralysis of the *suprascapular nerve* is also rare. It has been observed only in occasional cases (Bernhardt, Hoffmann, Sperling, Benzler, Köster, Göbel, Steinhausen, Valentin, Krahulck, Stein, Bernhardt, etc., and once by myself). It is produced by falls on the shoulder, or on the hand with contusion of the shoulder; in one case a chill was thought to be the cause. My case could be traced to carrying a heavy weight on the shoulder. Uhlich saw it follow gymnastics on the crossbar. It was attributed by Fischler (*N. C.*, 1906) to traction on the supra-scapular notch in forcible forward extension of the arm. The relation to cervical ribs, inferred from a case of Bernhardt's, is regarded even by himself as doubtful. He once saw it occur in a tabetic. It is frequently combined with paralysis of the circumflex nerve and with Erb's paralysis. It may also follow these, after the other muscles have regained their power of function. I have recently seen a combined paralysis of the circumflex and suprascapular nerves in the form of an occupation neuritis (see corresponding chapter) in a man who had to press down a signal lever forty times in the hour.

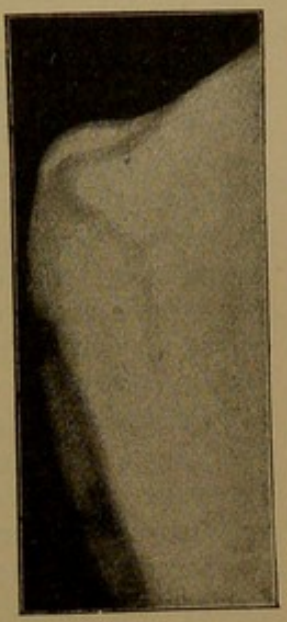

Fig. 217.—(Oppenheim.) Atrophy of the supra- and infra - spinatus, resulting from paralysis of the supra - scapular nerve.

The symptoms are *paralysis of the supra- and infra-spinatus* combined with atrophy. The atrophy of the infraspinatus is very pronounced. In my case the projection of the spine of the scapula was specially prominent (Fig. 217). The paralysis of the infra-spinatus is well shown by electrical examination. The supraspinatus has, according to Duchenne, the function of pressing the head of the humerus firmly against the socket when, during the elevation of the arm, it is subjected to downward traction from the deltoid and consequently has a tendency to become dislocated. It can also move the arm somewhat forwards and upwards. Paralysis of this nerve is characterised by impairment of this movement and especially by the rapid onset of fatigue (and pain) in raising the arm, carrying weights, etc. The tendency to dislocation has in some cases been distinctly evident in abduction of the arm; in other cases it has not been found. Steinhausen thinks that Duchenne has over-estimated the importance of the muscle with regard to fixation of the head of the humerus. Paralysis of the infraspinatus prevents external rotation, but the teres minor, according to Bernhardt, may mask this loss of function. In my case also this movement was simply weakened. Duchenne thinks that spinal movements, which, as in writing, sewing, etc., are associated with external rotation are interfered with.

Jorns (*M. f. U.*, 1899) has described a case of traumatic paralysis of the dorsalis scapulæ. As a result of paralysis of the rhomboids, the shoulder-blade was pushed upwards and outwards, so that the lower angle was separated from the spinal column; the inner margin stood away from the thorax like a wing.

Marcus contributes another case (*Ärzt. Sachverst*, 1905). Involvement of the rhomboids in extensive paralysis of the shoulder muscles has been somewhat frequently noted (see Bernhardt, *B. k. W.*, 1905).

We may mention a probably unique case in which a wound with a knife in the *supraspinous fossa* was followed by paralysis, limited to the plexus fibres for the *supinator longus* (Bernhardt). In a case of *musculo-spiral paralysis from a stabbing injury*, I saw all the muscles recover their power of function, with the exception of the supinator longus, isolated paralysis of this muscle being therefore the residue of a total musculo-spiral paralysis. In a case of obstetrical paralysis I have also seen persistent paralysis and atrophy limited to the supinators.

Paralysis of the Musculo-Spiral (or Radial) Nerve[1]

Of the brachial nerves, indeed of those of all the extremities, the musculo-spiral is the nerve most frequently paralysed. This is chiefly due to its peculiar course and its superficial position. It lies, especially where it passes round the upper arm, between the triceps and the humerus, then between the brachialis anticus and the supinator longus, and is very much exposed to pressure from without, the more so as it is not here covered by muscle and lies directly on bone which cannot yield to pressure. We can therefore understand how it is that Remak, out of 242 cases of paralysis of peripheral nerves of the upper extremities, found the musculo-spiral affected 105 times. This statement does not include cases where the nerve is involved as part of a paralysis of the plexus (see previous chapter); it refers only to paralysis of the nerve and its branches from lesions below the plexus.

In the great majority of cases the paralysis is of *traumatic origin*, and the nerve is usually injured as it passes round the upper arm. Simple pressure is here sufficient to destroy its power of conduction. *Pressure paralysis* is generally caused by the nerve undergoing compression during sleep from the head resting on the arm or the arm being pressed against something hard. It therefore occurs less often during the night, *i.e.* in bed, than in sleeping on the ground, on a board, a bunk, etc. But as a rule there is an additional factor. As I have already shown, it is chiefly individuals whose peripheral nerves are already weakened who are subject to this paralysis; especially *alcoholics*—and this is not due merely to the deep sleep into which they fall, but also to the pre-existing degeneration of the nerves, slight though it may be. Chronic *lead-poisoning* also creates a predisposition. Traumatic paralysis of the musculo-spiral nerve is more apt to develop during convalescence from febrile diseases, in cachexia, and in senility. The paralysis is therefore in many cases *toxico-traumatic* or cachectic-traumatic. In some cases it has been brought about by the clasp of the hand round the arm during sleep, or by carrying a load. It may also arise in surgical narcosis, not only in the way described by Braun, but from the upper arm being pressed against a hard substance (the edge of a chair in my case) during deep narcosis. Policeman's fetters applied to the upper arm may give rise to a unilateral or bilateral musculo-spiral paralysis. In a similar way too tight bandages, heavy burdens resting on the arms, iron pitchers, the handle of which is carried on the upper arm, etc., may cause compression. *Violent* action of the muscles, especially sudden *extension* of the arm, may also injure the nerve, so that it becomes completely

[1] For literature see specially E. Remak in Eulenburg's "Real-Enzyklopädie"; also the section on Neuritis, etc., in Nothnagel's "Handbuch," Bd. xi., and in Bernhardt, *loc. cit.*

paralysed, as in an abrupt throwing movement. I have treated a man who, while in danger of falling from a ladder, had forcibly thrown out his arm to catch hold of it, but had missed his object. The musculo-spiral paralysis was the result of the contraction of the triceps, perhaps also of traction on the nerve. In another the paralysis appeared while the patient was trying to stop a fly-wheel in motion (the same individual, formerly given to alcohol, had suffered a year previously from a toxico-professional paralysis of the median nerve). Gowers reports similar cases, and Gerulanos has recently shown that sudden violent contraction of the triceps muscle, especially of its external head, which lies directly upon the musculo-spiral nerve, may cause its paralysis. This paralysis may be produced in the same way during an epileptic attack (Adler).

In a case observed by Guillain-Courtellemont (*Presse méd.*, 1905) partial musculo-spiral paralysis was attributed to the contraction of the supinator brevis in the professional use of a conductor's baton.

Bernhardt states that it may be due to a fall on the back of the hand.

It hardly needs to be said that the nerve may be injured by a stab, a cut, a gunshot wound, etc. But it should specially be noted that it is not infrequently injured in *fracture of the humerus*, both from fragments or splinters of the bone of the forearm and from the callus. In a case under my care, the nerve was kept extended by a splinter, like strings over the bridge of a violin. In dislocation paralysis it is seldom affected alone, but sometimes along with the circumflex. On the other hand it may be the only nerve uninjured. It is not common for obstetrical paralysis to be limited to this nerve or to affect it chiefly, though I have observed this. "Amniotic" musculo-spiral paralysis also occurs. Cassirer[1] lately reported an interesting case of this kind, which I have had the opportunity of seeing. Spiller[2] interpreted a case in this way. In *crutch-paralysis* the nerve is rarely affected alone, as the paralysis is a total one and involves the triceps. Soca[3] ascribes it to injury of the root by traction. Strong pressure of this kind is usually due to the use of badly made, unpadded crutches.

As compared with the etiological importance of trauma, other factors are insignificant. *Overstrain* of the muscles innervated by the musculo-spiral nerve may give rise to paralysis when there is a predisposition, as I have observed, for instance, in a weaver whose work was associated with constant extension of the left hand (see chapter on Occupation Paresis). Féré describes an angler's paralysis of this character.

The paralysis is very seldom due to rheumatic influences. The poisons of infective diseases more frequently produce neuritis or degenerative atrophy of the nerve. Musculo-spiral paralysis has been noted in the course of *typhoid fever* (Bernhardt), in the *puerperium*, after *articular rheumatism* (Kast), and *pneumonia* (Varnali); here also the infective disease seems to make the nerve more sensitive, and the paralysis itself may be brought on by a slight trauma. I have seen it appear in an alcoholic during typhoid fever. It has been noted, usually in a very transient form, in rare cases of commencing tabes.

Toxic neuritis, with the exception of that due to lead, which is discussed elsewhere, is seldom confined to the musculo-spiral nerve. In chronic arsenical poisoning and argyria (Gowers) the neuritis may be

[1] *D. m. W.*, 1905. [2] *N. C.*, 1904. [3] *Nouv. Icon.*, xix.

limited to the muscles innervated by the musculo-spiral nerve. Michaut found it in opium smokers. A direct *chemical alteration* of the nerve in subcutaneous injections of ether (less often of chloroform, alcohol, antipyrin, osmic acid) into the forearm has repeatedly been found to produce paralysis of the extensors. Here the cause is not injury of the nerve by the syringe, but the inflammatory and degenerative changes caused by the ether.

Kausch regarded the musculo-spiral paralysis in a case which he saw as an *ascending neuritis*. I have also seen a case in which the paralysis developed after a wound in the thumb. The patient had treated it with urine, but it had not suppurated. It was difficult to be certain about the cause, however, as the patient had also had influenza (see p. 419).

The *symptoms* of musculo-spiral paralysis depend somewhat upon the site of the lesion. In the usual *pressure paralysis*, in which the nerve

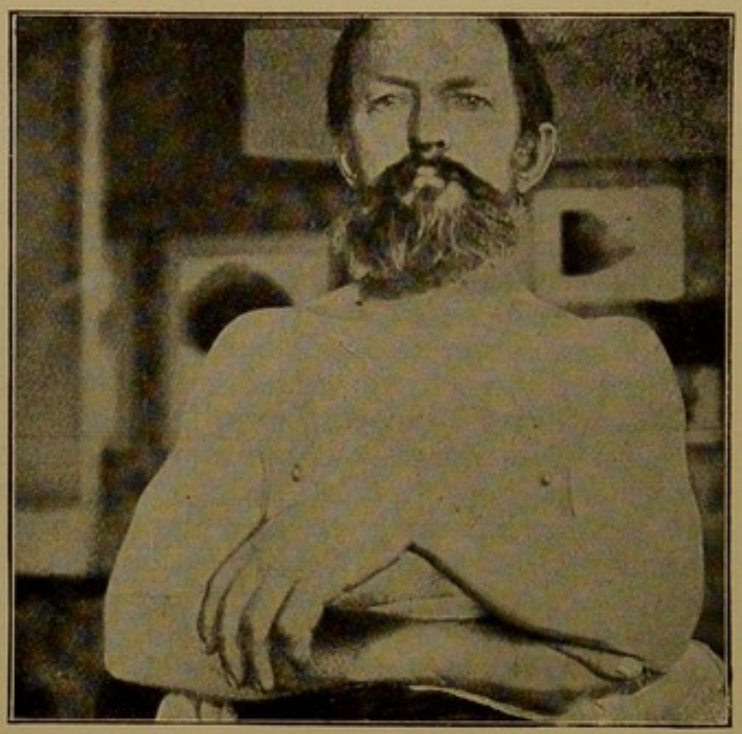

Fig. 218.—Position of the hand in musculo-spiral paralysis. (Oppenheim.)

is compressed after it has given off the branch to the triceps, there is paralysis of the *supinators*, the *extensors of the hand*, the *extensor communis digitorum*; with the *extensor indicis* and *extensor minimi digiti*, the *extensors* and *long abductors of the thumb*. Only the triceps and anconæus escape. The position of the hand may at once reveal the paralysis (Fig. 218). The hand is flexed almost to the maximum at the wrist-joint, and if it is changed from this position it immediately falls back into it. The fingers are also flexed at the metacarpo-phalangeal joints;[1] the thumb is in the position of opposition, and it has fallen somewhat forwards. The hand cannot be extended, nor can the basal phalanx of the fingers, whilst the extension of the other phalanges is not affected, as they

[1] Even when the hand is supinated to the maximum so that the surface of the hand is held upwards, the fingers are not necessarily extended, as they are kept flexed by the flexors. I draw attention to this because in one case of suspected simulation I made the error of thinking that under these conditions the fingers would of necessity follow the law of gravity.

are under the control of the non-paralysed interossei. This is most distinctly seen if the basal phalanges are passively extended. Separation and adduction of the fingers are retained, but on account of the flexed position of the fingers they can only be completely carried out when the hand and fingers rest on a support. In order to show that the lateral movements at the wrist-joint are affected, the hand must be brought into the position of extension, as even in health it is only in this position that these movements can be fully carried out.

Flexion movements of the hand and fingers are performed in a normal way. The *grasp of the hand* is, however, markedly *weakened*. This is due merely to the abnormal position of flexion of the hand and fingers, as the flexors can only be forcibly contracted, if, as the result of hyperextension of the hand, their points of origin and insertion are at as great a distance from each other as possible. Thus, even in musculo-spiral paralysis, the grasp of the hand is of the normal strength when the hand is kept passively over-extended. But the patient cannot forcibly push objects away from him (shoving a barrow, etc.). Abduction and hyperextension of the thumbs are absent or slight. As a rule there is no material weakness of the brachialis anticus, although it receives some branches from the musculo-spiral. Paralysis of the supinator longus produces slight weakening of the power of flexion of the elbow, and it is easily recognised from the fact that when the forearm is in a position midway between pronation and supination, forcible flexion—under resistance—does not cause the muscle to become prominent (Fig. 8, p. 17). Paralysis of the supinator brevis makes it impossible to supinate the hand when the forearm is extended ; it is therefore usually in the position of pronation, and supination may only be to a certain extent carried out in the flexed forearm by means of the biceps, or by an external rotation of the upper arm (infraspinatus). The triceps is hardly ever affected except in crutch, and sometimes in dislocation paralysis. Seeligmüller saw paralysis limited to the *triceps* in fracture of the humerus ; I have seen it in overstrain of this muscle, Gumpertz after an injury.

It rarely happens in pressure paralysis produced in the usual way that only some of the corresponding extensors are affected, as in Suchier's case (*D. m. W.*, 1902).

If the lesion is in the forearm, the supinators and the extensors of the carpus are intact. In *ether paralysis* as a rule it is only the extensor communis digitorum, or some of its divisions, *e.g.* the extensor indicis and the abductor longus pollicis, or not infrequently the main sensory branches also, that are affected. In a sabre cut of the forearm, which had injured the deep branch of the nerve, I found paralysis of the extensor communis digitorum, extensor indicis, the extensor of the little finger and of the long thumb muscles, whilst the radial extensors of the carpus and the supinators were spared. See also Leremboure (*Thèse de Paris*, 1905).

It is only in exceptional cases that the nerve is compressed below the point of emergence of the branches to the supinators. The supinator longus may remain intact in dislocation paralysis. In lead paralysis the supinators are invariably, and the long abductors of the thumb frequently spared.

In the usual form due to compression, the paralysis is generally simple, *i.e.* there are hardly ever any changes in the electrical excitability ; in exceptional cases there is simple diminution or slight increase of the

excitability. I have sometimes found exaggeration of the direct galvanic excitability and slowing in the tremor contraction, with otherwise normal conditions. On the other hand, even when the excitability is normal, *an electrical stimulus applied above the site of the lesion may have no effect* (Erb). Thus when the musculo-spiral nerve is stimulated in the axilla—at the upper end of the internal margin of the coracobrachialis muscle—it is only the triceps that contracts ; when Erb's point is stimulated the supinator does not contract. I have, however, seen occasional exceptions to this rule. In severe nerve lesions (wounds, lacerations, crushing by dislocated bones, etc.), there is of course reaction of degeneration. If the injury is of medium severity—crutch paralysis, slight forms of dislocation paralysis, in rare cases also pressure paralysis—partial reaction of degeneration is the usual condition. The amount of the reaction corresponds to the condition of the muscular nutrition ; in slight paralysis the muscles retain their normal size ; in severe cases they show more or less marked wasting, which is pronounced on the extensor side of the forearm. *Swelling of the tendon sheaths on the back of the hand* is occasionally noted, due perhaps, to the traction caused by the hyperflexion—and there may be swelling of the joint ; hyperostosis of one or more metacarpal bones is less frequent.

Affections of *sensibility* are usually exceedingly slight, and as a rule they are completely absent in pressure paralysis. The patient has sensations of formication and numbness in the area innervated by the radial nerve, especially on the dorsal surface of the thumb and the first metacarpal. These paræsthesiæ may, for a short time, precede the paresis. There is either no objective diminution of sensibility, or but very slight impairment in a circumscribed part of, or rarely in the whole area supplied by the radial nerve. Viannay's explanation that the sensory fibres run in the centre of the nerve and are therefore more protected from injury can hardly be accepted (see p. 408). Even in severe injuries and complete section of the musculo-spiral nerve, there may be no anæsthesia. Under these circumstances, however, sensory disturbances are usually present, and if the whole area of its distribution is affected there is anæsthesia on the back of the hand as far as about the middle ; on the dorsal surface of the thumb, including the lateral zone of its ball ; on the dorsal side of the first three fingers, excluding the dorsal surface of the last, or of both terminal phalanges (which are almost always innervated by the median nerve), and, on the forearm, along a narrow strip on the extensor aspect (inferior external cutaneous nerve, or N. cut. post. inf., on Fig. 27, p. 56). According to Head and Sherren,[1] such extensive impairment of sensibility is only to be expected if the musculo-cutaneous nerve is also injured in the forearm. But this area is usually intact when the nerve is injured immediately above the elbow. If the nerve is affected higher up, sensation may also be diminished on the upper arm in the area supplied by its internal cutaneous branch (N. cut. post. sup. rad. ; see Fig. 27, p. 56). In a case of complete musculo-spiral paralysis, due to fracture of the humerus, I found anæsthesia only on the dorsal surface of the first phalanx of the thumb.

Dejerine and Bernheim had the opportunity of making a pathological examination in a case of typical pressure paralysis. As was to be expected, the changes which they found were very slight.

[1] *Br.*, 1905.

Musculo-spiral paralysis may appear immediately in fracture of the humerus from the direct effect of the injury, or it may be due to the displacement of the ends of the fractured bone. The paralysis is then usually associated with pain, which is particularly severe when movement is attempted. Or it may have the character of a callus paralysis, *i.e.* the callus gives rise to the paralysis by compression of the nerve, by surrounding or over-growing it with bone or with masses of new-formed connective tissue. The onset is gradual, commencing usually with pain and paræsthesia, followed by atrophy and corresponding disturbances of electrical excitability, and ending in paralysis (rarely also anæsthesia).

The prognosis of musculo-spiral paralysis is *entirely favourable* in the majority of cases. In slight pressure paralysis, in which the electrical excitability is not diminished, recovery takes place—even without treatment—in a few weeks, four to six on an average, or, exceptionally, even in a few days. But several months may elapse before it begins. It is certainly quite unusual for this paralysis to become permanent, although I have seen it do so in one case, and Suchier has reported others. Crutch paralysis also tends to recover rapidly. In forms of medium severity two or three months, or even a longer period, may elapse before function is restored, and there are cases which falsify every expectation. The chances are less favourable in lesions of the musculo-spiral nerve due to stabbing, to fragments of bone, to the dislocated head of the humerus. If there is complete reaction of degeneration, the paralysis usually persists or recovers but slowly; recovery cannot be expected within many months. The paralysis caused by tight bandaging may also be severe. In complete section or laceration of the nerve, recovery can only be rendered possible if the ends of the divided nerve are united, and even then it requires considerable time (see pp. 414 *et seq.*). Ether paralysis generally completely disappears within a period of one to four months.

In our experience the prognosis is made graver in childhood by the addition of "habit paralysis" in Ehret's sense, or by the loss of corresponding motor-pictures, as I think. Thus I have treated a girl of seven who, nine weeks previously, had a pressure paralysis of the left musculo-spiral nerve. This appeared from the attitude, and the absence of corresponding voluntary movements, to be complete. I noticed, however, that after extension of the hand and fingers by electrical stimulation and the maintenance of this position for a time, the wrist assumed hyper-extension during a grasp of the hand, and that occasionally the hand or the fingers were extended in automatic movements. If, however, the child tried to carry out these movements to command, it extended the triceps or the flexors of the hand instead of these. That a true peripheral paralysis of the musculo-spiral had been present was shown by electrical examination, as the direct galvanic excitability was still increased and the contraction somewhat slowed (A.C.C. > C.C.C.). I could only assume that the child had, from want of exercise, forgotten the corresponding movements, a condition which was here facilitated by imbecility (previous epilepsy) and indolence. The paralysis was soon overcome by suitable treatment.

Ehret has, in our opinion, too narrow a conception of habit-paralysis.

As to *treatment*, the most essential points are given on pp. 413 *et seq.* As regards prophylaxis, the use of strong and continued pressure upon so sensitive a nerve should be avoided, especially from the application of ligatures, of Esmarch's bandage, etc. In subcutaneous injections of ether we should take care not to pierce through the fascia, and should pinch up a fold of the skin before introducing the needle.

We owe to E. Remak very precise directions as to electrical treat-

ment of musculo-spiral paralysis and data as to its results. He recommends for typical cases treatment with the stabile cathode : the cathode, measuring about 20-30 sq. cm., is applied to the site of the pressure—somewhat above and external to the usual site of stimulation of the musculo-spiral nerve in the upper arm—the other is laid on an indifferent point (sternum). The strength of the current, which should at first be weak, is gradually increased until the patient is conscious of less difficulty in the endeavour to extend the hand. For this a current of 6-8 milliampères is usually necessary. Remak has shown that the average duration is shortened by this treatment, the paralysis lasting from 12 to 20·5 days in treated, and in untreated cases more than thirty days.

Paralysis due to *callus*, especially if incomplete, may disappear under the influence of massage and electrical treatment, as I have seen. But as a rule it requires surgical treatment. I have even seen a complete paralysis of this kind, which persisted unaltered for one and half years, disappear under conservative treatment.

Nerve suture and *neurolysis* have frequently had good results, especially in the musculo-spiral nerve (Busch, Wölfler, Czerny, Oppenheim, Borchardt, Bräuniger, Purves, White, Reisinger, R. Mühsam, Bérard). Monod sutured the nerve after removing a tumour from it. Sick and Sänger saw paralysis of this nerve, due to traumatic defect, cured by suture of its peripheral end with the median. Even when the sectioned ends are widely separated Trendelenburg has had good results from suture after resection of the humerus.

The indications for surgical treatment have lately been thoroughly discussed by Riethus, Blencke (*M. f. U.*, 1903), Borchard (*Z. f. Chir.*, Bd. lxxxvii.), Auffenberg (*A. f. kl. Chir.*, 1907). Nurmann reports good results (*Dissert. Kopenhagen*, 1904).

In one case of callus paralysis of the musculo-spiral nerve recovery took place when, after a fresh injury, the bone was again fractured at the same spot.

Muscle grafting (see p. 217) has also been successfully tried in long-standing paralysis (Franke, Vulpius, Müller, Cappeln, Scheffler, Gray, Keen-Spiller). The flexor carpi ulnaris is specially used as the graft in this operation.

For the weakened action of the flexors of the hand and fingers, due to their flaccidity and flexed position, various apparatus have been recommended, among which that described by Heusner deserved special attention : a firm leather strap is bound round the fore-arm and hand as far as the base of the fingers, keeping the wrist-joint slightly extended, leaving the metacarpal of the thumb nearly free. On the back of the strap are fastened four indiarubber cords, which run into broad indiarubber bands, fixed round the basal phalanges of the four fingers. These keep the four fingers in a position of extension without opposing special resistance to the action of the flexors. This is described minutely and illustrated in the *D. med. Wochenschrift*, 1892, p. 115. Hoffa, Bogatsch, and others, recommend similar apparatus.

Paralysis of the Median Nerve[1]

Isolated paralysis of the median nerve is, owing to its protected position, an uncommon occurrence, and is almost exclusively of *traumatic*

[1] For literature on this and the following chapters, see Bernhardt, Nothnagel's "Handbuch," etc., xi.

origin. The nerve is often affected along with the other brachial nerves in paralysis due to *dislocation* and *compression*. It may also be paralysed by itself from the use of Esmarch's bandage. It may be injured in the upper arm, at the elbow, or in its further peripheral course by a blow, a cut, a stab, etc. It seems to be most frequently injured in its course through the fore-arm, especially near the wrist-joint, by splinters of glass, and so on. It is seldom injured in *fracture of the humerus*, more frequently in fracture of the *bones of the forearm*, either directly or through the pressure of the callus. Although late paralysis due to callus usually affects the ulnar nerve (see next chapter) it has also been observed in the median. Forcible contraction of the pronator teres may also produce the paralysis. I have seen paralysis limited to the sensory branches of this nerve, and lasting for a few days in a young man who, while dancing, had kept his hand fixed for hours in an over-flexed, pronated position. In a few cases the compression has been traced to cervical ribs. Gowers saw the paralysis follow severe distortion of the wrist-joint.

An isolated, non-traumatic neuritis of the median nerve is somewhat rare, but the so-called occupation pareses are generally due to an affection of this nerve. Thus, symptoms of paralysis in the parts supplied by the median are observed in laundry women who iron, in joiners, locksmiths, milkers (Remak), cigar-makers (Coester, Bittorf), carpet-beaters (Reinhardt), dentists, etc.; I have seen a partial median paralysis occur in tailors from continued use of shears. The so-called drummers' paralysis may, according to Bruns, affect the flexor pollicis (but also other muscles of the thumb). According to Düms and Steudel, however, it is not paralysis of this muscle which produces the symptoms, but laceration of the tendon of the extensor longus pollicis muscle. Probably both conditions occur. The *toxic* forms of polyneuritis sometimes also attack the median nerve. Puerperal neuritis most commonly occurs in the median and ulnar nerves. Tumours may develop in the median nerve and give rise to its paralysis.

Symptoms.—If the nerve is affected in the upper arm, the following muscles are paralysed: the *pronators*, the *flexores carpi* (with the exception of the flexor carpi ulnaris), the *flexor digit. sublimis and profundus* (except divisions of the latter for the last three fingers), the *opponens pollicis*, the *flexor pollicis longus et brevis*, the *abductor brevis*, and the *first two lumbricales*. If it is injured above the wrist-joint, the paralysis is confined to the small hand muscles. The position of the hand is not materially changed, but it is generally turned towards the ulnar side from the preponderating action of the flexor carpi ulnaris, and is kept slightly supinated on account of the paralysis of pronation. The flexion of the hand is weak and is accompanied by an inclination towards the ulnar side. The fingers cannot be flexed in the usual way at the first interphalangeal joints, whilst flexion of the terminal phalanges can only be carried out in the last three fingers. Flexion of the basal phalanges is not affected. The thumb is approached to the forefinger, is extended and lies alongside of it. There is no opposition of the thumb or flexion of the terminal phalanx. The patient cannot pronate the hand, and endeavours to compensate for the loss of pronation by internal rotation of the upper arm (when the lesion is seated high up on the median nerve). Objects cannot be properly grasped with the tips of the fingers or held firmly and forcibly.

In the cases which I have examined the paralysis of the two lumbricales was not characterised by any definite symptom, nor did I succeed in discovering changes of electrical excitability in these muscles if the interossei were intact.

Bittorf (*M. w. W.*, 1905) states, however, that he succeeded in finding such changes. Head also was able to stimulate the lumbricales in atrophy of the first interosseous. In a case of severe injury of the median nerve in the upper arm, which I recently saw, the paralysis, when examined several months after the injury, was complete in the whole area of the nerve and was associated with complete reaction of degeneration, although the three last fingers could be flexed with normal force at all the joints. The flexor digit. prof. apparently therefore entirely replaced the sublimis. It was difficult to explain, however, why there was reaction of degeneration in the whole flexor digit. sublimis and profundus. The view that the ulnar nerve was also originally involved and had again become capable of voluntary contraction could not, it is true, be rejected, but it is still remarkable that degenerative changes had developed in the flexor digit. prof. alone.

The condition of the electrical excitability is dependent upon the severity of the lesion (as in the case of the musculo-spiral nerve). Paralysis due to slight pressure is, however, much rarer in the median than in the musculo-spiral nerve ; the injury as a rule is more serious and therefore there are usually grave changes of excitability.

Pain, *hyperæsthesia*, and specially *diminution of sensibility* are usually present, and the anæsthesia may affect the whole area of innervation : i.e. *the palm of the hand as far as the fourth metacarpal, the palmar surface of the three first and the radial side of the fourth finger, as well as the dorsal surface of the second and third phalanges of the thumb (?), index, and middle finger*. There are very considerable individual variations as regards innervation of the skin on the dorsal surface of the phalanges. In one of our cases the dorsal surface of the thumb was not included in the area of anæsthesia, whilst the dorsal region of all three phalanges of the middle finger and the two end phalanges, as well as the adjacent area of the basal phalanx of the forefinger, were involved. In this case, it is true, the brachial artery was ligatured.

The radial and external cutaneous nerves also take part in the innervation of the external third of the ball of the thumb (Head).

There may be no sensory paralysis, and even when the nerve is completely divided such paralysis may be slight and limited to a small area, although the investigations of Head (*loc. cit.*) have changed our views on this question (see p. 408). Bernhardt found, in injury of the median nerve above the wrist-joint, definite sensory symptoms, whilst motility was almost intact in spite of the presence of reaction of degeneration. Since then I have found the same condition in several cases. Bernhardt at first thought it must be due to incomplete section of the nerve, but he has sought to explain the symptom in a later case by the theory that the ulnar nerve may vicariously replace the median by means of an anastomosis between its deep branch and the twig of the median which innervates the muscles of the ball of the thumb.

The paralysis which I have observed in cutters from the use of scissors is a partial median paresis which involves the flexors of the thumb and forefinger and the first interosseous and lumbrical, and is associated with diminished sensibility in part or the whole of the median area. In addition to the professional overstrain, there appeared to be, in one of my cases, compression or traction during sleep.

Vasomotor and *trophic* symptoms in the skin and nails occur much more frequently in traumatic neuritis of the median than of the musculo-spiral nerve. They have been described in detail by Weir Mitchell. The skin, especially in the later stages, is *cyanotic* and feels cold, and the sub-cutaneous tissue may be *infiltrated*. Vesicles resembling *herpes* and *pemphigus* sometimes develop and leave ulcers which are difficult to heal. There may be glossy skin, attenuation of the end phalanges, abnormal growth of nails, longitudinal striation of the nails, alopecia unguium (this formed the only symptom in a case where the nerve was imbedded in a cicatrix: it must be admitted that Head blames other factors for the altered growth of the nails). *Hyperidrosis* sometimes occurs, and more frequently anidrosis of the palm of the hand and of the fingers. In one of my cases of partial lesion of the median the trophic troubles extended to the nail of the ring finger, and the vasomotor symptoms (cyanosis) appeared on the palmar and dorsal surfaces of the second and third fingers.

Cassirer (*D. Klinik*, 1905), K. Hirsch (*D. m. W.*, 1906), and Wandel (*Z. f. N.*, xxxi.), have published interesting observations on the trophic changes after injury of the median nerve, especially of the atrophy of the bone of the end phalanges.

The *course* and *prognosis* depend on the severity of the lesion, and for these as well as for treatment the points already noted should be taken into consideration.

Head gives (*Br.*, 1905) very exact data as to the time at which sensory functions are restored after nerve suture. The first sign of improvement appears on the average on the sixty-fifth day. After about 200 days the analgesia has entirely disappeared, whilst it is only after an average of 387 days that sensation for light touch, etc., has returned in the whole area. The paralysis begins to disappear after an average of 272 days, coinciding as a rule with the return of faradic excitability. If the nerve suture is secondary, the improvement in motility commences at a much later period.

Paralysis of the Ulnar Nerve

This is more commonly observed than paralysis of the median nerve. The ulnar may first be injured along with the other brachial nerves, as in dislocation paralysis. In its further course it is specially injured along with the median nerve in the fore and upper arm in *trauma*, *fractures*, and their consequences. Isolated lesion of this nerve occurs in fracture of the internal condyle of the humerus, in supra-condyloid fractures, either from the primary injury or from the attempt at reduction, but it may also be due to a fragment of bone pressing, piercing, or crushing the nerve. Laceration is very rarely produced in this way. The lesion of the nerve may only be caused by the *callus*, by which the nerve is raised from its bed, flattened, and stretched, or in which it is imbedded. In a woman whom I treated, the callus paralysis did not develop until two years after the fracture, which was due to an abrupt movement. In another of my cases there was an interval of almost twenty years, and in a third twenty-five years elapsed between the injury (with callus formation) and the onset of the paralysis, which was brought on by overstrain or traction. In a woman who had suffered in childhood from a suppurative affection of the elbow-joint after smallpox, a neuritis of the ulnar nerve developed thirty years later. E. Weber (*Z. f. N.*, xv.) has described this case from

my Policlinic and has directed attention to these late paralyses, which had been but little studied, although they had been already described by Panas and Seeligmüller. Broca and Mouchet, Huet, Guillemain, and Mally have since reported cases of this kind. In many of these there was found a cicatricial tissue with which the nerve had become adherent.

See French literature in Guillemain-Mally (*Gaz. hebd.*, 1899); Huet (*Arch. de Neurol.*, 1900); Mouchet (*Gaz. des hôp.*, 1902); Vacquerie, (*Thèse de Paris*, 1902); Savariand (*Arch. gén. de Med.*, 1903).

The nerve is often injured independently. It may be incised or completely divided by a cut (broken glass), a blow, a stab, etc.

Slight pressure paralysis also occurs. The nerve may be compressed by the inner surface of the elbow or the condyle resting upon a hard support. This mode of origin is specially observed in persons who are emaciated and confined to bed. I have treated a strong workman, not addicted to alcohol, in whom a slight ulnar paresis developed when he fell into a deep midday sleep, resting upon the elbow of the right arm. The first attack ended in recovery in nine days; a second, which occurred some years later under the same conditions, was also slight. Cases of pressure paralysis during sleep have also been observed by Erb, Gowers, etc. Braun ascribes the sleep and narcosis paralysis of the ulnar nerve to the pressure exerted upon it by the head of the humerus when the arm is raised and abducted. This explanation does not, however, always apply to sleep paralysis. Dislocation and subluxation of the ulnar nerve with pain and paralysis is also described. It develops in individuals in whom the condyloid groove in which the nerve runs is too flat. A trauma, such as forcible contraction of the triceps, may then produce dislocation. The symptoms are caused by the neuritis which follows (Momburg,[1] Haim,[2]) In isolated cases, such as one described by Nasse, the symptoms of ulnar lesion may be traced to cervical ribs. A prolonged or *violent flexion* of the forearm may in some cases cause ulnar paralysis. Direct compression of the nerve by leaning on the hypothenar eminence and the simultaneous prolonged effect of cold has been blamed by H. Curschmann in one case. Occupation pareses (*loc. cit.*) are often limited to the muscles supplied by this nerve (Duchenne, Leudet, Ballet, Simpson, Lannois). To this class belong the paresis noted in glass-blowers and in wood-carvers (Bruns); a somewhat similar condition occurs in cyclists, bakers (Huet), and telephone operators (Menz). I have seen bilateral ulnar paralysis develop in a telegraphist who suffered from alcoholism and diabetes.

An interesting case is reported of a girl who from childhood had suffered from paralysis of the median nerve and had consequently, when she was ironing, been obliged to hold the iron with the last three fingers, with the result that she developed a neuritic paralysis of the ulnar nerve.

A primary spontaneous *neuritis* limited to this nerve is very rare, but it has been observed after acute infective diseases, especially typhoid (Nothnagel, Vulpian, Wolf, Liepelt). I have also seen a bilateral zoster-neuritis of this nerve. The ulnar is implicated in the paralysis of many forms of polyneuritis. The puerperal form may be confined to the ulnar nerve (Möbius), but the median is usually also involved. Syphilitic neuritis usually selects this nerve and is not infrequently limited to it

[1] *A. f. kl. Chir.*, Bd. lxx. [2] *Z. f. Chir.*, Bd. lxxiv.

(Ehrmann, Gaucher, Oppenheim). It may sometimes be detected from the fusiform swelling of the nerve trunk. The paralysis is more often due, however, to syphilitic meningitis and inflammation of the corresponding roots. Other forms of tumour, such as sarcoma, may develop on the ulnar nerve. Bowlby and Zum Busch describe the formation of a traumatic cyst. Cenas has observed an ascending neuritis. Compression caused by an anomaly of development in the humerus resembling exostosis is mentioned by Féré.

Symptoms.—In complete paralysis there is loss of function in the *flexor carpi ulnaris*, the *flexor dig. prof.* for the *three last fingers*, the *adductor pollicis*, the muscles of the *hypothenar eminence*, the *interossei*, and the *last two lumbricales*.

According to Bardeleben and Frohse the third lumbrical and the flexor digit. sublimis are sometimes innervated by the ulnar, and the adductor pollicis sometimes by the median nerve.

The patient in flexing the hand turns it towards the radial side. He cannot flex the terminal phalanges of the three last fingers, nor adduct the thumb, and its opposition is somewhat affected as the flexor pollicis brevis has a supporting action in the opposition of the thumb (Duchenne). The most evident motor troubles are those due to paralysis of the interossei and lumbricales; the basal phalanges cannot be fully flexed, nor the middle and terminal phalanges extended. Claw-hand develops on account of the preponderance of the extensor dig. communis and the long flexors of the fingers. The *claw position* (Figs. 9 and 10, p. 19) is most marked in the fifth and fourth fingers, diminished towards the second, while in the first and second slight extension of the two last phalanges may be carried out by the lumbricales innervated by the median. *Separation* and *adduction* of the fingers is impossible or is reduced to a minimum; the corresponding movements of the little finger are also abolished. In severe cases atrophy develops and is specially marked in the interossei and the hypothenar eminence.

If the conduction of the nerve is not altogether destroyed, extension of the phalanges may be possible although lateral movement of the fingers is absent. I found this in one among other cases of callus paralysis, which I examined ten years after the injury.

In another case of ulnar paralysis I noted the remarkable fact that the interossei and lumbricales were spared and could be stimulated by the electric current not from the ulnar, but from the median nerve. Whether there had been a pre-existing abnormal condition of innervation or whether the median had, since the onset of the ulnar lesion, replaced its function by means of an unusually developed anastomosis, I could not determine.

There is no explanation of Goldmann's case in which there were absolutely no motor disorders after section of the ulnar nerve (*Beitr. z. kl. Chir.*, Bd. li.)

As regards the electrical excitability we can only repeat what has been said in previous chapters. It may remain quite normal when the pressure paralysis is slight.

Sensory symptoms are seldom absent in ulnar paralysis. *Pain*, *hyperæsthesia*, and *anæsthesia* may all be present, the symptoms of irritation being specially frequent in cases in which a trauma had given rise to conditions which produced prolonged *compression* of the nerve. The *diminution of sensibility* may, if the nerve is injured in the upper arm or in the upper region of the forearm, be present in the whole area of innervation, therefore on the *palm of the hand* corresponding to the *fifth and the middle of the fourth finger*, on the *little finger* and the *ulnar side of the*

ring finger ; on the dorsal surface of the hand as far as the middle, and on the dorsal side of the fifth, fourth, and the ulnar half of the third finger in the region of the basal phalanx. The dorsal surface of the terminal phalanx of the middle finger is supplied by the median, and so frequently is the radial side on the dorsal surface of the terminal phalanx of the fourth finger.

In one case in which the diminution of sensibility on the ulnar side of the forearm extended almost to the elbow-joint, the internal cutaneous nerve was also involved. It should be remembered that the dorsal branch of the ulnar nerve passes over to the dorsal side at the boundary between the middle and lower third of the forearm, between the ulna and the flexor carpi ulnaris, so that cuts on the anterior surface in the lower third of the forearm do not affect the sensibility on the back of the hand and fingers. We must also remember that the dorsal surface of the terminal phalanx of the little finger, and part of the same area on the ring finger, receive twigs from the palmar branch of the ulnar nerve. The disturbances of sensibility are generally limited to a very narrow region, as we should expect from the anatomical conditions. Thus in complete ulnar paralysis the anæsthesia or hypæsthesia may be confined to the hypothenar eminences and to the little finger.

But according to Head this is true only of the disturbance of protopathic sensibility (p. 409), the extent of which after ulnar section is very variable, whilst as regards epicritic sensibility there is only slight overlapping between the median and the ulnar.

In some of my cases the patients complained of a distressing sensation of cold on the little finger. In one the skin of the ulnar region differed markedly from its surroundings by its deep red colour. Hesse noted lowering of temperature on the skin of the paralysed finger. Delay of sensory conduction was found by Erb. In a case of gunshot injury of the nerve there were merely sensory and vasomotor disorders and degenerative atrophy, no motor affections being present (Oppenheim). In one of my cases the thermo-anæsthesia was so complete that the patient burned his hand in warming it at a hot stove (the symptom is so rare in peripheral paralysis that I at first suspected gliosis).

I have also seen a case of slight pressure paralysis in which paræsthesia was the only subjective, and partial reaction of degeneration in the ulnar muscles of the hand the only objective symptom, the motor and sensory functions being otherwise normal.

Cutaneous trophic disturbances are not uncommon. A Dupuytren's contracture has occasionally been found (Eulenburg) in association with, and subsequent to a neuritis of the ulnar nerve (Oppenheim).

If the nerve is injured above the wrist-joint, the long muscles, viz. the flexor carpi ulnaris and the flexor digit. prof., are naturally exempt from the paralysis. This may occur even when the compression affects the nerve higher up (E. Remak).

In the rare cases in which the lesion affects only the deep palmar branch of the ulnar nerve (Bregman, *N. C.*, 1904), the paralysis is limited to the interossei, the third and fourth lumbricales, the adductor pollicis, and the deep head of the flexor pollicis brevis, the muscles of the hypothenar eminence being intact and sensibility practically unaffected.

In cases of slight pressure paralysis, recovery takes place in a few weeks. In severe cases the course is protracted and the conditions necessary for recovery are often obtained only by artificial means

(removal of fragments of bone, neurolysis, nerve suture, etc.), but these may bring about a cure even after the paralysis has persisted for years. Chaput, for instance, found nerve suture efficacious after fourteen years.

For exact data as to the period at which the anæsthesia and paralysis disappear after successful suture see Head (*Br.*, 1905). He has also studied the question of simultaneous injury of the median and ulnar nerves.

In dislocation of the ulnar nerve it may be fixed by a flap of bone periosteum obtained from the olecranon and made to roof over the condyloid groove. This operation has been successfully carried out by F. Krause, Cordua, Pauchet, Momburg, etc.

Appendix: there are congenital defects in the region of the small muscles of the hand which may produce and maintain a position characteristic of paralysis of the interossei. It is remarkable that such a process may, as I have seen, pass away during the first years of life, a fact which points to retarded development of these muscles (see p. 212).

Peripheral Paralysis of the Intercostal Nerves

Whilst we know little about peripheral paralysis of the superior intercostal nerves (Thomayer's observation seems to me doubtful), great consideration has been devoted of late years to paralyses of the inferior nerves, and the paralysis which they cause in the abdominal muscles. The exhaustive descriptions of Duchenne and Erb relate to paralysis of the abdominal muscles occurring in central, especially in spinal diseases. On the other hand, Bernhardt has pointed out the peripheral mode of onset in reference to a case of Taylor's. This affection was first thoroughly discussed by myself,[1] and since then similar experiences have been reported by Minkowski,[2] Lichtheim,[3] and Pelnár.[4]

Still, peripheral neuritis confined to the four inferior intercostal nerves is a rare disease.[5]

It may develop in association with herpes, or in malaria, typhoid, and perhaps also in alcoholism, gout, and diabetes.

The symptoms are unilateral or bilateral pain in the region of the abdomen, tenderness to pressure of the corresponding nerve stems, and paræsthesiæ. This is followed by unilateral or bilateral absence of the abdominal reflex, anæsthesia, and degenerative paralysis of the abdominal muscles. In unilateral paralysis, which is more common, the umbilicus deviates to the healthy side; the affected side is somewhat more prominent, and shows a globular swelling especially in coughing, screaming, straining, or on the attempt to move the trunk against resistance, and the umbilicus is drawn more towards the sound side (Figs. 18, 19, pp. 25, 26). Electrical examination shows complete or incomplete reaction of degeneration. The degenerative paralysis may be limited to individual muscles. As the paralytic symptoms are much more frequently of spinal origin, see the chapter on this subject.

Peripheral Paralysis of the Nerves of the Lower Extremities

The *lumbar* and *sacral plexuses* are much less frequently affected than the brachial, and the nerves of the leg are not by any means so often paralysed as the nerves of the arm.

[1] *Z. f. N.*, Bd. xxiv. [2] *D. m. W.*, 1905. [3] *D. m. W.*, 1906. [4] *Casop. lek.*, 1904.
[5] As to multiple syphilitic root neuritis of this region, see p. 307.

Few cases of paralysis of the *crural* and *obturator* nerves have been reported. The cause has usually been *tumours* arising from the spinal column, the retroperitoneal lymph glands, the pelvis and its viscera, and the femur. *Psoas abscesses* may also injure the crural, which is mainly endangered by the neighbourhood of the psoas. The neuritis of the crural nerve, which follows appendicitis, has been similarly explained (Raymond-Guillain). I have only twice had an opportunity of seeing a primary *spontaneous neuritis* of the crural nerve; in one case it was attributed to a wetting, and in the other it was of *gouty* origin. This etiology is also given by Riegel. Bilateral crural neuritis, associated with pentosuria, has been observed by Cassirer and Bamberger.[1] The symptoms of crural paralysis existed in one boy since birth (Fig. 219), and must be ascribed to a breech presentation. In alcoholic neuritis the sensory branches of the crural are often affected, but a *total bilateral crural paralysis* also occasionally occurs in *alcoholism*. Bruns saw crural paralysis appear with neuralgic pains in *diabetes mellitus*, and disappear under anti-diabetic treatment; in two other cases it affected the crural and obturator nerves and passed from one leg to the other. I have also seen similar cases, and I once found in the son of a diabetic a crural neuritis for which there was no evident cause. Isolated paralysis of the crural nerve very seldom develops as a result of infective processes.

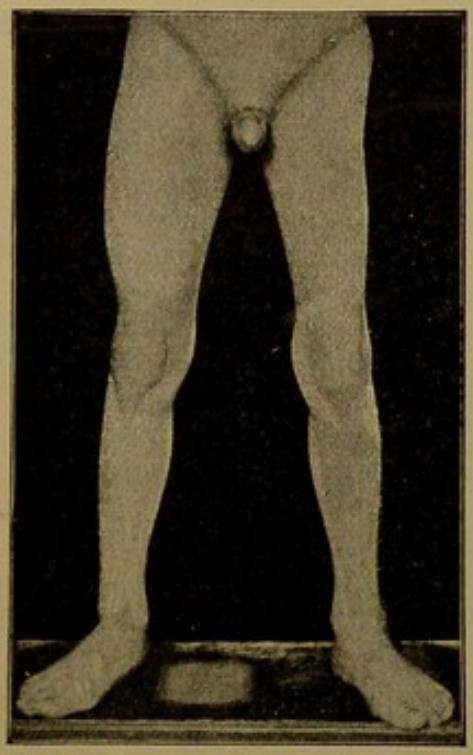

Fig. 219.—Congenital paralysis of the left crural due to breech delivery. (Oppenheim.)

The nerve is comparatively protected against injury, but traumatic paralysis has occasionally been noted in it, from direct injury and fractures of the bones of the thigh and pelvis, etc. This occurs in rare cases of reduction of a congenitally dislocated hip-joint (Bernhardt, *B. k. W.*, 1904).

Gumpertz mentions narcosis paralysis of this nerve; in his case the leg had been kept for a considerable time acutely flexed at the hip-joint. Turney saw it produced in this way in an operation for stone. The crural and obturator nerves were affected in Klempner's case (*N. C.*, 1906).

I have once seen this nerve become affected under the pressure of an *aneurism* from the femoral artery.

The *symptoms* are: 1. Paralysis of the *ileo-psoas*, which is absent when the nerve is injured outside the pelvis or below the origin of the branches which innervate this muscle in the pelvis, of the *quadriceps extensor cruris*, the *sartorius*, and the *pectineus*. Consequently when the paralysis is complete the hip-joint cannot be flexed nor the knee extended. In bilateral paralysis of this nerve the power of walking is greatly impaired; in unilateral paralysis the patient has to walk so as to avoid bending the

[1] *D. m. W.*, 1907.

knee. Paralysis of the pectineus (which, according to Bardeleben-Frohse, is innervated sometimes by the crural, sometimes by the obturator) is not complete and, as in the case of the sartorius, does not give rise to any definite symptoms. 2. *Anæsthesia* or *hypæsthesia* in the region of the *anterior and internal cutaneous* and of the long saphenous nerves (on the anterior and internal surface of the thigh, apart from its upper third, see Fig. 26, p. 55), on the inner surface of the leg, and the inner margin of the foot almost to the great toe. 3. *Absence of the knee jerk.*

We have no trustworthy observations as to the condition of the cremaster reflex in this paralysis.

Compression paralysis commences as a rule with symptoms of irritation, viz., pain that follows the tract of the crural and saphenous nerves. *Muscular atrophy* and changes of electrical excitability are always present if the nerve is severely affected.

As to treatment we would refer to the preceding chapter. Permanent paralysis may require transplantation of the flexors to the quadriceps tendon, etc., but this operation has hitherto been carried out only when the paralysis is of poliomyelitic origin (see p. 218).

Isolated paralysis of the obturator nerve is much less common (crushing from difficult labour, pelvic tumours, obturator hernia). It is characterised by paralysis of the *adductors* of the thigh—external and internal rotation is also affected—and *sensory disorders* on the *inner* surface of the thigh in the upper third, or even as low down as the knee.

I have seen Borchardt perform the appropriate operation of section of the anterior ramus of this nerve. In an examination of the patient which I made a few weeks later, I found loss of function in the adductors and internal rotators of the thigh, in which there was also reaction of degeneration, but the adductor magnus was quite intact and the pectineus also seemed not to be affected. It was difficult, however, to judge of this, as the case was one of Little's disease (see p. 186).

Bernhardt,[1] and Roth,[2] drew attention some years ago to an isolated disease of the external cutaneous nerve. They showed that pain, paræsthesia, and sensory symptoms occurred not infrequently in the area supplied by this nerve. The troubles appeared mainly, though not exclusively, in standing and walking, apparently because the fascia is then most strongly contracted. There was a more or less considerable objective diminution of sensation on the external surface of the thigh, especially in its inferior part. Hedenius [3] gives precise data as to the condition of the sensibility; he always found the sense of heat diminished. In a few cases (Roth, Esca) the impairment of sensibility extended to the anterior surface of the thigh, and the crural nerve (Neisser-Pollack [4]) was involved. The disease due to neuritis of the external cutaneous nerve, termed by Roth *meralgia paræsthetica*, and by others Bernhardt's affection of the sensibility or malum Bernhardtii, occurs mostly in men. Some of my patients were alcoholic; in a few of the others there were symptoms of neuritis in other nerves also. Other factors have also been blamed, namely trauma, overstrain of the legs, infective diseases, pressure from parts of the clothing, especially from waist-belts, adiposity, gout. The affection due to pressure on the nerves by tumours (Dopter) should not be included

[1] *N. C.*, 1895. [2] "Meralgia Paræsthetica," Berlin, 1895, S. Karger. [3] *N. C.*, 1903.
[4] *Mitt. aus. d. Grenzgebiet. d. Med.*, x. See also the review by H. Schlesinger in the *C. f. Gr.*, 1900.

here. In a review by Sabrazès and Cabannes,[1] who collected sixty-two cases from the literature, the disease was traced fourteen times to trauma, twenty-two times to an infective disease (syphilis, typhoid, acute articular rheumatism, etc.), eight times to alcoholism, twice to the effect of cold douches, and seven times to obesity. It also occurs in diabetes (Mohr). The relation of the disease to flat-foot has recently been pointed out (Pal, Kahane, Ehrmann).

Pathological changes were found in one case by Nawratzki,[2] viz., the signs of perineuritis and neuritis with marked atrophy of nerve fibres; the nerves showed even to the naked eye a fusiform thickening at the point where it passed below Poupart's ligament. The changes are generally much less marked. They have sometimes been entirely absent in cases where the nerve has been resected (Souques). Neisser and Pollack were able in one case to show that the fascia lata exerts pressure on the nerve which is removed by slitting the sharp margin. They are inclined to regard this as the general cause of the condition, which is usually not a serious one. In one of my cases it has existed for twenty-five years without any further symptom appearing. In another the subjective troubles disappeared in a few months under electrical treatment, but the anæsthesia can still be detected, nine years later. Naturally the prognosis becomes unfavourable when the symptoms are due to a central disease. A tumour which compresses the spinal roots of the nerve may produce symptoms of this kind. In the course of tabes also, pain and anæsthesia may appear in the region of the external cutaneous nerve. Brissaud observed a form of "intermittent claudication" (*q.v.*), localised in the area of the external cutaneous nerve and attributed to vascular disease.

As to treatment, anti-neuralgic drugs, electrotherapy, counter-irritants, sulphur baths, hot-air treatment (Götz), etc., are recommended. Occasionally it has been thought advisable to divide or to resect the nerve (Wandsbeck, Souques, Chipault, Sollier, Manclair, Brisard, E. Bramwell), or to slit the fascia lata that compresses it (Neisser-Pollack).

I have as a rule been able to dispense with this operation and have frequently relieved the patient, especially of his subjective troubles, by the methods named above. I once recommended resection, which was successful in a very intractable case.

Among the nerves of the *sacral plexus*, the *sciatic* is exposed to many injuries; but paralysis of this nerve involving all its branches is very seldom observed.[3] Almost all the factors which produce sciatic neuralgia (sciatica, see corresponding chapter) may give rise to symptoms of paralysis in the parts supplied by this nerve. These, however, rarely reach a high degree, and are much more often limited to a *degenerative paresis of the peroneal* (or *external popliteal*) *nerve*.

Fragments of bone in fractures of the lumbar vertebra, the sacrum, the pelvis, pelvic tumours, and exudations may compress the nerve-roots. For further information as to lesions at this level we would refer to p. 388 (diseases of the cauda equina). The paralysis which occurs during

[1] *Rev. de Méd.*, 1897. [2] *Z. f. N.*, xvii.

[3] With regard to recent literature on this matter we would refer to Bernhardt (*loc. cit.*); Daus, "Die Pathol. d. Peroneuslähmungen," *M. f. P.*, xiii.; Hösslin, "Die Schwangerschaftslähmungen der Mütter," *A. f. P.*, Bd. xl.

parturition (Basedow, Bianchi, Weir-Mitchell, Lefèbvre, Ballet-Bernard, Thomas, Hösslin, etc.) is of special practical interest. It is caused by the application of forceps, but it may in exceptional cases appear spontaneously, without artificial cause—especially in pelvic contraction (Hünermann, Charpentier), in generally narrow pelvis—as a result of the pressure exerted by the head of the child upon the nerve. There is usually vertex presentation; in one case there was delivery with after coming, in another with the head and face presentation. The sciatic is often so severely compressed during attempted reduction of congenitally dislocated hip-joint (Lorenz, Taylor, Bernhardt[1]) that symptoms of paralysis appear. Tumours may develop in and on the nerve itself (neuroma, sarcoma, syphiloma, etc.), and may cause a more or less complete interruption of its conduction; but we learn from a case of Buchanan's that conduction may remain almost unaltered, even when the nerve—the tibialis posticus in this case—is entirely surrounded. Neuritis due to infective diseases and intoxications has a special tendency to select the sciatic nerve and its branches. Gonorrhœa may cause neuritis of the sciatic nerve, but it more frequently gives rise to simple sciatica. Some cases reported by Lapinsky and others point to the relationship of sciatic neuritis and nephritis.

We should here refer to the remarkable fact that in cases in which compression or other injury affects the nerve in the pelvis, the paralytic symptoms are limited to the area of the peroneal nerve or are most marked in it. It has been assumed that the fibres for the peroneal nerve lie close together in the pelvis and rest directly upon the bones, being thus specially exposed to pressure paralysis.[2] Aeby showed that the fibres of the peroneal have already become grouped into a special nerve in the pelvis. According to the descriptions of Schwalbe, Bardeen, etc., a separation takes place even high up, although the two nerves still run in a common sheath. An unusually high division of the nerve into two main branches is also observed. Other authors suggest that the fibres of the peroneal nerve are specially sensitive to morbid influences and are less capable of resistance. D. Gerhardt showed that after the death of an animal the extensors of the foot lose their excitability more rapidly, and he proved experimentally that in lesions which affect the sciatic, the peroneal nerve is the first to become degenerated. All these are possible contributory factors. It is an established fact that infective and *toxic neuritis* is specially apt to involve the peroneal nerve. Thus Daus's review shows that practically all the infective diseases may be followed by peroneal paralysis. Puerperal neuritis may be limited to the sciatic nerve. Parametritic and septic processes in the pelvis may also spread to the sacral nerves, especially the sciatic (Leyden, Winckel, Pilliet, K. Mills, M'Donald, Guttenberg. For literature see Hösslin).

In its peripheral course it is exposed to traumatic influences of many

[1] *B. k. W.*, 1904.

[2] It has been pointed out that the peroneal nerve arises mainly from the *lumbo-sacral* nerve, *i.e.* from the branch of the plexus formed from the fourth and fifth lumbar and first sacral nerves, which lie directly upon the bones of the *linea innominata*, whilst the other portion of the plexus lies upon the pyriform muscle. But in these obstetrical paralyses other branches of the lumbo-sacral plexus, especially the superior gluteal nerve, which innervates the gluteus med. and min., and the obturator nerve, which crosses the linea innominata, may also be affected. M. Hofmann (*A. f. kl. Ch.*, Bd. lxix.) points out that the arterial supply of the peroneal nerve is much less favourable than that of the tib. post., so that it more easily becomes ischæmic in pressure. Other factors (superficial position, etc.), are blamed by Hartung (*M. m. W.*, 1906).

kinds. Complete or incomplete paralysis of the sciatic is produced by subcutaneous injections of ether, sublimate (in syphilis), antipyrin (Kühn), and often by the alcohol injections which have of late years been recommended in sciatica (Erb-Fischler), by stretching and other injuries, by dislocation of the hip-joint, and by fracture of the femur.

I have seen a case in which hysterical pain in the leg, erroneously diagnosed as sciatica or compression neuritis of the sciatic, was treated by surgical exposure of the nerve and cauterising with pure carbolic acid. The result was a most severe disease of the sciatic with complete degenerative paralysis of the peroneal, which, on inquiry some months later, I found to be still unchanged.

Among the nerves of the leg, the *peroneal* is much more often paralysed than the *posterior tibial*. In addition to the etiological factors already mentioned the following are to be noted. In fracture of the fibula the peroneal may be injured directly or by the formation of callus. I have seen a bilateral traumatic paralysis of this nerve in a bad accident in which both legs had been caught in a fly-wheel. Paralysis may result from traction on the nerve caused by jumping, or a false step (Charcot, Remak, Daus). Sudden forcible supination of the foot may specially cause traction by which, as Redlich thinks, the superficial peroneal is usually more severely affected than the deep branch. Raymond-Brühl describe a traction paralysis of this nerve. I have seen peroneal paralysis occur in an alcoholic person after simple energetic extension of the leg. One of my patients who suffered from sciatica attributed the development of the peroneal paralysis to rough massage. The peroneal and posterior tibial may be injured in operations in the popliteal space, as in Mally's case. Weinlechner saw narcosis paralysis of these nerves in laparotomy (high position according to Trendelenburg), as did Boucht. Few cases of peroneal paralysis due to the application of Esmarch's bandage (Wiesmann) and the pressure of stilts (Bergonié) have been reported. Gerhardt's observation, in which it developed in a man who had slept with his legs crossed, is also an isolated one. Jolly saw it result from fettering the legs.

Peroneal paralysis may develop in people who have to work in a kneeling position, such as potato diggers, asphalt layers, beetroot planters, pipe-layers, etc. (Zenker, Roth,[1] Bernhardt, Hoffmann, Kron, Schultz,[2] Curschmann [3]). Probably the cause here is compression of the nerve between the tendons of the biceps and the head of the fibula.

It may also develop in overstrain by working a sewing-machine (Charcot-Meige, Seeligmüller). In one of our cases it occurred suddenly in a badly nourished sewing-woman who had worked a whole day without intermission. The paralysis was degenerative and spared the peroneal muscles, whilst the sensory disturbances extended to the plantar surface of the foot and toes.

Overstrain in cycling may cause neuritis of the nerves of the leg, especially of the posterior tibial (Levi-Wormser).

Jolly saw a peroneal paralysis appear after acute articular rheumatism with chorea. It was observed by Finkelnburg in arthropathy of the knee-joint. In multiple neuritis, especially when caused by alcohol, the peroneal nerve is the most frequently affected, and the paralysis of the nerve may become permanent after the other morbid symptoms have disappeared (Oppenheim). Arsenical paralysis also frequently affects this

[1] *B. k. W.*, 1883. [2] *A. f. kl. M.*, Bd. lxxx. [3] *D. m. W.*, 1905.

nerve. If lead paralysis attacks the lower extremities, which often happens in childhood, it is localised in the muscles supplied by the peroneal nerve. Maczkowski describes neuritis of the peroneal nerves after carbonic oxide poisoning. Diabetic neuritis not infrequently affects this nerve (see section on Polyneuritis). I have once seen peroneal paralysis appear after appendicitis, in a tabetic patient.

Paralysis of the posterior *tibial* may be of *traumatic* or *toxic-infective* origin. In a child who had fallen through a cane-bottomed chair and remained hanging with the knee-joint in the frame, I found paralysis of this nerve, which did not completely disappear. Paralysis of the peroneal and posterior tibial nerves may develop in beetroot transplanters (Hoffmann).[1] In one of my cases it was limited to the posterior tibial, in another it affected the peroneal and posterior tibial of the left side and only the peroneal on the right, but the Achilles jerk was also absent. In an otherwise healthy man, in whom I found signs of neuritis (without paralysis) in both posterior tibial nerves, the cause was merely overstrain of the legs in climbing stairs, and an insignificant trauma from taking a false step in getting out of a horse tramway. The nerve is often implicated in multiple neuritis. In a case of dermatomyositis under my care, the affection, whilst producing severe œdema in the knee-joint, extended to the posterior tibial nerve.

Symptoms of Peroneal Paralysis.—The *extensors* of the foot and the toes, the *abductors*, and among the adductors the *tibialis anticus*, are paralysed. The point of the foot hangs down by its own weight ; the foot is in the equino-varus position ; it cannot be raised nor can the first phalanx of the toes be extended. If the paralysis persists, the foot may become fixed in the position of pes equinus by secondary contracture of the calf muscles. The interossei may undergo contracture and keep the proximal phalanges of the toes in a flexed position. Walking is affected, the point of the foot drags on the ground unless the patient over-flexes the hip- and knee-joints. The outer edge and the point of the foot first touch the ground. The paralysis is usually *degenerative.*

In neuritis of the peroneal, the paralysis may be partial, the tibialis anticus or the peroneus longus, for instance, being long spared, the deep or superficial peroneal nerves being affected independently. In neuritis of the peroneal the paralysis may even be limited to the extensor longus hallucis (Bernhardt, Oppenheim). A traumatic paralysis affecting only the deep peroneal nerve is described by Bartels ;[1] whilst the peroneal muscles and the interossei were normal in function, the tibialis anticus and the extensors of the toes were completely paralysed, and the foot was in the equinus position. Sensibility was intact. Bartels ascribed the paralysis to dragging upon the nerve at the point where it perforates the peroneus longus muscle. I have seen these symptoms develop after a sprain of the ankle and once after *brisément forcé* of the knee-joint. Isolated paralysis of the peroneus profundus has often been described. Maternal obstetrical paralysis is not infrequently limited to the peroneus profundus (Winckel, Vinay according to Daus).

The superficial peroneal nerve and its branches, on the other hand, are frequently affected by the occupation paralysis of the foot which sometimes develops in painful conditions (Ehret).

Neuritic paralysis commences as a rule with pain and paræsthesia,

[1] *Z. f. N.*, ix.

but in the toxic form these symptoms may be entirely absent. In the obstetrical form the most important symptom is pain in the nerve tract, which occurs with every pain or with every traction of the forceps, and which may be followed soon after the birth by paralysis (Hösslin). I have sometimes also observed the onset in spontaneous neuritis of peroneal paralysis with violent pain.

Sensibility may be completely intact, even when the affection involves the trunk of the peroneal nerve. When there is complete solution of continuity, however, symptoms are always to be expected which depend for their extension upon the site of the interruption (involvement of the external cutaneous). Head (*loc. cit.*) gives some data on this point. If anæsthesia is present, it is found on a small zone on the anterior surface of the leg above the crest of the tibia and external from this (in the lower half), as well as on the dorsal surface of the foot and toes, but not on the external and internal margin of the foot (see Figs. 31, 32, p. 58). There are, however, many individual variations (Frohse).

Vasomotor and trophic disorders are sometimes present.

Paralysis of the *posterior tibial* is indicated by the absence of *plantar flexion* of the foot and toes. If the popliteus is involved, internal rotation of the flexed leg is also affected. Paralysis of the interossei muscles may cause a claw position of the toes (pied en griffe) ; separation of the toes is also affected, but this may give rise to no material functional trouble. The patient cannot raise himself on tip-toe. The gait is markedly affected. Pes calcaneus and pes valgus may develop from preponderance of the antagonists (see pp. 21 *et seq.*).

In paralysis of this nerve the toe reflex takes the form of dorsal flexion—Babinski's sign does not in this case have the significance otherwise attached to it. The Achilles jerk may be abolished even although the neuritis is slight. I have often found this loss to be the only sign of nervous affection in alcoholism and diabetes, and have ascribed it to neuritis.

Sensation is diminished or absent on the postero-external surface of the leg, on the outer margin of the foot, the sole of the foot, and on the plantar surface of the toes.

Perforating ulcer has repeatedly been observed in compression, cutting, or gun-shot wound of this nerve, or of the sciatic nerve and its roots (Duplay, Fischer, Matthieu, Sonnenburg, Sattler,[1] etc.).

If the whole of *sciatic* is affected, the flexors of the knee are paralysed in addition to the muscles innervated by the peroneal and posterior tibial. According to Bardeleben-Frohse it also sometimes innervates the adductor magnus. The disturbance of gait is marked, but may be lessened by suitable supporting apparatus.

Division of the sciatic nerve may make suture of the nerve necessary, but so far it has only occasionally been performed on this nerve (Dallas-Prath, Cervera, Courtin-Bossuet). Paralysis of the peroneal and posterior tibial nerves should be treated according to the rules already given. It is particularly important to ward off secondary contractures by overcoming the pes equinus position, etc. If the paralysis is not cured, transplantation may be necessary.

Isolated paralysis of the *gluteal nerves* is extremely rare. They are, on the other hand, not infrequently affected in combination with the

[1] *W. kl. R.*, 1903.

sciatic in diseases of the sacrum and the pelvis—fracture, tumour, caries with abscess, as in cases of Laehr, Bartels (see chapter on diseases of the cauda equina)—and in maternal obstetrical paralysis (Lefèbvre, Hösslin). Paralysis of this nerve is manifested by paresis and atrophy of the gluteal muscles, of the pyriformis muscle, and the tensor fasciæ latæ. Extension movements in the hip-joint, as in climbing steps, etc., are lost, and so is the power of abduction and internal rotation of the thigh.

Peripheral Paralysis of the Cranial Nerves

Diseases of the first and second cranial nerves are so intimately related to cerebral diseases, that they will be discussed along with them.

Paralysis of the Oculo-Motor Nerves[1]

will be considered here only in so far as they are caused by lesions of the nerves themselves in their extra-cerebral course at the base of the brain or in the orbital cavity. It must, at the same time, be admitted that the limit between this extra-cerebral and the central course cannot be sharply drawn. These paralyses are frequently signs of a *general disease* or of a *disease of the central nervous system*; they are often of a *secondary nature*, *i.e.* they are produced by some morbid process in the neighbourhood and comparatively rarely represent a *primary*, independent disease. It is therefore advisable to consult, at the same time, the corresponding sections of this text-book, such as the chapter on opthalmoplegia (vol. ii.) and specially that included in the General Part (pp. 76-85).

Among the causes, cold should be mentioned first. *Rheumatic* oculo-motor paralysis is, it is true, more and more rarely diagnosed, as we have learned to recognise that paralyses of individual ocular muscles are frequently premonitory signs of a central nervous disease and are not uncommon consequences of an infective disease. It should, however, be remembered that exposure to cold may cause paralysis, almost always unilateral, of the abducens nerve, or of the oculo-motor or some of its branches. Such paralysis runs an acute course and usually terminates in recovery. A complete unilateral ophthalmoplegia due to rheumatism has also been described, and has been attributed to an inflammatory process in the region of the sphenoidal fissure. It is very probable that the rheumatic paralysis is really due to a *neuritis*, as there are many recorded cases which show that the ocular nerves may be paralysed along with the other nerves in multiple neuritis. I have had occasion in recent years to examine a number of such cases for the University Clinic for Eye Diseases, in which the definite etiology of chill, the development of the paralysis with pain, and the accompanying painfulness of the margin of the orbit and of the supraorbital nerve showed the peripheral-neuritic

[1] For literature see specially: Mauthner, "Die Lehre von den Augenmuskellähmungen"; *ibid.*, "Diagnostik und Therapie der Augenmuskellähmungen," Wiesbaden, 1889; Uhthoff, "Unters. über die bei der Syphilis d. Zentralnervensyst. vork. Augenstörungen," *Graefes Arch.*, 1893; Uhthoff, "Die Augenveränderungen bei Vergiftungen und Erkr. d. Nervensyst.," *Handbuch der ges. Augenheilk.*, xi., 1904; Marina, "Über multiple Augenmuskellähmungen," etc., Wien, 1896; Wilbrand und Saenger, "Die Neurologie des Auges," Wiesbaden, 1900 (*et seq.*); Bumke, "Die Pupillenstörungen bei Geistes- und Nervenkrankheiten," Jena, 1904; Schmidt-Rimpler, "Die Erkrankungen des Auges in Zusammenhang mit anderen Krankheiten," ii. Aufl., Wien, 1905 (Nothnagel's "Spez. Path. und Therapie"); E. Maddox, "Tests and Studies of the Ocular Muscles," 1898. Translated into German by Asher, Leipzig, 1903; L. Lewin und Guillery, "Die Wirkungen von Arzneimitteln und Giften auf das Auge," Berlin, 1905.

character. The same explanation seems to me to apply to a case in which an oculo-motor paralysis, accompanied by severe pain, developed in a non-syphilitic man after a mountain climb which had brought on great sweating and subsequent chill. Combined paralysis of the abducens and facial, of the oculo-motor, trigeminus, and facial ("multiple paralysis of cranial nerves," "multiple independent neuritis of the cranial nerves"), have also occasionally be attributed to a rheumatic neuritis (Hösslin, Möbius, Rad).

Ocular paralysis is very often of *infective* origin. The best known form is the *post-diphtheritic*. It affects the accommodation in both eyes as a rule, and not infrequently also the nerves to the external ocular muscles. It occurs in the abducens more often than in the oculo-motor, but it may involve the whole of the oculo-motor and even all the external ocular muscles. A trochlear paralysis is seldom observed under these conditions (Krauss). Although some of these paralyses (see section on post-diphtheritic paralysis) are of nuclear origin, changes, such as *hæmorrhages* and *degeneration*, have been found in the nerve itself, as we ourselves have seen. During and, more frequently, after *influenza* the ocular muscles (the ciliary muscle, the sphincter pupillæ, and also the external muscles) may be paralysed. Oculo-motor paralysis has been noted only occasionally in *acute rheumatism* (Michel, Bunzel, Pichler, etc.), and but rarely after *scarlatina*, *measles* (Dreisch, Simonin), erysipelas (Séville), pneumonia (Mauthner, Wadsworth), and mumps. It may also occur during typhoid (Ebstein, Emerson). I have once seen oculo-motor paralysis appear in the course of *acute nephritis*. The cases in which it is associated with zoster ophthalmicus (Brissaud, Hutchinson, Wyss, Fouchard, Désirat,[1] Casper[2]) appear to be mostly of neuritic origin.

Syphilis is one of the most common causes of this paralysis. It occurs usually either as a gummatous tumour or a basal gummatous meningitis which involves the ocular nerves, especially the oculo-motor, and causes paralysis of the whole nerve or some of its branches. There is also an independent *syphilitic neuritis*, and perhaps even a primary syphilitic atrophy of the ocular nerves. Finally *exostoses*, *syphilitic periostitis* of the base of the skull or the orbit, and *tuberculosis* may paralyse the ocular nerves. They may also be paralysed by abscesses in their neighbourhood, or perhaps by toxins derived from purulent foci at a distance. Richter[3] describes abducens paralysis in caries of the sphenoidal sinus. *Thrombosis of the cavernous sinus* may also affect the ocular nerves. Neuburger mentions the occurrence of ocular paralysis after great loss of blood.

The relation of this paralysis to disorders of menstruation is still doubtful. A case where it was attributed to this cause has been lately published by Winginroth. Feilchenfeld (*W. kl. R.*, 1906) has found accommodation paralysis after hot packs continued too long.

With regard to the *toxic origin* of ocular paralysis, we know a number of poisons which act upon the pupillary and accommodation nerves. *Chronic alcoholism* may undoubtedly have a paralysing effect upon the ocular nerves and may produce a neuritis in them (the nuclear ocular paralysis due to alcohol will be specially discussed). Diabetic paralysis of the ciliary and other eye muscles is not uncommon. Dieulafoy[4]

[1] *Thèse de Bordeaux*, 1902-1903.
[2] *A. f. Aug.*, Bd. xlviii.
[3] *Monatsschr. f. Ohr.*, 1903.
[4] *Presse méd.*, 1905.

has recently published some cases of this kind. This paralysis has also been observed in gout, but only in very rare cases (*e.g.* by Galezowski). *Lead poisoning* affects the ocular nerves only exceptionally. Hammer [1] has recently collected the cases of this kind. *Fish*, *meat*, and *sausage* poisoning first paralyse the ciliary muscle, then the sphincter pupillæ, and the external muscles of the eye (cases of Cohn, Leber, Guttmann, Scheby-Buch, Ermengem, David, Bylsma, etc.). Accommodation paralysis and mydriasis have also been ascribed to oyster-poisoning and auto-intoxication from the intestine (Panas). Ophthalmoplegia has occasionally been observed after mushroom poisoning (Weiss). It has also been ascribed to aconite poisoning. In one case Baas attributed the ophthalmoplegia to atropine which had been instilled into the eye. It is not certain whether nicotine has any effect in causing ocular paralysis (Hammer). In these forms of ocular paralysis, the disease is usually a nuclear one, of functional or organic nature.

The cases which have been published during the last few years describing the onset of ocular paralysis following *lumbar anæsthesia* with stovain and novocain are of very special interest.

I had an opportunity of examining one of the first of these cases, that of Loeser (*Med. Klinik.*, 1906). Other cases have been published by Adam, Feilchenfeld (*D. m. W.*, 1906), Landow, Mühsam (*D. m. W.*, 1906), Lang, Ach (*M. m. W.*, 1907) and others. The abducens was usually affected, the trochlearis less often. The paralysis generally appeared a day or two after the injection.

Lévy-Baudouin (*R. n.*, 1907) saw ocular paralysis produced in the treatment of trigeminal neuralgia by injections by Schlösser's method.

Compression of the nerve by tumours (tumour-metastases may also cause paralysis by their localisation in the ocular muscles themselves, as Elschnig has shown), aneurisms, meningitic exudates, hæmorrhage, fragments of bone, periostitic thickenings, inflammation of the cellular tissue of the orbit, etc., is a common cause of this paralysis. *Injuries of the orbit* and the skull (basal fractures) paralyse the ocular nerves by means of fracture and accompanying hæmorrhage, but contusions may also have a directly paralysing effect, even without any fracture of the bone, probably because, owing to some constitutional predisposition, they cause hæmorrhage within the nerves. An isolated trochlear paralysis due to this cause has been described by Klein. The pupillary fibres may be affected independently by contusion of the eye-ball and in injuries of the skull (Axenfeld [2]). Operations on the base of the skull, especially extirpation of the Gasserian ganglion in trigeminal neuralgia, have frequently led to injuries of the oculo-motor and abducens (Krause, Friedrich, Cushing, etc.). In a case which I was able to examine later, the abducens was also injured. *Surgical treatment* of diseases of the frontal sinus may endanger the superior oblique muscle. Pressure of the forceps during labour gives rise in rare cases to ocular paralysis affecting the levator palpebræ superioris and the superior rectus. *Arterio-sclerosis* of the basal cranial vessels may directly cause compression of the ocular nerves or may by the sinuosity of the main artery cause tension and traction of their branches, and thus give rise to crushing and strangulation of the nerves which pass over it. This, however, is an unusual mode of origin. I have seen the combination of trochlear paralysis with intermittent

[1] *Z. f. N.*, xxix.

[2] *D. m. W.*, 1906.

claudication. Frankl-Hochwart [1] found post-mortem a neuritis of the oculo-motor nerve in arteriosclerosis.

Primary hæmorrhages in the oculo-motor nerves are extremely rare, but cases have been observed which can only be explained in this way. Thus I have seen in a young, non-syphilitic man who suffered from profuse bleeding at the nose, the sudden onset of trochlear paralysis with vertigo and apoplectiform symptoms, which gradually disappeared. A paralysis of the left abducens occurred suddenly in a woman who had for years suffered from a left-sided hemicrania: at the height of a particularly severe attack, while she was sitting in a draught of air, the left side of the head became congested. Saenger found hæmorrhages in the oculo-motor nerve in tuberculous meningitis. Schlesinger [2] saw paralysis of several ocular muscles during a case of scurvy.

It hardly needs to be mentioned here that ocular paralysis is a common symptom in tabes, disseminated sclerosis, brain tumours, etc., and also in diseases of the pons and corpora quadrigemina. It is only exceptionally, as in a case examined by Dejerine and Petrén, that tabetic ophthalmoplegia is due to peripheral neuritis.

We have no definite evidence in favour of a reflex origin of ocular paralysis. A spastic mydriasis may be produced by foreign bodies in the ear and nose (Frenkel, Moos, Bonnier, Baudelier, Sabrèzes, *R. n.*, 1903).

Finally, there is a *congenital* and *hereditary* form of ocular paralysis, which affects some of the branches of one or both sides (levator palpebræ superioris, superior rectus), or less frequently all the external muscles of the eye (Gräfe, Mauthner, Möbius, Kunn, Gourfein, Bernhardt, Neurath, etc.). There is very little definite evidence as to their pathological cause. Absence or degeneration of the muscles has been found (Heuck); in other cases (Axenfeld) they were quite normal but the eye was fixed by a band of elastic tissue. Siemerling [3] noted nuclear atrophy in a case of congenital ptosis. This was in agreement with Möbius' view of "infantile nuclear atrophy." Heubner [4] found aplasia of the corresponding nuclear region in a case of this kind. I have seen several persons in whom other developmental malformations of the eyes were associated with the congenital oculo-motor paralysis. In the child of a woman who had bilateral coloboma and prognathism, I found mydriasis, absolute immobility of pupils, and paralysis of accommodation, as congenital symptoms.

It should be noted that this hereditary, familial, ocular paralysis may appear in later life, even in the fifth decade (Charcot-Dutil, Delord).

Axenfeld and Schürenberg [5] ("Zyklische angeborene Okulomotoriuserkrankung") observed a very uncommon combination of congenital oculomotor paralysis with spasms recurring regularly at short intervals in the levator palpebræ, sphincter iridis, and ciliary muscles. Fuchs has described a similar condition. Congenital oculomotor paralysis with continuous rhythmical changes in the pupils was observed by Lewinsohn (*D. m. W.*, 1907).

Harvey's statement (*Brit. Med. Journ.*, 1907) that the abducens nerve may be absent and that the external rectus muscle may then be innervated by the oculomotorius is worthy of note.

I might also point out that patients very often blame prolonged and excessive weeping as the cause of an ocular paralysis which sometimes affects the ciliary muscle, sometimes the sphincter iridis or an external eye muscle. Although I have generally discovered other causes, we cannot quite dismiss the possibility of such a causal relation. It is still doubtful whether overstrain of the eye muscles, *e.g.* of the accommodation, can lead to its paralysis, as Jacqueau and others think.

[1] *Obersteiner*, 1907.
[2] *D. m. W.*, 1907.
[3] *A. f. P.*, xxiii.
[4] "Über angeborenen Kernmangel," Berlin, 1901.
[5] *Klin. Mon. f. Aug.*, 1902, and *C. f. N.*, 1905.

I have frequently failed to find any cause for paralysis of the accommodation. This happened in some of the cases which I observed along with Michel and Helbron. In one, which Helbron has reported, we thought the cause to be a pyorrhœa alveolaris; in another we could only find a congenital malformation of the skull. Among 3000 cases of eye disease Helbron (*B. k. W.*, 1903) found 103 of isolated accommodation paralysis, of which thirty-seven were due to diphtheria.

Symptoms.—The symptoms of abducens and trochlear paralysis have already been described in the general part.

Total oculomotor paralysis is characterised by the following symptoms: *Ptosis*, paralysis of the *superior*, *inferior*, and *internal rectus*, the *inferior oblique*, the *sphincter pupillæ*, and the *ciliary muscle*. The upper eyelid droops so much that it covers the pupil. The lid can be raised slightly by overaction of the frontalis, but not if the eyebrows are kept unmoved. The eyeball can only be moved outwards, and every attempt to change its position moves it out to the external canthus, or outwards and downwards with some rotation produced by the action of the trochlearis. In course of time the eye comes to be permanently deflected to the outer canthus by secondary contracture of the external rectus. The pupil is of medium size and does not react to light. It can be further dilated by atropine. It does not contract for convergence. Illumination of the unaffected eye has no effect upon the pupil of the paralysed eye. The eye is easily dazzled. Paralysis of the external eye muscles sometimes causes a slight degree of *exophthalmos*. *Diplopia* is present in the whole of the field of vision, if the upper eyelid is raised, and is associated with the well-known characters. Where ptosis is absent, the patient spontaneously closes the eye in order to avoid the diplopia. A facial spasm may even develop from this closing of the eyelid, as I have seen. There may be some *pain* along with the paralysis, especially if it is due to rheumatism or compression.

The oculomotor nerve and the muscles which it innervates cannot be tested electrically, but Wertheim-Salomonson ("Psych. en neur. bladen," 1901) and Bregmann have, in degenerative paralysis, elicited a sluggish contraction in the levator palpebræ superioris by direct galvanic stimulation, and we have done the same in a few cases.

In incomplete paralysis of the third nerve, the corresponding movements are merely limited. It more often happens that some branches are completely paralysed, whilst others are merely paresed. *Partial oculomotor paralysis* is still more common. The paralysis may be limited to the superior rectus and the levator palpebræ superioris, or it may involve only one of the other recti, or all the muscles of the eye except the levator palpebræ superioris or the internal rectus, or finally it may be limited to the *internal* ocular muscles, *i.e.* to the ciliary muscle alone or in combination with the sphincter iridis.

In congenital ptosis a peculiar phenomenon of associated movements is occasionally observed, viz., the levator palpebræ superioris, which is not under voluntary control, can be brought into action by contraction of the jaw muscles, especially in opening the mouth or in contraction of the pterygoids of the same side, the eyelid being thus raised (Gunn, Helfreich, Bernhardt, Miller, Higier, *Z. f. N.*, xxi., etc.).

Sölder in the same way has seen the innervation radiating from the muscles which produce lateral deviation, etc., to the paralysed levator. G. Flatau has described a case from my "Polyclinic" in which convergence of the eyeballs occurred when the jaws were opened; the patient was hysterical. A. Westphal (*B. k. W.*, 1904) saw the eyeballs turn upwards in a case of paralysis of the levators under the influence of energetic contraction of the orbicularis.

As a rule *all* the muscles are more or less completely paralysed in diseases of the *ocular nerves*, whilst in nuclear affections there is usually unilateral or bilateral paralysis of some of the muscles, the others being intact. Thus a nuclear lesion is probable when there is bilateral paralysis of the sphincter pupillæ and the ciliary muscles, and also when these internal muscles act normally while all the external eye muscles on both sides are completely paralysed. This criterion is, however, *by no means absolute*, and a paralysis of some of the branches, *e.g.* of the sphincter iridis, may merely be the residue of a total peripheral (basal) oculo-motor paralysis, as I have repeatedly observed in syphilis. The sphincter iridis may also escape in basal lesions of the oculo-motor nerve, without warranting the assumption (with Adamük) that the pupillary fibres have an abnormal course. Dammron and Meyer, as well as Dejerine, found peripheral neuritis of the oculomotor nerves in a case in which the ocular paralysis exactly simulated a nuclear disease.

Bach is of opinion that according to recent experience the indications which were formerly regarded as characteristic of a nuclear ocular paralysis cannot now be always so interpreted, and that the diagnosis of a nuclear should often be replaced by that of a fascicular ocular paralysis.

The involvement of other, especially of neighbouring cranial nerves, points to the disease being a peripheral one.

Disease of the ocular nerves hardly ever in itself gives rise to associated paralysis. Occasionally, however (Thomsen, Ormerod), a paralysis restricted to the levators has been observed in basal disease of the oculomotor nerve. Fuchs and Silex [1] describe a progressive paralysis of the levator palpebræ superioris, appearing in later life, which they attribute to a primary myopathy of this muscle. Senile atrophy of the eye muscles has been reported by Thiele and Grawitz.[2] Hereditary ptosis sometimes develops after birth or even later. As to convergence and divergence paralysis, etc., see p. 79.

The *course* and *prognosis* depend essentially on the cause and character of the fundamental disease. So-called rheumatic ocular paralysis almost always terminates in recovery, and the form which follows acute infective diseases also generally disappears within a few weeks. This is specially the case as regards post-diphtheritic paralysis, although isolated cases have been reported in which the paralysis of accommodation did not recover (Mühsam, Helbron). Ocular paralysis occurring after influenza may be very obstinate. The syphilitic form affords on the whole a favourable outlook. If it has not been so long in existence that atrophy has developed, suitable treatment may bring about more or less complete recovery. In cases of injury the prognosis depends upon the severity of the lesion. In non-specific tumours the prognosis is unfavourable, especially as tumours at the base of the brain (except retrobulbar) are as a rule inoperable. According to present experience, paralysis following lumbar anæsthesia disappears rapidly and spontaneously. Ocular paralysis occurring in multiple neuritis almost always passes off, if the disease runs a favourable course. The ocular paralysis of tabes tends, especially in the first stages, to spontaneous recovery, but it may be a permanent symptom, or may even assume a progressive character. Diabetic ocular paralysis is often transient (Dieulafoy). *Isolated reflex immobility of the pupils* is a symptom of the

[1] *A. f. Aug.*, xxxiv. [2] *D. m. W.*, 1906.

gravest omen. It is in most cases a premonitory sign of tabes or paralytic dementia. It may also be the only sign of a *syphilitic brain disease.* It is rarely a symptom of chronic alcoholism.

The cases reported by Pilcz (*M. f. P.*, xxi.) are very remarkable and require careful consideration.

Schultze has found isolated immobility of the pupils in pneumonia, but in this case the immobility was apparently absolute. It cannot be doubted that reflex immobility of the pupils may also have a traumatic origin (Axenfeld). We do not, however, go so far as some French writers (Babinski, Brissaud), who always attribute isolated reflex rigidity of the pupils to syphilis. The *accommodation paralysis* which occurs spontaneously and is associated with mydriasis and usually with loss of the light reflex, is always of grave import. I know cases of this kind in which other signs of tabes or paralysis appeared even five to ten years later; but this paralysis is not invariably followed by a serious disease. Thus, after an interval of fifteen years, I have re-examined a patient who suffered from immobility of the pupils, probably due to previous cerebral syphilis, and could discover no fresh symptoms of the disease.

It should always be remembered that the symptom of mydriasis and immobility of pupils may be *artificially* produced by the use of atropine. I have seen a girl in whom I could at first find no cause for the maximum dilatation of the pupils, etc., until I discovered that she had regularly dropped atropine into her mother's eyes.

Treatment.—When there is a history of syphilis, causal treatment is urgently demanded. In my experience the most efficacious means of dealing with the rheumatic, infective, and toxic forms of ocular paralysis is that of *diaphoresis.* In the traumatic or rheumatic forms local *antiphlogistic treatment*, or *blood-letting* are suitable; vesicants (a fly-blister behind the ears) may also be tried. In diabetes, gout, etc., appropriate *diet* should be prescribed.

Electrical treatment of ocular paralysis has not been strikingly successful, but where causal treatment cannot be carried out, it should be employed. The galvanic method, is most recommended, the cathode being placed on the closed eye and the anode on the neck. Both electrodes may also be applied to the temporal region, or to the neighbourhood of the paralysed muscle; *e.g.* to the frontal region over the eye, etc., in paralysis of the superior rectus. The current should be strong enough to cause the facial muscles to contract on cathodal closure, and should be applied for two to three minutes. Faradic treatment is less employed. The attempt to stimulate the muscles directly by fine electrodes applied to the conjunctival sac should never be made. Subcutaneous injections of strychnine may in some cases have a curative effect.

In order to avoid the troubles arising from the diplopia, it is advisable to order spectacles with an opaque glass which will prevent the affected eye from seeing. The eye may also be entirely closed by a bandage. The use of strong prisms to unify the double images should be discouraged; when the distance between them is small, weak prisms may be employed. Gymnastic exercises are of doubtful value: they consist in gradually passing an object out of the field of single into that of double vision, whilst the patient endeavours to maintain his single vision as long as possible.

As to *surgical* treatment of ptosis and ocular paralysis, the text-books on ophthalmology should be consulted.[1] The patient usually contrives for himself some way of raising the paralysed eyelid. Thus, a young man suffering from double ptosis had fastened on to the frame of his spectacles a projecting bar which pressed upwards against the lids and kept them open. Ptosis spectacles of various kinds are recommended.

Periodic Oculomotor Paralysis [2]

Cases have been observed (Gubler, Camuset, Saundby, Möbius, Senator, Pflüger, Vissering, Charcot, Manz, Ballet, Schweinitz, Paderstein, Mingazzini, Strzeminski, Mering, Mathis, Kollarits, Russell, Lapersonne, Ryba,[3] Spiller-Posey,[4] Fischer,[5] etc.) in which there occurs from time to time, at regular, or, less frequently, at irregular intervals, a paralysis of the oculo-motor nerve, which within a few days, weeks, or even

Fig. 220.—Case of periodic oculo-motor paralysis during attack. (Oppenheim.)

Fig. 220a.—The same case during the period between the attacks.

months partially, or it may be entirely, disappears. Young people and *children* are specially affected; and no nervous heredity can as a rule be discovered. We have, however, seen this disease in a young man who suffered from stuttering, and revealed his neuropathic disposition by the presence of medullated nerve fibres on the disc of the same eye. Schilling [6] has studied and published the case which was sent to us by Michel (Figs. 220 and 220a). In another of my cases the father of the patient was an inebriate and the mother suffered from migraine. This paralysis, which always affects the same oculomotor nerve (and does not pass from one side to the other), is almost always associated with *headache* or pain in the eye, the brow, or in the whole forehead, corresponding to the side of the paralysis, and with nausea and vomiting. The headache has usually the character of an *attack of migraine*, is *repeated* every fourth week or at longer intervals, and is

[1] Of the recent works on this subject see, for instance, Landolt, "Arch. d'ophth.," 1903.

[2] See literature in Wilbrand and Saenger, "Die Neurologie des Auges," Wiesbaden, 1900; vol. i.; also Leclézio, "Contribution à l'étude de la Migraine ophthalmoplégique." *Thèse de Bordeaux*, 1904-1905.

[3] *W. kl. R.*, 1904.

[4] *Amer. Journ. Med. Sc.*, 1905.

[5] *Rev. of Ophthal.*, 1907.

[6] *M. m. W.*, 1903.

sometimes or always accompanied by oculomotor paralysis. As a rule the headache introduces the attack and disappears when the paralysis appears. Unlike typical migraine, the headache and sickness may last for a week. This paralysis usually affects the whole oculomotor nerve, but occasionally some branches are spared (those for the internal muscles, for instance). Indeed, the paralysis may be restricted to the levator palpebræ superioris (Knapp). Möbius appears to recognise only those cases in which there is unilateral total oculomotor paralysis. Diminution of sensation in the area of the *first branch of the trigeminus* was present in a few cases (Vissering, etc.). Karplus also saw involvement of the second. Hyperæsthesia was found by Kollarits in the area of the first branch ; in his case there was also a congenital amblyopia of the eye on the same side. Polyuria was observed by d'Astros during the decline of the attack.

There are further cases of *purely periodic* oculomotor paralysis, and others with *periodic exacerbations* (Senator). In the former the condition during the intervals was quite normal ; in the latter a paresis of the oculomotor nerve or of some of its branches (sphincter pupillæ, superior rectus, etc.) persisted and at times developed into total paralysis. The paralysis of the first attacks may also entirely disappear, but some of the muscles become permanently affected. In Schilling's case there was complete paralysis in the first attack ; in the intervals it only partially disappeared (Fig. 220A), to again become complete in each subsequent attack. Möbius does not admit Senator's distinction, as a certain degree of paralysis usually persists in the free intervals. It also appears from Mingazzini's review that the purely periodic form may develop into one of periodic exacerbations.

The authors do not say much as to the exciting causes ; in one of our cases the first attack occurred after a bath, the patient being at the same time excited by a thunderstorm, and it was associated with convulsions.

We know nothing definite as to the cause of this disease. Views differ even as to its site, some investigators, such as Brissaud, regarding it as a nuclear disease—Mingazzini as a nuclear or root disease—whilst the majority believe it to be a basal affection. In the cases examined post-mortem (Gubler (?), Weiss, Thomsen-Richter, Karplus), a morbid process was found on the oculomotor nerve trunk, in one case a plastic *exudate*, in the others a *new growth* (tubercle, fibrochondroma, neuroma). Many explanations of the disease have been suggested. Functional disorders, local hyperæmia, vascular anomalies, etc., have been suspected. Möbius, who at first assumed that the disease was of nuclear origin, thought later that the cause was an organic process, a new growth in the region of the oculomotor nerve. Schmidt-Rimpler is of the same opinion. The most plausible view seems to me to be the one based on Charcot's opinion, viz., that periodic oculomotor paralysis is allied to *hemicrania*, and like it is presumably due to *vasomotor* processes. The vascular spasm inhibits the flow of blood to the nerves and thus produces the paralysis. Or there may be paralysis of the vasomotor nerves, the distension of the vessels by blood producing compression of the nerve. Such attacks may be often repeated without causing injury to the nerve, but in the end *degenerative* and inflammatory processes ensue, and these preclude complete recovery. It is also comprehensible that such dis-

turbances of the circulation, frequently repeated, might be the exciting cause of exudative processes and new formations (especially fibrous). Strzeminski distinguishes between a functional form and one with an organic basis. Wilbrand and Saenger, like Marina, do not regard the symptoms as sufficient to constitute an independent disease. Luzenberger has suspected periodic swelling of the cavernous sinus; Plavec,[1] in the same way, blames the hypophysis. Charcot, remembering the close relation of the disease to migraine, speaks of an *ophthalmoplegic migraine*. A case of Seiffer's is also explained in this way. Karplus, Mingazzini, and especially Möbius do not accept this view. The latter emphasises the fact that the headache which accompanied the paralysis is not identical with typical hemicrania. He much prefers to make a distinction between hemicrania with oculomotor paralysis and periodic oculomotor paralysis. Cases of hemicrania have been observed (Oppenheim, Massalongo, etc.) in which there was temporary paralysis of a single eye muscle, *e.g.* of the levator palpebræ superioris, the sphincter pupillæ, etc. (see chapter on hemicrania).

Periodic abducens paralysis (Schweinitz) and periodic trochlear paralysis (Luzenberger) have been described. Bechterew's case of bilateral paralysis of the ocular muscles with periodic exacerbations does not belong to this section, nor Demicheri's case of alternating ocular paralysis. Trömner's case of ophthalmopleiga interna in ophthalmoplegic migraine also seems to me uncertain.

The *course* is in some cases progressive, but it should not be forgotten that the disease may be arrested or completely cured. The *prognosis* only becomes grave when, on long observation, the disease is seen to be progressing. The prognosis as to life is always doubtful, as the cause of the disease may be a new growth. The *treatment* should be similar to that of migraine and paralysis of the eye muscles.

Paralysis of the Trigeminal Nerve

Anatomy.—The trigeminus passes out of the pons or the ventral surface of the middle cerebellar peduncle by *two* roots, one small anterior, containing only motor fibres, the other posterior, distinctly larger, and purely sensory. They lie close together, the motor root running below and internal to the sensory. They pass through an opening in the dura mater external to the point where the abducens nerve pierces it, and enter a hollow space (cavum Meckelii) situated above the tip of the petrous bone, and formed by the dura mater on the upper surface of this bone. The cavity is, therefore, extra-dural. "It lies to the outer side of the cavernous sinus, and extends forwards and outwards from the impressio trigemini on the petrous bone to the inner angle of the sphenoidal (superior orbital) fissure, and to the foramen rotundum and the foramen ovale." In this space the posterior root passes into the *Gasserian ganglion*, from which again originate the three divisions of the trigeminus, whilst the anterior root passes to the inferior surface of the ganglion (without giving off fibres to it), and on its distal side joins the third division which arises from it. According to recent investigations, the sensory root of the trigeminus arises from the Gasserian ganglion and penetrates into the pons, descending hence into the spinal cord in order to form the spinal—previously termed the ascending—root. It therefore degenerates in a descending direction (Sherrington, Biedl, Bastanielli). With the first division (the *ramus ophthalmicus*) there pass to the eye or to the ciliary ganglion sympathetic fibres which innervate the dilator of the pupil (and the non-striped muscles of the upper eyelid). The second division (the *ramus supramaxillaris*) is connected by the sphenopalatine nerves with the *sphenopalatine ganglion* (Meckel's ganglion), which is united through the *Vidian* and great *superficial petrosal nerve* with the geniculate ganglion of the *facial* nerve. The third division is connected with the otic ganglion; from this originates the *small superficial petrosal nerve*, which sends a branch to the

[1] *Z. f. N.*, xxxii.

knee of the facial, and by the *tympanic* or *Jacobson's plexus*, in which it is continued, enters the *glosso-pharyngeal* nerve. Fine fibres are also found connecting the otic and the spheno-palatine ganglia with the Gasserian ganglion.

The first and second divisions of the trigeminal contain only sensory, the third contains also motor fibres. The first division innervates the skin of the head in the area marked 1 (Fig. 221) from the palpebral fissure to the vertex, also the conjunctiva, cornea, iris, the mucous membrane of the frontal cavity, and part of the nasal mucous membrane. The trigeminus also contains *secretory* fibres for the lachrymal glands. These, according to other authors (Goldzieher, Jendrassik, G. Koester [1]), arise from the facial, leave it along with the great superficial petrosal nerve, and reach the orbital branch through the sphenopalatine ganglion.

Koester does not regard the facial nucleus as giving rise to these fibres, but thinks they originate in the glossopharyngeal nucleus, or the pars intermedia. Landolt [2] leaves this question undeter-

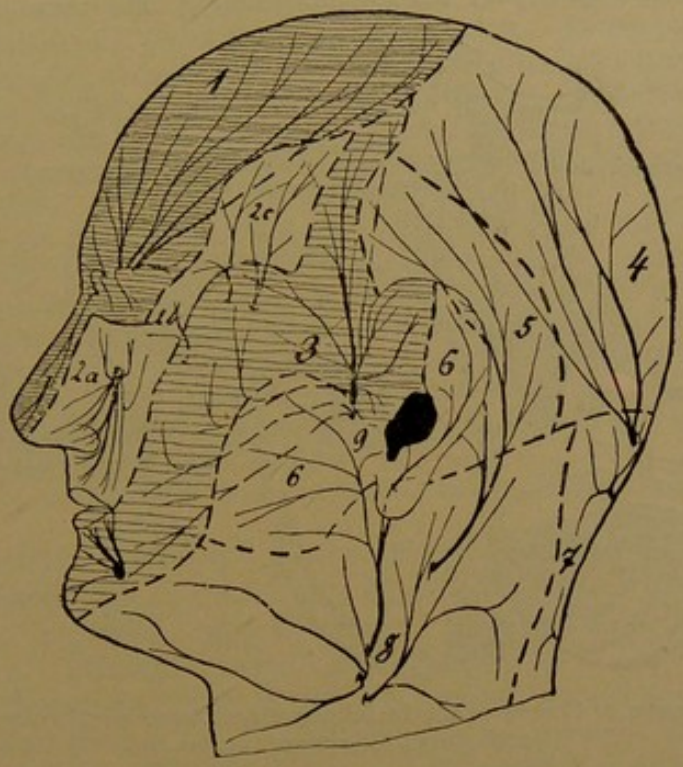

Fig. 221.—(After Frohse.) 1. First branch of Trigeminus, 2. Second branch of Trigeminus, 3. Third branch of Trigeminus. The cross-lines indicate the area of the first and third branches of the trigeminus, the black shows the area of the auricular branch of the vagus nerve in the outer ear. 2a, N. infraorb. 2b. N. zygomaticofac. 2c N. zygomatico-temp. 3. N. auriculo-temp.

For the other figures consult p. 57.

mined. Some authors (Saenger, etc.) assume a mixed innervation of the tear glands by the facial, trigeminus, and perhaps also by the sympathetic, and suppose that each of these nerves has a special function. Cushing [3] ascribes the diminution of the secretion of tears after extirpation of the Gasserian ganglion to the sympathetic.

The second division innervates the skin of the face between the palpebral and the oral fissures (2a, 2b, 2c, Fig. 221), the mucous membrane of the upper jaw, the naso-lachrymal duct, part of the mucous membrane of the nose, the palate as far as the palatopharyngeal arch (which is innervated by the glossopharyngeal nerve), the middle ear, and the antrum of Highmore, and contains sensory fibres for the teeth of the upper jaw, and probably also taste fibres.

The third division innervates the skin of the lower jaw, of the external ear and the cheeks, the underlip, and the lower teeth, and also contains motor fibres for the *masseter*, the *tensor tympani*, the *tensor palati*, the *mylohyoid*, and the anterior belly of the *digastric* muscles.

Frohse and Zander have shown that there is considerable variability in the distribution of the sensory cutaneous branches of the trigeminus. According to Frohse there is no spot of skin

[1] *A. f. kl. M.*, Bd. lxxii. [2] *Pfl. A.*, Bd. xcviii. [3] *Journ. Amer. Med. Assoc.*, 1905.

on the lateral surface of the face that is not innervated now by one, now by another of the nerves in question. Sometimes all three branches emulate each other as regards the innervation of a certain cutaneous area; at other times the cervical nerves and the auricular branch of the vagus take part, so that the spheres of innervation of the auriculotemporal, the great auricular, and the auricularis vagi overlap each other to a variable extent. The great auricular often innervates the skin of the whole region over the masseter and parotid. Zander established the remarkable fact that the median area of the face has a bilateral innervation, as the main branches extend beyond the middle line. F. Krause's [1] observations confirm this view. Davies in his exhaustive research (*Br.*, 1907) also arrives at similar conclusions. The views of Ivy-Johnson (Univ. of Penn., 1907), who after extirpation of the Gasserian ganglion found no change in the deep sensibility and endeavoured to explain the symptom by Head's theory, are also worthy of note. See also Pruschinin (*Inaug. Dissert.*, Berlin, 1906).

Vasomotor fibres also accompany the trigeminal nerve. This nerve has no real influence

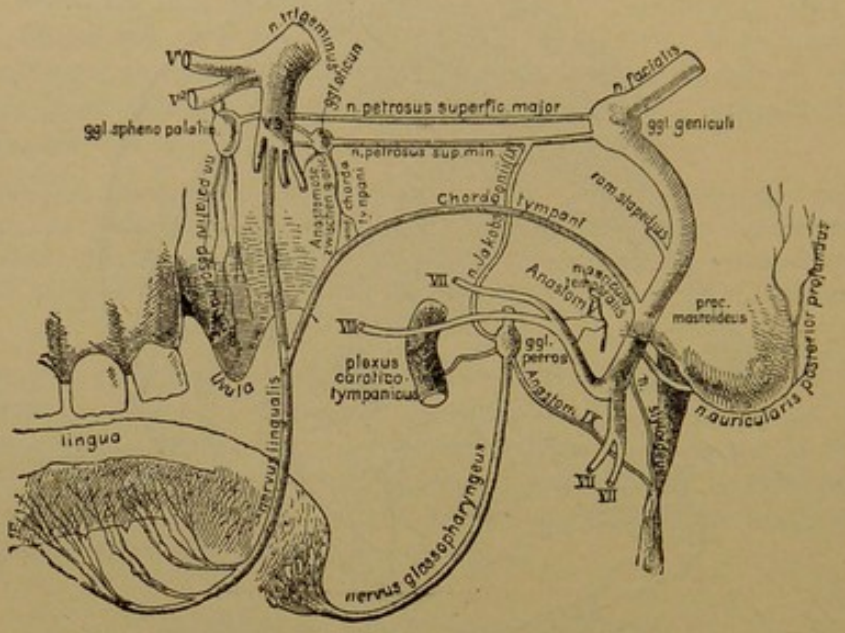

FIG. 222.—(After Leube.) Course of the facial nerve and its connections with the trigeminal and glosso-pharyngeal nerves.

upon the pupil, although Kreuzfuchs has recently assumed that a centripetal tract for dilatation lies in the first branch.

There is still great uncertainty as to the course of the *taste fibres* (see Fig. 222). Those for the anterior two-thirds of the tongue are contained in the *chorda typani*, those for its posterior part and the palate, in the *glossopharyngeal nerve*. The further centripetal course of these fibres is still doubtful. According to Schiff, Erb, Wallenberg, etc., the fibres of the chorda pass out of the *facial* by means of the *great superficial petrosal* nerve and the sphenopalatine ganglion into the second branch of the *trigeminus* and with it to the brain. Others, such as Ziehl and Müller, think the taste fibres of the chorda enter by means of the small minor superficial petrosal nerve and the otic ganglion into the third branch of the trigeminus, a view which is supported by observations of F. Krause and Kron. There are, however, published cases of central disease of the trigeminus without affection of the taste. According to other opinions (Brücke) the chorda fibres pass from the geniculate ganglion by means of the small superficial petrous nerve, the tympanic plexus, and Jacobson's nerve, into the petrous ganglion, and thence into the trunk of the *glossopharyngeal*, so that this may be regarded as a true nerve of taste. Landois thinks the fibres reach the glosso-pharyngeal by another way. Disturbances of taste in lesions of the glossopharyngeal have, however, only been observed in rare cases (Lehmann, Ziehl, Pope).

In one case only, that from my "Polyclinic" described by Cassirer (*A. f. A. u. Phys.*, 1899, Suppl.) had an affection of the glossopharyngeal nerve produced a total hemiageusia. This proves

[1] "Die Neuralgie des Trigeminus nebst der Anatomie und Physiol. des Nerven." Leipzig, 1896.

that the glossopharyngeal may contain all the taste fibres, whilst cases by Bálint (*N. C.*, 1905), Spiller (Univ. of Penn., 1903) and others show that taste may be conserved on the anterior third of the tongue although the glossopharyngeal is destroyed.

In sharp contrast to these theories is another which assumes that, in addition to the chorda fibres, the taste fibres of the peripheral glossopharyngeal also enter the trigeminus by means of the small superficial petrosal nerve (Gowers). This is supported by a case of Wallenberg's, who thinks with Kohnstamm that the taste fibres reach the solitary bundle through the pars intermedia. This view is contradicted by several observations, *e.g.* that of Bruns (*A. f. P.*, xx.). Finally the taste fibres have been assigned to the *facial* nerve or its pars intermedia (Lussana, Kohnstamm). Nageotte, and also Weigner (Anat., Hefte xxix.), trace the course of the taste fibres of the chorda through the pars intermedia.

We cannot here discuss the theories of Schultze, Stich, etc. Aural surgeons (Urbantschitsch, Schlichting) have concluded from their observations that all the taste fibres pass through the tympanic cavity. The fibres of the chorda and tympanic plexus are not infrequently injured in operations upon this region, thus producing an affection of taste; and according to Schlichting ageusia may appear in the anterior part of the tongue, varying in extent from one-third to four-fifths, in lesion of the chorda, and on the posterior parts of the tongue and palate in injury of the tympanic plexus. See also E. Maier (*D. m. W.*, 1904). Prévost has made experimental investigations of this kind.

The majority of the cases observed agree with the view of Erb and Schiff, but it cannot be doubted, especially in view of the evidence derived from surgical operations on the trigeminal nerve and Gasserian ganglion, that the course of the taste fibres is subject to *individual variations*. This is perhaps the reason why Cushing,[1] for instance, would deny the relation of the trigeminus to the conduction of taste, although he, like Gowers, has collected statistics of cases in which the Gasserian ganglion had been removed. Davies comes to a similar conclusion and suggests that the pars intermedia is the nerve for taste. Vintschgau, Köster, etc., assume that there are special fibres for the various qualities of taste.

Opinions regarding the course of the sweat fibres of the face are still at variance.

Primary isolated diseases of the *trigeminal* nerves are very rare, neuralgia excepted, but cases of this kind have been reported by Müller, Archer, Ferrier, Schmidt, and Gowers. Primary neuritis of this nerve is specially rare. Hirschl in one case regarded a paralysis of the whole trigeminus as rheumatic, and Kutner[2] reports a case of a similar kind, ascribing the symptoms to an inflammatory process in the Gasserian ganglion. Kaufmann, Hösslin, Rad, and Berger,[3] describe a "neuritis" of several cranial nerves, which involved the trigeminus. Gruber also regarded the affection as rheumatic in a patient who suffered from paralysis of the facial and motor trigeminus in the course of an otitis. Multiple neuritis very rarely extends to the fifth nerve and its branches,[4] *e.g.* in exceptional cases of post-diphtheritic paralysis. On the other hand, this nerve is frequently involved in morbid processes developing at the base of the brain, especially when these extend into the middle and posterior cranial fossa from the bones and meninges (fracture, inflammation, and new growths). The various branches may also be affected by diseases occurring in the sphenoidal (superior orbital) fissure or the orbit, in the sphenopalatine fossa, in the sphenomaxillary (inferior orbital) fissure, and in the upper and lower jaw.

Tumours and chronic meningitic processes at the base of the brain are specially apt to extend to the trunk of the trigeminus, the Gasserian ganglion, or the branches. This is particularly common in cases of gummatous meningitis. A *gummatous neuritis* of the fifth nerve has sometimes been noted. *Caries* of the bones of the base of the skull, especially

[1] *Journ. Amer. Assoc.*, 1905. [2] *M. f. P.*, xvii. [3] *N. C.*, 1905.

[4] Burr and M'Carthy found disease of the Gasserian ganglion in a case of multiple neuritis.

of the sphenoid bone, often gives rise to symptoms in the region of the trigeminus. Vaissade [1] described neuritis of the fifth nerve due to tuberculosis. The first division of the nerve is affected by tumours developing in the region of the sphenoidal fissure or arising from the pituitary body, by *aneurisms of the internal carotid*, by orbital tumours, by thrombosis of the cavernous sinus (in one case the compression was due to a varix of the sinus), etc.; the second and third divisions, by tumours on the floor of the middle cranial fossa and of the sphenomaxillary fossa.

Affections of the peripheral part of the trigeminus are often of traumatic origin. Injuries of the orbit, of the base of the skull, especially *basal fractures*, may cause laceration, crushing, and compression of the fifth nerve and its branches, apart from the fact that the peripheral branches in the face may be affected by any accident. The trigeminus and its branches have also, especially of late, been injured by surgical operations, whether resection, extraction by Thiersch's method, or excision of the Gasserian ganglion. Injections into the nerve stem or its neighbourhood by Schlösser's and other methods also undoubtedly tend to produce morbid processes of a neuritic kind in the affected nerves, although they seldom cause permanent injury.

It can only be mentioned here that the trigeminus is often affected in diseases of the brain and spinal cord, especially in tumours, hæmorrhages, softenings in the pons and medulla oblongata, and in tabes dorsalis, syringomyelia, etc. In diseases of the pons the sensory and motor portions may be affected, whilst in spinal cord diseases it is only the sensory roots that are involved as a rule.

Symptoms.—If all the divisions of the trigeminus nerve are involved there are disturbances of sensibility and motility, secretion, and frequently also trophic symptoms. The anæsthesia extends over the whole of the cutaneous area innervated by the nerve, to the mucous membrane of the conjunctiva, cornea, nose, mouth, tongue, cheek, and palate. Cases of F. Krause,[2] Cushing,[3] and others prove, however, that even after complete destruction of the fifth nerve the sensory symptoms may be limited to a very much smaller area than one would expect from the description already given of the distribution of this nerve. In the future Head's teaching will have to be taken into consideration in this relation. The secretion of tears is suppressed on the affected side. This seems only to occur, however, if the lesion of the nerves is in the sphenopalatine ganglion or in its peripheral distribution (see above). The mucous membrane of the nose also ceases to secrete, and the sense of smell may suffer on account of the resulting dryness. Krause agrees with Magendie's view that the trigeminus takes part in the function of smell. The insensibility of the conjunctiva causes absence of the reflex of the eyelids. The corneal reflex is also absent, and this symptom may, in my experience, be the first sign of disease of the fifth nerve. The sneezing reflex and the palatal reflex tend to disappear. The mucous membrane is insensitive to the fumes of chloride of ammonia and other irritating substances. A vessel placed between the lips is only felt upon the unaffected side and causes a sensation as if it were broken. The patient tends to bite his cheeks. The sense *of taste* is not always impaired. In some cases it has been

[1] *Thèse de Lyon*, 1902.

[2] *Loc cit.*

[3] "The Sensory Distribution of the Fifth Cranial Nerve," Johns Hopkins Hosp., 1904. See also Spiller and Frazier, Univ. of Penn., 1901; Davies, *Br.*, 1907.

found to be quite abolished, in others to be absent only on the anterior two-thirds of the tongue, and in a few to be normal (see above).

In an old gentleman there developed along with paræsthesiæ in the extremities and pain in the tongue, hemiageusia and hemianæsthesia of the left half of the tongue, and diminution of the tendon reflexes. I suspected that the case was one of atypical polyneuritis, mainly involving the lingualis, and due to some unknown toxin.

Of the *trophic* disturbances which sometimes accompany diseases of the trigeminus, the most common is *herpes*, but it seems to occur only in a certain form of neuritis. Neuritis of the ophthalmic division may lead to very painful *zoster ophthalmicus*, which may endanger the eye. The disease of the cornea known as *neuroparalytic keratitis* is most remarkable and most difficult to explain. It consists first in a dulling of the cornea, followed by formation of ulcers, perforation, and finally inflammatory destruction of the eyeball. This symptom, first described by Magendie, was thought to be due to the anæsthesia and to consequent injuries to the insensitive eye. This corresponded to the fact that it did not appear if the eye were kept closed and the entrance of any foreign body thus prevented. This did not, however, occur in every case ; and the keratitis was also not infrequently absent, although the anæsthesia was complete.

Meissner thought he had found the trophic fibres in a special bundle of fibres, section of which caused inflammation. This was also disputed (Senftleben). Later, vasomotor influences (Schiff) were thought to be responsible, and Gaglio has recently been led by the results of his experiments to attribute great importance to them. Other experimenters have shown that it is only when the lesion is in the ganglion and the roots arising from it that keratitis is produced (Gaule). On the other hand dryness of the cornea from the blepharoplegia (xerotic theory), and penetration of micro-organisms into the eye (mycotic theory) have been blamed, but we must not confuse the keratitis due to dryness with the neuroparalytic form. The latest view, which Charcot has advocated, is mainly based on the experiments of Turner and the experimental observations on man by Krause (Cushing, etc.). According to these, keratitis is not produced by paralysis of the trigeminus, nor by suppression of a trophic influence, but by an *irritative condition* in the nerve associated with inflammation. The keratitis is thus not neuroparalytic, but neuritic. The anæsthesia of the cornea makes it less capable of resisting injuries, so that traumatic affections heal more slowly, but section of the trigeminus does not produce any kind of trophic disturbance (see pp. 66 *et seq.*). The fact that this affection of the cornea is most often observed in neuritis and in compression of the nerve by tumours, etc., corresponds with this view. Wilbrand-Saenger[1] have accepted this theory on the ground of the material which they have collected and carefully revised, and have shown that the onset of neuroparalytic keratitis is not connected with lesion of any special site, but may be produced by a condition of irritation at any part of the trigeminal nerve.

Other trophic disturbances occur under similar conditions, viz., ulcerations of the mucous membrane of the cheeks and nose, etc. There may be also spontaneous loss of teeth, which, however, is hardly ever noted except in central diseases (tabes). It is doubtful whether trigeminal lesions can cause otitis through trophic or vasomotor influences

[1] " Die Neurologie des Auges," etc.

(Asher), as has been produced experimentally by Baratoux and Berthold. I have seen two cases which seem to suggest that lesion of the trigeminus may cause exudative processes in the antrum of Highmore. It is improbable that the so-called facial hemiatrophy is a result of disease of the fifth nerve.

Motor symptoms only arise in lesions of the *anterior roots* and of the third division of this nerve. This is injured in removal of the Gasserian ganglion. Paralysis of the motor trigeminus affects the muscles of mastication, the *masseter*, *temporal*, and *pterygoid* muscles, whilst involvement of the other muscles which it innervates does not as a rule cause any perceptible symptom. Paralysis of the masseter and temporal may be recognised from the fact that they do not contract in masticating or in closing the jaws. This is easily ascertained by palpation. Peripheral paralysis of these muscles is usually accompanied by changes of electrical excitability, such as diminution or reaction of degeneration. This would seem to be the case from the condition of the muscular excitability in direct faradic and galvanic stimulation, but it has only been reported in a very small number of cases (Marinesco and Sérieux,[1] Oppenheim, etc.). Later there is also marked atrophy. In unilateral paralysis the lower jaw may be moved towards the affected side and deviates slightly towards it in opening of the jaws. The subluxation of the joint on the affected side, observed by Remak during this movement, has not been found by Krause. Paralysis of the digastric and mylohyoid muscles does not give rise to obvious symptoms, but the floor of the mouth feels more flaccid than on the healthy side. There are no symptoms pointing to involvement of the tensor palati, but Müller and Schmidt mention lowering of the palatopharyngeal arch on the affected side. Paralysis of the tensor tympani gives rise, so far as we know, to no symptoms. In bilateral paralysis of the motor trigeminus the jaw jerk is absent.

There are cases in which unilateral atrophy of the masseter muscles occurs spontaneously and without any known cause (Werner, Remak).

The above description refers to complete paralysis of the trigeminus, as it occurs in total laceration or section of the nerve, and therefore in complete solution of its continuity. In *simple compression* the paralytic symptoms are incompletely developed and are associated with *symptoms of irritation* (neuralgic pains, hyperæsthesia, etc.), which may precede it. Many writers are inclined always to attribute trigeminal neuralgia to an inflammatory affection of the nerve or of the Gasserian ganglion (see corresponding chapter). These symptoms of irritation are then followed by diminution of sensibility, especially diminution of sensibility to touch and pain, which gradually pass into complete anæsthesia.

We need not describe the symptoms in affections of the separate divisions of the nerve. We must remember, however, from the point of view of differential diagnosis, that the distribution of the anæsthesia in disease of the nucleus into which the sensory root of the trigeminus passes, differs entirely from that due to disease of the peripheral branches (Lähr, Schlesinger, Sölder).

Irritation of the peripheral sensory branches of the trigeminus may cause reflex facial spasm, coughing, and vertigo.

In accordance with the views of Filehne and Exner on so-called senso-mobility, there has been noted in anæsthesia of the trigeminus a

[1] *Arch. de Phys.*, 1893.

certain reduction of the range of the movements of the facial muscles, which never becomes marked paresis.

In trigeminal neuralgia (*q. v.*) voluntary suppression of the movements which give rise to pain may simulate paresis.

The *course* and *prognosis* depend on the nature of the primary lesion and require no special discussion. Hirschl and Gruber noted recovery from a trigeminal affection which they regarded as rheumatic. But these cases, which are incompletely explained, are quite exceptional. *Treatment* must in the first instance be directed to the causal disease. From this point of view the possibility of a specific process should be always borne in mind. I have succeeded in several instances in curing the symptoms of a total trigeminal paralysis, even of a neuroparalytic keratitis, by *antisyphilitic treatment*. Opening of an abscess or extirpation of tumours may be indicated. Symptomatic treatment consists in giving narcotics for the pain and in electrotherapy.

With regard to prophylaxis, the eye must be protected against the danger of injury from foreign bodies, to which the anæsthesia of the cornea and conjunctiva render it liable.

Facial Paralysis [1] (*Prosopoplegia*)

Paralysis occurs more frequently in the facial than in any other single nerve. It is called peripheral when the lesion which causes it affects the trunk of the facial nerve after its emergence from the pons, in its intracranial or further course in the internal auditory meatus, in the aqueduct of Fallopius, after it leaves the stylomastoid foramen, or the peripheral branches of the nerve.

Amongst the *causes* of facial paralysis, the most important is *chill* (facial paralysis from *rheumatism* or *chill*). There is no doubt that it occurs in a great many cases (some 73 per cent.) in hitherto healthy persons as a direct consequence of a chill (draught, sleeping with open windows, washing the head). Recent observations (Minkowski, Dejerine-Theohari) make it probable that neuritis is usually due to some *infective* process. These authors have not found inflammatory changes in the neurilemma, etc., but a simple parenchymatous degeneration. Alexander [2] has, however, shown that in cases of simple degeneration of the peripheral nerve and the branches, there may be inflammatory changes in the geniculate ganglion. In any case this distinction between degenerative and inflammatory processes in the nervous system cannot be consistently carried out.

Diseases of the middle ear and *caries of the petrous bone* readily involve the facial nerve. It lies so close to the tympanic cavity and is separated from it by a lamella of bone so thin that the inflammation may simply extend to it, or it may be compressed by the pus, by masses of granulations, etc., inflammatory swelling or a sequestrum of bone inside the aqueduct of Fallopius. Tomka has shown that in the extension of otitic affections to the facial nerve, individually varying factors also play a part, *e.g.* width of the Fallopian canal, density of bone, congenital absence of the inner wall of the canal.

[1] Literature in Bernhardt, "Die Erkr. d. periph. Nerv.," 2. Aufl., Wien, 1902. Also the treatise of Fuchs (*Obersteiner*, 1907, and *W. med., Pr.* 1907).

[2] *A. f. Ps.*, xxxv. Mirallié (*R. n.*, 1906) has also published an anatomical investigation; also André-Thomas (*R. n.*, 1907) in three cases.

Facial paralysis may be superadded to an otitis, or both diseases may be produced simultaneously by the same cause—chill, infective disease (influenza, typhoid, etc.). This seems to be shown by a case described by Darkschewitsch and Tichonow,[1] who found in an otitic facial paralysis a simple neuritis of this nerve, which they thought they could not directly trace to the carious process. Gowers saw facial paralysis after *tonsillitis*. I have also seen it in a patient in whom the tonsillitis had been treated with applications of ice to the side of the neck; Hatschek, Dopter and others saw it after mumps, Garnier and Thaon after erysipelas. I once saw facial paralysis follow a mastitis. It occasionally develops after articular rheumatism with erythema multiforme. We should also mention the association of facial paralysis with herpes zoster; the symptoms of disease of the facial may either be directly due to the herpes or develop after it as a post-infective neuritis (Ebstein,[2] etc.). The cases in which the paralysis commences with fever and disturbances of the general condition (Oppenheim, Ferenczi, Ahlfors, etc.) point to an infective origin. *Gout*, *diabetes mellitus*, the *puerperium*, *diphtheria*, *leucæmia*, and specially *syphilis* also cause the paralysis. Syphilis usually affects the facial nerve at the base of the brain, the nerve being involved in a basal gummatous meningitis or by a gummatous tumour. But the syphilitic process may also compress it within the aqueduct of Fallopius. It is worthy of note that a facial paralysis, probably of *neuritic* origin, may appear even in the *early stage of syphilis*, a few months after the primary affection (Boix, Goldflam, etc.).

Other morbid processes at the base of the skull, such as *meningitis*, *new growths* and *aneurisms*, not infrequently extend to the facial and paralyse it.

It is now beyond dispute that injuries which affect it at any point of its course, such as sword or puncture wounds in the face, bullets or foreign bodies in the ear, and especially fracture at the base of the brain or hæmorrhages in the aqueduct of Fallopius give rise to paralysis. Knapp[3] describes paralysis of this nerve from pressure during sleep. Not a few cases are due to operations, especially on the petrous bone. Sub-cutaneous injections of antipyrin, given on account of a convulsive tic, resulted in a case of M. Bloch's in partial paralysis. In the use of Schlösser's method of injecting alcohol into the nerve sheath for the cure of convulsive tic, paralysis is always to be expected in the first instance. The mode of origin of the paralysis caused by the extraction of teeth is not clearly recognised (Frankl-Hochwart). In a case of Stocquart's inflammatory processes spread from the region of the wisdom-tooth to the nerve.

Multiple neuritis, especially the alcoholic form, may affect the facial, and produce a *unilateral*, or more commonly a *bilateral peripheral paralysis* of the nerve.

Facial diplegia is also observed, especially in diseases of the pons and medulla oblongata, in aneurisms of the basilar artery, in basal syphilitic meningitis, very rarely in bilateral diseases of the petrous bone and middle ear, and is sometimes due to rheumatism (Romberg, Mott, Stintzing, Oppenheim (see Fig. 225), Lévy, Sainton, Raymond, Pane-

[1] *N. C.*, 1893. Of recent works on otitic facial paralysis we may mention: Grivot, *Thèse de Paris*, 1903; Lannois-Pantet, *Revue de Méd.*, 1902; Stenger, *A. f. kl. M.*, 1904; Neumann, *W. m. W.*, 1906.

[2] *V. A.*, Bd. cxxxix.

[3] *M. f. P.*, xviii.

grossi,[1] etc.). Rad describes facial diplegia as one of the symptoms of multiple paralysis of the cranial nerves. A unilateral or bilateral facial paralysis may accompany head tetanus (*q. v.*).

Facial paralysis may be *congenital* or may be caused during labour, especially by the use of forceps. This *obstetrical paralysis* may be bilateral (Seeligmüller, Edgeworth). In one case (Vernier) it developed spontaneously in a breech delivery with premature rupture of the membranes. *Congenital* facial paralysis is usually bilateral and associated with oculomotor paralysis (Möbius, Bernhardt, Cabanne); it is rarely confined to one side (Stephan, Schultze, Bernhardt, Nonne, Comby), but individual muscles, such as the orbicularis oris, may be spared.[2] Thomas describes a combination of congenital facial diplegia and deafness. Remak mentions a defect limited to the platysma and muscles of the chin of one side. Congenital paralysis of the facial muscles may be associated with other anomalies of development, such as bifid uvula, astigmatism, curving or absence of some of the fingers (Möbius, Bernhardt). The combination with an arrest of development of the ear and petrous bone was noted by Marfand and Delille, Heller, and Goldreich-Schüller. Little is known as yet concerning the pathological conditions which cause this congenital facial paralysis. Möbius suggests a congenital nuclear atrophy, a view which is supported by the results of an autopsy by Heubner (see p. 468).

Finally, it should be pointed out that *neuropathic heredity* plays a part in the etiology of facial paralysis which should not be underestimated (Neumann). Charcot saw it occur in a number of brothers and sisters, as I have also, whose ages ranged from four to six. When there is a congenital tendency to the paralysis, some mental emotion may produce it. I have treated a patient in whom the paralysis appeared after a shock, and reappeared years later after another fright.

Facial paralysis is not related to any special age, but it usually occurs between the ages of twenty and thirty, rarely in childhood.

Symptoms.—The paralysis comes on suddenly over-night without prodroma, or previous symptoms may occur which are due to the primary disease (disease of the ear, brain tumour, etc.), and the development is then less rapid. The form caused by rheumatism or chill is sometimes preceded by *pain*, which may be present for a few days or even a fortnight before the paralysis appears (Webber, Testaz). These pains are felt in or behind the ear, in the face or neck, and are attributable to a simultaneous (neuritic ?) affection of the sensory nerves, especially of the trigeminal, occipital, and cervical nerves. In one of our cases paræsthesia of the sense of taste—acid taste on the same side of the tongue—preceded the paralysis for several days. Slight swelling of the face, mainly in front of and below the ear, is sometimes found at the onset of the disease; a diffuse swelling of the whole side of the face is less common. The affection rarely commences with fever, headache, vomiting, noises in the ears, etc., but I have repeatedly observed the following course of development in young children: acute onset of a feverish condition, which passes off in one to three days, leaving a complete facial paralysis with signs of a peripheral or pontine (?) paralysis, no ear disease being present.

[1] *Riv. speriment. di Freniatria*, 1903 (gives summary of literature).

[2] A full review of references to congenital facial paralysis may be found in the *Arch. de méd. des Enfants*, 1901. See also Heller, *Thèse de Paris*, 1903.

Gradual, progressive development of a simple peripheral facial paralysis is exceedingly rare; I have observed one case of this kind and Ziehen mentions another. In our case the etiology was quite obscure; Ziehen blames the prolonged effect of nitrous fumes. In another case seen by Schaffer and myself, which I mentioned in the third edition, further observation has shown that the cause was a tumour behind the angle of the jaw. It had grown so slowly that the facial paralysis was for years the only sign. There was at first some slight pain, but in the entire absence of a local condition—radiography also giving a negative result—no significance could be ascribed to it. It was only 2½ years later that other nerves became involved and that the tumour could be detected on palpation. Peripheral facial paralysis of gradual development should, therefore, be always regarded with suspicion.

The paralysis nearly always affects all the *facial muscles* innervated by the facial nerve. This gives rise to an asymmetry of the two sides of the face, which is apparent even during repose, and is more marked in old than in young people (Fig. 223, 224).

The wrinkles on the forehead, especially the transverse ones, can be seen only on the unaffected side; they suddenly cease at the middle line, the forehead of the affected side being smooth. The eye remains widely open, to such a degree that the surface of the mucous membrane of the lower lid may be visible and the tears do not reach the tear duct (Fig. 224). The nose deviates towards the healthy side, the naso-labial fold of the affected side is effaced, and the mouth much drawn towards the other side, whilst the angle of the mouth is lower and the mouth itself slightly open on the paralysed side.

The symptoms are much more marked on voluntary movement, and wrinkling of the forehead, closing the eyes, sniffing, and movements of articulation and facial expression can be made only on the unaffected side. The asymmetry is then greatly increased. The paralysis of the muscles of the lips is specially apparent in the attempt to laugh, in opening the mouth and showing the teeth. The lips cannot be closed on the paralysed side; the saliva, frequently also the food, escapes from the corner of the mouth. In doubtful cases it is advisable to test the strength of the muscles of the mouth by letting the finger be firmly sucked in the angle of the mouth or by trying to open the lips, which the patient attempts to hold firmly together. The buccinator does not contract, and the cheek is blown out in expiration. In chewing it no longer lies close upon the teeth.

In the attempt to close the eyes, the upper eyelid is lowered by relaxation of the levator palpebræ superioris, and the eyeball turns upwards and usually outwards until the cornea is hidden (Bell's phenomenon); but the palpebral fissure remains open on account of the paralysis of the orbicularis palpebrarum (*lagophthalmus*). It is usually somewhat more open than on the other side, even during rest. A secondary contracture of the levator palpebræ superioris is described by Dupuy-Dutemps and Berger-Loewy. The lid reflex is also absent, so that foreign bodies are not removed from the eye and conjunctivitis is thus produced. In sleep the eye remains partly open, and the palpebral fissure may be further narrowed by relaxation of the levator palpebræ superioris (Gruber, Herzfeld,[1] Vaschide-Vurpas). The contrary has also been observed, *e.g.* in paresis of the orbicularis the eyelid can only be shut in the waking condition (Hanke). If there is merely paresis of the orbicularis palpebrarum, the eye can be closed, but without any force; the slightest counter-pressure is sufficient to open the palpebral fissure.

[1] *B. k. W.*, 1901.

In one of my cases the orbicular paresis was so slight that the patient could shut the eyelids simultaneously, but there was no spontaneous or reflex winking, and as this gave rise to discomfort, he was obliged to make up for it by active movements.

The muscles of the ear and the platysma are not always involved. The facial has nothing to do with innervation of the soft palate (Gowers, Jackson, Lermoyez, Panier [1]). No importance should be attached to obliquity of the uvula, as it occurs in healthy persons. We can only speak of its paresis when the palatal arch is lower on one side and the soft palate becomes less tense in phonation, but this certainly is not found in uncomplicated facial paralysis. Articulation is affected at first, the labials being defectively formed. This condition, however, soon dis-

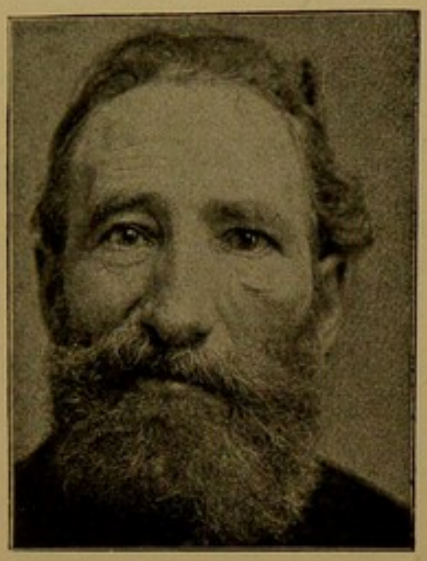

FIG. 223.—Left facial paralysis affecting all the branches. (Oppenheim.)

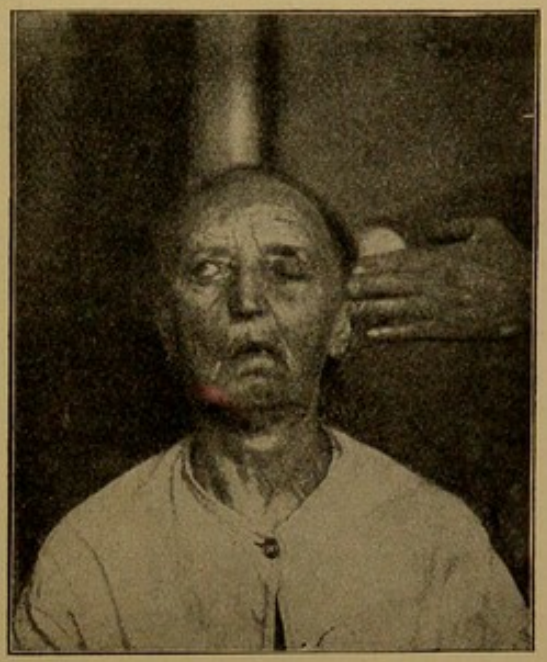

FIG. 224.—Incurable right facial paralysis in a woman aged 75. Attempt to close the eyes. (Oppenheim.)

appears. Deficient dilatation of the nostril may cause diminution of the sense of smell.

In many cases the sense of taste is diminished or absent on the anterior two-thirds of the same side of the tongue (from involvement of the chorda tympani [2]); the sense of touch is in rare cases slightly affected in this part of the tongue (Gowers, Bernhardt, Frankl-Hochwart, Adler, Biehl, G. Köster; the latter think they have proved experimentally that the chorda contains sensory fibres [3]). Total hemiageusia has been found in a few cases. There is frequently *affection of the secretion of saliva* on the paralysed side (Arnold, Romberg), usually diminution, less often increase

[1] *Thèse de Paris*, 1906. G. W. Jacoby (*R. of N.*, 1907) advocates the view that the relations of innervation to the palate only become of importance in paralysis of both facial nerves, *i.e.* in diplegia, but this does not correspond to my experience. An observation by Lachmund and an old one by Fragstein (*W. kl. W.*, 1903) may also be regarded in this sense.

[2] We have already referred on p. 477 to the fact, stated especially by aural surgeons, that disturbances of taste may be directly caused by lesions of the chorda in the tympanic cavity. Kniesow-Nadoleczy produced a metallic acid taste in the corresponding half of the tongue by stimulation of the chorda.

[3] Literature in the recent works of Scheiber, *Z. f. N.*, xxvii., and Donath, *N. C.*, 1906.

(G. Köster [1]). Köster has shown that disturbances of the *sweat-secretion*, especially anidrosis on the affected side, are amongst the most frequent symptoms of facial paralysis, which is in conformity with the statements of Vulpian, Raymond, and others, that the trunk of the facial contains fibres for the stimulation of secretion of sweat.

We have already referred to the *affection of the tear secretion* due to diseases of the seventh nerve above the geniculate ganglion.

The tongue is protruded straight, but there is sometimes apparent deviation due to the twisting of the mouth. Involvement of the stylohyoid and digastric does not produce any obvious symptoms, but Schultze has described a low position of the base of the tongue as a symptom of facial paralysis and is inclined to ascribe the symptom to paresis of these muscles. I have usually failed to find this sign.

The ear disease which accompanies facial paralysis may manifest itself by its own special symptoms. The geniculate ganglion in particular lies so near to the lower coil of the cochlea that lesions of this region frequently cause *nervous* deafness (Köster) as well as facial paralysis. In rare cases an abnormal acuity of hearing and sensitiveness to deep tones (hyperakusis, oxyokeia) form a symptom of facial paralysis (Roux, Lucæ, etc.). These are ascribed to paralysis of the stapedius, although on the other hand an abnormal or exaggerated contraction of this muscle has been blamed (Urbantschitsch). One of my patients complained of unpleasant sensations in the affected ear, difficult to define, and specially noticeable during mastication; another of buzzing in the ear on trying to close the eyes.

Herpes zoster in the area of the trigeminus and cervico-occipital nerves, and in the anterior two-thirds of the tongue (chorda tympani) are occasional complications. Œdematous swelling of the paralysed side of the face is also occasionally noted (Frankl, Hübschmann). In a few cases I have seen swelling of the glands in the course of facial paralysis (due to an infective-neuritic cause). Salomonson mentions atrophy of the facial bones in facial paralysis dating from early childhood, whilst Gowers thinks that, inversely, facial hemiatrophy (see below) may cause secondary involvement of the facial nerve.

The *electrical excitability* either remains normal (slight paralysis) or complete or partial reaction of degeneration develops during the first two weeks. Simple increase of excitability is the exception.

It has been observed by Petrina, Senator, Hoffmann, Bumke, and others, that in electrical stimulation of the healthy facial, the muscles of the affected side become contracted (the converse condition is rare). This symptom, which occurs chiefly in pontine facial paralysis, has been variously interpreted. Bernhardt refers to the interlacement of the muscles of the two sides of the chin, which Henle and Krause had already noticed, as a reason why a bilateral contraction may be produced by a unilateral stimulation of the muscle or its nerve. I have observed (*B. k. W.*, 1899, and Mohr, *B. k. W.*, 1900) this in persons who had suffered from childhood from facial paralysis, on stimulation of the unaffected side. There was prompt contraction of the facial muscles adjacent to the middle line, even although the current was so weak that it had no effect upon the muscles of the sound side. I ascribed this to *collateral innervation* from the healthy side, which Bernhardt has denied. Further experience, however, convinces me of the correctness of my own explanation, as opposed to that given by Bernhardt, Seiffer, and Lipschitz.

There could be no question in my cases of a reflex movement, as the contraction only followed the electrical stimulus as a short, lightning-like K.C.C.

Bergonié, whom my communication has quite escaped, describes the symptom as a new one.

[1] *A. f. kl. M.*, Bd. lxviii.

The symptoms of facial diplegia require no special description; they are illustrated by Fig. 225.

Diagnosis.—When facial paralysis is fully developed [1] the diagnosis usually affords no difficulty. It is, however, exceedingly important to determine the site of the lesion of the nerve. In this respect we must first distinguish sharply between the paralysis which is caused by a lesion of the *cortical centre* or of the tracts leading from it to the facial nucleus, and that due to an *affection of the nucleus* and of the peripheral tract to which it gives rise. Fig. 226, which is purely schematic, may serve to illustrate these conditions.

The tracts originating in the cortical centre of the facial meet the fibres rising from the arm and leg centres in the internal capsule, run with the motor tracts of the extremities of the opposite side of the body through the cerebral peduncle to the pons, in which they pass over the middle line to reach the facial nucleus (seventh nucleus), which lies in the neighbourhood of the abducens nucleus. From this nucleus arises the peripheral nerve, which at first runs beside the auditory (not shown in diagram). The tract from the cortex to the seventh nerve in the pons will be termed the *volition tract.* Facial paralysis due to lesion of this tract has the following characteristics: (1) the electrical excitability is not affected; (2) the superior facial, *i.e.* the branches to the forehead and eyes, is not paralysed or is merely slightly affected (lagophthalmos being produced in very exceptional cases), either because it possesses a special centre and tract—which I consider improbable—or because it is innervated from both hemispheres; (3) there is usually paralysis of the extremities of the same side. (It is only when this tract is affected on the short path from the site of its decussation in the pons to the nucleus that *alternating hemiplegia* occurs, *i.e.* paralysis of the facial on the side of the lesion, and of the extremities on the opposite side.) (4) The reflex excitability of the muscles supplied by the facial nerve is conserved.

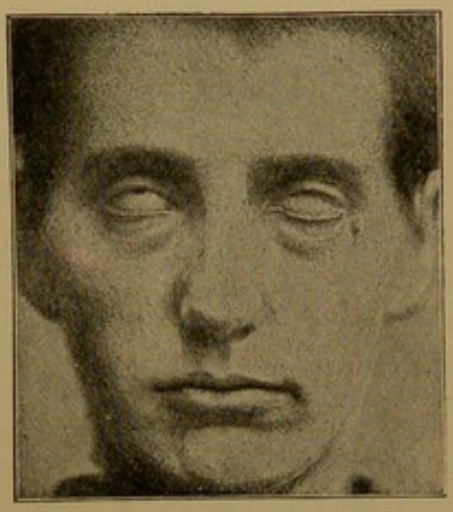

FIG. 225.—Expression in facial diplegia. (Oppenheim.)

For the symptomatology of supranuclear, ponto-facial paralysis, see section on pontine diseases.

Paralysis due to disease of the nucleus and the peripheral tract differs from supranuclear paralysis in the following characteristics: (1) that as a rule *all the branches* are affected, upper as well as lower, although even in pontine affections the upper facial is frequently spared; (2) that almost always—excepting only the slightest paralysis—there are *signs of nerve degeneration*, particularly the changes of electrical excitability already

[1] Slight asymmetry in the innervation of the face is not rare in healthy persons. In doubtful cases it is advisable to compare with earlier photographs. Such an asymmetry may also be due to the habitual expansion of one corner of the mouth, in smoking, for instance, especially a pipe (as Fritz Reuter has described in tramps). Absence of teeth on one side and atrophy of the alveolar process may cause asymmetry and simulate a difference of innervation. Rava (ref. *R. n.*, 1906) has exhaustively discussed the physiological differences of innervation of the facial muscles, etc.

described, as the nucleus forms the trophic centre for the muscles innervated by the seventh ; (3) that the *reflexes are abolished.*

There are also peripheral paralyses of the facial nerve which are confined to single branches. This is naturally most common in traumatic cases ; after excision of the submaxillary and retromaxillary glands, for instance, the lower branches are frequently paralysed (Camillo Fürst). I have seen a case in which only the branch for the frontalis muscle was permanently paralysed by operation. Mann and Bernhardt have shown that even under other conditions, particularly in congenital facial paralysis, individual muscles, *e.g.* the orbicularis oris and palpebrarum, may escape. Silex describes a paresis of indefinite origin, limited to the orbicularis palpebrarum.

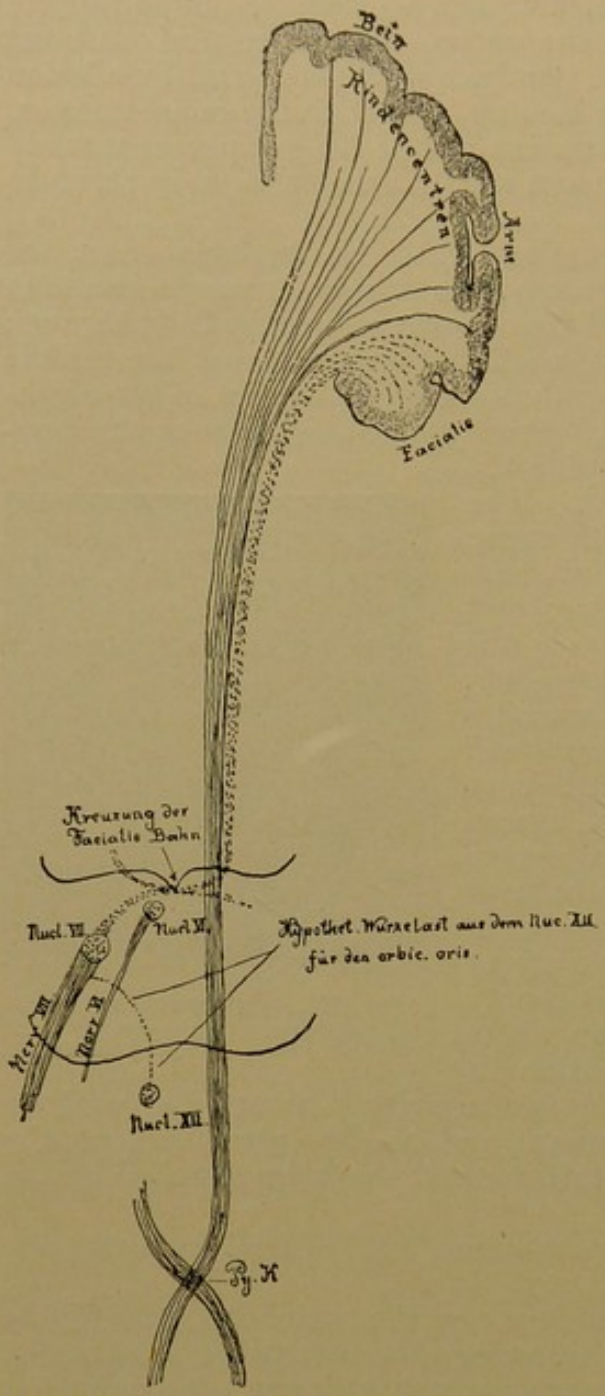

Fig. 226.—Diagram representing the motor nerve tract for the face and extremities. Rindencentren = Cortical centres. Bein = leg. Arm = arm. Facialis = face. Kreuzung der Facialis Bahn = decussation of the path from the facial centre to the facial nucleus. Hypothet. Wurzelast, etc. = the hypothetical root from the xii. nucleus to the facial for the orbicularis oris.

It may be difficult to distinguish between disease of the facial (the nucleus and root) in the pons and an affection of the nerve trunk, but the accessory symptoms almost always throw light upon the question, as the facial is hardly ever affected in the pons without other structures in this region being also involved, and diseases of the trunk of the facial are almost always distinguished from pontine affections by signs to be described below.

It is obvious that pontine facial paralysis should be usually accompanied by paralysis of the abducens nerve. I have, however, seen two cases in which there was a peripheral (rheumatic or infective) paralysis of the sixth and seventh of one side. Haskovec and others describe similar cases.

We may point out in passing that in diseases of the medulla oblongata the facial fibres for the mouth are as a rule affected along with the hypoglossus, a symptom which has given rise to the view that the hypoglossal nucleus is also concerned in the innervation of the orbicularis oris. This is indicated in Fig. 226, but is very improbable.

If the facial nerve is affected at the *base of the brain*, an *involvement of the auditory* and of other basal cranial nerves, and *general cerebral symptoms* (headache, vertigo, vomiting, etc.) will point to this localisation.

Erb's diagram, Fig. 227, gives a scheme for determining the site of the lesion in the further course of the nerve, but it is no longer regarded as sufficient, as even when one segment of a nerve is affected the fibres contained in it are not necessarily involved by the lesion to an equal extent. If the lesion lies in the part between one and two, the facial muscles are alone paralysed; between two and three, the taste fibres of the chorda are involved, the sense of taste is absent on the anterior two-thirds of the tongue, and the secretion of saliva is affected. If it lies between three and four, implication of the stapedius gives rise to hyperakusis. If the geniculate ganglion between four and five or the nerve above it are affected, then, according to former views, the soft palate of the corresponding side is paralysed, because the motor fibres for the muscles of the palate leave the facial in the tract of the great superficial petrosal nerve and reach the palate by means of the sphenopalatine ganglion and the descending palatine nerves. But, as already said, this view cannot be maintained. The condition of the tear secretion might perhaps support this opinion, as disturbances of this function are only to be expected when the nerve is affected in the geniculate ganglion or above it (Jendrássik, G. Köster). The combination of facial paralysis with nervous deafness is also in favour of this localisation; this factor must, however, be carefully considered. In a few cases of lesion of the facial below the stylomastoid foramen, ageusia has appeared; this has been ascribed to affection of recurrent fibres of the chorda (?). No indisputable case of ageusia in basal paralysis of the facial has been described.

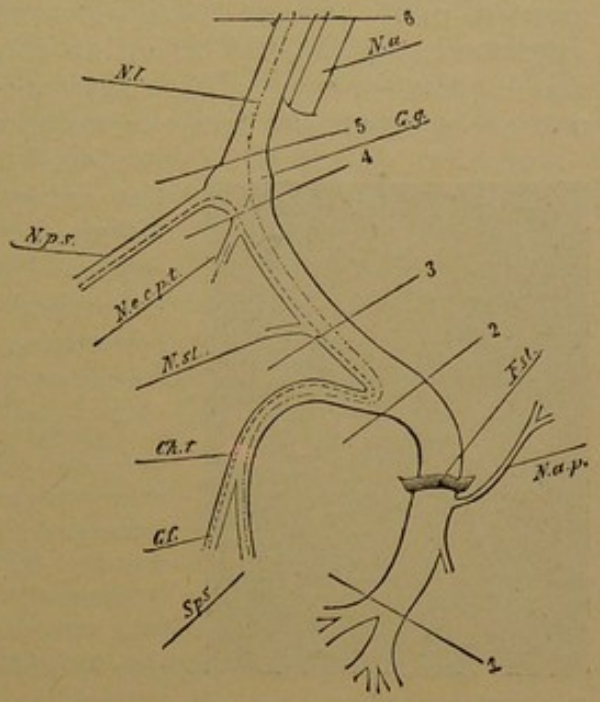

FIG. 227.—Diagram of the facial nerve trunk from the face of the skull to the pes anserinus. *N. f.* = facial nerve. *F. st.* = stylomastoid foramen. *N. a. p.* = posterior auricular nerve. *N. p. s.* = great superficial petrosal nerve. *N. e. c. p. t.* = branch to the tympanic plexus. *N. st.* = branch from facial to stapedius muscle. *Ch. t.* = chorda tymphani. *N. a.* = acoustic nerve. G. g. = geniculate ganglion. (After Erb.)

G. Köster specially notes that the various kinds of fibres collected into the trunk of the nerve possess a very varying power of resistance to the injuries which affect them; the most vulnerable fibres are the motor, the most resistant are the sensory fibres of the chorda, etc.

Course.—In slight cases the paralysis may disappear in a few weeks or even within one or two. In severe cases it persists for months or even remains permanent. If improvement occurs, and the patient again learns to move the muscles of the face, another complication not infrequently arises, viz., *contracture* of the hitherto paralysed muscles. The mouth is gradually twisted towards the previously paralysed side, the nasolabial

fold becomes deepened, the palpebral aperture is smaller on account of persistent tension and shortening of the muscles (Fig. 228). The unaffected side now appears to superficial observation as if it were paralysed. On testing the motility, however, especially in speaking, laughing, etc., it is at once seen that in spite of the contracture the active and emotional movements are limited on the side previously paralysed. The *associated movements*, which frequently accompany this contracture, may be confusing: in closing the eyes it is not only the orbicularis palpebrarum which contracts, but also the zygomaticus of the previously paralysed side, the angle of the mouth being thus excessively distorted. In a physician whom I treated for facial paralysis with ageusia, with the commencement of improvement there occurred on every effort at movement of the paralysed muscles, the sensation of a metallic taste on the corresponding side of the tongue. There may also be lachrymation at this stage; in one of my patients the eye watered specially during eating. Engelen has also seen this. I include the symptom in the category of associated movements, whilst Micas attributes it to an öesophagolachrymal reflex. Finally, other motor symptoms of irritation, in the form of contractures, or of *convulsive tic* (see corresponding chapter), frequently occur.

FIG. 228. — Contracture and associated movements in the previously paralysed right facial. (Oppenheim.)

In several cases I have seen tic on the healthy side follow the facial paralysis, a symptom which I ascribed to hyperinnervation of the muscles of this side when movement was attempted. Brissaud-Sicard-Tanon (*R. n.*, 1906) subsequently described this combination.

Some of my cases indicate that the same injury may affect both nerves or their nuclei, causing in one symptoms of irritation, in the other, of paralysis.

These *secondary* symptoms[1] which occur in the later course of facial paralysis are due to a condition of irritation in the nucleus, which is caused by futile and excessive attempts at innervation, or by marked secondary changes in the nuclei. Electrical treatment has also been blamed, but without justification, although one might imagine that the strong stimulus affecting the trigeminus would produce a condition of irritation in the facial nucleus (a convulsive tic not infrequently being superadded, *e.g.* to trigeminal neuralgia). But the contracture also develops in patients who have never had electrical treatment. It occurred in one case long after the paralysis was cured, following a painful operation on the eyelid of the same side. E. Remak points out that this spontaneous contracture in the previously paralysed muscles of the face occurred simultaneously with the blepharospasm, which, however, does not seem to me always to be the case.

A gradual and simultaneous onset of paresis and contracture is very

[1] In a patient who had bilateral blepharospasm due to conjunctivitis, I saw on the onset of a right-sided facial paralysis, a contraction of the lower jaw towards the left appear synchronously with the blepharospasm of the left eye. The stimulus, blocked in the tract of the right facial, was transferred to the motor trigeminus of the same side. Jolly saw facial diplegia appear simultaneously with movements of closing the jaw in the paralysed zygomatics, etc. As to associated movements and other symptoms we may refer to the explanation suggested by Lipschitz (*M. f. P.*, xx. Ergänzb., and *C. f. N.*, 1907).

uncommon ; I have seen it only in central diseases and in tumours which compress the nerve.

Négro (*Gaz. degli Osped.*, 1906) recently mentioned this form of apparently primary facial spasm, behind which the paresis was masked. Lachmund saw in one case an extension of the tic to the soft palate (*M. f. P.*, xxi.).

Recovery from facial paralysis may be incomplete, only some of the muscles, *e.g.* those of the mouth, regaining their power of function, whilst the closing of the eyes remains incomplete, and *vice versa*.

The fact should also be noted that facial paralysis, both the rheumatic and the otitic form, may *relapse*. The relapse more often affects the nerve of the other side, so that the term is not strictly correct.[1] In one case the relapse was due to recurrent otitis. I have observed this relapsing, alternating, facial paralysis as a family disease, associated with diabetes, in three members of one family.[2] It is doubtful (Möbius) whether there is a relapsing facial paralysis belonging to the same category as periodic oculomotor paralysis, as B. Rossolimo maintains.

It is not unusual, as I have found, for peripheral facial paralysis to be associated with an hysterical hemianæsthesia of the same side.

The *prognosis* is decided by the nature of the causal lesion. Where, for instance, the facial paralysis is due to an inaccessible tumour, or to caries of the petrous bone, recovery cannot usually be expected. In other cases, especially in rheumatic paralysis, the course is determined by the severity of the lesion. This is chiefly shown by the condition of the electrical excitability, and in this respect we can distinguish between a *slight*, a *severe*, and a *moderately severe* form of facial paralysis. It is slight when the electrical excitability is normal or merely slightly diminished after a period of about two weeks, severe when there is complete, and of medium severity when there is partial reaction of degeneration. Diminution of excitability is usually found after one week, exaggeration of direct galvanic excitability not until after two to three weeks. Slight paralysis passes off, as a rule, in about two to three weeks, paralysis of medium severity in four, six, eight weeks, and severe paralysis, if it disappears at all, only after three to six months. But there are not a few cases which show very irregular conditions and to which this rule by no means applies. In one severe case which I have treated, the first trace of active movement appeared after five months, and yet the improvement was substantial (see also p. 412). The view that prodromal pains afford data for the prognosis has no foundation (Bernhardt).

In catarrh of the middle ear, the prognosis depends partly on the curability of this disease. Chipault and Daleine have drawn up a series of axioms for determining the prognosis, but they are not unassailable. Paralysis due to cauterisation of the nerve with caustic or chromic acid in operations in the tympanic cavity is usually severe, but according to Jansen's experience (oral communication) tends gradually to disappear.

The acute, apparently infective form of facial paralysis in early childhood seems to be due to a process which, as I gather from one case, may endanger life. A case observed by Ahlfors (*Hygeia*, 1901) also points to this fact.

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[1] This form is discussed by Émile-Paul Petit in his thesis (Paris, 1905). See also Bernhardt (*N. C.*, 1899), Huet-Lejonne (*R. n.*, 1907).

[2] It was a specially interesting fact that two of these diabetics had attained the age of 88 and 90 years.

The prognosis seems to be very grave when the onset of a peripheral paralysis is slowly progressive (*vide supra*).

Treatment must at first be directed to the primary disease. Where *syphilis* is present or probable, specific treatment is appropriate, although it is by no means always successful, as even in syphilitics the facial paralysis may be due to a simple neuritis of the nerve. Cytodiagnosis (Noica) or serum diagnosis may give positive data as to the presence of syphilis. Recent rheumatic facial paralysis requires *diaphoretic* measures. It is advisable to employ local *blood-letting* (by leeches) at the point of emergence of the facial below the ear, or a *blister* at the same place or behind the ear. Gowers recommends *hot fomentations*. In the rheumatic and infective forms purgatives have sometimes a good effect in the first stage. In a case where the paralysis had followed acute articular rheumatism salicylate treatment proved useful.

Injury of the facial may necessitate *suture of the nerve* or its release from cicatricial tissue, if the site of the lesion is accessible. A tumour compressing the nerve should be removed if possible. Otitic facial paralysis requires expert treatment of the ear disease; it may necessitate paracentesis, a wide opening of the antrum, and evacuation of pus, granulations, sequestrum, or even opening of the aqueduct of Fallopius (Chipault, Grunert, Gellé, Moure, etc.). Chipault goes furthest in his suggestions for radical treatment.

As to nerve anastomosis, which has been repeatedly carried out by grafting the facial nerve upon the accessory or the hypoglossal, see p. 415. According to Bernhardt's[1] exhaustive work on this subject, the operation should be regarded as justified, although so far it has led rather to cosmetic results than to complete recovery from the paralysis. A certain degree of active movement certainly returns, but this is combined with associated movements in the region of the accessory nerves, or the facial movements can be executed only along with movements of the shoulder or tongue. Frazier, Tubby, Löhlein, Sick, Bardenheuer, Lafite-Dupont report particularly satisfactory results. The first sign of returning movement appears usually after six or nine months or may be still further delayed, but some years may elapse before the full effect is reached. On the whole, transplanation of the facial to the hypoglossal or accessory nerves may be recommended, but all the circumstances of the case should be taken into consideration. Plastic operations may also be carried out for the correction of asymmetry in cases of incurable paralysis (Hoffa). In lesion of individual branches, *e.g.* that to the mouth, section of the corresponding branch on the other side has even been carried out, in order to restore the symmetry (Wolff).

As regards *direct* treatment, electricity is strikingly successful in recent, and often in old-standing cases. In recent cases, stabile galvanic treatment of the nerve is specially suitable, the cathode (of 10 cm. square) being placed on the nerve, the anode on an indifferent site, or on the neck. A weak current (1-2 M.A.) with slow interruptions should be employed for two or three minutes. In a physician whom I treated in this way, the sense of taste, which had been absent on the corresponding side of the tongue, returned immediately after the first application. It may also be well to conduct the current transversely through the brain and the auriculo-mastoid fossæ. Later, labile galvanic treatment of the

[1] *Mitt. a. d. Grenzgeb.*, xvi.

paralysed muscle may be employed, if it reacts only to the galvanic current. Strong currents must be absolutely avoided, and special care must be taken in using the faradic current in severe cases. It is also advisable to discontinue electrical treatment as soon as the first sign of contracture appears. I have reason to believe that stimulation of the muscles from the mucous membrane of the mouth was successful in some cases. In many cases the sensibility to electricity is so great that one must begin with hardly perceptible currents and avoid all opening and closing. These are obviously cases in which a neuritis of the sensory facial nerves is associated with the paralysis. Electrical treatment should be applied at first daily, later every second day. I have succeeded in some cases in improving, to a certain extent, a facial paralysis dating from early childhood, by electrical treatment commenced at a mature age.

We have no effective remedy for the secondary contractures. Gentle massage, or stretching the cheeks by a ball of wood held in the mouth, are recommended. Electricity applied to the muscles on the unaffected side is of no use. In some cases I have materially aided the progress of the improvement by prescribing speech exercises, the corner of the mouth on the healthy side being fixed (firmly closed with the hand or a strip of adhesive plaster). Others have since then recommended similar methods.

When lagophthalmos is present, it is well to cover the eye to protect it from the inflammation produced by foreign bodies getting into it. In persistent paralysis an attempt has been made to cure the lagophthalmos by producing subcutaneous cicatrices round the palpebral aperture (Pflüger, Kuhnt, Hoffmann).

Primary and Isolated Peripheral Diseases of the Auditory Nerve

are rare in comparison with disturbances of hearing caused by diseases of the ear. The terminal ramifications of the nerve are comparatively often involved in cases of labyrinthine disease which may also be conducted from the middle ear. But the nerve trunk may also undergo many injuries at the base of the brain from morbid processes; thus caries of the skull bones, periostitis, tumours from the bones and meninges, aneurisms of adjacent cerebral vessels, may cause paralysis of the auditory nerve if it becomes compressed by or involved in an inflammatory process in the neighbourhood. Tumours may also develop from the nerve itself or from its sheath (see chapter: Brain tumours and neurofibromatosis).

A *rheumatic* paralysis of the auditory nerve is described, but seems to be extremely rare. A few cases have been explained in this way by Bing, Frankl, and, in particular, by Hammerschlag.[1] The auditory nerve has also been involved along with the facial and trigeminal or abducens and other cranial nerves, and a primary neuritis has been assumed to be the cause. Frankl-Hochwart [2] speaks of a cerebral polyneuritis with Menière's disease, and Hammerschlag and Berger [3] agree with him. From my own experience, however, I cannot sufficiently insist upon caution in the acceptance of this conception of a primary multiple neuritis of the cranial nerves, as basal tumours or some such lesion are usually present. Polyneuritis may of course involve the auditory nerve (Strümpell). A leukæmic infiltration of the nerve has been repeatedly noted.

[1] *A. f. Ohr.*, Bd. xlv. and lii.

[2] *Jahrb. f. P.*, xxv.

[3] *N. C.*, 1905. Forli (*N. C.*, 1905) and Schönborn (*W. kl. R.*, 1907) have also described similar cases.

Wittmaack[1] has recently insisted upon the occurrence of a toxic neuritis of the auditory nerve, usually bilateral, and affecting mainly its cochlear division. He regards this as analogous with optic neuritis, and as being produced in the same way. He had the opportunity of studying the disease and its pathological anatomy in relation to tuberculosis. He also found a neuritis or atrophy of the nerve in other infective diseases (scarlatina, typhoid, etc.). But on the whole it is doubtful whether *primary inflammation and atrophy of the acusticus* is ever an independent disease. Calcification or deposit of lime-salts has been found in the nerve.

Disease of the auditory nerve in tabes, disseminated sclerosis, and other central affections will not be considered here.

With regard to the *symptoms*, affections of the nerve itself cannot be sharply distinguished from affections of the labyrinth. The essential symptom in both is *nerve deafness*, which is usually associated with *subjective noises in the ear*, and often with *vertigo* and *disturbance of the equilibrium*. The latter are due to involvement of the semicircular canals or the vestibular nerve. We can understand, however, that there are diseases of the auditory nerve in which, during their whole course or a considerable part of it, there are only cochlear symptoms, as Wittmaack and Lachmund, for instance, have found in toxic acoustic neuritis, whilst in labyrinthine disease it is quite exceptional for the cochlea or the semicircular canals to be affected independently of each other.

Babinski would make use in diagnosis of the "galvanic-vertigo," *i.e.* the disturbance of equilibrium manifested by an inclination of the head to one side during the passage of a galvanic current through the head. Normally the head leans towards the side of the anode and in unilateral ear affections towards the affected side. This symptom, as he admits, is not constant. On the other hand in bilateral labyrinthine deafness from organic cause this vertigo and the lateral inclination of the head may be completely absent. Mann has confirmed Babinski's statements.

Inflammations, *hæmorrhages*, *sclerotic* processes may develop simultaneously in both labyrinths. Such affections may be due to *acute infective diseases* (typhoid, scarlatina, malaria, influenza, mumps, etc.), and to syphilis. Nephritis, diabetes, pernicious anæmia, hæmorrhagic pachymeningitis and leukæmia sometimes lay the foundation for disease of the labyrinth or auditory nerve (see above). But the morbid processes commonly spread to the inner ear from the surrounding parts, and most frequently from *epidemic cerebro-spinal meningitis*. There seems even to be variety of this disease, in which the inflammatory process is mainly confined to the labyrinths.

Cases are known in which a *hæmorrhage* in both labyrinths (after trauma) was the cause of sudden total deafness. The deafness due to poisons (quinine, salicylic acid, perhaps also alcohol and nicotin) may be of labyrinthine origin, although, according to Wittmaack, toxic neuritis is localised chiefly in the nerve itself, especially in the cochlear branch and in the spiral ganglion. He has succeeded in producing this process experimentally. Senile involution may extend to the labyrinth and cause diminished power of hearing, but senile atrophy also seems to affect the auditory nerve directly. The relations of labyrinthine disease to the symptoms of vertigo and disturbances of equilibrium will be fully discussed later.

[1] *Z. f. Ohr.*, Bd. xlvi. and liii.

Affections of the trunk of the auditory nerve are usually diagnosed by the associated symptoms. A basal disease is not as a rule limited to the auditory nerve, but tends to involve the facial or other cranial nerves, or the medulla oblongata, pons, and cerebellum, etc.

Toxic and infective acoustic neuritis is characterised by symptoms of deafness, usually bilateral, coming on with loud and usually constant subjective noises in the ear. It has mostly a rapidly progressive course (Wittmaack). Labyrinthine symptoms may be absent or may appear later. Other cranial nerves may be affected at the same time. The acute forms of this disease are accompanied by pain in the ear. As to the signs of nerve deafness and the methods of detecting them (Rinne's test, Weber's test, etc.), see pp. 71 *et seq.* (Compare also the section on general symptomatology of brain diseases). Lachmund[1] has recently published an exhaustive discussion of this question, containing much that is interesting and also much that is hypothetical.

He has attempted to transfer the idea of scotoma and hemianopsia to the auditory nerve and hopes that a reflex movement occurring in the tensor tympani muscle will prove to be a diagnostic sign, which can be utilised to determine the site of the disease in the auditory nerve, as the pupil reflex is for the optic nerve.

Subjective noises in the ears constitute a symptom which may occur in any disease of the auditory apparatus—in any of its segments. They tend to be specially obstinate in nerve deafness. Indeed, we may say that where there are no subjective noises in the ears, there is as a rule no nervous affection (Frankl). Nervous buzzing in the ears is very frequently due to neurasthenia or hysteria (*q.v.*). It should be remembered, however, that it may also arise from anæmia, congestion of the brain, and aneurisms of the cerebral arteries. Noises in the ears due to clonic spasm of the internal muscles of the ear are very rare. These, and the noises due to aneurisms, may also be heard on auscultation.

Another cause of deafness and *tinnitus* is the continuous influence of loud noise—in factory workers, smiths, engine-drivers, stokers, etc.

Loud noises in the ear extending to the head on both sides are most unfavourable as regards the prognosis (see chapter on Neurasthenia).

Text-books on otology should be consulted as to the details, the prognosis, and treatment. It may merely be stated that nervous noises in the ear may be materially diminished in some cases by the use of the galvanic current (anode on the ear of the affected side, cathode on any indifferent site, gradual increase and diminution of a weak current, during which the noises disappear or diminish. Each séance should last about ten minutes). As affections of the auditory nerve are often of syphilitic origin, iodide and mercury may be given. For the rest, derivative methods of treatment are specially advised.

Paralysis of the Glosso-Pharyngeal Nerve

Affections limited to this nerve have practically never been observed, and the vagueness which obtains as to its physiological position and its functions has not hitherto been elucidated by pathology.

Although it is proved that its peripheral branches receive and conduct the *sensations of taste* perceived on the posterior part of the tongue and

[1] *M. f. P.*, xx. Ergänz.

soft palate, some writers suggest that these fibres again leave the glossopharyngeal nerve in order to pass centrally into the trigeminus, whilst others think the fibres of the chorda also enter the central glossopharyngeus (see p. 476). Until recently no case had been observed which definitely proved that the posterior taste fibres were contained in the glossopharyngeal stem, or in the roots of this nerve. A case recorded by Pope, in which an aneurism of the vertebral artery pressing on the root of the glossopharyngeal caused a hemiageusia, does not seem to me convincing, since the pressure and its results may have involved other structures as well. But Cassirer [1] has lately described a case observed by us which shows that all the taste fibres may at times be contained in the glossopharyngeus.

Sensory fibres of this nerve reach the upper part of the pharynx, the tonsil, posterior palatal arch, the tympanic cavity and Eustachian tube, and the posterior parts of the tongue ; but part of this area is supplied by the trigeminus, so that the area innervated by the glossopharyngeal alone is not precisely limited. There is no doubt, however, that the glossopharyngeal is the main sensory nerve of the pharynx. This nerve takes a part in the *motor* innervation of the pharyngeal muscles, but that part cannot be exactly defined. Réthi thinks only the fibres for the stylopharyngeus arise from it. According to Kriedl the motor nerves of the œsophagus arise from the glossopharyngeal, but pass over into the vagus.

It is very probable that *secretory* fibres run to the parotid along with the glossopharyngeal. Kohnstamm thinks they originate in a special nucleus, viz. the nucleus salivatorius inferior.

If we exclude the diseases of the medulla oblongata and consider only the peripheral lesions which affect it, we find that inflammatory processes or tumours (in particular, syphiloma and aneurism) in the posterior cranial fossa at the side of the medulla oblongata involve it most frequently, but hardly ever by itself. It may also be injured directly or by periphlebitis in thrombosis of the jugular vein. Finally, it is sometimes affected in its extracranial course by injuries, compressing tumours, etc. So far as I am aware an injury limited to this nerve has not yet been observed.

Some time ago I saw a remarkable, but not quite convincing case of this kind. A lady consulted me for loss of taste in the pharynx and the posterior part of the tongue ; she had also noticed that she had no sense of temperature on these parts, and it was particularly distressing to her that she missed the refreshing sensation of the cool air in breathing and of water in swallowing. Objective examination showed also a very slight paresis of the palate and pharyngeal muscles with diminished reflex excitability, ageusia on the soft palate, pharynx, and the posterior part of the tongue, as well as loss of the temperature sense in these areas, whilst the tactile sense was conserved. The trouble had developed after influenza, indeed, as the patient stated definitely, after she had been treated with protargol injections into the nasal cavity. Although loss of taste and smell may occur after influenza, it would appear that in this case some chemical effect had produced the disease of the terminal branches of the glossopharyngeal nerve.

Degeneration of the nerve or its roots may, as I have found, occur in tabes.

[1] *A. f. Anat. u. Phys.*, 1899, Suppl.

The signs of *peripheral* affection of the glossopharyngeal are *anæsthesia* of the upper half of the pharynx, *loss of taste in the posterior part of the tongue, in the palate*, etc., and *difficulty of swallowing* from paralysis of some of the pharyngeal muscles, with *abolition of reflex excitability* on the pharyngeal mucous membrane. These symptoms also occur among others in post-diphtheritic paralysis, but in this case there are usually no disturbances of taste. Paralysis in some of the muscles of the palate on one or both sides is also occasionally noted in simple tonsillitis (Réthi, Erben).

It is stated that diseases of the tympanic cavity may produce a neuritis which may extend along the nerve of Jacobson and ultimately involve the glossopharyngeal. Taste disturbances have also been found in diseases of the middle ear and have been ascribed to the branches of the glossopharyngeal contained in the tympanic plexus (Urbantschitsch, Schlichting). In diseases of and operations on the tympanic cavity an increase of the secretion of saliva in the parotid has also been found, and it is attributed to a lesion of the glossopharyngeal fibres (Urbantschitsch). Kohnstamm mentions "parotid-paralysis" after section of the glossopharyngeal root. Pathology teaches us nothing as to the inhibiting influence upon movements of deglutition ascribed to the glossopharyngeal nerve.

Paralysis of the Vagus Nerve

Anatomy and Physiology.—There is no unanimity of opinion as to the region of origin of the vagus (and accessory) roots, especially as regards the *motor* root. The majority of recent writers (Meynert, Kölliker, Dees, Grabower, Bunzl-Federn, Gehuchten, Spiller, Kohnstamm [1]) regard the nucleus ambiguus as the motor vagus nucleus. Kölliker and others point to other cell groups. The posterior vagus nucleus is regarded by some writers as the sensory end-nucleus in which the sensory vagus roots anastomose (see text and figures in chapter on brain anatomy), but others (Forel, Monakow, Bruce, Gehuchten, Kohnstamm) trace the origin of motor or centrifugal fibres to this nucleus. Marinesco thinks the nucleus ambiguus is the motor nucleus for the transversely striated, the posterior nucleus for the non-striated muscles supplied by the vagus. He has lately along with Parhon (*Journ. de Neurol.*, 1907) tried to determine the intimate connections of the various ganglion groups to the different muscles. Kohnstamm has expressed a similar view; he terms it the visceral nucleus or the *sympathetic nucleus of the medulla oblongata*, and thinks fibres arise from it for the smooth muscles, the heart, and salivary glands. Some of these fibres enter the pars intermedia and pass by means of it into the facial and chorda. Kosaka and Yagita (*Neurologia*, 1905) have reached similar results and conclusions. The vagus root terminates or arises in the nucleus of the *same* side, but a few bundles pass through the raphe to the nucleus of the other side (Schwalbe, Bunzl-Federn).[2]

It is very probable that the fasciculus solitarius forms a common *sensory* root of the vagus and glossopharyngeus, which does not ascend from the spinal cord, as was formerly thought, but, originating from the root ganglia, passes into the medulla oblongata and then turns downwards. It is now generally termed the descending or spinal glossopharyngeal-vagus root. It ends in the nucleus of the solitary bundle,—the only sensory vagus nucleus,—into which enter the centripetal fibres belonging to the vagus, which run in the trigeminus and facial, or intermediary nerves (Kohnstamm).[3] Kosaka and Yagita conclude from their investigations that the solitary bundle enters into connection with the anterior horns of the spinal cord by means of fibres which pass downwards, and that these tracts have to do with respiration.

There is still great uncertainty of opinion in regard to the part played by the accessory nerve in the innervation of the muscles supplied by the vagus. Formerly the motor fibres for the laryngeal muscles contained in the vagus (and those also for the pharynx, stomach, etc.) were traced to the accessorius (Bischof, Longet, Bernard, Schiff, etc.). This view was opposed by

[1] Compare his latest work with Wolfstein in the *Journ. f. Psychol.*, viii.
[2] *M. f. P.*, v.
[3] *M. f. P.*, viii.

Grossmann and specially by Grabower.[1] The latter showed by experiment that the motor fibres for the larynx are contained in the lowest fourth and fifth root bundles of the vagus. He then demonstrated, by anatomical investigations carried out in my laboratory, that these fibres arise from the nucleus ambiguus, the accessorius alone having a nucleus in the spinal cord. According to his account, therefore, the so-called vagus accessory does not exist, the internal ramus being a part of the vagus nerve. This had already been suggested by Holl, and has apparently been recognised by most recent writers. Bunzl-Federn, from his experimental investigations, as well as Roller, Darkschewitsch, and Dees, trace the accessory nucleus up into the oblongata, and find fibres passing from it to the root of the vagus. Gehuchten also thinks the motor fibres for the larynx arise in the accessory nucleus. Kriedl from experimental investigations on apes gives us exact data as to the relations of the various root bundles of the vagus with the branches to which they give origin and the muscles which they control. Kohnstamm and others agree with him that the entire root area of the vagus (or vagus-glossopharyngeus-accessorius-system) is divided, regardless of its peripheral course, into three bundles, the superior (glosso-pharyngeus), the median (vagus), and the inferior (vagus accessorius nucleus).

The vagus has a very widespread area of innervation; it extends to the pharynx, larynx, heart and lungs, the œsophagus, abdomen, and even to the intestine. By means of the auricularis vagi, the vagus fibres reach the fundus of the external auditory meatus. The vagus or vagus-accessory plays an important part in the *innervation of the palate*; some writers even regard it as the only motor nerve of the palate. A branch of this nerve, the *pharyngeal*, along with the glossopharyngeal (and sympathetic) forms the pharyngeal plexus, which innervates the muscles and mucous membrane of the pharynx.

Of the two laryngeal nerves, the *superior laryngeal* innervates the cricothyroid muscle, perhaps also the thyro-epiglottic and aryepiglottic folds of mucous membrane, that of the epiglottis as well as that of the entrance to the larynx as far as the glottis. The sensory distribution does not seem to be strictly unilateral. According to Hedon the superior laryngeal also contains vaso-dilator and secretory fibres for the laryngeal mucous membrane. The inferior recurrent laryngeal innervates all the remaining laryngeal muscles and the part of the mucous membrane below the rima glottidis. Opinions still differ as regards the part played by the inferior laryngeal nerve in sensory innervation.

Section of the vagus in animals produces delayed and deepened respiration from interruption of the fibres which have a reflex exciting action upon the respiratory centre. The superior laryngeal contains centripetal fibres, stimulation of which makes the breathing slower and deeper, or causes cessation of respiration and occlusion of the rima glottidis. The pulmonary branches contain motor fibres for the non-striped muscles of the bronchi, sensory fibres (which excite cough) for the bronchi and lungs, others, which when stimulated have an inhibiting action upon the inhibitory heart fibres, and therefore cause a rapid pulse, as well as the centripetal fibres already mentioned to the respiratory centre. Bilateral section of the vagus in animals causes death, as particles of food reach the lungs on account of inadequate closing of the larynx (Traube). Schiff states that a neuroparalytic hyperæmia develops from paralysis of the vasomotors in the lungs. Bilateral section of the vagus may be followed by no symptoms, if the nerves are not divided simultaneously but successively, a considerable period being allowed to elapse between the operations (Nicolaides). Eichhorst regards the vagus nerve as a trophic centre of the heart muscle, its section producing fatty degeneration. Mollard-Regaud and others do not agree with him.

The œsophageal branch innervates the muscles and mucous membrane of the œsophagus. The vagus contains both *secretory* and vasomotor fibres for the gastric mucous membrane, section of the main trunk of the vagus producing hyperæmia in it. P. Maass shows that both vaso-constrictor and vaso-dilator fibres go to the coronary vessels, the vasodilator being mostly contained in the vagus. The vagus also sends *motor* fibres to the stomach. According to Bischoff, Batelli, and others, however, these come from the accessorius, but from the roots, it is true, which Grabower and others ascribe to the vagus. It is maintained that the vagus sends not only motor but also inhibitory fibres to the œsophagus and stomach (Kronecker-Meltzer, Openchowski, Langley), whilst other writers (Courlade-Guyon) regard the sympathetic as the inhibitory nerve. The groups of nerve-cells in the stomach wall have also been considered to be automatic centres for movement and secretion (Mering-Aldehoff), but they may be influenced from the central

[1] *C. f. Phys.*, 1890, and *A. f. Laryng.*, 1894. See also Onodi, "Die Anat. und Physiol. der Kehlkopfnerven," Berlin, 1902; Hudovernig, *N. C.*, 1904, and *Journ. für Psych.*, ix.

nervous system. Trophic disturbances—ulceration of the gastric mucous membrane—have been attributed to injury of the vagus (Lorenzi). Carion and Hallion noted dilatation of the stomach after section of the vagus. Symptoms of spasm of the cardiac orifice of the stomach and dilatation of the œsophagus are also observed in vagus disease and attributed to this nerve (Kraus, *Internat. Beitr. z. inn. Med.*, Richartz, *D. m. W.*, 1905). Intestinal movements are partially under the influence of the vagus. It contains *inhibitory fibres* for the heart movements, which run in the lowest root bundle (Cadman, Gehuchten), but it also has to do with the acceleration of the heart. We may call to mind the interesting observation that some individuals can accelerate the action of the heart by directing their attention to it. Slight vagus stimulation increases the diastole, stronger stimulation stops the heart in diastole. Weak vagus stimulation may also, according to the observations of some physiologists, be followed by acceleration of the heart beat. Our ideas as to the influence of the nervous system upon the action of the heart have lately undergone many changes, due chiefly to the investigations of Engelmann, His, Romberg, Krehl, etc. Engelmann, in particular, maintains that the action of the heart muscles is independent of the nervous system; indeed, he even questions whether motor nerves reach the heart muscle at all, and he thinks that the nerve fibres and ganglion systems contained in the heart are sensory in nature. Much opposition has been raised to this view by Kronecker and others. With regard to the depressor nerve, the work of Cyon and Ludwig and of Köster-Tschermak (*A. f. An.*, 1902, Suppl.) should be referred to. Finally, the vagus is thought to have an influence upon the renal secretion.

Etiology.—The vagus may be injured at various points in its course by morbid processes developing in its neighbourhood, but it is seldom affected by a primary neuritis. A rheumatic form of *neuritis*, limited to one or both recurrent nerves, is occasionally mentioned. The vagus is also sometimes involved in multiple neuritis, especially in the alcoholic form. Diphtheritic paralysis comparatively often invades this nerve. As a rule the paralytic symptoms are due to neuritic or degenerative changes in the nerve and its ramifications (P. Meyer, Vincent, etc.). Signs of vagus or recurrent paralysis have been observed in typhoid (Lublinski, Zur Helle, Weil), pneumonia (Schroetter, Botkin), scarlatina (Gottstein), malaria (Schech), cholera (Matterstock), influenza (Schmidt, Krackauer, Réthi, Lähr), gonorrhœa (Engel-Reimers, Lazarus), and other infective diseases.

Poisons other than alcohol may also affect the nerve; the most important are chronic *lead* intoxication, and *arsenical* poisoning (Imbert-Gourbeyre).

I have seen a case of acute neuritis of the vagus nerve in a brass-founder who worked with lead, zinc, and phosphorus. Disease of the vagus and recurrent laryngeal has also been found in experimental chronic lead poisoning in the horse (Thomassen).

Paralysis of the vocal cords is observed in acute poisoning with atropin and morphia. Hæmorrhage into the vagus nerve was seen by Reichel in phosphorus poisoning. Atrophy of the vagus has occasionally been found, for which no cause was discovered.

The vagus symptoms occurring in tabes are usually of bulbar origin, but they may be due, as I have shown, to degeneration of the nerve itself. The vagus paralysis caused by diseases of the medulla oblongata (tumours, softenings, hæmorrhages, bulbar paralysis, tabes, disseminated sclerosis, etc.) is considered elsewhere.

I was able to trace a recurrent paralysis in cervical ribs to a gliosis which was also present (see p. 435).

This nerve is somewhat frequently affected in its *intracranial* course in the base of the skull—by *meningitic exudation*, *hæmorrhages*, *tumours*, *vertebral aneurisms*, *periostitis*, and carious processes. In these basal

affections it is usually affected in common with other cranial nerves, especially with the glossopharyngeal, accessory, and hypoglossal. A lesion involving all these nerves gives rise to a characteristic group of symptoms. *Arteriosclerotic* diseases of the vessels in the posterior cranial fossa, especially of the vertebral and inferior cerebellar arteries, may directly involve the vagus by pressure and traction. In thrombosis of the transverse sinus and the jugular vein the vagus is sometimes involved (Stacke and Kretschmann, Schwarze, Kessel). Suppuration in the neighbourhood may extend to any part of the nerve (Martius).

Trauma and operations on the neck not infrequently injure the tenth nerve. It is especially often injured in ligature of the carotid and excision of tumours. The discussions on this matter have been reviewed by Deibel,[1] Traumann,[2] and Weidner.[3] Gunshot-wounds, which almost always affect other nerves, such as the hypoglossal and the sympathetic along with the vagus, are also described (by Hirsch, for instance). In other cases the vagus or the vagus recurrent may be compressed by *tumours in the neck*, or in the mediastinum, especially glandular tumours and aneurisms of the aorta (also of the carotid and subclavian). In nineteen out of sixty-nine cases, Syllaba Lad[4] found the cause of paralysis of the recurrent laryngeal nerve to be atheroma of the aorta. Recurrent paralysis is also observed in mitral stenosis (Ortner, Krauss, Hofbauer, Alexander), and is ascribed to the dilatation of the auricle or tension of the ligamentum arteriosum Botalli and the pressure thereby exerted on the nerve, and is also found in patent ductus arteriosus (Schrötter[5]). Ohm[6] found it in pneumothorax and traced it to displacement of the heart, as did Lublinski.

The vagus or recurrent paralysis which appears occasionally in tuberculosis has been sometimes ascribed to compression of the nerve by the thickened pleura, but more frequently it is due to greatly enlarged lymph glands. There may no doubt be, however, a simple neuritis of this nerve in the course of tuberculosis, such as is observed in other nerves. The recurrent laryngeal is also involved in other affections of the lungs and pleura (Bäumler, Unverricht, Landgraf). The paralysis of the muscles of the vocal cords which occasionally develops in largyngitis is usually of muscular origin. Some of the paralyses occurring in infective diseases are also apparently of this nature.

The vagus symptoms (aphonia, palpitation of the heart, respiratory troubles, etc.) appearing in the *functional neuroses* (hysteria) are of central nature and are discussed elsewhere. Gerhardt mentions a benign form of recurrent paralysis due to over-exertion. Finally, *tumours* on the nerve itself, especially neuromata, are noted in isolated cases.

Symptoms.—The symptoms depend to a certain extent on the site of the lesion. Signs of total vagus paralysis are seen specially in the processes which spread to it at the base of the skull, but in these cases other cranial nerves, particularly the superior root of the eleventh and usually also of the ninth and twelfth, are almost always involved. If the lesion is limited to one side, then unilateral *paralysis of the palate, larynx and pharynx* are the typical symptoms ; if the twelfth nerve is involved

[1] " Über die traumatische Vagusparalyse beim Menchen," Berlin, 1881 (*Inaug. Diss.*).
[2] *Z. f. Chir.*, xxxvii. [3] *Z. f. Chir.*, xxxvii. [4] *Arch. bohém. de méd.*, 1902.
[5] *Z. f. k. M.*, Bd. xliii. Félix also gives a review of the causes of recurrent paralysis, *Semaine méd.*, 1905. Dege treats the question of traumatic recurrent paralysis (*B. k. W.*, 1906).
[6] *B. k. W.*, 1905.

paralysis and atrophy of the same side of the tongue supervene.[1] The soft palate hangs loosely down on the affected side and does not move in phonation ; speech is nasal, the difficulty of swallowing which arises from the unilateral pharyngeal paralysis is usually not great, but in a case of Harmer's, for instance, swallowing of solid food was impossible. Erben found protrusion of the posterior wall of the pharynx on the paralysed side, the hyoid bone and the larynx being displaced towards the healthy side. But this symptom, due to flaccidity of the pharyngeal muscles, is inconstant (Möbius, Oppenheim). A wing-like displacement of the pharyngeal wall towards the healthy side in the attempt to swallow has sometimes been noted (also by Oppenheim). The vocal cord occupies the intermediate or cadaveric position and does not move either in phonation or in respiration. The degenerative character of this paralysis has only occasionally been shown by electrical examination. Anæsthesia of the pharynx and larynx (and of the external auditory meatus) is but rarely found. It is hardly possible to pick out from these symptoms those which are related exclusively to disease of the vagus. The same symptoms, excepting of course the lingual paralysis, are observed in injuries of the vagus high up in the neck. But the paralysis of deglutition is as a rule very slightly marked in extracranial vagus affections.

As regards the special symptomatology of paralysis of the palate the symptoms due to paralysis of single muscles are described in the general part of this book, and also by Mann (*Z. f. Ohr.*, Bd. xlvii.).

The combination, very remarkable and difficult to explain, of paralysis of the right side of the palate and larynx and the left vocal cord was seen by myself in a young girl, the affection developing apparently after influenza. In two cases of basal disease I found paralysis of the palate, not on the corresponding but on the opposite side, a condition which I had difficulty in explaining.

The symptoms arising from the *heart* are not constant in unilateral affections of the vagus ; there is sometimes slowing, but much more often *acceleration* of the heart's action, for instance, in compression by tumours (Hayem, Riegel, Stix, etc.), and especially after section. In unilateral division of the vagus all the symptoms, even the laryngeal paralysis, may be absent (Weidner, Gurfein, Reich).[2] Of course the divided nerve has often been previously injured by compression or infiltration. *Respiratory disturbances*, in so far as they are not of laryngeal origin, occur principally in central diseases and bilateral lesions of the nerve. There is sometimes slowing, sometimes acceleration and irregularity of breathing. Occasionally a slow pulse with marked acceleration of the breathing is found in vagus affection. Egger noted respiration as slow as three to four breaths a minute in a tabetic, and he put it down to bilateral vagus paralysis, judging from the experimental results of Herzen and Pawloff. In no case of unilateral section of the nerve did the disturbances of respiration become permanent (Traumann).

[1] The question as to the site of this unilateral paralysis of palate, larynx, neck, and tongue has been much discussed (Semon, Gowers, Schech, Harmer), but there is no doubt that it is due either to radicular or peripheral affections of the corresponding nerves, a nuclear origin of this group of symptoms never having being certainly noted. Compression by tumours, syphilis, or injuries are the causes, but the etiology is not always clear. The unilateral paralysis of palate and larynx, well known to earlier writers (Jackson, Beevor, Oppenheim), has been termed the Avellis' symptom (see Camillo Poli : " Sulla sindrome di Avellis," Siena, 1906), but we have been able to discover no justification for this name.

[2] Bruns, *Beitr. z. Chir.*, Bd. lvi. The author states that on the contrary traumatic excitation of the vagus produces severe symptoms from the heart and respiration. See also H. Schlesinger, *W. kl. R.*, 1908.

Amongst the other symptoms of irritation and paralysis ascribed to the vagus, but which only occasionally occur in peripheral paralysis of this nerve, we may mention vomiting, bulimia, loss of the sense of hunger and thirst, abdominal pains, diabetes (Henrat). In one case vomiting occurred when the exposed vagus was touched. The pneumonia sometimes observed after division of the vagus in man is always a broncho-pneumonia. There are no symptoms pointing to paralysis of the vasomotor nerves to the lung.

Atony and dilatation of the œsophagus seem occasionally to be symptoms of vagus paralysis (Kraus, Richartz, etc.); Bálint observed atony and motor insufficiency of the stomach.

The most important among these symptoms is *laryngeal paralysis*. This is most frequently an independent symptom, both in lesions affecting the vagus itself and more especially the inferior recurrent laryngeal nerve. *Recurrent paralysis* is characterised by the following symptoms: the vocal cord of the same side remains always in the cadaveric position (the vocal cords after death occupy a position about midway between adduction and abduction [1]), and is immobile during both phonation and respiration (see p. 89). If the paralysis is unilateral, phonation can be carried out by the healthy vocal cord being moved across the middle line and thus closing the rima glottidis fairly well. The voice is not necessarily greatly altered, but it is usually somewhat hoarse and rough, or in total paralysis has the character of a falsetto. Abduction of the healthy vocal cord is also sufficient for the dilatation of the rima glottidis in inspiration, but deep inspiration is usually accompanied by stridor, and coughing is as a rule noiseless.

Grossmann maintains that his experiments show that the vocal cord occupies the median position, and he ascribes this to the action of the cricothyroid; this, however, is disputed by F. Klemperer, Chiari, Herzfeld, Dubois-Raymond, Katzenstein, and others from experimental and clinical observations. Grabower has seen it in the median position, but in his experience it rapidly passes into the cadaveric position, as the cricothyroid at first acts as a tensor of the vocal cord in place of the paralysed internal thyro-arytenoid muscle, and after a few days becomes itself paralysed (*A. f. Laryng.*, vii., and *B. k. W.*, 1906). See further Sinnhuber (*B. k. W.*, 1904, and *A. f. kl. M.*, Bd. lxxix.) and the discussion in the *B. k. W.*, 1906, Nr. 43. and 44; also Rosenbach (*B. k. W.*, 1906, Nr. 46).

In *bilateral recurrent paralysis*, which is but rarely caused by a peripheral lesion of the vagus nerves (compression from large tumours, scalding of the œsophagus in Benenati's case, etc.), the symptoms are severe. There is complete *aphonia*; the glottis is not closed in coughing, and breathing is not only affected by the insufficient width of the glottis, but also by the fact that in inspiration the vocal cords are aspirated, drawn close together, and the rima glottidis is thus closed; hence the long-drawn *inspiratory stridor* and the *dyspnœa*. It is not rare for injuries affecting the vagus (central or peripheral) and the recurrens to cause paralysis of the posterior crico-arytenoids (abductors) alone; there is then *respiratory laryngeal paralysis* in normal phonation. A secondary contracture of the extensors of the vocal cords may then develop, which may increase the difficulty in breathing. Compression or constriction of the recurrens may also produce this condition. Krause regards it as a *primary* contracture of the adductors (lateral crico-arytenoids), but this is not the correct explanation, although we do not question the occurrence of a

[1] See, however, the work of Rosenbach in *B. k. W.*, 1906.

primary adductor contracture. The symptom is much more likely due to a primary paralysis of the abductors (Rosenbach, Semon), by the lesion affecting the nerve. The fact so often noted in pathology is here again repeated, that an injury affecting a nerve (mechanical, toxic, infective) by no means affects all its fibres to the same extent.

Semon distinguishes three stages in progressive recurrent paralysis: 1. that of isolated posticus paralysis, characterised by lessened amount of abduction of the vocal cord; 2. that of posticus paralysis with secondary contracture of the adductors, characterised in addition by fixation of the vocal cord in or close to the middle line; 3. that of complete recurrent paralysis with cadaveric position.

Semon and Horsley (*Intern. Centralbl. f. Laryngol.*, xi.) found experimentally that after the death of the animal the electrical excitability was first lost in the abductors. Donaldson and Hooper came to similar conclusions. According to Fränkel and Gad, in gradual freezing of the nerve the functional disturbances first occur in the abductors. Frese found this with regard to the effect of chemical agents. It is stated by Risien Russell that the fibres for the posterior cricoarytenoid muscles form a separate bundle in the recurrens. In the horse the fibre bundles for respiration and phonation have a separate course in the vagus and recurrens (Onodi). Finally, Grabower thinks he found differences in the conditions of the nerve endings between the postici and the adductors and a smaller number of fibres in the posticus (*B. k. W.*, 1904). Broeckaert, who admits the correctness of Semon's law for central diseases, ascribes the earlier paralysis of the abductors in peripheral recurrent paralysis to the fact that the posterior cricoarytenoids are naturally more feeble muscles; Kuttner, however, disputes this explanation. There is a case by Saundby in which lesion of the nerve was first followed by paralysis of the contractors of the glottis, but an observation so isolated cannot overthrow the Rosenbach-Semon law, as Rosenbach himself maintains (*B. k. W.*, 1906). The discussion as to the innervation of the adductors in quiet breathing, the occurrence of the median position in recurrent paralysis, etc., is not yet closed (Kuttner-Katzenstein, Dorendorf, Grabower (*B. k. W.*, 1904), Kuttner (*A. f. Laryng.*, Bd. xviii. and xix.), etc.).

Recurrent paralysis has not infrequently been observed after infective diseases (typhoid, diphtheria, etc.).

A bilateral paralysis limited to the phonators is practically always of central origin, and due less frequently to organic affection of the medulla oblongata than to hysteria. Organic diseases cause either respiratory paralysis alone or usually a combination of phonatory and respiratory paralysis. Incomplete paralysis of the phonation muscles is also noted, however, in organic nervous diseases, such as disseminated sclerosis, bulbar paralysis, etc. Pure recurrent paralysis may occur also in hysteria.

Isolated paralysis of the *superior laryngeal nerve* has been reported only in a very small number of cases, after injuries and operations on the neck. It manifests itself by paralysis of the cricothyroid—defective approximation of the thyroid and cricoid cartilages in phonation—hoarse, deep voice, rapid fatigue in intonation—and anæsthesia of the laryngeal mucous membrane. Movement of the epiglottis may be abolished by paralysis of the thyroaryepiglottidean muscle, although the relation of the nerve to this muscle is questioned, *e.g.* by Gerhardt. Moeser and Dorendorf describe a higher position of the posterior segment of the margin of the vocal cord on the paralysed side. The latter thinks there is also outward rotation of the arytenoid cartilage in the condition of rest.

Holger Mygind (*A. f. Laryng.*, xviii.) mentions defective adduction and folding of the anterior half of the vocal cord.

Some authors think that the inferior laryngeal participates in the sensory innervation of the larynx. Massei (*B. k. W.*, 1906) in particular holds that anæsthesia of the entrance to the larynx

is a constant symptom of recurrent paralysis, and Onodi teaches that the innervation of each side is bilateral. Avellis (*A. f. Laryng.*, xviii.) believes this also of the superior laryngeal nerve. Kuttner and Meyer (*A. f. Laryng.*, xix., and *B. k. W.*, 1907), however, strongly oppose Massei's view.

Remak (*B. k. W.*, 1903) ascribes to the sensory fibres of the superior laryngeal a part in the galvanic deglutition reflex, and regards its absence as a sign of their paralysis, but B. Fraenkel disputes this.

Bilateral paresis of the thyroarytenoid muscles, along with paralysis of the posterior cricoarytenoids, has been noted in *lead poisoning*, as has unilateral paralysis of the vocal cords.

The degenerative character of recurrent paralysis has been made obvious to me [1] in some cases by the fact that electrical stimulation of this nerve at the neck by the use of the strongest current produced no results. Normally it elicits adduction of the vocal cords.

This is not the place to discuss the so-called vagus neuroses : paroxysmal tachycardia, nervous asthma, etc. (see chapter on neurasthenia).

Treatment may sometimes be directed by causal indications. In such cases iodide and mercury are often efficacious when syphilis is the cause. I have seen a recurrent paralysis disappear under specific treatment, and Steinhaus has had similar success in a case of bilateral recurrens paralysis. Vagus affections in alcoholic paralysis make it necessary to prescribe the alcohol (wine, brandy), which would otherwise be withdrawn, as well as other stimulants. In lead paralysis of the larynx the general poisoning must be treated by baths, counter-irritants, and iodide of potassium.

The removal of a tumour compressing the vagus or the treatment of an aneurism may dispel the signs of the vagus lesion, such as the recurrent paralysis (Landgraf, Litten), but spontaneous remissions and recoveries of this paralysis occur in aortic aneurisms (Groszmann, Berent [2]). Absorption of lymphoma is sometimes brought about by the use of preparations of iodide, arsenic, and strengthening diet, or by the use of the X-rays.

In section of the vagus there may be a question of transplantation of the nerve or anastomosis with a neighbouring nerve, as has been done experimentally by Calugareanu and Henri.[3]

The result of electrical treatment of laryngeal paralysis due to organic disease is doubtful. However, some cases of apparently rheumatic paralysis of the recurrens have been described in which the cure was attributed to faradisation. It is always advisable to limit it to the percutaneous electrical stimulation. Intralaryngeal stimulation may greatly aggravate the respiratory difficulty in posticus paralysis, as it has both a direct and reflex effect upon the tensors of the vocal cords. In order to excite the recurrent laryngeal externally, a button-shaped electrode (cathode) is placed between the inner margin of the sternomastoid and the larynx at the level of the cricoid cartilage, and deep pressure is applied to the nerve. The unaffected nerve can be successfully excited by the closing of a strong galvanic current.

Gymnastics and massage of the larynx are also recommended as being successful : by pressure on the posterior part of the thyroid cartilage the arytenoid cartilage and the vocal cords are approximated, whilst the

[1] *A. f. P.*, xviii. [2] *B. k. W.*, 1904. [3] *Journ. de Physiol.*, 1900.

patient is requested to phonate. This method can only have a material result in hysterical phonation paralysis (see chapter on hysteria).

Posticus paralysis may necessitate tracheotomy.

Paralysis of the Spinal Accessory Nerve

According to the opinion expressed in the previous chapter, we are not justified in speaking of an inner branch of the spinal accessory, the vagus accessory, as these fibre bundles belong from their origin to the vagus. Some recent writers, however, are inclined to assign to the accessory, besides its spinal nucleus, which arises from the external ramus, other cell groups in the oblongata, and to include the corresponding root bundle with the accessory, so that this question cannot yet be regarded as definitely disposed of.

The *external* ramus, or accessory nerve, innervates the *sternomastoid* and *trapezius* muscles. The former is almost entirely under the control of the accessory and receives no nerve fibres, or very insignificant ones, from the second and third cervical nerves. But the cervical nerves take a greater part in the innervation of the trapezius, both of its clavicular and more particularly of its acromial portions (Sternberg, Schulz), so that a lesion which paralyses the spinal accessory does not always cause complete paralysis of the trapezius. The share taken by the cervical nerves in the innervation of this muscle appears indeed to vary individually. This partly explains the circumstance that the observations of various writers (Remak, Bernhardt, Schmidt, Schlodtmann,[1] Laehr,[2] Cassirer, etc.) do not entirely agree as to the condition of the middle part of the trapezius in paralysis of the nerve. In one of my cases, in which the spinal accessory was divided on both sides and a large part of it excised for spasm of the muscles supplied by the nerve, the sternomastoid had completely disappeared, but the trapezius, although it was markedly weak and atrophied, still contained muscle bundles capable of contraction. There was only a slight degree of rotation of the scapula. Schultz[3] ascribes only the lower portion of the trapezius entirely to the accessorius.

Apart from diseases of the upper cervical region, which may destroy the nucleus and its roots and thus give rise to symptoms of paralysis in the muscles supplied by both spinal accessory nerves (superior cervical myelitis, progressive muscular atrophy of spinal origin, cervical gliosis, etc.), the following are the chief causes of peripheral paralysis : *caries* of the upper cervical vertebræ with *compression* of the nerve roots by tubercular granulomata and abscesses, *tubercular and syphilitic pachymeningitis*, *new growths*, and *meningitic exudations* in the region of the foramen magnum, lesions extending from the petrous bone into the foramen jugulare[4]; further, *injuries* of the nerve at the neck (especially during operations on tumour for the relief of accessory spasm, etc., as in a case I saw in which the accessory was injured and paralysed in resection of cervical ribs), or compression of the nerve by tumours. Of late years the nerve has been divided in the operation of nerve anastomosis for incurable facial paralysis (Faure, Kennedy, Bernhardt-Körte, Stewart-Ballance, etc.).

A primary neuritis of the nerve also occurs.

[1] *Z. f. N.*, v. (Literature). [2] *N. C.*, 1899. [3] *Z. f. N.*, xxii.

[4] Leroux's thesis (Paris, 1902), "La névrite spinale d'origine otique," treats mainly of symptoms of irritation of the muscles supplied by this nerve.

It is not yet known whether the accessory paralysis noted in some cases of *tabes* (Martius, Ilberg, Ehrenberg, Seiffer, Oppenheim) is of central or peripheral origin.

Affection of this nerve is usually *unilateral*, but bilateral disease has been occasionally observed, the vagus being frequently and the hypoglossal occasionally affected, especially in lesions at the base of the skull, in the foramen jugulare, or directly after their emergence from it. In one case of extirpation of a tumour at the neck, the sympathetic, hypoglossal, and accessory (at the foramen jugulare) were divided.

The *symptoms* are, so far as our present knowledge goes, exclusively motor: there is paralysis of the sternomastoid and of the trapezius, the latter, however, being in the majority of cases incomplete. In this case there is not necessarily deformity. This only arises when a secondary contracture develops in the muscle of the unaffected side. The symptoms of paralysis of the sternomastoid are inability to turn the head or the chin completely towards the opposite side, and the absence of visible tension of the sternomastoid during the effort to make this movement, and also in deep inspiration. I have also occasionally seen the omohyoid muscle become unusually prominent in speaking, etc. In bilateral sternomastoid paralysis the head easily falls backwards and it cannot, especially in the horizontal position, be brought forward without assistance. In one case which I examined, however, this movement could be carried out with undiminished power.

The anomalies of the position of the shoulder-blade and the motor disturbances due to complete trapezius paralysis have already been described on p. 13. We shall merely mention that the incompleteness of the paralysis found in many cases is specially revealed by the *absence of rotation of the shoulder-blade* (Schaukelstellung). Paralysis of the trapezius somewhat limits the power of moving the arm, especially of elevating it. This movement may be painful. The paralysis is, except in the slightest cases, degenerative, and is characterised by the well-known changes of electrical excitability.

The combination of unilateral paralysis of the vocal cord or recurrens, and of the trapezius and sternomastoid of the same side, was formerly ascribed to simultaneous affection of the so-called internal and external branches of the accessorius, as in a case by Lermoyez and Laborde (*Ann. des mal. de l'oreille*, 1901). It may be well explained by a lesion limited to the corresponding nuclei or root bundle, or by disease of the accessory and vagus nerves of the same side, if we suppose that only the laryngeal branch in the latter is affected.

The *prognosis* is given in accordance with the primary disease.

Treatment is specially successful when the cause is a syphilitic process or a slight trauma. The paralysis should be treated by electricity. For bilateral paralysis of the trapezius Gaupp recommends a supporting apparatus to draw backwards the shoulder girdle which has sunk forwards, and enable the unaffected muscles (serratus, deltoid, etc.) to exert their full strength and to cure the pain due to traction.

E. Remak[1] points out that the functional disorders occurring in section of the accessory are less serious if the operation is performed high in the neck than if it is made near the entrance of the nerve into the trapezius, as in the latter case the nerve has already become united with branches from the cervical nerves. Observations by Sternberg, Neisser, Laehr, Krähemann,[2] etc., corroborate Remak's view.

[1] *B. k. W.*, 1888 and 1892.

[2] *Inaug. Diss.*, Leipzig, 1903.

Paralysis of the Hypoglossal Nerve

The twelfth nerve is much more often affected by morbid processes in its intracerebral and bulbar than in its peripheral course. Out of seventy-nine cases which Ascoli [1] collected from the literature, only one-third had a peripheral cause. The intracerebral nerve tract, which goes from the cortical centre to the nucleus in the medulla oblongata, accompanies the tract for the extremities (at least within the cerebrum), so that hypoglossal paralysis of the same side is an almost constant accessory symptom of hemiplegia. It is never accompanied by atrophy of the tongue, as the trophic centre is contained in the hypoglossal nucleus of the medulla oblongata.

Diseases of the *medulla oblongata* usually involve the hypoglossal nucleus and the roots on *both sides*, and cause a bilateral atrophic paralysis of the tongue, which is almost always associated with paralysis of other cranial nerves. A *unilateral* nuclear disease is not, however, very rare.

Peripheral hypoglossal paralysis is mainly caused by morbid processes in the posterior cranial fossa, which injure the nerves at this site. Tumours of the base of the skull, meningitic exudations, basal hæmorrhages, carious processes may have this effect. As the hypoglossal lies near the vagus and accessory, these nerves are also compressed, as a rule at the same time (and on the same side), by tumours and exudations near the medulla oblongata, the result being the triad of symptoms already described, viz. paralysis of the palate, of the larynx, and of the tongue (the spinal accessory also being often involved). *Aneurisms* of the vertebral arteries may also in this way affect the twelfth cranial nerve. It may be injured as it passes through the anterior condyloid foramen by syphilitic (Lewin) and carious processes (Oppenheim, Lüschow,[2] etc.). Paralysis of this nerve has also been noted in caries and dislocation of the first cervical vertebra. M. Brasch describes basal fracture with involvement of the hypoglossal canal as a cause of paralysis limited to the hypoglossal nerve. In a case of Dupuytren's the nerve was compressed at this site by hydatid cysts.

It is also occasionally injured in common with the tenth and eleventh cranial nerves after it leaves the cranial cavity. It may be paralysed by wounds in the neck, by the *compression* of tumours or injuries during their *removal*. Cases of this kind are described by Hutchinson, Weir Mitchell, Schüller, Bernhardt, Remak,[3] Traumann,[4] Paget, Ascoli, Biancone,[5] Debove,[6] etc. An unavoidable paresis of this nerve has recently been produced as the result of a surgical anastomosis between it and the facial (Körte, etc., see p. 415).

A simple isolated neuritis does apparently occur, though very rarely (Erb,[7] Montesana, Marina,[8] Hoffmann,[9] Panski.[10] In several of the cases regarded as such, there was a previous acute infective disease (scarlatina, angina), usually involving the cervical glands. It is doubtful whether hemiatrophy of the tongue may also have a *toxic origin* (lead, arsenic, alcohol). Pastorvich [11] has recently described a case in which alcoholism was apparently the cause.

[1] *Il Policl.*, 1897.
[2] *Inaug. Diss.*, Greifswald, 1884.
[3] *B. k. W.*, 1888 and 1892.
[4] *Z. f. Chir.*, 1893.
[5] *Riv. speriment. di Fren.*, xxix.
[6] *Presse méd.*, 1903.
[7] *A. f. kl. M.*, 1885.
[8] *N. C.*, 1896.
[9] *N. C.*, 1899.
[10] *N. C.*, 1903.
[11] *Riv. speriment. di Fren.*, xxvii.

Hemiatrophy of the tongue has also been observed in tabes, syringomyelia, disseminated sclerosis, either alone as a congenital symptom, or combined with atrophy of other cranial nerves or muscular defects, and as one of the symptoms of facial hemiatrophy. In Heubner's case, in which a hypoplasia of the nerve nuclei was histologically proved, the hypoglossal nucleus was involved. Gierlich has described a similar case.

Peripheral hypoglossal paralysis is characterised by paralysis and atrophy of the corresponding half of the tongue. As it lies on the floor of the mouth the tongue does not deviate towards the healthy side, or does so at the tip only. Traumann ascribes this to the fact that the tonus of the longitudinal muscles causes slight shortening of the unaffected side. Gowers mentions that in the mouth the root of the tongue is higher on the paralysed side than on the other, as the result of absence of tonic contraction of the posterior fibres of the hypoglossus. I have occasionally seen this, but have also failed to find it under the same conditions. Traumann blames the loss of function of the genioglossus, the tonus of which maintains the tongue in its normal position. Inside the mouth the tongue as a rule cannot be moved completely towards the affected side, so that it is difficult to touch the upper jaw, the teeth, or the palate on this side with the point of the tongue, to move particles out of the cheek-pouches, etc. This paresis is seldom very pronounced. When protruded the tongue deviates towards the paralysed side, and the raphe forms a curve, the concavity of which is turned towards the paralysed side (see Fig. 38, p. 87, and Fig. 78, p. 154). Dinkler[1] describes the characteristics of the deviation in peripheral hypoglossal paralysis as follows: deviation towards the affected side with curving of the point towards the unaffected side when protruded; deviation towards the unaffected side when the (root of the) tongue is drawn back. The deviation of the protruded tongue is due to paralysis of the genioglossus, contraction of which moves it towards the opposite side. The curving of the raphe is perhaps caused by paralysis of the interior muscles of the tongue (longitudinal, transverse) which contract only on the unaffected side. In partial hypoglossal paralysis there may be no deviation of the tongue when protruded.

The paralysis is accompanied by atrophy. On the affected side the tongue is wrinkled and shows strong fibrillary tremors; it is narrowed and feels soft and flaccid. Electrical examination shows reaction of degeneration, which is usually not quite complete. This may also be sometimes detected on the floor of the oral cavity (genioglossus).

The tongue can be easily moved passively towards the other side, in contrast to hysterical hemispasm of the tongue, in which an attempt at this movement is met with resistance (E. Remak).

Involvement of the *external laryngeal muscles* innervated by the ansa hypoglossi (sternohyoid, sternothyroid, and omohyoid) is but rarely reported (Möbius, Remak). The motor fibres for these arise practically in the upper cervical roots, so that lesions affecting the nerve above the entrance of these fibres cause no paralysis of these muscles. It would be revealed by atrophy of the muscular layer covering the thyroid cartilage and the lateral displacement of the larynx in swallowing.

It is stated (Tooth) that the hypoglossal nucleus takes part in the innervation of the orbicularis oris muscle, but we have only one case pointing to this, that reported by Bruggia-Matteucci

[1] *Z. f. N.*, xiii. See also Flesch, *M. m. W.*, 1908.

(*Arch. ital. p. l. mal. nerv.*, 1887), so that we still remain very sceptical regarding it; of course this may be an unusual individual relation.

Functional disturbances due to unilateral paralysis of the tongue are not usually grave, and the mobility of the tongue is not necessarily essentially affected. The speech disturbance is slight even in complete hemiglossoplegia (difficulty in pronouncing *x* and *sh*, according to Dinkler), but it is marked when the paralysis is bilateral, even though it is incomplete. Difficulty in swallowing and masticating only occurs in bilateral hypoglossal paralysis, but it is very rarely of peripheral origin.

A favourable course has occasionally been observed in syphilitic and traumatic hypoglossal paralysis. Rheumatic (post-infective) neuritis may also recover (Marina, Panski). I have also seen recovery of hemiatrophy of the tongue in vertebral caries. Wiersma [1] noted the disappearance of a hypoglossal paralysis, due to suppuration of the glands.

From a *therapeutic* point of view there is little to add to the measures recommended for paralysis of the other cranial nerves. Wölfler [2] successfully employed nerve suture in section of the hypoglossus in a case of suicide.

Multiple Neuritis (Polyneuritis) [3]

We owe our knowledge of this disease mainly to Leyden [4] and to Duménil, Lancereaux, Leudet, Eichhorst, and Eisenlohr, who had described cases of it, even before him. Its causes are very numerous. They are for the most part *toxic*. Among the poisons which produce it, alcohol must be assigned the first place. The great majority of cases of multiple neuritis which we have had the opportunity of seeing in this country (Germany) are due to alcoholism. It is not only the spirit drinker who is affected, but also the excessive beer drinker; the wine drinker is less susceptible. *Lead paralysis* is also a form of multiple neuritis, but its symptoms give it a quite special character. *Arsenical* poisoning not infrequently gives rise to paralytic symptoms, which, as a rule, are due to multiple neuritis. It is only in very exceptional cases that the disease can be traced to poisoning with *copper*, *zinc* (?), *mercury*, *carbonic oxide*, *bisulphide of carbon*, *anilin*, *phosphorus*, to meat or sausage poisoning, etc. It is not certain whether chronic nicotine poisoning may cause a polyneuritis; Baccelli gives a case which he attributes to this.

Infective diseases are also important causes of multiple neuritis. It may appear in the course of, and more especially after *typhoid*, *small-pox*, *scarlatina*, *influenza*, *erysipelas*, *pneumonia*, empyæma, *acute articular rheumatism*, parotitis, gonorrhœa, dysentery, measles, Pasteur's inoculation for rabies (Darkschewitsch), *whooping-cough and diphtheria*, but we have still to ascertain whether this post-infective paralysis is always due to a peripheral neuritis. A *septicæmic* and *puerperal* form of this disease is also described.

In a small number of cases there had been previous *gastro-intestinal* disorders (Wagner), obstinate constipation, purulent bronchitis (Min-

[1] *N. C.*, 1899.

[2] *D. m. W.*, 1905.

[3] For the most important references to the literature see E. Remak (and Flatau), "Neuritis und Polyneuritis," Nothnagel's "Spez. Path. u. Ther.," xi., T. 3, " Wien, 1900. Also Raymond, "Maladies du Système nerveux," 1889, 1894, 1897.

[4] *Charité-Annalen*, 1878; *Z. f. k. M.*, i., and "Die Entzündung der peripherischen Nerven." Zwei Vorträge, Berlin, 1888.

kowski), and the cause of the polyneuritis was believed to be an *auto-intoxication*. Thus Poljakoff and Choroschko ascribed the disease to the bacterium coli. Jaundice and cirrhosis of the liver have occasionally been regarded as causing the disease (Kausch, Gerhardt, Gouget, Larrier, and Roux).

Numerous observations have proved that *tuberculosis* causes not only slight conditions of degeneration in the peripheral nervous system, which remain clinically latent, but also severe inflammatory and degenerative processes with the clinical features of polyneuritis. The disease can be brought into relation with *syphilis* in a few cases only, but there is no doubt that a multiple neuritis may be caused by syphilis. Polyneuritis may also be due to *malaria*.

Its relation to *diabetes mellitus* is shown by clinical and pathological observations. *Gout* has also been included among the causes (Gowers, Ebstein, Gruber, Thomayer, etc.), but so far as I know no case of polyneuritis unquestionably due to gout has been described. The existence of uræmic polyneuritis (Crocq, Nogués-Sirol, Dunger) seems to me still uncertain.

In the cases in which the disease occurred without apparent cause, and corresponded in development and course to the type of an acute infective disease, there was probably some *infective* agent which acted directly upon the nervous system. The theory has been propounded that the micro-organisms of pneumonia, acute articular rheumatism, cerebro-spinal meningitis, etc., produce under certain conditions an immediate polyneuritis instead of the corresponding infective disease.

Beri-beri (kakke) is probably an endemic form of multiple neuritis. Eisenlohr has pointed out that even in Germany cases may occur with remarkably exaggerated frequency at certain times and places. An epidemic has also been described by Hammond, Boudurant, etc.

Severe forms of *anæmia, cachexia, and senility* may produce degenerative processes in the peripheral nervous system which correspond to definite clinical types of polyneuritis. The senile forms developing in old age (Oppenheim)[1] are perhaps in part due to arteriosclerosis. In a number of cases the disease has been ascribed to vascular disease—arteriosclerosis, obliterating arteritis (Oppenheim-Siemerling,[2] Joffroy-Achard,[3] Gombault, Lorenz, Schlesinger, Lapinsky,[4] etc.)

Finally, it has in many cases been attributed to *chill*. It is hardly admissible that this can produce the disease in individuals upon whose nervous system no other agent has been at work. But it is certainly often an exciting *factor*. Thus I have seen it frequently occur in alcoholics immediately after a severe chill.

It should be pointed out that the *combination* of the above factors[5] is pre-eminently calculated to produce multiple neuritis, such as combined alcohol and lead intoxication, the *combined effect of alcoholism and infective diseases* (pneumonia, tuberculosis, etc.). But the causal connection cannot always be clearly recognised, as in my experience persons suffering from multiple neuritis are peculiarly susceptible to other infective diseases. I have thus seen tonsillitis, diphtheria, pneumonia, tuberculosis, influenza, typhoid, etc., occur during the course of multiple neuritis.

[1] *B. k. W.*, 1893.
[2] *A. f. P.*, xviii.
[3] *A. d. Méd. exp.*, 1889 and 1890.
[4] *Z. f. N.*, xiii.
[5] Compare Oppenheim, *B. k. W.*, 1891.

The disease most frequently affects those in middle life, from the age of twenty-five to fifty. If we except the diphtheritic form, the disease is very rare in childhood, but I have occasionally observed it in children of from four to six. Perrin [1] and Thomas-Greenbaum [2] have recently studied the polyneuritis of childhood. Old age is less susceptible ; it is almost exclusively the cachectic and senile forms which occur at this time of life.

Heredity and the *neuropathic diathesis* do not play an important part in this disease. There is of course a family form of multiple neuritis or an allied affection, " the interstitial and progressive hypertrophic neuritis of childhood " (Dejerine, Sottas, Rossolimo), which, however, occupies a special position (see p. 255).

Symptomatology.—It will be well to choose a definite form of multiple neuritis, indeed the most common of all—*alcoholic neuritis*—as a type, and to add to its description the special characteristics of the others.

Alcoholic neuritis or alcoholic paralysis has, as a rule, an *acute* or *subacute* development. It may accompany or follow delirium tremens. The alcoholism may form merely the basis, some exciting cause—a chill or febrile illness—bringing on the disease. The temperature is sometimes high at first, and fever may also occur during the later stages, but an afebrile course is not uncommon. As a rule the patient at first suffers from *paræsthesiæ* and *pain*. He complains of a feeling of tingling and numbness in the feet and the finger-tips, of dull or irritating—rarely lancinating—pain in the extremities, especially in the legs. This pain is often of slight intensity, but in many cases it is very severe. It is aggravated by movement, by pressure on the nerves and muscles, and sometimes even by contact with the skin. Then *weakness* very soon appears, either mainly in the legs or entirely limited to them. The weakness rapidly increases—within a few days or weeks, less often in the course of months—so that the patient can either not walk at all or only with difficulty. Let us make the *examination* at this stage.

The patient may be quite conscious, or he may be *delirious*. In addition to the symptoms of chronic alcoholism, which include the gastric troubles and the tremor, we find the following :

In the legs there is usually a certain degree of wasting, but this may be absent at first or may be masked by œdema, and later by excessive development of fat. The feet usually occupy the *pes equinus* position. The muscles are very *flaccid* and soft. Pressure upon them is *painful* ; the calf muscles are often peculiarly sensitive to pressure, as are also the nerves, especially the peroneal, posterior tibial, and crural nerves. The nerves are rarely felt to be swollen. Passive movements are quite free, but they may be so painful that they cannot be fully carried out.

The *tendon reflexes* are absent or can be only slightly or indefinitely obtained by Jendrássik's method. Their exaggeration, which is reported by Strümpell, Möbius,[3] Werner, Brissaud,[4] Buck, and others, is found only in the rarest cases and in the slightest forms, or at the commencement of the disease, and perhaps also as the result of special complications. The paralysis is usually incomplete, not affecting all the muscles of the extremities, but being limited to those supplied by individual nerves. The nerves rarely escape and they are generally the

[1] *Arch de méd. des enf.*, 1902.
[2] *Jour.. Amer. Med. Assoc.*, 1907.
[3] *M. m. W.*, 1886.
[4] *Journ. de Neurol.*, 1902.

first to be affected (Fig. 229); the posterior tibial nerves are frequently involved, the anterior crural, etc., sometimes. The paralysis either affects all the muscles supplied by a nerve equally or it may spare some of them, *e.g.* the tibialis anticus in paralysis of the *peroneal* nerves. It is quite exceptional for the proximal muscles of the extremities to be exclusively or most severely affected. In such cases this condition is found oftener in the lower than in the upper limbs. In severe cases and at the height of the illness, a more or less complete *paraplegia* may be caused by involvement of all the nerves. Even then the *peripheral* character of the paralysis is almost always evident from its preponderance in the muscles supplied by certain nerves (*e.g.* the peroneal).

Another and most remarkable peculiarity of this paralysis is its *degenerative* nature. Muscular degeneration is always present, and if it is not always apparent to the eye, changes of the electrical excitability are hardly ever absent. The *reaction of degeneration* is complete or partial, more frequently the latter; there may be loss of faradic reaction, or in some muscles simple quantitative diminution of the excitability. The reaction of degeneration may even be perceptible in nerves that are not paralysed.

Fig. 229.—Bilateral peroneal paralysis in alcoholic neuritis. (Oppenheim.)

Popow (*N. C.*, 1901) describes an early onset of these symptoms of degeneration, even before the appearance of subjective disorders.

The upper extremities are either not involved at all or are paralysed to a much less extent. It is unusual for the disease to commence in the upper extremities; but its extension to them may be of great diagnostic value in cases in which there is a diffuse paresis (or even paraplegia) in the lower extremities. There are great differences in the extent of involvement of the various neuromuscular areas. Thus the neuritis may be confined to one extremity or to single nerves in it; it may involve single nerves of different limbs—the same nerves or different ones—or, as in the majority of cases, it may extend over a great part of the peripheral nervous system.

In the arms, the *musculo-spiral* nerve is the one that is chiefly affected, but other nerves may be involved or indeed be the ones most severely attacked. Here we meet the astonishing fact that some of the muscles under the control of one nerve may be paralysed, whilst the others retain their power of movement. The supinators, and often also the abductor longus pollicis, may be intact, whilst the other muscles supplied by the musculo-spiral are completely paralysed. The extensor communis digitorum may at first be the only one affected. In the legs and arms it is the *distal parts, i.e. the muscles which move the feet and hands*, which are the

first, or may be the only ones that are paralysed. This degenerative paralysis, which almost always has a symmetrical distribution, produces such a striking clinical picture that a probable diagnosis can often be made at the first glance.

If the patient can still walk, his gait usually exhibits the characteristics due to bilateral peroneal paralysis. But as there is often also weakness in muscles supplied by other nerves, the gait may be difficult and unsteady.

The motor weakness is sometimes associated with *ataxia*, which in rare cases is very pronounced. Voluntary movements and the gait are then correspondingly modified. An attempt has been made to distinguish an *ataxic* form of multiple neuritis (the *peripheral neuro-tabes* of Dejerine [1]) from the motor form. But it is rare for the disturbances of co-ordination not to be accompanied by symptoms of paralysis.

It is not usual for the ataxia to affect the arms, but in one case I have seen this so pronounced that the patient, when trying to touch her nose with her eyes shut, did not come within a foot of it. When *ataxia* is present, there may be *spontaneous movements*, small or even marked jerks of which the patient is usually unconscious.

Sensory disorders are as a rule less pronounced than the motor weakness, but they are rarely entirely absent. They develop even more markedly than the paralysis in the periphery of the extremities. A diminution of sensibility for all kinds of stimuli is not uncommon in the extremities, but there may also occur a very peculiar and almost pathognomonic *combination of anæsthesia and hyperæsthesia*, especially an anæsthesia for touch with hyperalgesia for painful stimuli. The sense of touch and of position may be considerably diminished, and in addition there may be hyperalgesia for the prick of a needle. *Vice versâ*, there may be diminished sensibility to pain along with exaggerated sensibility to touch. This hyperæsthesia is found chiefly on the *sole of the foot*, and it may materially affect the power of walking. Diminution of the sense of position in the toes is sometimes the only objective sensory disturbance. We should also remember that there may be diminished sensibility at one part of the skin (*e.g.* on the dorsum of the foot), whilst on another (the sole of the foot) there may be hyperæsthesia. Slowness in the conduction of sensations, after-sensations, etc., is repeatedly found. Like the motor symptoms, the anæsthesia is generally less marked on the hands; slight diminution of sensation at the finger-tips may be the only sign of involvement of the nerves of the upper extremities.

The *cutaneous reflexes* are usually diminished or absent; if there is hyperæsthesia they may be elicited to an exaggerated degree in the non-paralysed muscles.

In a few cases the anæsthesia and ataxia may be the most prominent symptoms, the paralysis being less in evidence; we then have a condition which corresponds to Dejerine's peripheral neuro-tabes. The degenerative paralysis can usually be detected, at least in circumscribed areas. In a case of this kind I found, on close examination, weakness of the extensor longus hallucis with reaction of degeneration, although at first it had seemed as if there were only sensory disturbances and absence of reflexes.

[1] *Compt. rend.*, T. xcvii., and *Arch. de Phys.*, 1887; *Arch. de méd. expériment.*, 1889; *Semaine méd.*, 1893, etc.

We must always remember that the so-called " acute ataxia " (see p. 321) may have its origin in multiple neuritis.

Vasomotor, secretory, and trophic symptoms are occasionally present. *Hyperidrosis* on the feet and hands, is most frequent. *Œdema* is not uncommon. It develops at the distal parts of the limbs, or at times over the affected nerves and muscles. In a few cases there was, in the course of or at the onset of the disease, *swelling of the joints*, due to extravasation of fluid. The condition may resemble articular rheumatism. The skin on the feet is often *very red* and feels abnormally warm. Glossy skin is occasionally found.

Cutaneous eruptions, formation of tumours, etc., are less common. Lépine describes the eruption of vesicles with hæmorrhagic contents, and Neisser cutaneous hæmorrhages (*M. m. W.*, 1905). There was symmetrical gangrene in a case reported by Lépine and Porot (*Lyon méd.*, 1905).

One of my patients developed, in addition to the polyneuritis, an extensive *cutaneous lupus*, which persisted after the polyneuritis was cured. Fraenkel (*D. m. W.*, 1896) saw multiple granulomata, of unexplained nature, occur during the illness; I had later an opportunity of examining this case, when it reminded me of the picture of dermatomyositis (*q. v.*) or neuromyositis, but it was very peculiar even from that point of view. Only in one particularly severe case have I seen the articular affection lead to considerable new formation of bone and to ankylosis of some joints. Muscular hypertrophy, mentioned by H. Curschmann (*M. m. W.*, 1905), occurs only in rare cases.

The *functions of the bladder and intestine* are usually unaffected. This forms an important distinction between this disease and the affections of the spinal cord with kindred symptoms. There are, however, exceptions to the rule, and weakness of the bladder or precipitate micturition may be present. One of my patients who suffered from a severe typical polyneuritis had to use the catheter during the first five or six days; the weakness of the bladder then disappeared, whilst the polyneuritis persisted for a few months, and then completely passed off. Incontinence of urine and fæces occurring during the delirium or in the condition of mental confusion and stupor which not infrequently accompany the illness, being due to this mental condition, are of no pathognomonic value. When, however, this symptom persists although the patient is quite conscious, as in the case just quoted, it suggests a complication, an involvement of the spinal cord, which, however, is not necessarily of grave significance as regards the prognosis of the case as a whole. This also applies to impotence. Amenorrhœa may develop (Buzzard). Girdle sensation is not one of the symptoms of multiple neuritis, though it is mentioned in rare cases.

The functions of the *brain* and cranial nerves are frequently affected. The *mental* disturbances which accompany alcoholic neuritis (Korsakow's[1] " polyneuritic psychosis ") consist mainly in a condition of *confusion* and *forgetfulness*, the chronological order of events disappearing entirely from memory and long-past experiences being transferred to the present. This is particularly evident as regards incidents in the immediate past, showing an almost entire loss of the memory. There are also memory-hallucinations and memory-deceptions of the strangest kind, as well as illusions and hallucinations. For example, a patient who has been confined for weeks to bed will imagine he has been driving out the day before, meeting acquaintances at a certain place, and seeing relatives long since dead. He speaks at random, and entirely fails to recognise the situation. These

[1] *Wjest. psich.*, ii., ref. *N. C.*, 1887.

delusions, however, are not permanent ; they are easily suppressed and may reappear again for a time. This psychosis has been brought into analogy with Meynert's amentia. Charcot opposed the view of a polyneuritic psychosis, but mental disturbances of the kind described are found also in polyneuritis after typhoid, influenza, and in the puerperium (Blocq-Marinesco, Köhler, Collatz, Redlich, Tiling, Soukhanoff). I have observed them in a case for which I could discover no other cause than a continued use of pyramidon. Jolly's [1] suggestion that this psychosis should be called by Korsakow's name has found general acceptance. Gudden states that it is not connected with polyneuritis, but may occur independently of it, and Mönkemöller [2] shows that the symptoms of neuritis may be very insignificant.

Of the cranial nerves, the *oculo-motor* are comparatively often involved. Paralysis of the abducens, of the oculo-motor or of some of its branches, occasionally occurs, but there is hardly ever reflex immobility of the pupils. It must be admitted, however, that chronic alcoholism may also produce this symptom, as the cases of Raimann, Mönkemöller-Bonhöffer, and Raecke-Meyer show. Nystagmus is present in not a few cases, as I was the first to note. The optic nerve is rarely involved, but both *neuritis* and *partial atrophy* (especially pallor of the temporal half of the disc) are occasionally found. Uhthoff [3] has seen many cases of this kind and has studied their pathology. The visual disturbance is generally of the nature of a *central scotoma* (especially for colours). This is found not only in alcoholism, but it has been noted in a case of multiple neuritis of *carcinomatous* origin.

Facial diplegia occasionally appears, as in several of my own cases.

The affection of the *vagus and phrenic nerves*, which occurs in not a few cases, is of special importance. Involvement of the vagus is indicated by acceleration of the pulse (in rare cases by slowing) and by respiratory troubles ; affection of the phrenic by paresis or paralysis of the diaphragm. The nerves are usually also tender to pressure and the phrenic may show loss of electrical excitability. The power of masticating and swallowing is rarely impaired in polyneuritis, but bilateral paralysis of the masseters is described in one case by Gaspero [4] and in another by Auerbach. In a few of my own cases there were very marked bulbar symptoms. I have also occasionally, though not often, observed paralysis of the vocal cords. In a few cases I have also noted a systolic murmur and a dilatation of the heart at the height of the disease. Strümpell mentions involvement of the auditory nerve.

Polyneuritis limited to the cranial nerves—multiple paralysis of the cranial nerves of a neuritic nature—has been occasionally observed (Hösslin, Mannaberg, Hammerschlag, Rad, Rudinger [5]), but the symptoms are usually due to a basal process which has involved the cranial nerves.

With regard to the *course* and *prognosis*, we should note the following facts : Alcoholic neuritis has almost always an acute or subacute course. The disease reaches its height in a few weeks or months. It then remains stationary for a period as long or even longer, and then as a rule gradually

[1] *Charité-Annalen*, xxii.

[2] *Z. f. P.*, Bd. liv. and lvi. See also Brodmann, *Journ. f. Psych.*, i. and iii. ; Bonhöffer, " Die akuten Geisteskrankheiten der Gewohnheitstrinker," etc., and *M. f. P.*, xv. ; Meyer-Raecke, *A. f. P.*, Bd. xxxvii. ; Bödeker, *A. f. P.*, Bd. xl. ; Knapp, " Die polyneuritischen Psychosen," Wiesbaden, 1906, etc.

[3] *A. f. Ophth.*, 1886, Bd. xxxii. and xxxiii. [4] *M. f. P.*, xiv. [5] *Jahrb. f. Psych.*, xxii.

disappears, the nerve last affected being the first to recover. There are cases with a very *violent course and high fever*, in which the prognosis is almost always unfavourable. They may end fatally in eight to fourteen days. The course may then correspond to that of *Landry's paralysis* (*q. v.*). Eichhorst speaks of a neuritis acutissima progressiva. An almost *apoplectiform* onset has also been observed in exceptional cases (Dubois, Dejerine, Westphal [1]). If the development is less rapid, the prognosis will be practically decided by the general condition, the intensity and distribution of the paralysis, and the involvement of the cranial nerves. If there is marked *marasmus* or a condition of exhaustion due to an infective illness, life is always in danger. The symptoms of involvement of the vagus and phrenic nerves make the prognosis very much graver, but even in such cases a favourable termination is possible, as I [2] have found in several instances. If the legs only are affected, there is more prospect of recovery than when the paralysis extends to the arms and to the muscles of the trunk. The outlook is more favourable when the paralysis is limited to the distal segments of the limbs than when it implicates all the muscles of the extremities.

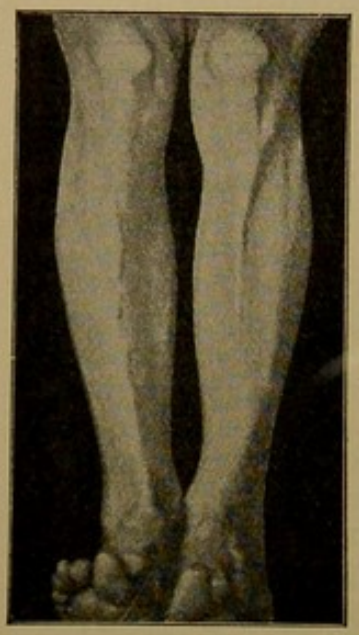

Fig. 230. — Paralysis and atrophy of the left tibialis anticus muscle, all that remains of an alcoholic polyneuritis, otherwise practically cured. (Oppenheim.)

The disease has a chronic course only in exceptional cases, and is then either chronically progressive or undergoes successive exacerbations. There is also a relapsing form of polyneuritis. Such cases have been described by Oppenheim,[3] Sherwood, Eichhorst, Sorgo, Schlier, Thomas, and others. In some of these the disease returned every year about the same time.

In the majority of cases the course is favourable, a more or less complete recovery being the result. But convalescence may be very slow. The symptoms of irritation, the pain and tenderness to pressure, usually disappear first, but hyperæsthesia may persist for a long time and may be a very distressing symptom during convalescence when the patient attempts to walk. I have seen patients in whom the symptoms of paralysis had entirely disappeared, but who for months found the greatest trouble in trying to walk on account of the hyperæsthesia of the soles of his feet. A period of one or several years may elapse before the paralytic symptoms entirely disappear. But I have also seen severe cases in which they had completely passed off within a few weeks. It is not uncommon for some of the muscles to remain permanently affected, and this residual paralysis has been specially noted in the peroneal region (Oppenheim, etc.). In one of my cases the only motor disturbance left was paralysis of the left tibialis anticus muscle (Fig. 230). Contractures may develop in the antagonists of the paralysed muscles, especially those of the calf, which may persist after the paralysis has gone and prove very intractable. In one of my patients a process of ossification took place in the tendons and the joints, and gave rise to ankylosis and restriction of movement very

[1] *A. f. P.*, Bd. xl. [2] *B. k. W.*, 1890. [3] *A. f. kl. M.*, xxxvi.; *Z. f. kl. M.*, xi.

difficult to treat. It should also be remembered that the mental weakness may, although it seldom does, persist after the paralysis has disappeared. Unfortunately there are often *relapses*, due not only to renewed action of the same agent, but also to other injuries (cold, overstrain, a fall).

Lead Paralysis [1]

This is characterised as a peculiar form of multiple neuritis by the fact that it is usually confined to the muscles supplied by a certain nerve and hardly ever involves the sensory fibres. The cause of this paralysis is chronic lead-poisoning, which is common in those employed in lead-mines, in compositors, varnishers, painters, file-cutters, pipe-layers, tinsmiths, accumulator-workers, etc. Potters who make glazes containing lead, and weavers who work with lead-weights, are also exposed to the intoxication. The poisoning is less often produced by drinking water from lead pipes, by the use of cosmetics containing lead (rouge), of snuff containing lead (especially Russian), of playthings made with lead (trumpets), by washing out utensils with lead shot, or by bullet wounds. I have seen it in persons who soldered up tins in a cannery. Recently it has been found (Raudnitz, Hahn) that it may also be produced in children by the use of lead plasters, Hebra's ointment, etc.

Fig. 231.—Paralysis of the extensors of hands and feet (probably hereditary lead paralysis). (Oppenheim.)

As a rule the onset of the paralysis is preceded by other symptoms of lead poisoning, especially *lead-colic*, sometimes pain in the joints and muscles (*arthralgia*), or symptoms of brain disease due to the lead-poisoning (*encephalopathia saturnina*).

In such cases we frequently find a sign of the lead-poisoning in the form of the so-called *blue line*, the margin of the gums close to the teeth being of a blackish-blue colour. The appearance of basophile granular erythrocytes in the blood may be a diagnostic sign (Grawitz,[2] Frey,[3] etc.). *Anæmia* is usually present and sometimes *cachexia*, arterio-sclerosis, and nephritis. But the lead paralysis may be the first and only sign of the poisoning.

It is a fact to be noted that lead intoxication may produce morbid conditions even in the descendants. The children of lead workers are specially subject to *epilepsy* (Berger). Other neuroses and organic diseases of the nervous system have also been observed (Legrand, Roques, Seeligmüller). I had occasion to see a case of lead paralysis (Fig. 231) which could only have been acquired through heredity (published by Anker, *B. k. W.*, 1894). It affected the *radial* and peroneal nerves in the typical way. The lower extremities seem to be particularly often involved in the lead paralysis of childhood, as Putnam, Newmark, Bernhardt (*N. C.*, 1905), and Labastide (*Thèse de Paris*, 1902) have remarked.

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[1] Literature in Remak, *loc. cit.*

[2] "Klin. Path. d. Blut.," iii. Aufl., 1906.

[3] *D. m. W.*, 1907.

Both upper extremities are usually affected at the same time, although one (generally the one most used) may be attacked before the other). The paralysis not unusually shows itself mainly in the right arm. It develops in the course of a few weeks, or less often it has an acute onset directly after an attack of colic. The *extensors* of the *wrist* are mostly, and in many cases exclusively, affected. If the paralysis is very slight or recent, it may be limited to the *extensor communis digitorum* and even to some branches of this muscle, so that, *e.g.*, only the third and fourth fingers can be extended. In completely developed cases, however, the extensores carpi are also paralysed (the extensor carpi ulnaris being sometimes spared), as well as the extensor and especially the long abductor of the thumb.

The hands and fingers are flexed, and when passively moved from this position they immediately return to it. This position is somewhat characteristic (Fig. 232). Owing to it, the power of flexion of the

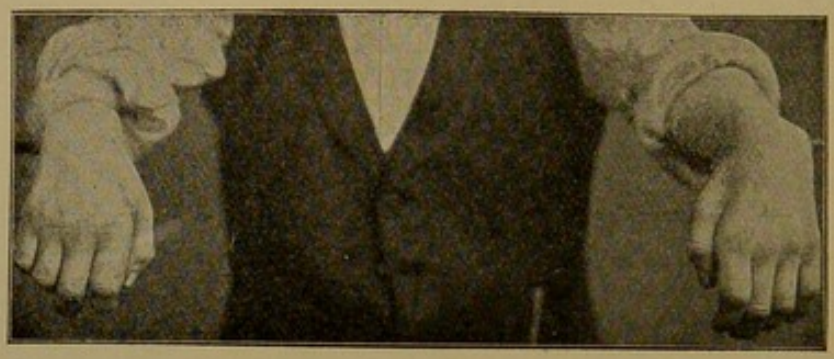

Fig. 232.—Position of hands (wrist-drop) in case of lead-paralysis. The interossei were involved in addition to the extensors of the hands and fingers. (Oppenheim.)

fingers or the grasp of the hand is greatly diminished, but it becomes normal if the hand is passively lifted into a position of extension.

We may regard it as the rule that in typical cases of lead paralysis the *supinators*, especially the *supinator longus*, are spared, as well as the *triceps*. It not infrequently happens, however, that the paralysis is not limited to the musculo-spiral nerve, but extends to the small muscles of the hand supplied by the median and ulnar nerves, most frequently to the muscles of the thenar eminence, paralysis of which tends to precede that of the abductor pollicis longus, and occasionally also to the interossei, where, however, it is seldom complete. The deltoid muscle is not infrequently affected along with the extensors.

There are also *atypical* cases of lead paralysis, involving the supinators, and almost always simultaneously the arm muscles, viz., the biceps, brachialis anticus, and deltoid (Remak's upper-arm type). The paralysis may begin in the small muscles of the hand, or may remain limited to them (Dejerine-Klumpke,[1] Bernhardt, Merklen-Guiard[2]).

Localisation may be influenced by the nature of the patient's occupation, by overstrain of certain groups of muscles (toxic-professional paralysis).

Lead paralysis is always *degenerative*. The atrophy of the muscular tissue may be recognised from the flattening of the muscles. It is always revealed by the reaction of degeneration, which is sometimes present even

[1] "Des polynévrites," etc., Paris, 1889.

[2] *Arch. de Neurol.*, xvii.

in muscles which are not involved in the paralysis. These changes of electrical excitability become markedly evident some eight days after the onset of the paralysis. Fibrillary tremor is usually, and saturnine tremor often present.

There is sometimes *swelling* over the joints of the hand, due to thickening of the tendon sheaths of the long extensors of the fingers. Protrusion of the metacarpal bones is less common.

Sensibility is not affected, and paræsthesia and pain is usually absent.

In atypical cases, in which the paralysis develops in nerves which have been overstrained by work, I have found sensory disturbances also, *e.g.* in the circumflex nerve in a man who worked with lead and also carried sacks, in the ulnar region in a solderer ; but such cases, which are beyond the range of typical lead paralysis, are rare.

The lower extremities are *very seldom* affected. The extensor cruris and ileopsoas may be transiently paralysed, but typical lead paralysis in the legs is that involving the *peroneal* nerve, the tibialus anticus muscle being intact (E. Remak). G. Koester noted a localisation in the region of the small muscles of the foot. Finally, there is a *generalised lead paralysis* in the muscles of the arms, legs, and trunk, with an acute feverish onset following encephalopathia saturnina.

The *diagnosis* can easily be made in typical cases, as a similar distribution of the paralytic symptoms is exceptional in neuritis of other origin (alcoholic paralysis), and is very rare in poliomyelitis. In atypical cases the diagnosis will be confirmed by the anamnesis and by the detectable signs of lead-poisoning (blue line, lead tremor, etc.).

It should be noted that lead-poisoning may also produce symptoms of paralysis in the muscles supplied by the cranial nerves, such as the *laryngeal* (paralysis of one vocal cord, paresis of the adductors and the abductors). Prolonged acceleration of the pulse and respiratory disorders may show that the vagus is involved. Debove mentions paralysis of the facial muscles and nystagmus, but this is exceedingly rare. The *optic* nerves may be independently affected (amblyopia without any changes, optic neuritis or atrophy) or may become involved in the course of an *encephalopathia saturnina*.

This term has been applied to the most varied kinds and groups of *brain symptoms* caused by lead intoxication. It includes true *apoplexy* (hemiplegia and aphasia) due to hæmorrhages, less frequently to softenings, also *transient focal symptoms*, the pathological cause of which is not precisely known, *e.g.* amaurosis, hemianopsia, etc., and more especially *delirium, coma, and convulsions* of the type of general or more rarely of localised epilepsy. Whilst these disturbances have mostly an acute onset and either disappear rapidly or lead to a fatal termination (death occurs usually in coma or during the convulsions), there are others which have a chronic development, or follow a chronic course after an acute onset. These include *hysteriform* symptoms ; especially *hemianæsthesia* with corresponding sensory disturbances, *spasms* of the hysterical type, hemiparesis resembling functional paralysis, etc. *Epilepsy* and *psychoses*—including a curable form resembling paralytic dementia and perhaps also a true form of this mental disorder—must in some cases be regarded as products of chronic lead-poisoning. The lead may also have a *direct* action upon the brain and thus cause general cerebral and focal symptoms, and particularly those peculiar neuroses which are allied to hysteria. At the same time individual cranial nerves, such as the optic, may also show pathological lesions. Lead may also injure the cerebral vessels and produce an *arteritis*, which causes *hæmorrhages* into and *softening of the brain* ; finally, it may give rise to *nephritis* and thus produce uræmic brain symptoms. All these factors may be operative at the same time. A. Westphal [1] has discussed this in detail in the thesis which he has written under my direction.

The prognosis as to life is favourable, should no serious complications

[1] *A. f. Psych.*, xix. Also : Meillère, " Le Saturnisme," etc., *Thèse de Paris*, 1903 ; Hübner, *Inaug. Diss.*, Berlin, 1904.

(lead cachexia, nephritis, etc.) be present; it is also good as to recovery in the first stages, if the paralysis is not very extensive and not of long standing. Even the presence of reaction of degeneration does not justify a grave view of the case, as recovery is the rule when the conditions admit of proper care and the absolute removal of the injurious poison. But if the paralysis has occurred repeatedly, the prognosis is essentially more grave. Relapses may be caused not only by a fresh intoxication, but even by other influences, such as overstrain of the muscles.

Arsenical Paralysis

This is less frequent than the other toxic forms of polyneuritis already described, but yet sufficiently so to call for the reminders which of late have been repeatedly expressed, of the necessity for careful dosage in prescribing arsenic.

This paralysis usually follows *acute* poisoning. In many cases the arsenic or Paris green has been taken with suicidal intent. But arsenical paralysis may also develop in the course of or after subacute and chronic poisoning, as in cases where the drug is prescribed for chorea and other affections (Brouardel, Barrs, Raymond, Colman, Karplus, Kron, etc.).

The morbid picture may, from cases observed by Erlicki-Rybalkin,[1] Henschen,[2] Jolly,[3] Facklam,[4] Raymond,[5] etc., and a number of my own, be outlined as follows :—

The *gastro-intestinal* symptoms, which are rarely absent, as in a case of Kron's, are followed within a few days or weeks by symptoms of the nervous disease. These usually commence with *pain and paræsthesia* in the *feet* and *hands*, or in the legs and arms. In the subsequent course of the disease these form also an essential part of the condition. They are rapidly joined by the *atrophic paralysis*, which also affects chiefly or entirely the *distal* segments of the extremities. The extensors (peronei, musculo-spiral) are usually most affected, but the paralysis attacks the flexors and the small muscles of the hand[6] much more often than in alcohol- or lead-poisoning. It generally has the character of tetraplegia ("paralysie chiropodale"), its distribution being as a rule equal, the paralysis not affecting one extremity more than another. Electrical examination shows *reaction of degeneration* and great diminution of excitability.

The muscles and nerves are tender to pressure. Cutaneous sensation is almost constantly diminished, and *anæsthesia* or *hypæsthesia* is repeatedly found, especially on the feet and legs and the hands and fingers. The pulse is sometimes rapid. Rise of temperature is seldom noted. *Mental* symptoms—failure of memory and confusion—may appear in the course of the illness. Epilepsy and amaurosis are much less frequent.

Ataxia is common; it may be the most prominent symptom, but is usually associated with paralysis. A distinction has been drawn between a motor and an ataxic form of arsenical polyneuritis. As the knee jerks are almost always absent, the cases characterised by ataxia may show a remarkable resemblance to tabes, as Dana[7] specially points out (arsenical pseudotabes). Bladder troubles may also occur (Kron).

[1] *A. f. P.*, xxiii.
[2] *N. C.*, 1894, and *Upsala läk*, xxix.
[3] *Charité-Annalen*, xviii. and xxii.
[4] *A. f. P.*, xxxi.
[5] *Nouv. Icon.*, ix.
[6] I do not, however, go so far as Raymond (*R. n.*, 1906), who regards this and the pruritus as pathognomonic.
[7] *Br.*, ix.

Trophic disorders of the skin, such as eruptions of herpes, pemphigus, etc., pigmentation suggesting Addison's disease, glossy skin, loss of hair, etc., are comparatively common. Herpes zoster has also occurred in several cases in which arsenic had been used as a medicine. Geyer noted these trophic changes, particularly pigmentation in the supra-clavicular region, hyperkeratosis on the hands and feet, etc., in epidemic poisoning due to the use of drinking water containing arsenic in Reichenstein in Silesia. Similar observations were made in a recent epidemic in England due to beer containing arsenic (Reynolds,[1] etc.). The bulbar nerves are hardly ever involved (Müller [2]).

The *prognosis* is on the whole favourable. The symptoms of paralysis usually gradually disappear, the muscles last affected being the first to regain their mobility, but the convalescence may last for months or years.

FIG. 233.
Paralytic contracture in arsenical paralysis.

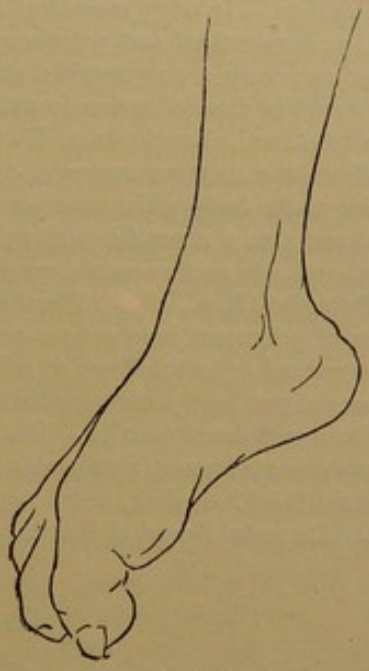

FIG. 234.
(After Erlicki and Rybalkin.)

In a few cases secondary contractures have developed in the antagonists of the paralysed muscles (Figs. 233 and 234).

Life is not usually endangered ; in one case pneumonia supervened, with fatal results. Death occasionally followed paralysis of the heart.

We are less well informed with regard to the polyneuritis caused by other poisons. This is true especially of *mercurial* poisoning. Only a small number of clinical observations correspond to the experimental results of Letuile [3] and Heller,[4] which moreover Brauer has been led by his own investigations to dispute. In the majority of cases in this group (Forestier,[5] Leyden,[6] Engel, Gilbert [7]) there had been previous syphilis for which mercury had been prescribed ; but this was not so in the cases reported by Kétli, Spillmann Etienne,[8] Faworski,[9] and Spitzer. The existence of a

[1] *Brit. Med. Journ.*, 1900, and *R. of N.*, 1905.
[2] *Wien. med. Presse*, 1894.
[3] *Arch. de Physiol.*, 1893.
[4] *D. m. W.*, 1894.
[5] *La Méd. mod.*, 1890.
[6] *D. m. W.*, 1893.
[7] *D. m. W.*, 1894.
[8] *Revue de méd.*, 1895.
[9] *N. C.*, 1890.

syphilitic polyneuritis (not of specific, but of syphilotoxic origin) cannot be doubted. Cases of this kind have been described by Schultze, Buzzard,[1] and Oppenheim.[2] See also the chapter on Landry's paralysis. Cestan[3] has lately published indisputable cases, in some of which the resemblance to lead paralysis was striking, except that the supinators were also involved. The disease develops very rapidly, within a few weeks after the appearance of the primary affection. On the whole it is a very rare affection. It is worthy of note that polyneuritis in syphilitics may be aggravated by the use of mercury, as Minkowski, Engel, and I have observed.

Symptoms of neuritis and polyneuritis may also occur in *poisoning with bisulphide of carbon* (Delpeche, Mendel, Laudenheimer, G. Köster,[4] Guillain-Courtellemont,[5] Merlin,[6] etc.). The picture may be dominated by mental disorders and other symptoms due to injury of the central nervous system. In a few cases the neuritis was ascribed by Dufour, Soupault, and Français to benzine, or benzotoluol and petroleum-ether poisoning. It seems to me still doubtful whether sulphonal can cause a polyneuritis, as Erbslöh (*Z. f. N.*, xxiii.), thinks.

The neuritis due to carbonic oxide poisoning (Bourdon, Leudet, Brissaud, Rendu, Litten, Lereboullet-Allard, Meczkowski, W. Sachs, Schwabe, Masssanek, Croizet[7]), is usually localised, *i.e.* it affects one or more nerves of one extremity. In one unusual case it affected the nerves of the side of the body which was paralysed owing to a focal brain disease. There is a central and neuritic, and also a myositic paralysis of this origin (Soelder). Neuritic symptoms following the use of phosphoric acid creosote, prescribed in tuberculosis, were noted by Loewenfeld (*C. f. N.*, 1903), Babinski (*R. n.*, 1905), Wertheim-Salomonson (*N. C.*, 1906), and Chaumier.

Cases of polyneuritis in copper-poisoning are few and uncertain (Suckling, Murray). Lewin is very sceptical as to this form of poisoning.

It has not yet been ascertained whether the paralytic conditions in poisoning by shell-fish, etc., are due to a peripheral neuritis. See Thesen (*Arch. f. exp. Path.*, 1902).

I have repeatedly seen symptoms of polyneuritis (and other conditions of poisoning) in persons who use *artificial hair dyes*, and I regret that, in spite of a suggestion which I made several years ago to the Imperial Board of Health, this question has hitherto received so little consideration.

The Infective Forms

The clinical picture of these forms corresponds in its main features to that described with regard to the toxic forms.

A *rise of temperature*, even to 40° C. (104° F.), is frequently noted at the commencement of the illness, and may even occur during the later course. Delirium, enlargement of the spleen, and albuminuria are occasionally observed, and disturbances of the general condition, such as loss of appetite, headache, etc., are almost constant. Severe gastric symptoms, *e.g.* prolonged vomiting, diarrhœa, jaundice, were present only in exceptional cases. Profuse sweating is a still less common symptom.

With regard to the paralysis, there is little to add to the facts already stated, but we shall refer to some details in the forms now to be discussed.

Diphtheritic Paralysis

This is the most common form of polyneuritis due to an infective cause.[8] According to Woodhead's London statistics, 1362 cases out of 7832 of diphtheritis were associated with paralysis.

[1] *Lancet*, 1885. [2] *B. k. W.*, 1890. [3] *Nouv. Icon.*, xiii. [4] *Z. f. N.*, xxvi.
[5] *R. n.*, 1904. [6] *Thèse de Paris*, 1905. [7] *Thèse de Paris*, 1903.
[8] Trousseau, Maingault, Tardieu, Donders, Legrande du Saulle, Wilks, Williamson, West, Leube, Grainger-Stewart, Ziemssen, Schirmer, Bernhardt, and others have contributed to the

We may distinguish between a *localised* and a *generalised* form of this paralysis, but the boundary is not sharp and the two forms blend into each other. Slight as well as severe cases of diphtheria may result in paralytic conditions which usually appear two to three weeks after the illness, less often during it. The paralysis is naturally more common in children, but adults are not less susceptible. The most ordinary form of diphtheritic paralysis does not correspond to the type of a polyneuritis, but is limited to the small groups of muscles which move the *soft palate*. During or immediately after the illness, nasal speech and difficulty in swallowing become apparent. Fluids are returned through the nose and solid food is swallowed with some difficulty. Direct examination shows that the soft palate hangs loosely down and cannot contract during phonation. There is usually anæsthesia of the palatine muscles and loss of the palatine reflex. The paralysis of the muscles of the palate is generally atrophic, but it may be difficult to demonstrate the reaction of degeneration. It is unusual for the palatal paralysis to be limited to one side, as Aubertin-Babonneix,[1] have described. As a rule, the paralysis disappears spontaneously within a few weeks.

In a considerable number of cases the paralysis extends, first to the *ocular muscles*, less frequently to the muscles of the *pharynx* and *larynx*, or equally to all these areas. Among the ocular muscles, the ciliary is earliest and most often affected. *Paralysis of the accommodation* is revealed by the sudden onset of difficulty in near vision. The pupil reflex is almost always conserved, and the accommodative reaction may be intact, although accommodation is absent. The *abducens* may be involved on one or both sides ; the oculo-motor or even the whole of the external ocular muscles are less frequently involved. Trochlear paralysis has once been observed (Krauss).

Paralysis of the pharyngeal muscles causes *trouble in swallowing* or even complete paralysis of deglutition. In these cases the muscles which move the epiglottis are also sometimes involved in the paralysis. Along with this there may be *anæsthesia* and *absence of the reflexes* in the mucous membrane of the larynx and pharynx ; the epiglottis is not depressed, and there is great danger of particles of food getting into the air passages. The paralysis less often extends to the area of the *recurrent laryngeal*, causing hoarseness and aphonia. The *affection of the nerves of the heart*, which occurs in some cases, is revealed by slowing and later by accleration and irregularity of the pulse. Aubertin found as few as seventeen pulsations per minute. The respiratory muscles also are sometimes paralysed.

Even in these localised forms of diphtheritic paralysis the tendency of the disease to spread to distant nerves is sometimes shown by the fact that the *knee jerks are absent* (Rumpf, Schulz, Bernhardt [2]), although the extremities are otherwise quite normal. The Achilles jerk can usually not be elicited (Rolleston).[3] These cases form the transition to the generalised forms, which are characterised by the *appearance of motor and sensory symptoms in the extremities*. These symptoms usually follow the local paralyses just described, or that of the soft palate only. After the paralysis of the palate has disappeared and the other paralytic symptoms

elucidation of this paralytic condition. The collected literature up to 1900 will be found in Remak (*loc. cit.*). Of the later papers we may mention those of Aubertin (*Arch. gén. de Méd.*, 1903), Babonneix (*Thèse de Paris*, 1904), and Raymond (*Arch. gén. de Méd.*, 1905).

[1] *Gaz. des hôp.*, 1902. [2] *V. A.*, Bd. xcix. [3] *Br.*, 1905.

have partially gone, the patient complains of weakness, paræsthesia, and pain in the legs, or in all four extremities. These troubles increase from day to day, and are accompanied by corresponding objective symptoms, *e.g.* disturbances of *movement*, of *sensation*, and of *co-ordination*.

In some cases the clinical picture is dominated by sensory symptoms, especially at the distal parts of the extremities, by ataxia, and uncertainty of gait. The resemblance to tabes is then very striking, but in the great majority of cases the paralysis is the most prominent feature. In addition to a diffuse paresis there is a *degenerative paralysis* with the complete or middle form of the reaction of degeneration, which mainly affects the peripheral parts of the limbs (peronei, etc.). The degenerative nature of the paralysis is, however, not always distinctly marked. Walking is exceedingly difficult or impossible. Westphal's sign is always, and Romberg's symptom frequently present. The anæsthesia or diminution of sensibility is also most marked at the distal segments of the limbs ; the sense of touch, of position, and of locality is as a rule considerably diminished, but hyperalgesia may exist or may come on later. The cutaneous reflexes are sometimes exaggerated.

There are generally no disorders of the functions of the bladder and intestine, though they have occasionally been mentioned, as by Katz [1] and Englisch.

These symptoms of paralysis reach their height within a period of one to three months, while the paralysis of the palate, larynx, eyes, and pharynx have already disappeared. In rarer cases the trunk and intercostal muscles and the diaphragm are involved ; affection of the tongue and facial muscles is also uncommon. I have found facial paralysis with partial reaction of degeneration in one case. Ebstein observed attacks of apnœa lasting for hours, and necessitating artificial respiration, in a case of generalised diphtheritic paralysis. Paralysis of the cervical muscles may be so complete that the head cannot be held upright. In a child whom I treated, the lumbo-pelvic muscles were also affected, causing lordosis, and the child climbed up his limbs in rising from the ground. Œdema was observed in uncommon cases, as by Kraus.

Hansemann [2] has made known to us a particularly severe case by describing the history of his own illness. Here the development of the disease had been preceded in the course of some years by various infective processes, *e.g.* scarlatina, repeated attacks of tonsillitis, septic infection, pleuro-pneumonia, etc.

On the eighteenth day after the onset of the diphtheria, *paralysis of the soft palate* and *tachycardia* supervened. These were followed by *sensory disturbances*, which spread over the mucous membrane of the lips, tongue, and cheeks and over the skin of the face and head. The sense of *smell* and *taste* were diminished, the movements of the *lips* and *tongue*, *swallowing*, *masticating*, the *speech* and the *voice*, were affected and the *ocular muscles* were involevd. The sensory troubles then spread to the legs ; the knee jerks disappeared, and the legs were affected by weakness, *anæsthesia*, and *ataxia*. Eventually there was *complete motor paralysis* and *atrophy* in many muscles of the arms and legs, entire absence of the sense of position, girdle sensation, dyspnœa, etc., as well as spontaneous movements, which the patient did not feel, but saw. Improvement did not set in until three months after the onset of the disease. In the examination which I made at the end of four months, there was still considerable quantitative diminution of the electrical excitability, but no reaction of degeneration. In January of the following year Hansemann was cured, but he still complained of paræsthesia in the ulnar region.

[1] *A. f. Kind.*, xxiii.

[2] *V. A.*, Bd. cxv.

I found in one case that the sensory trigeminus was involved, a fact which I recognised when I saw that the child did not remove a foreign body (hair) from the eye ; I examined and found anæsthesia and absence of reflexes in the conjunctiva and cornea.

The duration of the affection depends essentially upon its severity and extent. Slight, localised paralysis may recover in a few weeks, but the severe, generalised forms may persist for many months, and may even last for a whole year, though that is very rare. Thus in a case reported by Ziemssen complete recovery only took place after eight months, and even a year after the onset of the disease there were considerable disturbances of electrical excitability, in particular faradic reaction of degeneration. We may consider a period of three months as the average duration of the generalised paralysis.

The *prognosis* is entirely favourable as regards paralysis limited to the palatal and ocular muscles. Recovery is always to be expected. But even in severe cases, if life is retained, the prognosis as to recovery is good. Involvement of the vagus constitutes a danger to life. Paralysis of the heart is unfortunately no uncommon occurrence. The deglutition paralysis may also be an ominous symptom, as it frequently gives rise to inanition or still more often to broncho-pneumonia. Paralysis of the respiratory muscles, especially of the phrenic, is also a grave sign (Pasteur). Where these and other complications, such as myocarditis or nephritis, are absent, complete recovery may be expected.[1] Of thirty-two cases of this kind which I observed (up to 1903), and the course of which I was able to follow, I only saw five end fatally. In these the heart was affected, and in one there was a complication with nephritis. The patients were aged from three to six. The adults who suffered from diphtheritic paralysis all recovered : these included fifteen men and four women, the ages varying from fifteen to twenty-eight. In the fatal cases, death may be expected early ; if the first six weeks are well past, the danger is slight.

Some writers, such as Deguy (*Rev. mens. des mal. des enfants*, 1903), Berthelot (*Thèse de Paris*, 1904), and Rolleston (*R. of N.*, 1906) regard early onset of the palatal paralysis within the first few days or weeks as an unfavourable prognostic sign, as it points to the diphtheria being of a malignant form.

Our experience is that post-diphtheritic paralysis has not become less frequent since the introduction of Behring's treatment, but we would point out that many more children now survive the early stages and that this may perhaps account for the paralysis being more frequently observed (Slawyk,[2] Woollacott, Goodall). Wettstein states on the ground of Ransom's experiments and his own comparative investigations, that post-diphtheritic paralysis has become rarer since the introduction of serum treatment, and may be entirely prevented by proper application of the method. Lublinski mentions that, when serum treatment is used,

[1] I have seen diphtheritic paralysis of the soft palate become permanent in an imbecile child. B. Fränkel also mentions a case of persistence. In a few cases of generalised paralysis I found absence of the palatal reflex after the other symptoms had disappeared. According to Mühsam and Helbron, the accommodation paralysis may also remain. It is doubtful whether a case of persistent bulbar paralysis described by Harris (*Brit. Med. Journ.*, 1903) should be included here. A combination of post-diphtheritic hemiplegia and post-diphtheritic paralysis here described, such as I have seen in one case, is very unusual. Here the hemiplegia appeared first, and as the paralysis which supervened extended to the extremities, the knee jerk was conserved on the hemiplegic side, and absent on the other.

[2] *Charité-Annalen*, xxiii.

generalised paralysis more frequently occurs without any preceding localised paralysis (soft palate, etc.). This had been noted even before the era of serum treatment, although extremely seldom. Perrin thought the cause in one such case was septic infection.

On the other hand it is maintained (Comby) that the serum may also have a beneficial effect upon the existing paralysis. On this question see the interesting experiments of Rosenau-Anderson (ref. *R. of N.*, 1907).

As a rule, the physician has to deal with cases of pharyngeal diphtheria, but the paralysis may also develop when the diphtheritic process is localised elsewhere, *e.g.* in the vulva, intestine, or umbilical cord (Gassicourt, Roger). I have also, in a case of a wound in the forearm infected with diphtheria, seen paralysis of accommodation appear before the paralysis became generalised.

In a few cases the post-diphtheritic paralysis appeared without being preceded by any of the early symptoms of the disease (Boisserie,[1] Senator, Escherich, Guthrie). Henoch, however, is doubtful of these cases and suspects that the pharyngeal diphtheria had been overlooked. In some rare instances (Bourges, Merklen-Broc) paralysis of a diphtheritic character followed a simple tonsillitis.

The cases reported by Eisenlohr[2] of an infective form of multiple neuritis, in which there was no question of diphtheria, resemble those in the group just described in so far that the muscles of the palate and of deglutition, which are hardly ever implicated in the non-diphtheritic forms, were paralysed. Paralysis of the soft palate and the laryngeal muscles has also been observed after *erysipelas*. I have seen infective forms of polyneuritis, in which the abdominal muscles were mainly affected.

The disease of beri-beri (also known as kakké) which occurs in the tropics, is, according to the investigations of Scheube, Bälz, Grimm, Rumpf-Luce, Okada, Wright,[3] and others, a form of polyneuritis (or polyneuromyositis), characterised by marked affection of the heart and vascular nerves (œdema). Glogner reports having found malaria plasmodia or similar bodies in the blood of these cases. Däubler and others deny this. Wright attributes it to a special micro-organism. The polyneuritis which occurs in this country occasionally very much resembles this type (Rosenblath). Normann is of opinion that the disease now and then appears even in Europe.

For the sake of completeness we must also refer to *leprous neuritis* and its peculiar symptomatology (see p. 386). Polyneuritis due to malaria has been observed by Singer, Gowers, Eichhorst, Ewald, Baumstark, Sacquépée-Dopter, Luzatto, Boinet, Oppenheim, etc.

The neuritis which appears during and after *typhoid* is usually, according to the observations of Nothnagel, Leyden, Bäumler, etc., limited to a single nerve, such as the ulnar, peroneal, long thoracic, etc., but it may extend to several, *e.g.* to both ulnar nerves (Bernhardt), and may finally correspond entirely to the type of a *polyneuritis* (Gubler, Nothnagel, Raymond, Curschmann, Etienne, Foix, etc.). In one case under my observation the paralysis was much more marked in the abdominal muscles and the ileopsoas than elsewhere, whilst changes of electrical excitability were also present in the nerves of the lower extremities. In cases reported

[1] *Gaz. hebd.*, 1881. [2] *B. k. W.*, 1887. [3] *Br.*, 1903, and rep. *N. C.*, 1905.

by other writers there was generally a paraplegia, often with marked involvement of the peronei. We can here only remark in passing that paralysis of the nerves, differing in character, may result from typhoid (Landry's paralysis, myelitis).[1]

The polyneuritis which not infrequently follows *influenza* (E. Remak, Mills, Eisenlohr, Putnam, Diemer, Bonnet, Oppenheim, etc.) does not require special description. Cestan and Babonneix have devoted a comprehensive description to this form. Polyneuritis after whooping-cough has been described by Möbius, Mackay, Guinon, Simionesco, etc. (exhaustive thesis by Valentin [2]). Edens [3] saw the disease follow measles.

Puerperal Neuritis

has become well known from the investigations of Möbius, Kast, Lunz, Tuillaut, Leroy, D'Etiolles, Remak, Joffroy, Mader, Eulenburg, Reynolds, Saenger, Palowski, Sinkler, etc. We owe to Hösslin [4] an exhaustive study which comprises the literature. We may distinguish between a localised and a generalised form. The former chiefly affects the median and ulnar nerves, but may be confined to the circumflex and supra-clavicular nerves or to individual nerves of the leg. The generalised form spreads over the extremities and cranial nerves and may closely resemble the type of post-diphtheritic paralysis. Optic neuritis also occurs (Schanz, Saenger). It may take the form of Landry's paralysis.

The cause is evidently some infective agent (puerperal infection, septic infection). This polyneuritis may appear during pregnancy, especially in patients suffering from hyperemesis gravidarum. Saenger thinks the cause is an autointoxication related to the physiological process of pregnancy, whilst others, *e.g.* Huber, blame infective material arising from a previous puerperium or an infective disease of the uterus.

Hösslin thinks that a macerated fœtus or the retention of portions of the placenta may play a part.

Septic polyneuritis has hitherto been little discussed, but Kraus has recently collected all that is known about it.

There are severe and slight forms of this disease. Although the prognosis is grave in the former, complete recovery is possible even there.

There can be no doubt, according to the observations of Engel-Reimers, Eisenlohr, Allard-Meige, Lustgarten, Bloch, Muratoff, Lesser, Speranski, Raymond-Cestan (the comprehensive theses of Adeline, Delamare, Evrard,[5] etc.), that neuritis and polyneuritis may develop after gonorrhœa. The disease seems to be localised mainly in the nerves of the lower extremities; it may be associated with vasomotor and trophic symptoms.

It has been shown by the investigations of Pitres-Vaillard,[6] Oppenheim-Siemerling,[7] Collela,[8] etc., that degenerative processes in the peripheral nerves may occur in the course of *tuberculosis*. Whilst these pathological changes do not necessarily give rise to clinical symptoms, it is

[1] See the résumé of Friedländer, which refers specially to the diseases of the central nervous system in typhoid, *M. f. P.*, v., vi., and vii.

[2] Paris, 1901. [3] *B. k. W.*, 1904. [4] *A. f. P.*, Bd. xl.

[5] *Thèse de Paris*, 1904. [6] *Rev. de Méd.*, 1896. [7] *A. f. P.*, xviii.

[8] "Ann. di clin. di mal. ment.," etc., Palermo, 1903.

evident, on the other hand, that symptoms of a localised and generalised affection of the peripheral nervous system may be produced by the neuritis of tuberculosis. In the majority of published cases (Joffroy, Strümpell,[1] Eisenlohr, Rosenheim,[2] Anglada, Frenel, Steinert,[3] etc.) the morbid picture corresponded to that of a "symmetrical amyotrophic polyneuritis," but was not characterised by any special symptoms attributable to the primary tubercular cause. Moreover, it is pointed out with justice by E. Remak, that in most of the published cases the cause was a combination of alcoholism and tuberculosis. This was the case also in the majority of my own cases.

Among the forms of polyneuritis due to *autointoxication*, the *diabetic* is the most important. It is characterised by *atrophic* paralysis commencing with violent neuralgic pain, and it has a remitting course, usually ending in recovery. It affects chiefly the regions supplied by the *crural*, *obturator*, and *peroneal* nerves. I have seen it also in the nerves of the upper extremity. The paralysis of the crural and obturator nerves may have passed off before the peroneal becomes affected. Electrical examination usually reveals partial reaction of degeneration. Sensory symptoms are frequently present, and Westphal's sign has been found in many cases. On the whole the clinical picture (see p. 160) may greatly resemble that of tabes dorsalis (diabetic pseudo-tabes). The prognosis is favourable, but the illness may be severe.

The form of neuritis which appears in *old age*—senile polyneuritis—to which many years ago [4] I drew attention, is distinguished by its insidious course and the insignificance of the sensory symptoms of excitement. The paralysis is also slight as a rule and does not affect the cranial nerves. Some cases, however, show (Stein [5]) that these criteria are not always present. Recovery is possible in senile polyneuritis—I have observed it in several cases—but the arteriosclerosis which is usually present constitutes a danger to life, and the patient in some cases succumbs later to apoplexy (Stein). The polyneuritis of old people may often be traced to senile arteriosclerosis and other forms of vascular disease.

It has been shown by ourselves and others that polyneuritis may develop from carcinomatous cachexia. In a severe case of this kind the optic nerves were affected in addition to the nerves of the extremities (Miura [6]). A miliary carcinosis may also develop in the peripheral nerves (Oberthür-Mousseaux, Raymond [7]).

. . . . . .

Pathological Anatomy of Multiple Neuritis.—Inflammatory and degenerative changes in the peripheral nerves form the pathological basis of this condition. The neuritis develops chiefly in the *peripheral* ramifications of the nerves, the muscular and the sensory cutaneous branches. The signs of inflammation are as a rule less marked than those of degeneration and atrophy. In many cases swelling and redness of the nerves and hyperæmia of the nerve sheaths is apparent, even to the naked eye. Hæmorrhages are less frequent (Eichhorst, Dejerine).

[1] *A. f. P.*, xiv. [2] *A. f. P.*, xviii. [3] *D. m. W.*, 1904. [4] *B. k. W.*, 1893.
[5] *M. m. W.*, 1897. [6] *B. k. W.*, 1891. [7] *Arch. de Neurol.*, xvii.

Perineuritic and interstitial changes are generally absent or slight, whilst the nerve fibres themselves show signs of a more or less complete atrophy. All the transitions between Gombault's[1] slight periaxillary neuritis and total degeneration of the nerve may be found, but Stransky,[2] in particular, would distinguish strictly between this periaxillary neuritis of toxic origin and Wallerian degeneration. Symptoms of regeneration are also noted (Gudden). Fig. 235 shows disintegration of the myelin into clots and granules, such as we find notably often in the muscular branches of the peroneal nerve. Fig. 236 B represents the transverse section of a nerve in interstitial and parenchymatous neuritis (compare with Fig. 236 A).

The peroneal nerves and their ramifications and the saphenus major are usually markedly affected, and, when the upper extremities are involved, the branches of the musculo-spiral, etc. The pathological anatomy generally corresponds well with the clinical symptoms. It is only in very acute cases (see Landry's paralysis) that although the paralytic symptoms are extremely severe the changes in the nervous system are slight or even negative.

The fine changes in the axis cylinders are described by Marinesco (*R. n.*, 1906).

FIG. 235.—Parenchymatous neuritis. Stained with osmic acid (teased preparation).

The peripheral character of the process is, however, not established with regard to all the forms of paralysis described. Alcoholic paralysis is undoubtedly due to polyneuritis. But even this affection may be associated with slight central changes in the spinal cord, *e.g.* partial atrophy of the anterior horn cells or a *poliomyelitic focus*, *diffuse or disseminated* inflammatory conditions such as I,[3] Leyden, Pal, and others have found in several cases, and which were evidently due to the action of the poison, but were much too slight and incomplete to be regarded as the primary cause of the symptoms. The mental symptoms, however, prove that multiple neuritis may be associated with a brain affection, and this has also been confirmed by the pathological investigations of Bonhöffer, Ballet, Faure, Wehrung,[4] Boedeker,[5] etc. The vagus symptoms and the oculo-motor paralysis have in some cases been traced to central, nuclear processes, to hæmorrhagic inflammation of the nuclei of the nerves (Thomsen [6]).

Although alcohol chiefly attacks the peripheral nervous system and produces the most marked changes in it, it may also simultaneously affect various other segments of the central nervous system.

In recent observations on polyneuritis (alcoholic, diphtheritic, etc.), by means of the finest methods of investigation—chiefly those of Nissl and Marchi—changes have been almost constantly described in the central nervous system (Marinesco, Bikeles, Sano, Goldscheider-Moxter,

[1] *Arch. de Neurol.*, 1880-1.
[2] *Journ. f. Psych.*, i.
[3] *Z. f. k. M.*, xi.
[4] *A. f. P.*, xxxix.
[5] *A. f. P.*, xl.
[6] *A. f. P.*, xix.

Preisz, Schlesinger, Luce, Heilbronner, Winkler, Bruns, Batten, Halban, Utchida, Cole,[1] Köster,[2] Homén,[3] etc.). There are notably diffuse and disseminated degenerations in the roots of the spinal and cranial nerves, in the *posterior columns* and anterior cornua, and fine changes in the cells. These are found also in the *nervous elements* of the cerebrum and cerebellum and in the spinal ganglia (Philippe). But a few only of these changes can be regarded as equivalent to the peripheral changes and attributed directly to the agents which produce the neuritis. Certain changes, including both those revealed by Nissl's method in the anterior horn cells, and some of those shown by Marchi's method in the posterior roots and posterior columns, have been interpreted as the simple results of the peripheral neuritis (see pp. 124 *et seq.*). But it is now recognised by most writers—and even at the commencement of the Nissl and Marchi era I had expressed my own reservations—that great caution is necessary in estimating these changes, and that above all they should not without further evidence be regarded as equivalent to degeneration.

The peripheral site of the *lead paralysis* is shown by the majority of the published pathological investigations. The poison does, however, sometimes exert its deleterious influence upon the spinal cord, and gives rise

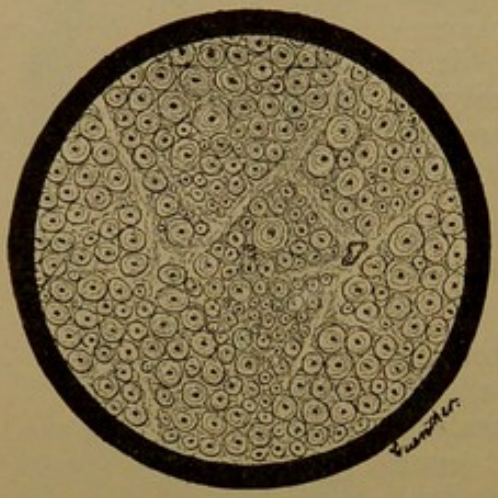

FIG. 236 A.—Transverse section through a normal nerve.

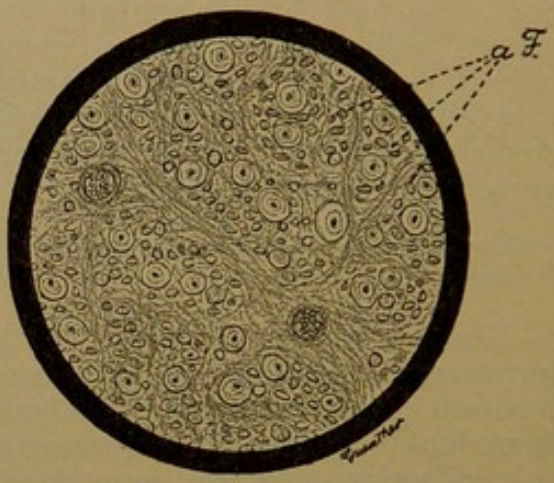

FIG. 236 B. (compare with a.)—Transverse section through an atrophied nerve. *a F.* atrophied fibre.

to a severe anterior poliomyelitis, as in a case described by myself [4] and some less marked cases of Zunker,[5] Oeller, Monakow,[6] and Onuf. That it also directs its influence towards the brain is most markedly shown by the symptoms of encephalopathia saturnina. The one nerve which is constantly involved is the musculo-spiral, but only some of its fibres are affected. Atrophic paralysis has been experimentally produced in animals (inhaling lead salts), and its cause has been found to be a focal poliomyelitis (Stieglitz [7]).

The nature of *arsenical paralysis* is as yet little understood. The few published pathological reports (Alexander, Henschen, Erlitzki, and Rybalkin), and in a higher measure the clinical nature and the course of the disease, point to a peripheral neuritis, although changes have also been found in the spinal cord and have possibly in a few cases been the predominant element.

It can hardly be doubted, judging from the investigations of P. Meyer,[8] Gombault, Arnheim,[9] Oppenheim,[10] and others, that the neuritic pro-

[1] *Br.*, 1902. [2] *Loc. cit.* [3] *Z. f. kl. M.*, 1903. [4] *A. f. P.*, xvi., 1885.
[5] *Z. f. k. M.*, i. [6] *A. f. P.*, x. [7] *A. f. P.*, xxiv. [8] *V. A.*, 1881, Bd. lxxxv.
[9] *A. f. Kind.*, xiii. [10] *A. f. P.*, xviii.

cesses constitute the essential basis of *diphtheritic paralysis*. The toxin here acts first upon the nerves (of the palate and pharynx) in the immediate vicinity of the infective focus; it then passes into the laryngeal and oculo-motor nerves and finally becomes still further disseminated. That the diphtheritic toxin has first a local effect upon the nervous system is shown by a case in which the paralysis remained limited to the abdominal muscles after diphtheritic inflammation of the umbilicus in a newborn child. Accommodation paralysis may, however, be the first result of an intestinal diphtheria. Symptoms of an interstitial and parenchymatous neuritis have been found in the nerves, and occasionally hæmorrhages, *e.g.* in the oculo-motor nerves. *Neuritis nodosa* has also been noted. Buhl found micro-organisms in the nerve sheaths. Arteritic processes were also discovered after death from diphtheritic paralysis. Klimoff describes lesion of the ganglia of the heart. We cannot admit that diphtheritic paralysis may be due to a primary myositis, even although marked changes have been found in the muscles and indeed were in one instance limited to them..

Experimental investigations recently made by Crocq, Foulerton-Thomson, Ransom, etc., have it is true revealed neuritic processes resulting from the infection, but they also showed changes in the roots, the meninges, and especially in the spinal cord itself (myelitic foci). Babonneix,[1] in particular, thinks he has experimentally proved the ascent of the neuritis from the morbid focus into the central organs. Pathological investigations on man carried out by modern methods, such as those by Sano,[2] in my laboratory, by Bikeles,[3] Preisz,[4] Katz,[5] etc.—as well as some older ones (Dejerine, Gombault)—have revealed degenerations, by no means inconsiderable, in the spinal roots and in the cord itself. Occasionally the results were negative, as in cases by Kohts and Hasche.

From all that has been said it is clear that *the diphtheritic poison acts upon the whole nervous system, producing the most marked changes now at this site, now at that, but most often in the peripheral nerves, and that it may also have a toxic effect upon certain areas without producing structural changes in them.*

The degenerative processes found in the nerve-cells of the central organs after acute fatal poisonings cannot be regarded as the basis of the typical paralytic conditions, as Raymond has again recently stated (*Arch. gén. de Méd.*, 1905). Bolton (*R. of N.*, 1903) found changes in the nervous nuclei of the medulla oblongata in acute diphtheritic toxæmia.

The peripheral nature of *tubercular, senile, and cachectic polyneuritis* has been placed beyond doubt by the investigations of Pitres and Vaillard, Oppenheim and Siemerling.

It cannot be doubted that conditions of central degeneration may develop simultaneously. Sand in one case found an association of tubercular polyneuritis and combined systemic disease of the spinal cord (*N. C.*, 1904), but this is quite an unusual complication.

Pathological investigations into the question of diabetic neuritis are but few in number (Marinesco,[6] Lapinsky, etc.). In Findlay's case phthisis was also present.

It is not probable that the micro-organisms themselves give rise to the

[1] *Revue mens. des mal. de l'enf.*, 1904. Orr and Rows (*Brit. Med. Journ.*, 2417) come to similar conclusions.

[2] *Journ. de Neurol.*, 1896.

[3] *Obersteiner*, 1894.

[4] *Z. f. N.*, vi.

[5] *A. f. Kind.*, xxiii.

[6] *N. C.*, 1903.

nervous degeneration in cases of multiple neuritis from an infective cause. The nervous system is usually affected by the introduction of toxic material, chemical bodies in the form of alcohol, lead, etc., into the organism. This is shown by the investigations of Homén-Laitinen,[1] Dopter, and Lafforgue, and has been proved as regards diphtheritic paralysis by Brieger, Fraenkel, and others. Similar evidence is also given by the results of bacteriological examination in polyneuritis, but these on the whole are very rare. Positive results were obtained by Glogner and Wokenius. See chapter on Landry's paralysis.

The *diagnosis* of multiple neuritis is generally an easy matter, especially when the paralysis is entirely of the peripheral type. Difficulties arise mainly from two directions. When the anæsthesia and the ataxia are most prominent, the clinical picture may so resemble that of tabes dorsalis that even an expert physician may occasionally fail to establish a definite diagnosis. Usually, however, the acute onset, the evidence of a toxic or infective cause, the absence of bladder symptoms, the immobility of the pupils (which has been observed only in rare cases of alcoholic neuritis, by Oppenheim, Eperon, Pándy, Kramer, etc., although the data of Raimann, Mönkemöller, and others differ from ours on this point) give definite indications for the differentiation. The diagnosis of multiple neuritis is also well founded if there is marked hyperæsthesia of the nerves and muscles, if the degenerative paralysis is associated with ataxia, etc. Exceptional cases of neuritis have, however, been described, in which the course was chronic, the tenderness to pressure very slight, the symptoms of paralysis absent, and in which bladder troubles and girdle sensation were occasionally present, so that even experienced physicians were led to diagnose tabes. It is well in every doubtful case where alcoholism is pronounced to decide in favour of neuritis, but to remember that even alcoholics may suffer from tabes (and also from combined systemic degeneration). I have occasionally seen perforating ulcers in alcoholic neuritis. It may be noted that gastric crises do not occur in alcoholic neuritis. Attacks of vomiting may be present, but these are usually not painful, are easily treated, and are never so intense as the crises of vomiting in tabes. Korsakow's psychosis decides in favour of alcoholic neuritis. This form of dementia has a superficial resemblance to paralytic dementia, but on close observation it cannot be mistaken. E. Meyer and Raecke have indeed maintained that a similar group of mental symptoms may occur in the course of paralytic dementia. Treatment in hospital, including the employment of suitable measures and especially the withdrawal of alcohol, will always lead to a definite result. The fact should also be kept in mind that spinal-cord diseases of another kind, such as the affection of Goll's columns in a case of Vierordt's, may develop from alcoholism.

It is only in a very small number of cases that lumbar puncture and serum reaction will be required to establish the differentiation (see p. 161).

It should further be remembered that at the height of acute infective diseases, of pneumonia at least, both the light reflex of the pupils and the knee jerks may be absent (F. Schultze,[2] Pfaundler, Lüthge[3]).

In cases in which trophic-motor symptoms or degenerative paralyses

[1] Finsk. läkar, 1896; *N. C.*, 1898, and Homén, *Acta. soc. scient. Fenn.*, 1902.
[2] *A. f. kl. M.*, Bd. lxxiii. [3] *M. m. W.*, 1902.

are the most marked features the affection may greatly resemble *anterior poliomyelitis*, both in its acute and sub-acute forms. In neuritis, however, sensory symptoms, viz., violent pain, paræsthesia, and at least slight diminution of sensibility in the feet and the finger-tips, are almost always present, or have originally been so. Lead paralysis occupies a unique position in that it affects exclusively the motor elements in the nerves, but other poisons and infective products may apparently act in this way. I have seen a few unimpeachable cases of polyneuritis in which pain and sensory symptoms were entirely or almost entirely absent. Tenderness of the muscles to pressure also appears in poliomyelitis, but intense tenderness of a nerve trunk on pressure, not due to any mechanical cause (traction on the nerve by the dragging of the paralysed arm, etc.), is a definite proof of neuritis. Further, the paralysis in acute poliomyelitis is seldom so symmetrically distributed, but is more often confined to one extremity and follows another type of distribution (see corresponding chapter). If the illness has developed acutely, a reappearance of the fever in the later stages, and renewed attacks of further symptoms of paralysis are in favour of neuritis. Finally, involvement of the cranial nerves is so unusual in poliomyelitis, that it also points to neuritis. Thus in a case in which there were no objective sensory symptoms, Raymond regarded the tenderness of the nerves to pressure and the facial diplegia as deciding the diagnosis in favour of multiple neuritis. This disease is certainly present where there are inflammatory changes in the *optic* nerve.

Raymond [1] regards polyneuritis, poliomyelitis, and Landry's paralysis as diseases which cannot be sharply differentiated from each other, as they have the same etiology. He admits, however, that a distinction on clinical grounds is expedient and necessary.

There is a chronic form of polyneuritis which, from the slightness of the symptoms of sensory irritation and paralysis, may greatly resemble chronic poliomyelitis or progressive muscular atrophy. Grinker (*Journ. Amer. Assoc.*, 1907) described such cases. Barnes (*Br.*, 1905) should also be referred to.

Eisenlohr has pointed out that *trichinosis* may give rise to symptoms similar to those of neuritis (and myositis), as it results in pain, paralysis, and œdema, which may be accompanied by atrophy in the muscles with reaction of degeneration, and even by Westphal's sign. The diagnosis can usually be made from the history of the disease : gastro-intestinal troubles first appear, then fever and severe muscular pains, followed by the characteristic muscular swelling, difficulty in moving the eyes, which are surrounded by œdema, disorders of speech, of voice, dyspnœa, etc. Great increase of the eosinophile cells and diazo reaction are also among the symptoms (F. Müller [2]). As to the relations of neuritis to polymyositis and the differentiation of these two affections, the corresponding chapter should be consulted.

I have seen a few cases of diffuse sarcomatosis of the cerebro-spinal meninges which at the commencement simulated the picture of polyneuritis.

The *periarteritis nodosa* described by Kussmaul and Maier, may, according to the observations of Schrötter, Freund, and others, give rise

[1] "Leçons sur les mal. du syst. nerv.," 1897 and 1901. See also Rosenberg, "Die Differentialdiagnose der Pol. ant.," etc., *Inaug. Diss.*, Heidelberg, 1890.

[2] "Der Ausbau der klin. Untersuchungsmethoden." *Z. f. ärztl. Fortbild.*, 1906.

to a clinical picture resembling that of polyneuritis. The reports on this very rare disease are as yet too few in number to permit of the establishment of diagnositic criteria of recognised validity.

Compare Benda, *B. k. W.*, 1908.

Syphilitic phlebitis (E. Neisser,[1] Buschke) should also be remembered from a diagnostic point of view.

Although, as I have found,[2] there is in neuropathic individuals marked tenderness of the peripheral nerves to pressure, it seldoms attains such a degree as in real neuritis, and there are no signs of interruption of continuity in the nerves, etc.

Treatment.—Treatment of multiple neuritis is one of the most grateful tasks of the neurologist. First of all the cause of the disease must be discovered and the injurious agents removed. Alcohol must be unconditionally withdrawn. This principle should only be departed from if there are signs that the heart is weak, when wine and brandy should be prescribed in suitable doses. In lead-poisoning, the patient must be absolutely prohibited from working with material containing the lead; his body must be carefully cleansed, especially the teeth, nails, etc., and any clothing which he wore at his work must be removed,[3] etc. Elimination of the lead from the organism will be assisted by the use of *iodide of potassium*, by stimulation of the intestinal functions (*aperients*, sulphates), by *diuresis*, and by baths, especially *sulphur* ones.

If the source of the poisoning lies in the organism itself, it is occasionally cut off by the removal of putrid, septic masses, by the stimulation of excretion, and above all by exciting *diaphoresis*. Diabetic neuritis does not, it is true, always disappear with the abatement of the glycosuria, but the fact that the neuralgia diminishes under the effect of a diet which lessens the secretion of sugar shows that the neuritis should be treated by strict *antidiabetic diet*. Malarial neuritis is usually cured by quinine. If the polyneuritis is due to syphilis, mercury should be carefully prescribed. This may fail, however, or may even have an unfavourable effect; the other methods recommended for the treatment of polyneuritis should then be immediately adopted. It is also advisable to make the diagnosis of syphilitic neuritis with *great reserve*, to look for other causes, and to adapt the treatment to them. Thus I have quite recently seen a case of severe alcoholic polyneuritis which was not recognised, and which on account of previous syphilis was treated by antisyphilitic methods, with very injurious results, whilst the treatment whch I instituted led rapidly to definite recovery. Change of climate has been specially efficacious in treatment of beri-beri.

In all forms of neuritis a *strengthening diet is important*. Milk-food, easily digested meat and fat (cod-liver oil), should be given at frequent intervals. In *diphtheritic* paralysis of deglutition, in order to prevent the penetration of particles of food into the trachea and to maintain the patient's strength, we should have recourse to feeding by a tube, and where necessary to nutrient enemata. Ziemssen recommends that the following food should be given by the tube four times a day: ½ to 1 litre of milk soup into which is stirred fine flour, 2 to 4 eggs, sugar and port-wine, alternating with ½ to 1 litre of concentrated meat broth, mixed with 4 tablespoonfuls of meat juice, 2 to 4 eggs, and one glass of port wine.

[1] *D. m. W.*, 1903.

[2] *Journ. f. Psychol.*, i.

[3] With regard to prophylaxis, see L. Lewin, "Die Hilfe für Giftarbeiter," *B. k. W.*, 1905.

Other nourishing preparations, such as somatose, may be added to these fluid foods. If vomiting is caused by the introduction of the tube, the patient should be laid on his stomach, the head hanging out of the bed. For weakness of the heart it may be necessary to use *stimulants* : wine, brandy, or subcutaneous injections of camphor (camphor 1·0 to 5·0 ol. amygd. dulc., filling the syringe several times). Faradisation of the heart region is also recommended. If there is bronchitis without sufficient expectoration, *artificial increase of expiration* by pressure on the lower parts of the thorax may be wonderfully helpful. Blood-letting should be entirely avoided.

Comby (*R. n.*, 1906) believes that injection of the diphtheritic serum has also a beneficial effect upon the paralysis, but this is contradicted by Guinon-Paton and others.

In the first stages of polyneuritis, *diaphoretic measures* are to be very specially recommended. This recommendation is unfortunately not yet sufficiently attended to, as I have found in many cases which have come under my treatment after the acute stage has passed. When the condition of the patient's strength permits of it, the secretion of sweat should be stimulated by hot baths carefully given ; otherwise—especially when the heart is weak—it should be attained by wet-packs and woollen coverings, by introducing hot air underneath the bed clothes, by the use of a hot-air apparatus, along with hot drinks, and finally by the administration of aspirin. Diaphoresis may be maintained for one to two hours. I have obtained splendid results with this treatment, even in severe and long-standing cases, and I have at my disposal a very large number of similar observations. Careful supervision is of course necessary when the patient is weak, and in a few cases the sweat cure can only be borne if employed every second day. I have occasionally had to prolong this treatment for many months before recovery was complete.

Drugs can usually be entirely dispensed with, but in the infective forms of polyneuritis *preparations of salicylates*, salol, and especially aspirin, already mentioned, are suitable. If the pain is violent and is not relieved by the application of warm fomentations or Priessnitz's pack, the well-known anti-neuralgic drugs may be tried, and if these fail, *morphia*. I have almost always been able to avoid giving morphia, and hitherto I have not required to use sub-arachnoid injections of cocaine or the Sicard-Cathelin method.

During the first stages *absolute rest in bed* and a comfortable position is necessary ; a single overstrain of the affected nerve may cause considerable aggravation. Passive movements must be avoided as far as possible. These precautions are specially needful in diphtheritic paralysis if there are signs of affection of the heart or vagus. The patient must *never raise himself* in bed. Involvement even of the laryngeal and pharyngeal branches of the vagus, calls for the greatest care, as paralysis of the heart may supervene rapidly and unexpectedly. The room must be well aired, or the patient may be carried in his bed into the open air.

In *alcoholic paralysis* the patient should be treated, not at home but in hospital, as only then can he be properly watched and alcohol be absolutely cut off. The influence of chill must be avoided as carefully as possible.

If the neuritis has reached its height, and the condition become stationary or shown the first signs of improvement, *electrical and mechanical*

treatment become advisable. So long as symptoms of irritation persist, *stabile galvanic treatment* is to be recommended, which may later be replaced by the *labile* and *faradic stimulation of the muscles*. In many cases in which the electric current had no effect, I have seen almost immediate improvement follow the use of *massage*. It must be used with *extreme care* and suited to the individual sensibility. It should be commenced with gentle rubbing and stroking in the neighbourhood of the affected nerves and muscles, the manipulations being very gradually strengthened. Active and passive gymnastics may be of real benefit, but all overstrain must be most carefully avoided.

Subcutaneous injections of strychnine ($\frac{1}{30}$-$\frac{1}{25}$ grain several times a day) are often of use in persistent paralysis. Injections of carbolic acid (2 per cent.) or of carbol-morphia into the neighbourhood of the affected nerves have also been recommended.

Baths, no longer at a high temperature, but *mild* baths of 90°-96° F., are also helpful in the later stages. When the conditions permit of it and there is no danger involved in moving the patient, a course of baths at Nauheim, Oeynhausen, Kreuznach, Wildbad, Wiesbaden, Teplitz, Aix-les-Bains, etc., may be advised, but only after improvement is far advanced. This is specially suitable as an after-treatment. Peat-baths and local peat fomentations may also be used. A winter residence in the *south* may have an excellent effect. In the stage of *convalescence*, *hydrotherapeutic methods* are beneficial, but they should be limited to local friction of the extremities with damp cloths, commencing always at mild temperatures.

Measures should be taken early to prevent the development of paralytic contractures, especially in young persons; as soon as the pes equinus position becomes apparent, pressure of the bed clothes should be prevented by a *wire cage*. A heavy sand-bag should be placed so that it will press the point of the foot upwards. If contractures have developed when the patient comes under observation, they must be treated according to the well-known methods. Tenotomy is seldom necessary.

Residual paralysis may be cured by transplantation, but this operation should only be decided upon when the paralysis has lasted so long as to show that it is definitely incurable.

Appendix

I. Landry's Paralysis, Acute Ascending Paralysis

In the year 1859, Landry described the following very characteristic group of symptoms: A healthy person, after slight prodromal subjective troubles (general feeling of malaise, paræsthesiæ in the extremities, etc.), develops flaccid paralysis in the legs, usually at first in one only, but extending within a few hours or a day to the other. After the paralysis has become complete in the legs, it passes to the muscles of the trunk, and within a few days reaches the arms, which also develop a condition of flaccid paralysis. Thereafter the muscles of deglutition, articulation, and respiration are affected, and death follows in a few days or weeks, with symptoms of suffocation, except in slight cases, in which the symptoms disappear in order, the muscles last affected being the first to regain their mobility.

According to Landry's description, there are also slight sensory symptoms, but he emphasises chiefly the absence of *muscular atrophy* and its electrical signs, and the *absolutely negative results of post-mortem examination.* The course of the disease may be modified to this extent, that the bulbar symptoms may appear first, followed by the paralysis of the arms, etc.

Although Landry's data were essentially confirmed by the observations which first followed his (Pellegrino-Levy,[1] Westphal,[2] Bernhardt,[3] Kahler and Pick,[4] Ormerod,[5] Eisenlohr,[6] and others), and although not a few cases were described which exactly tallied with this description, further investigations have shown that its limits had been drawn too narrowly, that cases belonging to this category may differ in manifold ways from Landry's type, and that the only characteristic which is common to them all is the flaccid paralysis which extends in *rapid succession* from below upwards, *i.e.* from the legs over the trunk and the arms to the bulbar nerves (or, much more rarely, inversely, from above downwards).

With regard to the symptomatology, the following details have been established :—

The paralysis commences in the majority of cases in one or both lower extremities. Paræsthesiæ may occur at the same time. It makes rapid progress, and may become complete paraplegia within one or a few days. It is *flaccid*, and usually associated with *loss of the tendon and cutaneous reflexes.* Pain is entirely absent, or present only during pressure on the muscles and nerves and during passive movement. There is rarely any complaint of spontaneous pain. The paralysis spreads upwards *within a few days*, first to the muscles of the pelvis, then to those of the abdomen, back, shoulders, and thorax. It then reaches the arms, which become completely paralysed. Finally, the muscles of the lips, tongue, palate, pharynx, and respiration are affected, speech becomes indistinct and difficult, and there is trouble in swallowing. Severe respiratory troubles, simple rapid or Cheyne-Stokes breathing and signs of paralysis of the diaphragm supervene, and death follows with symptoms of asphyxia on the eighth to tenth day, less frequently even on the third or fourth day, and in rare instances after some weeks. In many cases, however, recovery sets in in the way described above ; convalescence may then extend over a long period.

If the affection is ushered in by the bulbar symptoms, death may supervene before the paralysis has spread to the extremities. Thus in a case of this kind described by Howard,[7] death occurred after twenty-nine hours. It is only in exceptional cases that the arms are first affected, or all four limbs simultaneously.

The condition of the *sensibility* varies in the different cases. We may regard absence of marked disturbances as the rule. There is never complete loss of sensation, but a slight diminution of sensibility for some or all qualities is frequently present at the ends of the extremities. A delay in the conduction of sensation has occasionally been noted. The muscles generally retain their *normal size* and react promptly to the electrical current : this is particularly true of the rapidly fatal cases, and has been found also in a few of longer duration. But in some cases which otherwise

[1] *Arch. gén.* i., 1865. [2] *A. f. P.*, vi. [3] *B. k. W.*, 1871.
[4] *A. f. P.*, x. [5] *St Barth. Hosp.*, xxviii.
[6] *V. A.*, Bd. lxxiii. ; *C. f. N.*, 1883, and *D. m. W.*, 1890. See also literature in Remak, *loc. cit.*, and Eulenburg's *Realenzyklopaedie*, xii.
[7] *Brit. Med. Journ.*, 1898.

correspond exactly to this type, there is *muscular degeneration*—manifold disturbances of the electrical excitability, quantitative diminution, and partial and complete reaction of degeneration being present. I found a peculiar condition of the electrical excitability in one case of this kind; the nerves and muscles reacted even to weak electrical stimuli, but the intensity of the contraction could not be increased by increasing the intensity of the current; their minimum and maximum were very nearly approached, and even application of the strongest current could not elicit a marked contraction; occasionally, in fact, a strong current elicited no contraction at all, or a weaker contraction than that produced by a current of less intensity (compare chapter on myasthenic paralysis). Investigation of an excised particle of muscle revealed wax-like (vitreous) *degeneration*. These phenomena disappeared with recovery. In a few cases regarded as Landry's paralysis, which assume a favourable course, a muscular atrophy has remained as the residue of the disease (Immermann,[1] Etienne).

The *functions of the bladder and intestine* are not as a rule affected, but there are exceptions, and complete paralysis of the sphincters has been observed in cases which have been included in this group.

No cranial nerves other than those mentioned are involved; oculomotor paralysis only (diplopia, accommodation paresis) has appeared in exceptional cases. Unilateral or bilateral *facial paralysis* has been several times described. Paralysis of the laryngeal muscles also occurs (Kapper).

The *mind* almost always remains clear; it is only when there is high fever and when signs of a general septic infection have been present that consciousness may be impaired. The *temperature* is *normal* in the great majority of cases, but attacks of fever have occasionally been reported. Profuse sweating is often mentioned, œdema less frequently (as in a case of Soltmann's [2]).

It is well to consider the *etiology* and the *pathological anatomy* together. Even Landry had the impression that the disease was due to *poisoning*. This view is accepted by almost all the writers, and Westphal has thoroughly discussed its basis. The following facts point to its being a toxic or infective disease:

In several cases there was *enlargement of the spleen*, swelling of the lymph glands, hæmorrhagic foci in the lungs and intestine, *albuminuria* or nephritis. Chantemesse and Ramond [3] observed an epidemic in an asylum in the form of a paralysis, which at least in its onset and extension revealed a near relationship to Landry's paralysis. The cases of Baumgarten,[4] Curschmann,[5] Centanni,[6] Eisenlohr, Remmlinger,[7] Oettinger and Marinesco,[8] Marie and Marinesco,[9] Bailey and Ewing,[10] Buzzard,[11] etc., are specially significant. In that described by Baumgarten there had probably been anthrax infection, corresponding bacilli being found in the blood and the tissue fluid. Curschmann describes a case in which—although there had been no typhoid—in addition to the intestinal changes characteristic of typhoid, typhoid-bacilli, which could be grown in pure cultures, were found in the spinal cord. Centanni found interstitial

[1] *A. f. P.*, xvii. [2] *Jahrb. f. Kind. N. F.*, i. [3] *R. n.*, 1898. [4] *B. k. W.*, 1895.
[5] *Verhandl. d. Kongress. f. in Med.*, Wiesbaden, 1886.
[6] *Zieglers Beitr.*, viii. [7] *Soc. d. Biol.*, 1896. [8] *Semaine méd.*, 1896.
[9] *Semaine méd.*, 1895. [10] *New York Med. Journ.*, 1896. [11] *Br.*, 1903.

neuritis associated with bacilli in the endoneural lymph-space. Eisenlohr was able in a case of Landry's paralysis to demonstrate various kinds of bacilli, and in one case, which, however, does not strictly belong to this group, various kinds of staphylococci in the central nervous system, and to trace the clinical symptoms to a mixed infection. Remmlinger found the streptococcus longus; Marinesco found diplococci, some of which were enclosed in leucocytes. In the case he examined with Marie he discovered a micro-organism resembling or corresponding to an anthrax bacillus, which was chiefly contained in the vessels. Chantemesse and Ramond in acute ascending paralysis found a species of proteus in the blood, the cerebro-spinal fluid, and the tissues, which produced in animals a disease with paralytic symptoms which ended fatally. In a case of acute descending bulbar paralysis, which certainly belongs to this class, J. Seitz [1] found the Fränkel-Weichselbaum bacterium, which was scattered through the brain stem and the spinal cord in large masses, following the lymph tracts of the tissue. The nervous elements were unaltered. The author thinks that the micro-organisms had found their way by the naso-pharynx to the brain. Roger and Josué found a virulent pneumococcus in one case, as did Courmont and Bonne.[2] Macnamara and Bernstein succeeded in cultivating a tetracoccus, and Sheppard-Hall a streptococcus from the blood and cerebro-spinal fluid (*R. of. N.*, 1907). F. Buzzard,[3] found a kind of coccus in the dura mater, which produced a flaccid paralysis in animals. On the other hand, cases of Landry's paralysis have been described quite recently in which bacteriological examination yielded absolutely negative results (Seifert, Schultz, Thomas, Kapper, Workman, Hunter [4]).

The pathological appearances are very indefinite and differ greatly in different cases. In many they were absolutely *negative*, as was found by Landry, Westphal, etc. Such observations have recently been made by Ormerod and Prince, Seifert, Kapper, Hun,[5] Girandeau-Levi,[6] etc. Goebel [7] and Burghardt [8] also found only slight changes. In some cases there were *disseminated foci of inflammation* or exudation and capillary hæmorrhages in the medulla oblongata (especially in the pyramidal tracts); in others similar or identical changes were found in the spinal cord (Eisenlohr, Schultze,[9] Gombault, Kétli, Hlava, Immermann, Mönckeberg,[10] etc.). Wappenschmidt attaches much importance to the hyaline thrombi which he discovered, as, according to Recklinghausen and Klebs, these may be due to the action of bacterial toxins. There was occasionally *swelling of the axis cylinders* in the white matter of the antero-lateral columns. In other cases the condition was regarded as the slightest form of poliomyelitis, or in view of its clinical characteristics, as a form of acute ascending poliomyelitis. In one case degeneration of the anterior roots was the sole change. Widal and Le Sourd describe neuritis of the roots as the only alteration in one case.

Since attention has been directed to the peripheral nervous system, neuritic processes have been frequently discovered and are regarded by some writers as the basis of the disease (Dejerine-Goetz, Nauwerck,[11] Barth, Ross, Putnam,[12] Klumpke, Boinet,[13] Rolly,[14] Pelnár,[15] and others).

[1] *D. m. W.*, 1897. [2] *Arch. de Neurol.*, 1899. [3] *Br.*, 1903 and 1907.
[4] *R. of N.*, 1906. [5] *New York Med. Journ.*, 1891. [6] *R. n.*, 1898.
[7] *M. m. W.*, 1898. [8] *Charité-Annalen*, xxii. [9] *B. k. W.*, 1883.
[10] *M. m. W.*, 1903. [11] *Zieglers Beitr.*, 1889. [12] *Boston Med. and Sug. Journ.*, 1889.
[13] *Gaz. des hôp.*, 1899. [14] *M. m. W.*, 1903. [15] *Z. f. k. M.*, xxxii.

A combination of neuritis with myelitic or poliomyelitic processes has also been found (Krewer,[1] Mills-Spiller,[2] Guizetti, Knapp and Thomas,[3] etc.), and this has given rise to the theory that Landry's paralysis was produced by the rapid extension of a neuritic process to the spinal cord and oblongata (Krewer).

Recent investigations by means of finer methods have almost always given positive results, the changes predominating in the spinal cord, especially in the grey matter. These changes may be inflammatory and vascular (vascular disease, hæmorrhage, exudation, thrombosis, softening, infiltration, etc.), or they may affect the nerve cells, which, however, are seldom markedly atrophied. Such conditions have been described by Marinesco, Bailey and Ewing,[4] Courmont-Bonne, Wappenschmidt, Thomas, O. Reusz,[5] E. Bramwell,[6] Lohrisch,[7] Mann-Schmaus,[8] Marinesco,[9] Workmann-Hunter, etc. The finer the method employed, however, the greater as a rule is the necessity for caution in the interpretation of the changes, as I have already emphatically stated in several parts of this work with regard to the methods of Nissl and Marchi.

It is obvious, in view of the varying results of pathological investigation, that we cannot build our nosological conception of this disease upon such a basis. The clinical symptoms are fairly well characterised and are undoubtedly due to infective causes and *toxins*, which in the majority of cases are bacterial products. We do not, however, know of a specific vehicle of infection in Landry's paralysis. The disease may develop from diphtheria, typhoid, small-pox, anthrax, influenza, pneumonia, whooping-cough, the puerperium, gonorrhœa (?), malaria (?), and probably also from septicæmia and other unknown infective processes. Walker [10] and Buchanan saw it occur in chronic cystitis. It may apparently develop in the course of rabies or after the Pasteur treatment of this disease (Rendu, etc.). One of my patients, who was a groom, developed Landry's paralysis as the result of a wound from the kick of a horse suffering from septicæmia (compare H. Behner, *Inaug. Diss.*, Berlin, 1898). Hey [11] saw it follow suppuration of a finger. In another case of my own, there had merely been severe diarrhœa. *Autointoxication* from the intestine was regarded as the cause in a few cases, *e.g* by Kapper, who found skatol and indol in the urine. Alcoholism, and especially syphilis, are included among the etiological factors.

It is doubtful whether the ascending paralysis observed by Rosenfeld in the course of exophthalmic goitre should be included here. Observations by Lohrisch and others show that it may supervene upon a tabes.

It appears also from a communication by Spillmann (*Rev. méd. de l'Est*, 1905) that a similar group of symptoms may develop in uræmia.

It is uncertain whether penetration of the micro-organisms themselves into the spinal cord, the medulla oblongata, and the peripheral nervous system are capable of producing the disease. It is probable that the active poison injures the motor nerve tracts in the spinal cord, the medulla oblongata, and the peripheral nerves, that it may have a paralysing effect without producing recognisable lesions in the nervous

[1] *Z. f. k. M.*, xxxii.
[2] *Journ. Nerv. and Ment. Dis.*, 1898.
[3] *Journ. Med. Sc.*, 1898.
[4] *New York Med. Journ.*, 1896.
[5] *Charité-Annalen*, xxiii.
[6] *R. of N.*, 1905.
[7] *A. f. P.*, Bd. xl.
[8] *A. f. kl. M.*, Bd. lxxxix.
[9] *R. n.*, 1905.
[10] *Brit. Med. Journ.*, 1895.
[11] *M. m. W.*, 1904.

system, but that the attack upon it occasionally leaves visible (microscopic) traces, which can be detected sometimes in the motor tracts and centres of the medulla, sometimes in the peripheral nerves, and sometimes in both simultaneously. Whether the structural changes are more pronounced in this or that part must depend upon the character of the virus, the intensity and acuteness of its action, and also upon the individual disposition (greater susceptibility of this or that region). As a rule it affects the trophic centres and tracts so slightly that there is no muscular atrophy. But there is no rule of any kind in this respect.

This view, expressed in the first edition of this text-book, has been accepted by most recent investigators.

Gowers thinks that the fibrous net in the grey matter of the anterior horns, and the terminal ramifications of the pyramidal tracts in this region are specially affected. We should in that case have expected an interruption of the motor tracts, without trophic disorders and spastic symptoms, but this is merely an unfounded theory.

The cases which correspond exactly to Landry's type are clearly distinguishable clinically from other well-known diseases. Those which differ from this type for the most part closely resemble *multiple neuritis*, an affection which has in common with Landry's paralysis a toxico-infective basis. It is not, however, correct to identify the two completely and to regard Landry's paralysis as an extremely acute polyneuritis. Still less are we justified, we think, in regarding it as acute poliomyelitis, although with Raymond,[1] who regards acute anterior poliomyelitis, polyneuritis, and Landry's paralysis as a morbid unity and as merely different manifestations of the same disease (the cellulo-névrite aigue antérieure), we recognise its close genetic relationship. Etienne and Martinet [2] also adopt this point of view. On the other hand the separation of Landry's paralysis into three different forms, bulbar, spinal, and peripheral, according to this presentation, cannot be carried into practice.

There is a form of acute ascending myelitis and meningomyelitis (*e.g.* on a syphilitic basis), which in its development and course reveals a certain resemblance with acute ascending paralysis, but which differs in its symptomatology. Buzzard and Russell have described one such case.

F. Buzzard (*Lancet*, 1907) gives valuable data as to the differential diagnosis. A complicated case published by Jacob (*N. C.*, 1907) is not clear.

Taylor points out that some of the cases described by American writers and termed Landry's paralysis, such as the case of Bailey-Ewing, should be regarded as acute poliomyelitis of the adult. He thinks that Landry's paralysis can no longer be considered as a morbid entity, and that the term should be reserved solely for the cases corresponding to the old type of Landry (Taylor-Waterman [3]).

Affections resembling Landry's paralysis have been lately produced in animals by the introduction of micro-organisms into the circulation of the blood (Thoinot and Maselli).

The prognosis of the disease is unfavourable as regards life. A fatal termination is to be particularly expected in the very violent cases which spread rapidly to the medulla oblongata. Death may also occur after some weeks in cases with less rapid course. We are particularly justified in anticipating recovery when some of the symptoms, notably

[1] *Malad. du Syst. nerveux*, 1897. [2] *Thèse de Paris*, 1897. [3] *Brit. Med. Journ.*, 1902.

the bulbar, have begun to pass off. In three out of eight cases which I had an opportunity of treating, there was complete recovery. In these the course was somewhat protracted, whilst in the fatal cases it was very rapid (in one there was tuberculosis, in the second pneumonia, and in the others the etiology was uncertain).

Some French writers (Brissaud,[1] Sicard-Brauer [2]) have lately attempted to find indications for the prognosis from the cytological conditions, as the presence of lymphocytosis and numerous polynuclear elements shows the primary lesion to be a central myelitis and thus points to a grave prognosis (?).

Treatment.—Remedies of very various kinds have been recommended. Counter-irritation, *e.g.* the application of the cauterising iron to the back, is warmly advocated. It seems advisable to adopt diaphoretic and antiphlogistic treatment in certain cases. In a few with a history of syphilis, mercurial treatment had a beneficial effect. Soltmann has quite recently reported a successful case of this kind. As to the permanent therapeutic value of lumbar puncture, we have as yet no adequate experience. Among the drugs used, *ergotin* has been found to be specially useful (ergotin 1·25, aq. cinnam. 60·0, in teaspoonful doses every hour); in one very severe case recovery is said to have been induced by this drug.

Whether the blood-serum treatment will prove of any service in this form of disease the future alone will show.

II. Acute and Chronic Polymyositis

Our knowledge of this disease is of recent date. We owe the first fundamental observations to Wagner,[3] Hepp,[4] and Unverricht [5]; subsequently Strümpell,[6] Loewenfeld,[7] Senator,[8] Lorenz,[9] Kader,[10] and others have contributed to the investigation of this affection. I [11] have also made communications upon the subject.

Other forms of myositis, which were already known to the earlier writers (Virchow, Froriep), such as chronic interstitial myositis resulting in the formation of cicatricial tissue, purulent myositis or abscess of the muscle, and progressive myositis ossificans, need not be considered here.

Polymyositis may occur at any age. The youngest of my patients was eight years old, the oldest was a woman in the fifties. The onset of the affection in childhood is mentioned by A. Schüller.[12] Men are somewhat more frequently affected than women; this distinction was not apparent amongst my own cases.

Among the causes of polymyositis, the most important are *infective processes*. The disease sometimes develops in *tubercular* individuals. It has been occasionally observed in the *puerperium* (Winkel, Waetzold, Unverricht). I myself have seen such cases. It may undoubtedly occur after *influenza*, *tonsillitis*, and *acute articular rheumatism*. Its relation to the latter has been specially considered by Sahli, Rosenbach, Leube, Risse,[13] and Edenhuizen.[14] Jessen [15] noted it after measles. Cases

[1] *R. n.*, 1906.
[2] *R. n.*, 1906.
[3] *A. f. kl. M.*, Bd. xl., 1887.
[4] *B. k. W.*, 1887.
[5] *Z. f. k. M.*, xii.
[6] *Z. f. N.*, i.
[7] *M. m. W.*, 1890.
[8] *D. m. W.*, 1894, and *Z. f. M.*, xv.
[9] Nothnagel's "Spez. Path. u. Ther.," xi.
[10] *Mitt. a. d. Grenzgeb.*, ii.
[11] *B. k. W.*, 1899 and 1903.
[12] *Jahrb. f. Kind.*, viii., Ergänz.
[13] *D. m. W.*, 1897.
[14] *A. f. kl. M.*, Bd. lxxxvii.
[15] *B. k. W.*, 1904.

reported by Servel,[1] Eichhorst,[2] Clerk-Dandoy, Ware,[3] and others point to a gonorrhœic form of myositis. Senator suspected an autointoxication arising from the gastro-intestinal tract. Emphasis has in some cases been laid upon the gouty diathesis. The occurrence of a myositic form of carbonic oxide paralysis is illustrated by a case of Soelder's. In two of my cases the disease developed after a forced "Kneipp cure"; and although in one of these tonsillitis was one of the first symptoms, I would ascribe to the excessive cold the significance at least of an accessory cause. Gowers[4] places this factor in the foreground of the etiology.

Although from the evidence of the cases to which we have access we may attribute a prominent part in the etiology to infective processes, most writers draw a sharp distinction between polymyositis and the purulent forms of muscular inflammation. This view has been opposed by A. Fraenkel,[5] and still more decidedly by Kader. They hold the separation of the purulent from the non-purulent forms to be an artificial one, since the latter is also a metastatic or *septic* affection of the muscles. Pointing to the results of bacteriological investigation by Waetzold and Bauer[6]—who found the staphylococcus pyogenes in polymyositis—and the fact, generally admitted, that septic processes may run their course without any trace of suppuration, Kader would regard all the forms of myositis (serous, interstitial, and purulent) as a single morbid species, viz., *septic myositis*. Still, for the present, it appears to us right that polymyositis should be distinguished from muscular abscess, just as we would maintain the differentiation of non-purulent encephalitis from cerebral abscess, although the two affections are closely related to each other.

Micro-organisms have also been found in a few recent cases (Georgiewsky, Körmöczi[7]).

The disease has usually an *acute*, but not a sudden onset. It commences as a rule with disturbances of the general condition. The patient feels exhausted, and has a dull feeling of heaviness in all his limbs; this is followed by headache, vertigo, and gastric symptoms. From the first or after a few days, pain comes on in the *muscles* of the extremities and of the trunk. It is described as dragging and irritating, and it restricts active movements. The muscles affected gradually become completely *powerless*, and the patient lies in bed paralysed and helpless, unable to move a limb.

The proximal parts of the extremities—especially the *muscles of the shoulder and upper arm*—are usually more severely affected than the distal, so that the fingers can be moved, although the shoulder- and elbow-joints are absolutely immobile. The muscles are *very tender to pressure*, and passive movements cause pain. In one of my patients the pain was first localised in the joints, which, however, did not swell. It then passed suddenly to the muscles and settled in them.

Swelling of the muscles and of the soft parts and the skin above them is one of the most important *objective* symptoms. The œdematous or tensely solid infiltration of the skin and subcutaneous tissue may be so great that it is impossible to judge of the condition of the muscles. This

[1] *Thèse de Bordeaux*, 1900. [2] *D. m. W.*, 1899. [3] *Amer. Journ. Med. Sc.*, 1901.
[4] *Wien. med. Presse*, 1899. [5] *D. m. W.*, 1894. [6] *A. f. kl. M.*, 1900.
[7] *Orvosi Hetilap*, 1902.

swelling occurs chiefly over the muscles most affected, and is therefore most marked on the trunk segments of the limbs, in the shoulder, the upper arm and elbow-joint, on the thigh, etc. This unusual localisation of the œdema is specially characteristic. It is also frequently noticeable in the face, chiefly in the region of the eyelids. The arms are as a rule more severely affected than the legs. Where the muscles can be palpated, they are sometimes felt to be firm, sometimes soft and "pulpy," and a kind of pseudo-fluctuation and circumscribed swelling has been noted. The inflammatory process may spread to the tendon-sheaths. Contractures, *e.g.* in the biceps, may develop as a result of the atrophy of the muscles.

The *skin* is generally *reddened*, and sometimes hot. The redness may suggest erysipelas, as not infrequently it is specially marked in the skin of the face. *Exanthema* resembling roseola and urticaria have been observed. Unverricht therefore speaks of a *dermatomyositis*. The skin may later peel or desquamate, and may assume a cicatricial appearance or even a sclerodermic condition. I have occasionally observed glossy skin and a non-œdematous swelling of the subcutaneous tissue. *Hyperidrosis* is frequently present.

The muscular affection rarely involves the muscles of respiration and deglutition, of the tongue, jaw, diaphragm, heart, and bulb. Extension of the myositis to the *respiratory muscles* causes more or less difficulty in breathing. Involvement of the deglutition muscles makes it difficult or impossible to take food. Tachycardia and symptoms of weakness of the heart muscles have been noted in several cases (Loewenfeld, Bauer, Lorenz,[1] Jolasse,[2] Oppenheim, and others). Fajersztain[3] mentions disturbance of articulation. Ptosis and paralysis of the eye muscles have also been observed. On the other hand the myositis may in mild cases be limited to the muscles of one extremity, to one or both legs, an upper arm, or even to one muscle. A localised form of the dermatomyositis has also been observed (Oppenheim, Schlesinger[4]). Stomatitis and tonsillitis were present in many cases. In a few of the cases under my treatment the mucous membrane of the mouth, larynx, and pharynx was involved to such a marked degree (redness, swelling, ulceration) that the term of *dermatomucosomyositis* appeared to me to be appropriate.[5] The affection had also spread to the conjunctivæ and to the external auditory process. Iritis was present in one case. The mucous membrane may, however, be quite unaffected, as shown by Korniloff[6] and others.

The mind as a rule remains clear, but the fever and heart weakness may give rise to delirium and confusion, especially towards the end of life. In one of my cases visual hallucinations were very marked. The sensory nerves otherwise act normally. Sensibility does not seem to be affected in typical cases.

The electrical examination is associated with great difficulty on account of the severe swelling of the soft parts. It reveals *quantitative diminution* of the excitability (especially direct), amounting to entire abolition, without any qualitative changes. But in a few cases there

[1] This author has recently published an exhaustive discussion of the heart symptoms (*D. m. W.*, 1906).
[2] *Mitt. aus der Hamb. Staatskr.*, 1897. [3] *Gazet. lekarsk.*, 1899. [4] *C. f. Gr.*, 1900.
[5] This experience is confirmed by Streng, *Z. f. k. M.*, Bd. liii. [6] *Z. f. N.*, ix.

were no disturbances whatever of the electrical excitability; these may have been merely slight forms of the disease.

The *tendon reflexes* are diminished or quite abolished when the corresponding muscles are affected; the *cutaneous reflexes* may be normal.

The *temperature* is usually increased during the whole course of the illness, and it may rise above 40° C. The pulse is correspondingly accelerated, but tachycardia may occur independently of the fever. Rise of temperature is by no means constant in chronic cases. Enlargement of the spleen has sometimes been found. Hæmorrhage from the internal organs, especially from the intestine (Buss), has been observed as an accessory symptom.

The illness may last for weeks, months, and even for a space of one to two years; it has therefore an *acute*, a subacute, and a *chronic* course. It may terminate in recovery in a few weeks, or in death after some weeks or months. In two of my cases with a protracted course, fresh exacerbations occurred somewhat acutely from time to time. An intermittent course is also described (Waetzoldt, Laquer, Herzog, Sick[1]). *Remissions* are not uncommon in chronic cases.

Asphyxia or broncho-pneumonia are the usual causes of death. A complicating nephritis may prove fatal. Life is also endangered by involvement of the heart muscle. One of my patients died with the symptoms of paralysis of the heart, signs of heart weakness having developed during the last weeks of life. Bauer has demonstrated the involvement of the heart muscle (hæmorrhagic myositis) by pathological investigation, as Jolasse has also done.

The prognosis as to life, so far as our experience goes, is grave, although recovery is by no means impossible, and is to be chiefly expected in cases which from the first have assumed a mild course. Reports of cases with a mild and favourable course will undoubtedly increase when we become more familiar with the disease. This expectation, which was expressed in the first edition, has already been fulfilled, as numerous cases of a benign character have been described in recent years (B. Lewy,[2] M. Levy-Dorn,[3] Herz,[4] Hnatek,[5] Christen,[6] and others). I[7] have also been able to show from my own experience that even in the severe diffuse forms of the disease, the prognosis is not so unfavourable, as out of ten cases only two ended fatally, whilst five recovered (under diaphoretic treatment consistently carried out). Neubauer also reported recovery in a severe case of acute polymyositis, as did Unverricht, Buss, and Georgiewsky. In the circumscribed form, on the whole, a favourable course is to be expected.

The primary *pathological cause* is either a *parenchymatous* or more commonly an *interstitial myositis*, extending over the whole or a great part of the muscles of the body. The affection can generally be recognised macroscopically from the swelling, discoloration, and infiltration of the muscles, in which hæmorrhages are often also found. Some authors think there is a special hæmorrhagic form of polymyositis. The changes most commonly reported post-mortem are *serous* infiltration, gelatinous appearance, *doughy*, fragile, soft, or firm consistency, a mottled look, a greyish-yellow colour of the muscular tissue, etc. Fig. 237 shows a transverse section of a muscle from a case of this kind

[1] *M. m. W.*, 1905. [2] *B. k. W.*, 1893. [3] *B. k. W.*, 1895. [4] *D. m. W.*, 1894.
[5] *Wien med. Presse*, 1905. [6] "Korresp. f. Schweiz," 1903. [7] *B. k. W.*, 1903.

described by Wagner, in which the round-cell infiltration of the interstitial tissue is very marked. Figs. 238 and 239 are made from preparations which I obtained in one case from an excised portion of the living muscle.

As regards the *differential diagnosis*, confusion with *trichinosis* is the most likely mistake. The symptoms of the two diseases resemble each other so closely that polymyositis has been termed pseudo-trichinosis. Trichinosis, however, affects a number of individuals who have eaten meat from the same animal. Here also the gastric symptoms are most prominent, especially at the onset. The muscles of the eyes, pharynx, and larynx are mainly affected, and are the site of violent pain, whilst the œdematous swelling in the face and eyelids develops early. Great increase of the eosinophile cells in the blood and diazo-reaction also point to trichinosis (Fr. Müller).

I have drawn attention to the clinical relationship of *dermatomyositis* and *sclerodermia*, and to the consequent difficulty in diagnosing them from each other. Mild cases may be confused with *muscular rheumatism*, but swelling of the muscles, discoloration of the skin, which is often present, and rise of temperature, etc., reveal the true character of the disease.

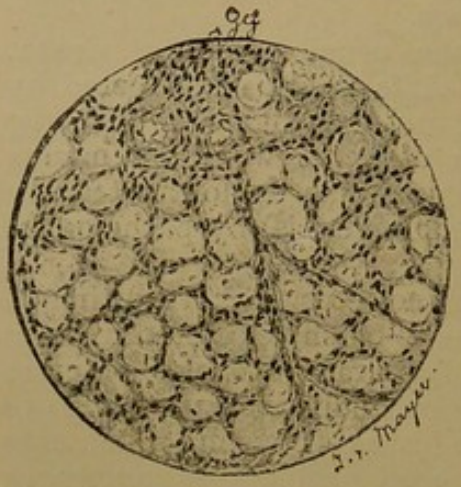

Fig. 237. — Interstitial myositis. Transverse section of muscle. (From a section stained by Wagner with alum-hæmatoxylin in Oppenheim's Collection.)

The symptoms of polymyositis are also closely allied to those of *polyneuritis*, and a combination of the two affections, a *neuromyositis*, is not uncommon, as Senator in particular has shown. Fajerstain also found that the nerves were implicated in a case which he examined post-mortem, and a case of Damsch's [1] also seems to belong to this group. The conditions, however, differ from each other in this, that in neuritis there is almost always tenderness of the nerves to pressure, sensory disturbances, and the paralysis is accompanied by RD., whilst the muscular swelling and the œdema are less prominent.

Syphilitic myositis may further correspond to polymyositis and may greatly resemble the disease under discussion (Herrick). It should be noted that myositis ossificans may have a relapsing febrile course, each attack being followed by the process of ossification. Some rare cases show (Schultze, Oppenheim-Cassirer [2]) that there are forms of polymyositis in which the affection terminates in progressive or permanent muscular atrophy.

In one of my cases the diagnosis of "akinesia algera," and in a second that of spondylitis or chronic rheumatism had been made elsewhere, and in none of my cases had the condition been previously recognised and correctly diagnosed. Apparently the disease is not a well-known one.

There is another form of myositis caused by over-strain or trauma, which is limited to *single muscles*. Strümpell, for instance, observed it in the lower extremities of an organist, who had to use the pedals

[1] "Festschrift Orth.," 1903; abstract *B. k. W.*, 1903. [2] *Z. f. N.*, x.

for hours at a time. I have seen it occur after an exhausting march. It would appear from Scheffer's [1] investigations, that over-fatigue may

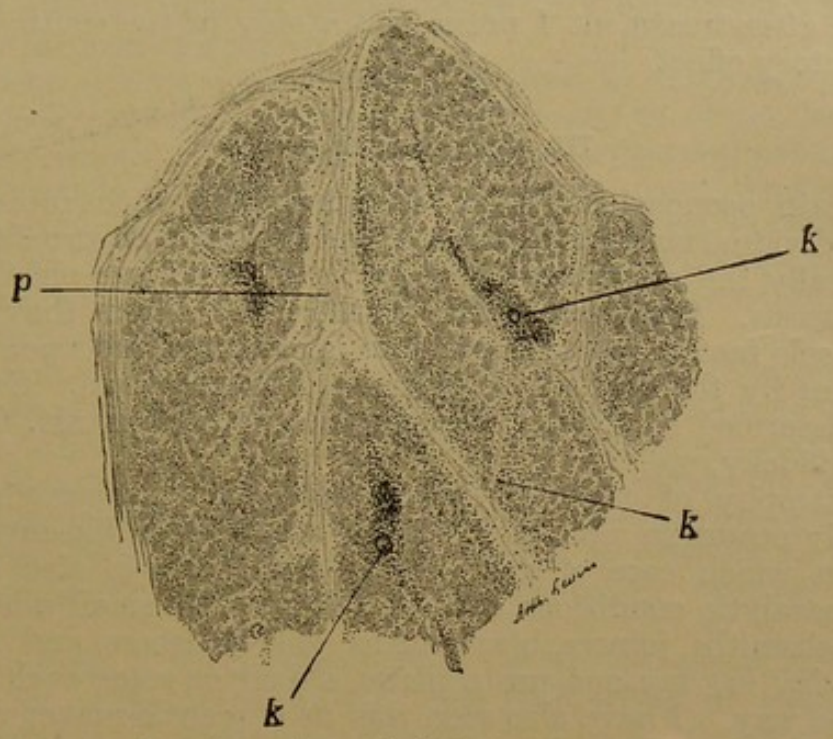

Fig 238.—Transverse section of muscle, excised during life, in acute polymyositis. Stain: alum-hæmatoxylin. *k* = round-cell infiltration. *p* = thickened perimysium. (Oppenheim.)

produce structural changes in the muscles. The prognosis of this localised form is favourable. The affection, however, often produces permanent

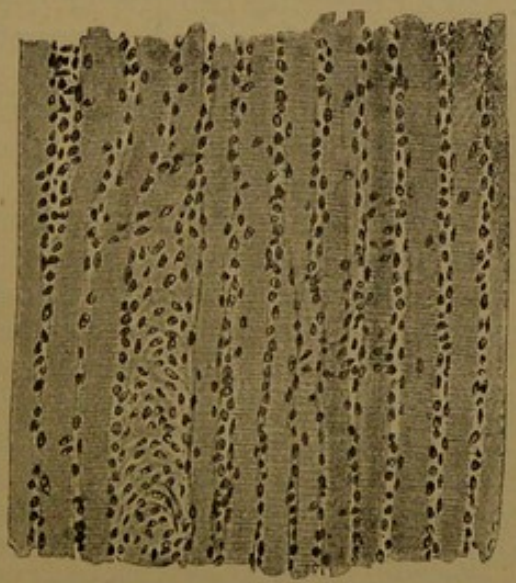

Fig. 239.—Longitudinal section from same case as 238. Stain: carmine-alum-hæmatoxylin.

induration and atrophy of the muscle, but it has no effect upon the general health. Hackenbruch [2] has thoroughly investigated this form, which is of special interest to surgeons.

Treatment is practically the same as in neuritis.

[1] *W. kl. R.*, 1903.

[2] *Beiträge z. klin. Chir.*, 1893.

In the severe cases above mentioned in which recovery took place, I made vigorous use of *diaphoresis*, followed later by thermo-massage and electricity. Wet packs also seemed to be beneficial in a previous case. For after-treatment I prescribed a stay in the south, which had a very salutary effect.

III. Occupation Pareses [1]

In certain occupations which are associated with prolonged *over-strain* of individual muscles, these sometimes undergo an *atrophic paralysis*. It is naturally the *small muscles of the hand* which are chiefly attacked by this paresis. Careful analysis of the cases shows that over-strain is not the sole factor, but that pressure is often exerted upon the nerves and muscles by the tool which is grasped in the hand. Cases of this kind are described by Berger, Remak, Leudet, Bernhardt, Oppenheim, Baraks-Doilideky, etc.

Thus atrophy of the first interosseous and *opponens pollicis* not infrequently occurs in women who iron,[2] and also in file-cutters, paper-glaziers (in which case a toxic factor—the lead—is also concerned). Atrophic paralytic conditions often develop in the small muscles of the hand in locksmiths, joiners, blacksmiths, gold-polishers, and other workmen who, day in, day out, use a plane, a drill, or some such instrument in the same way. I have also seen this in a mantle-maker who had to work with thick needles. I was able to trace a cutter's paralysis in the median nerve to forced continuous use of the scissors. Ulnar paralysis due to over-strain has been observed in oarsmen, and in glass-workers who have to lean the inner surface of the elbow upon a high table, also in wood-engravers (Bruns), bakers, and telephone operators (Menz). Raymond-Courtellemont (*R. n.*, 1904) describe neuritis of the nerves of the hand in coachmen. W. Salomonson found an occupation atrophy in the form of atrophy of the interossei associated with hypæsthesia in diamond cutters. Atrophy of the thenar eminence may even be due to prolonged writing or playing on the zither; atrophy of the *interossei* to cigar-rolling (Coester [3]). Paralysis and atrophy of the muscles supplied by the median may be a complication in milker's spasm. Steiner [4] observed neuritic symptoms in the median nerve in sand-moulders and in post-office officials who were occupied in sorting letters. Hoeflmayer describes occupation neuritis in the subscapular and axillary nerves in cabinet-makers and skin-dressers. Professional serratus paralysis in tailors' cutters is mentioned by Claude-Descamps.[5] In drummers we have a paralysis of the extensor pollicis longus, usually involving the small muscles which extend the end phalanx of the thumb; less frequently paresis of the flexor pollicis (Bruns,[6] Zander). Recent cases, however (Steudel, etc.), corroborate Düm's [7] view that in most of such cases there is merely inflammation and laceration of the tendon sheaths. I have seen atrophy of almost all the muscles of the hand or fingers in a man who worked a printing-press, and had to grasp the lever with his hand. A weaver whom I treated showed symptoms of paralysis of

[1] See literature in E. Remak (*loc. cit.*).

[2] In an ironer whom I treated, and who had for years suffered from traumatic paralysis of the median nerve, an occupation paresis developed in the over-strained ulnar nerve.

[3] *B. k. W.*, 1884.

[4] *M. m. W.*, 1905.

[5] *R. n.*, 1906.

[6] *N. C.*, 1891 and 1895.

[7] "D. milit. Zeitschr.," 1896.

the right triceps muscle, due to the fact that he had to extend the forearm about 20,000 times a day. I have also seen paralysis of the deltoid, supra- and infra-spinatus of similar origin in a railwayman who had to press down the arm of a signal.

Occupation paresis occurs much less often in the lower extremities, but some of the paralytic conditions in the nerves of the leg (peroneal and tibialis posticus) which occur in occupations carried out in a crouching position, such as digging potatoes (Zenker), transplanting beetroot (Hoffmann, etc.), peat-cutting (Kron), working a sewing-machine, belong to this group. In addition to overstrain, pressure exerted on the nerves by bending the knee is an injurious factor. Traction on the nerves may also play a part.

Occupation pareses are not infrequently accompanied by slight pain, paræsthesia, and mild sensory disturbances. The only explanation of this fact is that the pressure affecting the peripheral nerves has produced a neuritis. We cannot always definitely determine whether the muscular atrophy is merely a consequence of this neuritis or is the direct result of overstrain. But it seems to me that in addition to the *neuritic* form there is an occupation paresis of *myositic* origin. Bittorf [1] advocates this view.

The occurrence of this paresis is favoured by *alcoholism*, *lead-poisoning*, *marasmus*, etc. There are cases of this kind which we must regard as a toxico-professional paresis (Oppenheim [2]). In a lady whom I treated, the small muscles of the hand became atrophied when during her convalescence she began wood-carving.

In my experience also, occupational overstrain may so injure the nerves that some slight trauma (pressure, traction) may subsequently give rise to an acute onset of the paralysis. An acute anterior poliomyelitis in childhood is apt, according to our experience, to leave a predisposition to these forms of atrophic paralysis. Thus I have treated a man who on account of poliomyelitic paralysis of the right leg had to support himself with the right hand upon a stick which he grasped firmly ; this resulted in an atrophic paralysis of the muscles of the right hand, which almost entirely disappeared in hospital with care and electrical treatment.

These conditions have usually a chronic or subacute development. I have occasionally, however, seen an acute or relapsing onset (see above).

The *prognosis* of occupation paresis is essentially *favourable*. If the patient can take care of himself in time, and get rid of the injurious element, the affected muscles almost always recover. The disease appears only in very rare cases to be the starting-point of a progressive muscular atrophy.

From a *therapeutic* point of view, particular stress should be laid on complete rest and on sparing the affected muscles. The electric current may also be employed.

[1] *M. m. W.*, 1905.

[2] *B. k. W.*, 1891, and *A. f. P.*, xviii. This is confirmed by other writers, such as Guillain, Edinger, and Steiner.

The Neuralgias

Literature: Text-books by Erb, Eulenburg, Gowers, Seeligmüller, Pentzoldt-Stintzing, etc., and by Bernhardt, "Die Erkrankungen der peripherischen Nerven," 2nd edition, Wien, 1904. For further data see sections on the different forms of neuralgia.

General.—By neuralgia we understand severe pain which comes on in attacks and is felt in the main trunk of a nerve or in its branches. The patient usually traces the course of the pain with his finger, as in most cases it is either confined to one point or extends superficially from it. It is only in rare cases, where the terminal ramifications of the nerves are involved, that the pain is not felt along the line of the nerve, but is confined to certain points or small areas.

Neuralgia may occur at any age, but it is very rare in childhood, and most common in middle life. In Bernhardt's experience, men much more often suffer from neuralgia than women. *Puberty*, *pregnancy*, the *puerperium*, and the *climateric* temporarily increase the predisposition.

The causes are very numerous. One of the most important is the *neuropathic disposition* (Anstie). Individuals with hereditary and congenital nervous dispositions are most susceptible.

Exhausting illnesses, and loss of blood and lymph, may lay the foundation of neuralgia, and *anæmia*, *cachexia*, *senility*, and *arterio-sclerosis* are very favourable to its development. The importance as regards the nervous system of morbid processes in the vessels has recently been exhaustively discussed by Lapinsky.[1] Constipation may favour the onset of neuralgia. *Infective diseases*, both from their exhausting effect upon the organism and by introducing into the blood *toxic* products which directly injure the nervous system, may be the cause. Malarial neuralgia is undoubtedly due to a virus. This is the probable origin of the forms occurring after typhoid, smallpox, influenza, gonorrhœa, etc. I have seen a typical supra-orbital neuralgia appear in a boy during the incubation stage of measles and disappear with its eruption. O. Rosenbach describes neuralgia occurring during the commencement of typhoid. Within the last few years an epidemic form of neuralgia has occasionally been reported (Wille, Reilly [2]). It has for a long time been known thus to appear in numerous cases at the same time and place in the form of herpes zoster, and this is in accordance with the fact that Head has been able to trace an analogy also from the pathological point of view between this disease and acute anterior poliomyelitis (see section: herpes zoster). It is very doubtful whether a true neuralgia is caused by syphilis, as Fournier, Obolensky,[3] and others think. But a syphilitic or gummatous neuritis may for a considerable time present the characters of a neuralgia.

The etiological importance of many *poisons* has been established. Lead, mercury, copper, alcohol, nicotin, arsenic, etc., are among the causes of neuralgia. The form which develops in diabetes mellitus, less frequently in arthritis, may also be due to chemical poisons. This is supported by the fact that a relation is sometimes noted between the intensity of the pain and the amount of sugar eliminated, and that a gouty neuralgia may disappear immediately after an attack of gout.

[1] *Z. f. N.*, xiii. [2] *New York Med. Rec.*, 1899.
[3] *B. k. W.*, 1894. See also Hervouet, *Thèse de Paris*, 1903.

The neuralgia which occasionally appears in the course of nephritis may possibly have a similar explanation (Oppozzer, Berger), but Lapinsky[1] has been able to trace it to disease of the vessels of the nerves.

Chill, mental and physical *exhaustion*, and *emotional excitement* are also regarded as causes of neuralgia ; as a rule they act merely as exciting factors. The rheumatic diathesis favours its occurrence, and rheumatism and neuralgia may be combined. A few writers (Stevens) attribute an important part in the etiology of neuralgia to overstrain of the ocular muscles and anomalies of refraction.

Injuries of the nerves and their branches, especially gunshot wounds (Michell, Keen) and traction and compression by cicatrices, ulcers, aneurisms, varicose veins (Henle, Quenu[2]), often give rise to neuralgic pain, but it is extremely difficult in such cases to distinguish between true neuralgia and the neuritic-degenerative diseases of the nerves. Where some exciting cause in the periphery, arising from a cicatrix, a decayed tooth, inflammation of cell tissue, or an affection of a distant organ (uterus, floating kidney, nasal dysmenorrhœa, etc.) has given rise to reflex disturbances characterised as neuralgic, we are certainly justified in regarding the condition as a true (idiopathic) neuralgia. But when the affection develops in the area innervated by the trigeminus, we cannot always determine whether the local process has merely given rise to reflex pain, or whether an inflammatory or infective irritation arising from it has been transferred to the nerve.

The pain of neuralgiform character occurring in the course of organic nervous diseases, in tabes, syphilitic meningitis, tumours of the base of the brain and the spinal cord, etc., is to be distinguished from neuralgia.

Symptomatology.—The pain comes on in *attacks*, which may last from one or more minutes to several hours. But even during the attacks the pain is not continuous ; it consists of single jerks or throbs, which are described as stabbing, boring, tearing, burning, etc. It is not generally so rapid as lancinating pain, but lasts some seconds or minutes. In the intervals pain is completely absent ; it is only in exceptional cases that a dull sensation of pain persists, the attacks forming exacerbations of this. They come on spontaneously or are brought on by *movement* of the part of the body in which the pain is situated, by the effect of cold air, by taking food, by mental excitement, by coughing, straining, sneezing, etc.

The attack is often accompanied by *secretory*, *motor*, *and vasomotor* symptoms. Flow of tears and saliva, local secretion of sweat, contractions (*e.g.* facial spasm in trigeminal neuralgia), pallor, and still more often redness of the skin, are the ordinary accessory symptoms of the attack. Circumscribed œdema is less common. When the trouble is of long duration, swelling and infiltration of the skin and soft parts, and even of the periosteum and bones, may appear. Persistent œdema, permanent dilatation of the cutaneous vessels, erythema, etc., are occasionally observed. The relation of herpes to neuralgia calls for special discussion (see section : intercostal neuralgia). Loss of the hair, greyness, or other changes in its colour are uncommon symptoms.

Paralysis is not one of the symptoms of neuralgia, but the pain may give rise to restriction of the active movements and may thus cause a kind of pseudo-paresis. A simple atrophy of the muscles, due possibly

[1] *N. C.*, 1898. [2] *Traité de Chirurgie*, ii., and *Arch. de Neurol.*, xxxiii.

to deficient supply of blood, also occurs (Nothnagel). Some of the symptoms, especially those of motor excitement, are doubtless of reflex origin.

The pain is situated in the main trunk of a nerve or in some or all of its branches. At the height of an attack it may radiate to branches hitherto intact, or into other nerves. More rarely the corresponding nerve on the unaffected side is involved at the height of the paroxysm. A kind of *transfer*, or passing of the pain to the other side—spontaneously, or as the result of electrical or surgical treatment—occasionally appears as a transient symptom. I have not indeed observed this in true neuralgia. The skin is often hyperæsthetic in the affected parts, especially to light contact, whilst deep pressure will have a soothing effect. Slight diminution of sensibility (never anæsthesia) more rarely develops in the parts supplied by the affected nerve, and usually only in the later stages (Nothnagel).

In many cases *pressure points* are found in the course of the affected nerves, *i.e.* circumscribed spots at which pressure is felt as pain. These points, first described by Valleix,[1] usually correspond to the site at which a nerve branch emerges from a canal in a bone, or to a site at which it can be pressed against a solid tissue (bone, ligament). It is seldom sensitive to pressure throughout its whole course. Pressure points are also often found on the spinal column, corresponding to the origin of the affected nerve (Trousseau).

At the height of the attack *slowing of the pulse* and vomiting may occur, but these are very rare.

When the disease lasts for a long time the *general health* of the patient is apt to be affected. Loss of appetite and digestive troubles appear, the nutrition is affected, and there may be a considerable degree of *cachexia*. The *mind* is also threatened. Simple depression, excitability, disinclination for activity are ordinary consequences of neuralgia; these seldom develop into marked psychoses, but chiefly into delirium and melancholia (Griesinger's neuralgic dysthymia). In severe and intractable cases the pain not infrequently leads the sufferer to attempt suicide.

Some forms of neuralgia are characterised by qualities peculiar to themselves. The form which is due to *malaria* is distinguished by its *typical* course, and periodicity. The attack comes on at certain hours, ceases at a certain time, and returns after an interval of one to four days. Malarial neuralgia does not, however, always show this intermittent course, and simple neuralgia may also assume this type. I have found this specially so in the neuralgia of hysterics. There is also a *nocturnal neuralgia*, or a true *hypnalgia*, *i.e.* a neuralgia which only comes on during sleep, and is directly produced by it (Oppenheim [2]).

Malarial neuralgia affects mainly the *supraorbital*, *sciatic*, and *occipital nerves*. The neuralgia of *diabetes* usually attacks the *trigeminus*, especially the third branch, and the *sciatic*. Its *symmetrical* distribution is also characteristic. In gout the trigeminus and the sciatic are favourite sites for the neuralgia. The neuropathic disposition seems to exaggerate the tendency to neuralgia chiefly in the supraorbital and intercostal nerves. Influenzal neuralgia usually selects the supraorbital nerve.

The nature of neuralgia.—We have no definite knowledge as to

[1] *Traité des névralgies*, 1841. [2] *B. k. W.*, 1899.

the pathological basis of the disease, and we should therefore be justified in dealing with this chapter under the heading of functional neuroses. The fact that a neuralgia may persist for years, more than ten in fact, without producing any sign of an organic lesion of the nerves is evidence against an organic disease. On the other hand a slight *neuritis* may give rise to the symptoms of *neuralgia*, and some forms of neuralgia, especially of the sciatic nerve, give rise, even at an early stage, to symptoms which we are accustomed to attribute to neuritis. In any case we must, on account of the typical cases and forms of neuralgia, distinguish it from neuritis, but we must admit that the boundary is not a sharp one, and that there are cases in which we can hardly say with certainty whether we are dealing with neuralgia or neuritis. It is probable that the primary cause is *fine disturbances of nutrition in the nerve*, in its sheaths, in the nervi nervorum, and that these may under certain conditions develop into *neuritis and perineuritis*. It is not impossible also that the pain may in many cases originate from the roots and the central origin of the nerves, especially from the spinal ganglia and their equivalent Gasserian ganglia (see chapter on trigeminus and intercostal neuralgia). It is certain that the pain may arise from the sensory nerve tracts of the cerebrum and the sensory cortical centres.

The criteria which distinguish neuralgia from neuritis are as follows : In the latter the pain is usually continuous, although it may undergo exacerbations at times ; in neuralgia it comes on in attacks. In neuritis the nerve is sensitive to pressure throughout its whole or great part of its course ; in neuralgia there are merely tender points, or no tenderness to pressure of any kind. In neuritis there is sometimes swelling of the nerves, which is absent in neuralgia. In neuritis the pain is as a rule rapidly followed by symptoms of paralysis, atrophy, and anæsthesia ; in neuralgia these are entirely absent, or they appear only in the later stages and are never very intense. Neuritis is generally an acute, neuralgia more frequently a chronic disease.

With regard to the *differential diagnosis* of neuralgia, the following points may also be mentioned. The diagnosis of neuralgia must never be made before a thorough examination has excluded an organic disease, and the possibility that the pain is due to organic changes in the nerves or the central organs. Inflammatory processes and new growths in the region of the nerve roots are specially apt for a considerable time to simulate neuralgia. The diagnosis can only be established when symptoms appear which point to interruption of the continuity in the affected nerves, and especially to involvement of the anterior roots and the spinal cord. Symptoms of irritation which might have a reflex origin, such as muscular contractions, do not exclude neuralgia. Chipault,[1] who saw spasms of the abdominal muscles occurring in secondary affection of the lower dorsal roots extending from the meninges, would regard the disease, which he terms " radiculites meningopathiques," as one distinct from neuralgia, but the differentiation can only be practically carried out when signs of paralysis have appeared. For similar cases reported by Lortat-Jacob, etc., see the remarks in the chapter on sciatica. From the diagnostic point of view we should consider specially tabes dorsalis, tumour of the spinal or cerebral medulla, syphilitic meningitis, ulcers in the tract or in the neighbourhood of the affected nerves, aneurisms,

[1] *R. n.*, 1902.

and so on. Disseminated sclerosis may sometimes give rise to neuralgic pain; I have seen a case in which intractable trigeminal neuralgia was among the first symptoms of the disease. On the other hand one is apt too rapidly to diagnose neuralgia in *hysterical* individuals. Although a true neuralgia may develop in one suffering from this neurosis, there is merely as a rule a *pseudo-neuralgia*, *i.e.* pain which has either a *purely mental origin* or is produced by some peripheral excitement, so slight that only the morbidly affected mind would react to it with neuralgic pains: it is therefore, a *psychalgia*. This may usually be recognised from the fact that every mental change is capable of influencing the pain, of increasing it for a time, of allaying, producing, or abolishing it, according to the idea and the emotion to which it gives rise. It is very often evident that the pain comes on and increases under the influence of self-observation and disappears when the attention is diverted. These pseudo-neuralgias are also not so strictly confined to the nerve tracts; they appear in patches, have a diffuse distribution, affect *segments of one side of the body*, and are accompanied by corresponding symptoms of hysteria. The difficulty of diagnosis may, however, be increased by the fact that a true neuralgia may in predisposed individuals give rise to a reflex hemianæsthesia of the "hysterical" type; this, however, rarely occurs. I[1] have further been able to show that the points tender to pressure are not infrequently found in hysterical and neurasthenic individuals, and are due, not to a mental or general, but to a local hyperæsthesia which affects the nerves only.

The term of neurasthenic pseudo-neuralgia has been used (Jendrássik),[2] but the criteria given are essentially those which I had previously applied to psychalgia, although they were not otherwise sharply defined. The conception of neuralgia has been still further widened and has been used to include pain occurring at certain bony points or parts of the joints, which reveal no local alteration. These conditions are usually due to hysteria (see above). Other factors may, however, be at work. Remak and Bernhardt[3] describe pain located in the external condyle of the humerus, especially on the right side, which comes on chiefly during movement of the hand and fingers, and on pressure. This affection, termed *epicondyalgia*, is in no way allied to neuralgia, and may be due to overstrain of the muscles which arise from this part of the bone. It should probably be classed with the occupation neuroses. Periostitic irritative conditions possibly also play a part in the production of the pain (Remak).

Myalgia is as a rule distinguished from neuralgia by the site and distribution of the pain.

Course and prognosis.—Neuralgia may have an acute onset, may last for a few weeks and then permanently disappear. This is not precisely the ordinary course. It often persists for months and years, but may show remissions of considerable duration. In some cases the attacks of pain occur seldom and last but a short time, whilst the free intervals may extend over a long period. In others the attacks come on in rapid succession and the intervals are limited to a few hours or days.

The prognosis is comparatively favourable in recent cases with an acute onset, when the patient is young and in a well-nourished condition.

[1] *Journ f. Psychol.*, i. [2] *D. m. W.*, 1902. [3] *N. C.*, 1896.

The neuralgias which follow acute infective diseases are usually less intractable. The outlook is graver in cases where the constitution is weak, in conditions of exhaustion, in old age, in chronic poisoning, when the disease is of long standing and the pain of great intensity. A neuropathic heredity affects the prognosis of typical neuralgia unfavourably.

Treatment.—Thorough investigation and examination is the necessary preliminary in treatment. If the cause is deficient nutrition, loss of strength, or a bad state of the blood, *improvement in the general health* and nourishing diet may of themselves be sufficient to effect a cure. On the other hand the neuralgia may be due to too rich a diet, and an excessive use of alcohol and spices. Neuralgia very often appears when a sudden change has been made from a diet rich in albumen to a vegetable one. This fact should receive every consideration. The diet should be *mixed*, and not stimulating; it should be adapted to the existing condition of nutrition. In a few cases under my observation a careful *lowering diet* has been successful; in some other particularly obstinate cases, in which every method had failed, Weir-Mitchell treatment was efficacious. Cod-liver oil is particularly good for thin persons.

We need not insist upon the great importance of regulating the diet in diabetic and gouty neuralgia. Abrupt transitions and an excessively monotonous diet should, however, be avoided. Treatment of the uric acid diathesis by regulation of the diet and alkaline waters, etc., may be of use.

The susceptibility to rheumatic influences may be combatted by frequent washing with cold water and a mild cold-water cure. This treatment is also recommended for neuralgia itself, and is even regarded as the most efficacious method of treatment by Winternitz and Buxbaum, who specially commend the Scotch douche (see chapter on sciatica).

Regulation of the bowels is an important point in the treatment of neuralgia. Cold-water enema, rhubarb, and castor-oil are often more useful than nerve tonics and sometimes cure even longstanding neuralgia. If these are not successful, a course of treatment at Kissingen, Marienbad, or Homburg may have a good effect.

In persons of sedentary habits, the prescription of regular and sufficient exercise in the open air, cycling, riding, gymnastics, etc., may have a beneficial influence. Where exhaustion is the probable cause, absolute rest, and even prolonged confinement in bed, is indicated.

If the nerve is embedded in a cicatrix, or the pressure is due to an ulcer or callus, surgical interference is required

Recent neuralgia sometimes yields rapidly to *diaphoretic* treatment. Even in old and intractable cases, some of my patients have been satisfied of the beneficial influence of hot or vapour baths, or of hot-air treatment. *Local bloodletting* may also have excellent results in such cases.

Malarial neuralgia yields as a rule to quinine. Large doses of 1-2 grammes (15-30 grains) are usually necessary, but I always recommend that the individual reaction to the drug should first be ascertained by small doses, and that when it is given for a long period the condition of the organs of hearing and sight should be carefully observed. Quinine is best taken a half to one hour before the attack is due. Should it fail, arsenic may be efficacious. In anæmia *preparations of iron* and perhaps *iron* combined with *arsenic* should be given, provided that it does not

interfere with the taking of food. Tinctura ferri pomati, the liq. ferri albuminati, Blaud's pills, the tincture ferr. comp. of Athenstaedt, hæmatogen, sanguinal, etc., are preparations which may be recommended.

If there is a history of syphilis a course of antisyphilitic treatment, especially with iodide of potassium, is indicated, even should the neuralgia be apparently idiopathic.

If the cause is metallic poisoning, baths (especially sulphur baths), *diaphoretic treatment*, and saline aperients, along with nourishing food are frequently efficacious. The most important precaution is of course the avoidance of further poisoning by removal of the cause, change of occupation, etc. Indifferent warm springs, and carbonic-acid brine baths have occasionally been of practical service in other cases, especially in those of rheumatic origin.

For the pain *soothing ointments* of opium, belladonna (1 : 10), cocain (1 : 20-30 vaselin), chloroform (with ol. hyoscyami and ol. olivæ, in equal parts), anæsthesin (1·0 : 10·0 vaselin), bromokoll, etc., have been employed. They are not very helpful, but are harmless. *Counter-irritations* and *blisters* are much more efficacious and often curative in their effect, even in old cases. We may use veratrin, 0·5 : 20, aconitin, 0·5 : 20, concentrated hydrochloric acid, menthol, and guaiacol (a.a. 1·0 to absol. alcohol. 10·0), or *cantharides*, and *points de feu* (cauterisation with a hot button-cautery). We may also mention mesotan, which should be used with great caution, rheumasan, and glycosal. In slight and recent cases, mild counter-irritation, such as cantharides laid upon the painful points or even applied in the form of a line over the nerve by means of collodium cantharidatum, or the *papier Fayard*, is often successful. In severe and longstanding cases the cauterising iron is often an excellent remedy. It is sufficient to produce one or more superficial scars. The local application of *heat* in the form of hot fomentations, steam, sand-bags, sand-baths, the hot-air apparatus according to Tallermann and others, proves to be a soothing and not infrequently a curative measure.

In many cases cold has an alleviating influence. The momentary cooling of the skin by external applications of methyl chloride or ethyl chloride (Debove, Reddard) may have a palliative effect.

Electricity is an important aid to treatment. The galvanic current is specially recommended, particularly with the *stabile application of the anode*. The positive pole is placed upon the affected nerve, its point of origin, or upon a pressure point; the negative pole upon an indifferent site. It is advisable always to commence with weak currents, *e.g.* 0·5-2·0 milliampères, with electrodes averaging about 10 cm. square, and, if this does not soothe the pain, to increase the strength of the current. The current should be turned on and off by means of a rheostat, and should never be suddenly interrupted. The application should last 1 to 5 minutes, but a longer period, amounting even to 30 minutes, is also recommended. The faradic current may also be used, especially the *faradic brush*, which produces counter-irritation. The brush is applied directly over the point at which the nerve emerges, or upon a pressure point, or a double brush may be used—two brushes placed directly over the nerve—the current being increased to the limit at which the patient can bear it, even to 0 on the register. If tender points are found on the spinal column, it is advisable to bring them also under the

influence of the anode. I have seen excellent results from galvanic anode treatment in recent cases; in several recovery immediately followed 10 to 20 applications. In chronic and old-standing cases this treatment occasionally leads to improvement, but is usually a failure. In a few which proved particularly intractable, *cataphoric* treatment with cocaine or chloroform and a suitable electrode of diffusion proved successful.

Special electrodes should be used for this treatment, which are provided with a container for holding drugs. These electrodes will conduct the positive current. They are placed firmly upon the nerve, and a weak current of ½ to 2 milliampères is conducted through them.

Where this treatment fails, *static electricity* may be tried; the brush discharge-current and the spark-current are recommended. I have never seen this treatment fail in true neuralgia. The use of prolonged but very weak galvanic currents has proved beneficial in some cases, and so has the electric bath. On the other hand very strong prolonged currents, with the use of large electrodes (30-50-100 MA. and more, with electrodes of 200-500 qcm. duration of 15-20 minutes and even of an hour), are recommended, particularly by French writers (Bergonié, Dubois, Bordier, Delherm). It need hardly be mentioned that the greatest care is necessary, especially in treating the head. Of recent years, treatment with the D'Arsonval currents and with electric light or the Volta light has come into vogue, but as yet there is no conclusive evidence as to its value. In almost all the cases of true neuralgia in which I recommended this treatment, it was of no effect. Russian physicians, such as Gribojedow, report wonderful recoveries under the light treatment. The magneto-electrical method (systems of Konrad, Trüb, etc.) has received excessive praise. In the majority of my cases it has entirely failed but I have twice seen an obstinate neuralgia in old men (by chance both Russian officers) recover under this treatment. In one, opium was given at the same time, and in the other aconitin.

As to the *radium* treatment of neuralgia, we have as yet but little experience; I have used the substance for several years, but have as yet come to no definite conclusion, as my cures were usually induced in cases of psychalgia. The use of the X-rays is advocated by a few writers.

In psychalgia I [1] employ a method of treatment of my own, which by systematic exercises aims at diverting the attention of the patient from the site of the pain. I shall describe this method under the treatment of hysteria.

Massage is of great benefit in sciatica, and of very little in trigeminal neuralgia. Vibratory massage may also be included among the curative agencies. I have not much personal experience as to the effect of Nägeli's manipulations.[2]

Treatment by climate. Although we cannot attribute great success to this it may frequently be advisable to recommend residence at the seaside, in the high mountains, in warm and equable climates.

Drugs are prescribed to cure the disease, to alleviate the pain, or to deaden it by producing narcosis. Of the remedies which have proved useful in neuralgia, the following may be mentioned :—

[1] "Zur Psychotherapie des Schmerzen," *Therap. d. Gegenwart*, 1900.
[2] "Therapie von Neuralgien und Neurosen durch Handgriffe," Basel, 1894.

Quinine: Commence with small doses and increase these to 2·0-4·0 grammes (or 30 to 60 grains) a day, when the first dose is not effectual and the drug can be borne (but with care).

Arsenic: This is best given in the form of Fowler's solution—4 to 6 drops in a dose—or of arsenic acid in the form of pills, but we may also prescribe one of the waters containing arsenic (Levico, Roncegno). Other preparations, particularly the metarsenic acid anilid or atoxyl used subcutaneously (0·04-0·1) and cacodylic-acid salts and ferrous arsenate, have come into vogue of late years.

Sodium salicylate, 3·0-6·0 grammes (45-90 grains) per day in aqueous solution.

Salol in similar doses. *Salipyrin*, 0·5-1·0 gramme (7½-15 grains).

Bromides. *Iodide of potassium*, 0·3-0·5 gramme (4½-7½ grains) several times a day.

Ol. Therebinthinæ, 5-10-15 drops in gelatine capsules.

Tinct., gelsemii, 5-15 drops several times a day.

Aconitin nitr., 0·0001 (of a solution of 0·05 gramme in 25 grammes of distilled water; one drop may be given ten times a day, increasing to 8 drops per dose). Cannabine tannate 0·2-0·3 gramme.

Of the newer drugs we may mention: *antipyrin* 0·5-1·0, *phenacetin* 0·5-1·0, *lactophenin* in the same dose, *antifebrin* 0·2-0·5, *analgen* 1·0, *exalgin* 0·1-0·2 in alcoholic solution, *methylene blue* in doses of 0·01-0·08 subcutaneously, or internally 0·1 (1·0 the largest daily dose) with addition of nutmeg in gelatine capsules, *butylchloral* (7·5, glyc. 20.0, aq. dest. 130·0, a tablespoonful every ten minutes), *pyramidon* (0·2-0·5-1·0), *aspirin* (0·5-1·0), etc. etc.

None of these drugs can be absolutely depended upon; their use is always of the nature of an experiment, and the great majority of them require careful supervision in order to prevent poisoning. I would specially recommend the greatest caution in the use of phenacetin and antifebrin. I have seen a great deal of good from the use of *pyramidon*, occasionally even in severe deep-seated cases. I can also recommend *aspirin* as being very often efficacious. I commence with doses of 0·5, and when the effect is inadequate I rapidly increase it to 1·0 (3 or 4 times a day). I have often found *trigemin* (0·3-0·5) beneficial. I have little experience of *kryofin*. Dana describes a very complicated treatment by drugs, in which he uses strychnine, iodide of potassium, and other remedies, along with rest in bed.

Prolonged use of arsenic in small doses should always be tried in intractable cases. In severe cases all these drugs fail, and the intensity of the pain necessitates the administration of *morphia*. This is most efficacious in subcutaneous injection into the neighbourhood of the affected nerve, and morphia is said to have not merely an alleviating, but a curative effect in neuralgia. Care should be taken not to give the syringe into the hands of the patient or his relatives, and not to repeat the injection frequently at short intervals. When morphia is not sufficient, a combination of morphia and atropine has been recommended. Cocaine, which has lately been greatly lauded by French writers, especially by Brissaud and Verger,[1] or the less poisonous allied preparations (eucain, tropococain, etc.), may also be tried.

Ether and bichloride of methyl are recommended in the form of

[1] *Rev. de méd.*, 1904.

sprays, but there is not much to be expected of this method in true neuralgia.

Nor can we promise much from the subcutaneous use of osmic acid (1 to 2 drops to a syringe of a 1 per cent. solution) and carbolic acid. Lately, however, good results have been reported from osmic acid by Anschütz, Murphy, and others. This remedy should be brought as nearly as possible into direct contact with the affected nerve.

There has come into use, particularly within the last few years, a method introduced by Schlösser[1] and Lange,[2] in which a quantity of fluid containing alcohol or small doses of a narcotic drug (1 ‰ β-eucaine or 2 ‰ tropococaine in 8 ‰ ClNa-solution, or the latter alone) is injected into the nerve or its immediate neighbourhood. For the advantages and technique of the treatment see the chapter on trigeminal neuralgia and sciatica. Further reports as to its benefits, mostly favourable, are contributed by Opitz,[3] Alexander,[4] Umber,[5] Rüdiger,[6] Krause,[7] Strümpell-Müller, and others.

We must also point out some of the older methods which bring the soothing drugs into direct contact with the nerve-roots. These include the so-called "rachicocainisation" or injection of cocaine into the subarachnoid space of the spinal cord by a method corresponding to Quincke's between the laminæ of the lower lumbar vertebræ, and epidural injection through the sacrococcygeal ligament after Sicard and Cathelin.[8] By these means very small quantities—about 0·001 to 0·01 or 0·02—of a sterilised solution of cocaine or tropococaine are introduced. Cathelin and Strauss[9] both think that they have observed a sedative effect from the use of physiological salt solution (7·5 sodium chloride to one litre of water), and they recommend its use.

French and Italian authors in particular have reported palliative and curative results obtained by these methods in neuralgia of the spinal nerves (Widal, Sicard, Hallion, Marie-Guillain, Souques, Achard, Pasquier-Lévi, Suffit-Delille, Marri, Durand, Cavazzani, and especially Cathelin). In their experience, epidural injection is undoubtedly the least dangerous method.

Strauss follows Cathelin in recommending the use of a platin-iridium needle of 6 cm. in length and 1 mm. thick, which is heated before each puncture. The patient lies on his left side, with the legs drawn up so that the sacrum is prominent and the ligament between the sacrum and the coccyx is made tense; two small bony prominences which form a triangle, with the last sacral segment lying above them, bound the space formed by this membrane and may serve to localise it. This minor operation should of course be carried out with strict antisepsis.

The sedative and curative effect of Schleich's infiltration-anæsthesia has been lately much praised by R. Bloch and Alexander. Peritz also reports successful results from it.

It is sufficient here to draw attention to the injection of air into the neighbourhood of the nerve, which has been specially recommended for sciatica by Cordier (*Lyon méd.*, 1902), Vigue (*Thèse de Lyon*, 1902), etc.

The ultimate resource of treatment in neuralgia is *surgical intervention*—at least it should be so. Krause[10] indeed warns us against undue delay, and thinks the knife should be applied rather than that

[1] *B. k. W.*, 1906.
[2] *D. m. W.*, 1905.
[3] *Klin. Therap. W.*, 1907.
[4] *Z. f. phys. und diat. Th.*, 1906-1907.
[5] *Ther. d. Geg.*, 1906.
[6] *Med. Klinik.*, 1906.
[7] *D. m. W.*, 1906. See also the work of Schultze, Schlösser, and others in the *D. m. W.*, 1907 (18), and Ostwalt (*B. k. W.*, 1906), Windscheid (*D. m. W.*, 1907), and the discussion following.
[8] "Die epiduralen Injekt. durch Punkt. des Sakralkanals." German by Strauss, Stuttgart, 1903.
[9] *B. k. W.*, 1903.
[10] *A. f. kl. Chir.*, Bd. xliv.; also *D. m. W.*, 1893; *M. m. W.*, 1901, and "Die Neuralgie des Trigeminus," etc., Leipzig, 1896.

the patient should be led into the clutches of morphinism. Bruns goes still further and recommends that severe cases should immediately be handed over to the surgeon ; in this he certainly far overshoots the mark.

Simple section of the nerve—neurotomy—has almost always merely a transient effect. This treatment has therefore been practically abandoned. *Neurectomy, i.e.* excision of a larger portion of the nerve, is much more helpful. *Nerve-stretching*, first used by Billroth and Nussbaum, has also had a curative effect in many cases. But all these methods are uncertain, and relapses are to be expected in the greater number of cases.

Dege (*D. m. W.*, 1906) reports the ultimate results of division of the trigeminus as shown by Krause's numerous cases. According to him the operation as a rule merely gives immunity from pain for a period between ½ to 3½ years, and the duration of this period differs according to the branch of the nerve affected.

The method of nerve extraction or exairesis advocated by Thiersch [1] and Witzel, in which the nerve is gripped by a special clamp and twisted out with all its branches, has been very succsssful, although relapses are by no means excluded by this method (Angerer [2]).

In neuralgia of the trigeminus many cases have been treated by resection of the branch of the nerve at the base of the skull, and lately the Gasserian ganglion itself has been excised along with the adjacent portion of the nerve (W. Rose,[3] F. Krause,[4] Hartley, Horsley, Doyen, Keen-Mitchell,[5] Keen-Spiller, Lexer,[6] Hutchinson,[7] Friedrich, Cushing,[8] Renton, Holmgren, Williams, Tiffani, Bartlett, Murphy-Neff, etc.). The method of subdural operation advised by F. Krause has specially come into vogue, and he has also the credit of being the first to remove the ganglion (1893). This bold operation has generally been most successful in accessible cases and has often been the means of effecting a cure. Krause has carried it out twenty-nine times in patients between thirty and seventy-two years of age (Horsley even in an old man of eighty), and has had only a few cases of death as against a number of complete recoveries, which by lasting for several years—seven or eight—have proved to be definite. This number had increased in 1906 to forty-nine. Even in 1902 Türck was able to collect from the literature 201 cases with 156 permanent recoveries. The after-effects of removal of the trigeminus were in Krause's experience quite insignificant. He never found it give rise to neuroparalytic keratitis, whilst others, *e.g.* B. Keen (three out of whose eleven operations were fatal), Frazier, and Spiller saw this condition and loss of the eye follow the operation in several cases. Oculo-motor paralysis (Friedrich, Krause ; the latter once saw total ophthalmoplegia follow the operation), facial paralysis, transient aphasia, and other symptoms, such as pulsating exophthalmos (Tertsch) have occasionally been caused by the operation. Transient contraction of the pupils, attributed to the sympathetic, and other oculo-pupillary symptoms, are more frequently mentioned. Moreover, other surgeons, such as Friedrich, Garrè, Keen, and Cushing, have in

[1] *Verhandl. des xviii. Kongr. der D. Ges. f. Chir.*
[2] *A. f. kl. Chir.*, Bd. liii.
[3] *Lancet*, 1891.
[4] *D. m. W.*, 1893, etc.
[5] *Phila. Med. and Surg. Rep.*, 1894.
[6] *A. f. kl. Chir.*, Bd. lxv.
[7] 'The Surgical Treatment of Facial Neuralgia," London, 1905.
[8] *Journ. Amer. Med. Assoc.*, 1905. See also Sherman, *ibid.*, 1904 ; L. Prat, *Thèse de Paris*, 1903 ; Bardescu, *Spitalul*, 1904.

contrast to Krause, observed relapses which they ascribe to regenerative processes. In two of my patients who underwent this operation, relapses soon occurred, which I attributed to the fact that the neuralgia was localised in the segments of the trigeminus which lay on the proximal side of the ganglion. Perthes ascribes the regeneration in his case to a remaining portion of the ganglion. Hudovernig describes persistence of the pain after excision of the ganglion. Krause's operation has been modified by Dollinger, Lexer [1] and others ; the latter reports over fifteen cases upon which he operated, mostly with favourable results.

Section of the sensory root of the trigeminus between the ganglion and pons has been recommended and successfully carried out by Spiller-Frazier (*Univ. of Penn.*, 1901, and *Journ. Amer. Med. Assoc.*, 1904) ; Gehuchten (*R. n.*, 1904) also advocates the operation, which seems to be a rational one. As to Bardenheuer's neurinsarcoclesis (*M. m. W.*, 1903 ; *Z. f. Chir.*, Bd. xlvii.) see the following section.

Ligature of the carotid has been performed, but it is hardly to be recommended, nor is removal of the superior sympathetic ganglion as carried out by Chipault (Cavazzani, Poirier, Delbet, etc.).

Finally, it should be remembered that an attempt has been made to cure neuralgia in the spinal nerves by section of the corresponding sensory roots within the spinal canal (Bennet, Abbé, Chipault, Demoulin,[2] Horsley, Prince [3]). Nothing can be definitely deduced from the reports at our disposal, as to the indications for and the value of this procedure. Only so much is clear, that operation upon the spinal cord is dangerous in a high degree, as in several cases it has been followed by Brown-Séquard paralysis.

Neuralgia of the Trigeminal Nerve (Tic Douloureux, Prosopalgia, etc.).

Of all the nerves the trigeminus is most liable to neuralgia. Among 717 cases which Conrad [4] collected, it was attacked 239 times. This may be due to the extensive area which it innervates, to the abundant ramifications of the nerve, to its exposed position, and to its course through numerous narrow bone caniculi. The etiological factors above mentioned also apply throughout to neuralgia of the fifth nerve. Here *neuropathic heredity* plays a particularly important part, and in many cases it is the only assignable cause of the trouble. Malarial neuralgia and that following acute *infective diseases* (especially influenza) also affect the trigeminal region, particularly its first branch. This is true also of the toxic forms. Whether there is a rheumatic neuralgia of the trigeminus, such as Leube describes, or whether the chill acts only as the exciting cause, we cannot definitely say. Acute articular rheumatism may also assume the guise of neuralgia.

The condition frequently arises from morbid processes in the mouth, the nasal and frontal cavities, especially from *carious* teeth, abnormal formation of the teeth, exostoses on these or on the alveolar process, infective products which make their way from a carious tooth into the nerve (Conet), abnormal condition of the tooth-pulp (Boennecken [5]), from chronic catarrh, swelling, and new growths of the *nasal mucous*

[1] *A. f. kl. Chir.*, Bd. lxv. [2] *Gaz. des hôp.*, 1895. [3] *Br.*, 1901.
[4] *Inaug. Diss.*, Bonn, 1889. [5] *B. k. W.*, 1893.

membrane, or from *catarrh of the frontal sinus*. I have sometimes seen it develop after operations within the naso-pharynx. Swelling of the periosteum within the bony canals through which the branches of the fifth nerve pass, dilatations and inflammatory-degenerative processes in the vessels which accompany it may be the cause. Moos [1] found trigeminal neuralgia to be due to an exostosis on the posterior wall of the external auditory meatus. The neuralgia which not infrequently occurs in the alveolar process of toothless jaws (névralgie des édentés) is attributed to an ostitic process in the alveola, by which the nerve endings are irritated. According to Jarse, extraction of a tooth may give rise to neuralgia.

An ocular disease (conjunctivitis, iritis, glaucoma, anomalies of refraction) may also bring on the neuralgia; it is less often due to a catarrh of the middle ear. In a few of my cases *prolonged stay in an overheated room* has been blamed.

Senile and arterio-sclerotic neuralgia most commonly affect the trigeminus.

In a few cases aneurism of the internal carotid was the cause (Romberg).

Neuralgia of the fifth nerve is hardly ever bilateral (except in diabetes), very rarely affects all three branches of the nerve, and as a rule involves one or two, or even one twig only of these branches. Neuralgia originally limited to a small area supplied by one branch may later extend to several others, and *vice versâ*; it may also leave an area and pass over to other nerves, but this is not usual. The pain is generally of *great severity*, so intense that robust men describe it as unbearable. It is compared to the piercing of a hot wire, or the boring of a pointed knife. The patient presses his hand against his cheek, avoids any movement of the facial muscles, and is entirely under the domination of the pain. Its intensity, however, varies with the individual and the time. At the height of the attack, the pain may radiate into other branches and even into other nerves (irradiation). The throbs of pain may come on singly or may be constantly repeated during several minutes or even during several hours. This is usually associated with a *flow of tears* from the eye of the affected side, sometimes also with increase of the *nasal secretion* or a *flow of saliva*.[2] *Spasms of the facial muscles*, less often of the masseters, *redness of the face*, *œdematous swelling*, chemosis, or even hæmorrhage, *e.g.* in the gums, have been observed at the height of the paroxysm. When it is of long duration diffuse swelling of the skin, of the soft parts, and the periosteum, may also develop. Spasm of accommodation, deafness, narrowing of the field of vision, hallucinations of taste, mental disorders (Griesinger, Laquer,[3] Mondino, Krafft-Ebing) are mentioned among the rare complications of neuralgia. Herpes is more common, especially the frontal form; it may also affect the conjunctiva and result in inflammatory, even destructive processes in the eye. It seldom extends to the mucous membrane of the mouth and tongue. Cases in which neuroparalytic ophthalmia occurs in the course of the disease should not be regarded as pure neuralgia.

[1] *B. k. W.*, 1884.

[2] Trendelenburg (*D. m. W.*, 1903) describes a case in which at the height of the attack 10 gr. of saliva was ejected from the right Wharton's duct into the arch.

[3] *A. f. P.*, xxvi.

The changes in the colour of the hair already mentioned are comparatively frequent in facial neuralgia. Facial hemiatrophy is rare.

The *first branch* is particularly often affected (*ophthalmic neuralgia*) —according to Bernhardt in two-thirds of all the cases—and in it chiefly the *supraorbital nerve* (*supraorbital neuralgia*). The pain is situated above the eye and extends as far as the margin of the hair, the coronary suture, and even as far as the parietal region. The tender point is found at the supraorbital foramen. In malaria the neuralgia is so often localised in this nerve and is of such a typically intermittent nature that this has been specially termed the *intermittent larval* form. Influenzal neuralgia also affects mostly the supraorbital nerve. According to Seeligmüller supraorbital neuralgia due to affection of the frontal sinus is also characterised by a typical course. In some cases the whole area of the first branch is affected ; the pain radiates into the eye, the eye-lids, and towards the nose, and pressure points are to be found on the upper eyelid, at the canthus, and the nose. In neuralgia of the first (sometimes also of the third) branch, a painful point is occasionally discovered on the parietal eminence.

There is a form of neuralgia which is confined to the eyes (*ciliary neuralgia*) : the intense pain is localised in the eye or behind the eyeball ; there is a lachrymation, redness of the conjunctiva, photophobia ; and there may also be an affection of the optic nerve. This is specially observed after diseases of the conjunctiva and cornea, in glaucoma, after injuries or over-strain of the eye, and in anomalies of refraction.

Neuralgia of the *second branch* (*supramaxillary*) is situated chiefly in the *infraorbital nerve ;* its site of emergence at the infraorbital foramen is the most constant pressure-point. Not infrequently the alveolar or superior dental nerve is affected alone. The pain is felt in the upper jaw, or deep in the antrum of Highmore. If the whole branch is involved in the neuralgia, the pain is felt in the cheek, nose, upper lip, the region of the malar bone, and the temple. Pressure points are then usually present on the temple—at the anterior end of the temporal muscle—on the malar bone, where the malar branch emerges on the gums of the upper jaw, etc. The naso-palatine branch is on the whole very seldom affected.

Neuralgia of the *third branch* (*inframaxillary*) gives rise, if all the branches are affected, to pain in the lower jaw, in the tongue, in the region of the chin and temple, and in the ear. It is usually confined to the inferior alveolar nerve. Pressure-points are accordingly found at its site of entrance into the glenoid canal and the site of emergence of the mental nerve Neuralgia may also be limited to the lingual nerve.

There are slight and severe forms of trigeminal neuralgia—some of which are completely cured in a few weeks, and others which last for many years, even for tens of years. We may thus distinguish between acute and chronic forms of this neuralgia. I have treated cases in which the attacks appeared every year, generally at certain times, and again disappeared in a few weeks or months. Atmospheric conditions seem to be important. There are some persons who suffer from attacks only every second year. In the most severe cases the pain is so violent that when morphia loses its effect and the operations, to which the patients most willingly submit, prove unavailing, suicide puts an end to life and suffering. This, however, has been extremely rare in my

experience. These severe neuralgias, which Trousseau terms *névralgies épileptiformes*, occur specially in old age. They are usually associated with loss of strength, as the amount of food taken is reduced to a minimum on account of the exacerbations of pain which are caused by eating.

The pain comes on spontaneously, but is particularly apt to be excited and increased by *movement* of the facial and masseter muscles, and therefore by speaking and eating. Very many of my patients complained that they could not put a handkerchief to the nose or touch the moustache without being afflicted with most violent pain. On the other hand firm pressure is often soothing. Mental excitement may also produce the pain.

The *diagnosis* is usually a simple matter. Ordinary toothache does not follow the line of the nerve, and it comes from a decayed tooth or from a definite point of the jaw, at which the pain-points are found, instead of at the site of emergence of the nerve. I have, however, known many cases in which a masked dental trouble, overlooked by the dentist, caused pain in the corresponding branch of the trigeminus, then in the whole area of the nerve, and in rare cases also in that of the other side, which only disappeared definitely after the condition of the tooth had been attended to (extraction, stopping). Zang, Hesse, and others describe similar cases. I must emphasise the fact that some affections of the teeth may be overlooked, even by experienced and celebrated dentists. In doubtful cases the aid of the X-rays should not be neglected. Periostitis and diseases of the bone are associated with pain of diffuse extent and sensitiveness in the whole area of the parts affected.

As to the hysterical and neurasthenic pseudo-neuralgias, the points mentioned on p. 554 with regard to differential diagnosis may be referred to. It should be specially noted that these algias are not confined to the area of distribution of a nerve or of a branch of a nerve, but are indefinitely localised, are often bilateral, and extend beyond the area of the trigeminus. Here also the alleviating influence of mental diversion is particularly marked. And further, speaking and eating do not give rise to the pain, but as a rule soothe or dissipate it.

It may be difficult to determine whether the neuralgia is real, idiopathic, or symptomatic. Sclerotic processes at the site of emergence of the trigeminus, tumours in the region of this nerve, in the neighbourhood of the Gasserian ganglion, aneurisms of the carotid, etc., may for a time be masked by the symptoms of neuralgia. Later, however, other signs, viz., pain within the skull, vascular murmurs in aneurisms, symptoms of brain pressure, paralysis of the cranial nerves in tumour, etc., etc., almost always appear, which reveal the primary disease. A few cases have been known in which the cause of a disease which appeared to be trigeminal neuralgia was discovered to be a tumour, such as a cholesteatoma, on the Gasserian ganglion or in its neighbourhood (Romberg, Little, Schuch, Krause-Benda, Spiller, Hagelstam, Lexer, Verger et Cardenac,[1] Hofmeister-Meyer[2]). I have seen cases of cerebellar tumour or neuroma or fibroma of the auditory nerve, in which the trigeminal neuralgia preceded the development of the other symptoms for a considerable time. In one case a calcareous concretion, which pressed upon the supraorbital nerve, gave rise to the neuralgia. Recent investigations have also revealed changes in the branches of the trigeminus

[1] *R. n.*, 1905; see also discussion here on differential diagnosis. [2] *Z. f. N.*, xxx.

or in the Gasserian ganglion in simple neuralgia. Thus Dana [1] found a disease of the vessels of the nerve, Putnam,[2] sclerotic processes in the nerve, Horsley, Rose, Saenger,[3] Krause, Keen, Spiller and Schwab,[4] sclerotic and degenerative changes in the cells and fibres of the Gasserian ganglion. But Krause rightly points out that the significance of these conditions is open to question, as they were found in individuals in whom the trigeminus had been subjected to many surgical operations. Coenen [5] came to the same conclusion; he examined the ganglia extirpated by Lexer and found them unaffected unless operations had taken place upon the peripheral branches. Vascular changes are certainly not generally the cause of neuralgia, but are perhaps a result of it, as, according to our own experience (and to the observations of Thoma,[6] Dehio, and others), arterio-sclerosis may develop in consequence of prolonged vaso-motor disturbances.

There is a form of neuralgia which is limited to the nerves of the scalp (cranio-neuralgia), and which, as I have seen in several cases, may be associated with *alopecia*.

Otalgia (tympanic neuralgia) is on the whole a disease of which we know little. This neuralgiform pain in the ear occurs in diseases of the pharyngeal mucous membrane, the teeth, and the temporo-maxillary articulation. It may apparently also develop on an infective basis (B. Baginsky,[7] Kaufmann).

Neuralgiform pains without any organic cause occur also in the mastoid process (Schwartze), but it is questionable whether they should be regarded as neuralgia. The auricular branch of the vagus nerve is usually the site of the pain. In cases of this kind which I have seen, hysteria was the cause, and the pain promptly yielded to suggestive treatment.

Treatment.—Treatment should be preceded by a thorough examination of the teeth, the bones of the face and jaw, the nose and the eye. Usually, before coming under treatment, the patient has attempted to arrest the pain by having the teeth, which are often quite sound, extracted. In any case it is advisable to remove decayed teeth, if they are sensitive to pressure or tapping, and especially if we can bring on an attack by touching or shaking them. I have seen a case of trigeminal neuralgia, associated with spasms in the facial, masseter, and cervical muscles, which disappeared after the extraction of a carious tooth. If the teeth are sound, exostoses or periostitic processes on the jaws may be the cause. The neuralgia of toothless persons has sometimes yielded to resection of the alveolar process.

Chronic catarrh, or swelling of the nasal mucous membrane, requires local treatment. Two of my patients stated, however, that they only suffered from severe neuralgia after the nose had been examined or treated with instruments.

In neuralgia arising from the frontal sinus, the *nasal douche* (with warm water, weak solution of salt, potassium chloride, or boracic acid, and possibly with the addition of cocaine) is specially recommended. Errors of refraction should be corrected by glasses.

It is sometimes possible, as shown by Gussenbauer's [8] experience, to cure the neuralgia by giving aperients and cold-water enemata.

[1] *Journ. Nerv. and Ment. Dis.*, xvi.
[2] *Boston Med. Journ.*, 1891.
[3] *N.C.*, 1895.
[4] *Journ. Nerv. and Ment Dis.*, 1903.
[5] *A. f. kl. Chir.*, Bd. lxvii.
[6] *A. f. kl. M.*, Bd. xliii.
[7] "Eulenb. Realenzyklop.," xiii.
[8] *Prag. med. Wchnschr.*, 1886.

Otherwise treatment of trigeminal neuralgia should follow the principles laid down on pp. 555 *et seq.* Electricity is a valuable aid. Where direct treatment (galvanic, faradic, static, cataphoric) of the affected nerves is unsuccessful, galvanisation obliquely through the skull (corresponding to the position of the Gasserian ganglia) or of the cervical sympathetic should be tried. A pressure-point is occasionally found on the spinal column, on the highest cervical vertebra, and in such cases the application of the anode to these parts may be beneficial. In a few instances prolonged application of the galvanic current, continued for an hour, has been successful. Frankl-Hochwart [1] has used the double-brush electrode with benefit in trigeminal neuralgia. Electrical treatment, however, is very often a failure. The use of electric-light baths and the galvanic light have not proved absolutely successful. The magneto-electrical method has failed in the great majority of my cases, but in a few cases it appeared to give a favourable direction to the course.

With regard to drugs, salicylate of soda, quinine, arsenic, and iodide of potassium have proved most successful. Cocaine dropped into the conjunctival sac, or painted in a 5-10 per cent. solution over the nasal mucous membrane, may soothe the pain. *Butylchloral* has sometimes a good effect even in small doses of 0·1-0·2. Inhalation of amylnitrite (two to four drops on a handkerchief) during the attack may sometimes arrest it. Aconitin, narcein, napellin, neurodin, extr. stramonii. bromidia (one to two teaspoonfuls), citrophen, trigemin, and many other drugs have been recommended. Among the latest, *pyramidon* has been very specially advocated. Next to it *aspirin* has done me the greatest service. In some very severe cases, in which operation had already been resolved upon and the patient had meditated suicide, this drug, in doses of 1·0, three to four times a day, was the means of cure in one instance and of prolonged improvement or complete intermissions in many others. Trousseau has in the most intractable cases used quinine and opium in the fullest doses (even 8-10 gr. a day). Schleich advocates the subcutaneous local application of a solution of 0·1 cocaine, 0·02 morphia, 0·3 sod. chlor., and 100 aq. dist.

Of recent years Schlösser [2] and subsequently Ostwalt [3] have published important results from alcoholic injections made at the site of emergence of the trigeminal branch at the base of the skull. They use a syringe of special construction, with which they penetrate behind the alveolar process of the upper jaw along the great wing of the sphenoid to the foramina of the base of the skull. Ostwalt gives a description of the very difficult technique. The fluid injected is composed of 80 per cent. alcohol with 0·01 cocaine or stovaine. Recovery took place in some cases after two to four injections. But it is generally necessary to repeat the treatment within a few months or years.

A case first cured by Schlösser, which came subsequently under my care, proved to be one of psychalgia.

Compression of the carotid may arrest the attack. Massage of the

[1] *Wien med. Bl.*, 1888, and *Z. f. k. M.*, xvii.

[2] "Bericht über die xxxi. Vers d. ophth. Ges. Heidelb.," 1903, Wiesbaden, 1904; also *B. k. W.*, 1906.

[3] *B. k. W.*, 1906; see also Laporte, *Thèse de Paris*, 1905; Brissaud-Sicard-Tanon, *R. n.*, 1907.

cervical sympathetic (Rossander),[1] Nägeli's method, etc., have not produced any noteworthy results.

Finally, trigeminal neuralgia is the chief field for *operative* treatment. Surgical treatment is indicated when the other and less radical measures fail. We have then to consider the advisability of *neurectomy*, which results more often in remissions than in recovery, of nerve *extraction*, after Thiersch, which in the judgment of many surgeons (Krause alone declines to follow this method) is of the greatest service, and finally in the most intractable cases, and in those where the symptoms point to involvement of all the branches and to a high position of the disease, of *intracranial resection* of the nerve or of the *Gasserian ganglion* after Krause and others. We have already alluded to the results of this treatment and to its dangers (p. 560). Conclusions as to the methods are given in the writings of Vogt, Schede, Angerer, Lexer, Frazier, Hutchinson, Cushing, and especially in the monograph of F. Krause.

Bardenheuer takes the nerve branch out of the bone caniculus, which he chisels out, and makes for it a soft resting-place upon the muscle. He has found this successful, and so has Grabowski.

Occipital Neuralgia

Of the four superior cervical nerves, the great occipital is most often affected independently. Occipital neuralgia is, however, much rarer than trigeminal neuralgia. Out of 15,000 cases of neuralgia there are, according to Remak, some fifty of the occipital form. The other branches —the small occipital, the great auricular, the subcut. colli, and the supraclavicular are still less often affected. I have treated several cases of this kind in which the neuralgia involved the whole area supplied by these nerves, and in which pressure-points were found not only at the point of emergence of the great occipital—midway between the mastoid process and the highest cervical vertebræ—but also at the posterior margin of the sterno-mastoid muscle, just where the nerve passes round it. According to F. Krause,[2] occipital neuralgia frequently extends to all these nerves.

The pain is often limited to the great occipital, but is then more frequently bilateral than unilateral. It therefore passes from the neck over the occiput into the parietal region. The pain is usually very intense, not always intermittent, but sometimes continuous with spasmodic exacerbations. It is aggravated by every movement of the head, by laughing, sneezing, coughing, and by rapid walking. The head is therefore generally carried stiffly, and is inclined backwards, or to the side. The most constant pressure-point corresponds to the site of emergence of the great occipital.

Among the *accessory symptoms* we may mention hyperæsthesia of the skin of the occiput, loss of hair in this region, and swelling of the glands in the neck. Seeligmüller adds narrowing of the pupil and redness of the ear on the most affected side during the attack, noises in the ear, gastric disorders.

During the attack sneezing, watering of the eye, etc., may occur. These symptoms are explained by the anatomical relation of the occipital nerve to the trigeminus. A combination of

[1] *Hygiea*, 1885.

[2] *Beit. z. klin. Chir.*, xxiv.

neuralgia with symptoms due to the cervical sympathetic, which may apparently be traced to a morbid process in the superior cervical ganglion of the sympathetic, is described by Johnson.[1] Reflex spasms in the neck muscles were noted by Paccinotti and others.

I have occasionally found tubercula dolorosa on the cervical nerves.

Among the *causes* of this neuralgia we should specially mention carrying heavy loads on the head, trauma and chill, infective diseases (malaria, typhoid, cerebro-spinal meningitis, influenza, tonsillitis[2]), *gout*, and arthritis deformans. The neuralgia which occurs in the last-named disease is probably always *symptomatic* (compression of the nerve as the result of the disease of the vertebræ). Leidy[3] mentions occipital pain as a symptom of uræmia. The occipital pain which appears in caries of the highest cervical vertebra should not be included here; it may indeed fully correspond to the picture of occipital neuralgia, but it is due to a neuritis caused by trauma or compression. The strict avoidance of certain movements in this affection, the tenderness on pressure on the vertebra, the swelling in its neighbourhood (tubercular granulations, abscess), the crepitation which sometimes exists, the anæsthesia which supervenes later in the occipital region, and eventually the symptoms pointing to involvement of the spinal cord, of the roots and the nerves originating from the medulla oblongata, generally permit of a correct diagnosis. In rheumatism of the neck muscles, the muscles themselves are sensitive to pressure, and the pain has a diffuse distribution; it generally increases in bed, attacks the shoulder muscles, etc.

Hysterical pain in the neck is the most frequent cause of confusion. Hysterics often complain of pain in the neck. It is generally stated, however, that the pain does not occupy the neck region alone, but spreads upwards from the back, even from the lower region of the back, over the head and into the eyes. The pressure-points are not limited to the site of emergence of the occipital nerve. The hyperæsthesia of the skin, of the head, and neck is particularly marked. The psychogenic origin of the pain is almost always revealed by the effect of suggestion. Other signs of hysteria are also present.

The *prognosis* of pure occipital neuralgia is on the whole favourable, but exceedingly obstinate cases do occur, as shown, *e.g.*, by a case of Jastrowitz.[4]

In recent cases, local massage with soothing ointments, moderate blood-letting, a hot fomentation, or Priessnitz's compress, and diaphoretic measures should be tried. A vapour bath may give immediate relief. The galvanic current (especially the stabile anode treatment) is also recommended; E. Remak has again recently laid special stress on its merits. If weak currents are not effectual, a wonderful effect may sometimes be obtained by the use of very strong currents, conducted through the highest cervical cord or directed straight to the nerves by the electrode being placed on the occipital pressure-point. Stimulating ointments, cantharides, local painting with tincture of iodine, and in specially obstinate cases the button cautery, may also be employed.

Quinine is particularly valuable in the intermittent malarial form, and often in ordinary neuralgia. For further details, we would refer

[1] *New York Med. Journ.*, 1894.

[2] Vincent attributes the relation between tonsillitis and occipital neuralgia to unusual anastomoses between the sensory pharyngeal nerves and the occipital nerve or the root of the second cervical nerve.

[3] *Journ Nerv. and Ment. Dis.*, 1897.

[4] *D. m. W.*, 1898.

to the methods of treatment already discussed. Nerve-stretching has also been applied to this nerve (König, etc.). Surgical treatment has recently been specially developed by F. Krause. By his method the superior cervical nerve is sectioned as close as possible to its origin, and the peripheral branches are then subjected to exæresis by Thiersch's method. Henkind also reports the successful use of this method.

Neuralgia of the Phrenic Nerve

We have little trustworthy knowledge of this form of neuralgia. The pain follows the course of the nerve, viz., from the diaphragm, through the thorax (between the pericardium and pleura), and up towards the neck, and is accompanied by a feeling of oppression. It may extend to the shoulder of the same side. Pressure-points are found apparently at the point of insertion of the diaphragm, and on the nerve itself at the site where it is electrically stimulated. Jousset describes as a constant pressure-point a site close to the sternum, corresponding somewhat to the bone and cartilage connection of the fifth rib.

During the attack respiration is usually difficult and painful. Masticating and swallowing may also excite the pain.

This neuralgia most frequently accompanies diseases of the heart, the pericardium, and the large vessels. It also occurs in tuberculosis, as Reynaud (*R. n.*, 1902) has again recently stated. When these affections are not present, the prognosis is favourable. In one case which I have seen, the neuralgic pain in the phrenic was the result of fracture of the clavicle. In another of my cases it appeared to be the intermittent larval form; the attacks came on only during night. Crespin (*Gaz. des hôp.*, 1897) thought the trouble was due in a few cases to malaria, or to enlargement of the spleen, as did Claude (*Thèse de Montpellier*, 1903). A bilateral occurrence of this neuralgia has also been described. The diagnosis can very seldom be made with absolute certainty.

Brachial Neuralgia

Neuralgia of the brachial plexus affects the whole or part of the area of the four lower cervical and the first dorsal nerves. It very rarely keeps strictly to the course of one brachial nerve, although as a rule one of these, *e.g.* the musculo-spiral, internal cutaneous, or more commonly the ulnar nerve, is chiefly affected. More often there is a vague distribution of the pain, which cannot be sharply localised; it occasionally radiates to the upper intercostal and the shoulder nerves and even to the trigeminus. It appears either only in spasms, or it may be constantly present in a less degree, increasing from time to time to considerable intensity. It is described as boring, tearing, lightning, burning (causalgia). The patient instinctively grasps the arm and tries to put it into a position in which all pressure or traction will be avoided. It eases him to a certain extent to support it with the unaffected hand, or carry it in a sling. As movement generally aggravates the pain or brings on the paroxysm, all muscular action is avoided.

Pressure-points are frequently found on the affected nerves, especially on the musculo-spiral, where it passes round the upper arm, on the ulnar (between the internal condyle of the humerus and the olecranon), on the median in the elbow or above the wrist-joint, and on the circumflex at the point where it gives off the main branch. The cutaneous branches may be tender as they emerge through the fasciæ. The pressure produces not only pain, but also paræsthesiæ in the area of distribution of the nerve, so that the patient can often show exactly its anatomical course, and can describe the ramifications of the nerve in the periphery.

Pressure-points are sometimes present above or adjacent to the spinous process of the lower cervical vertebra. The less common cases in which the pain occurs only in certain movements (playing the piano, manual work, ironing, etc.) should be distinguished from neuralgia and regarded as a form of occupation neuroses (see above).

Paræsthesia (formication), pallor and redness of the skin, hyperidrosis, and rarely herpes occur as *accessory symptoms* of the neuralgia. The patient usually complains of a feeling of heaviness and numbness in the arm. Severe *trophic* symptoms, such as we find specially after gunshot wound of the nerves, always point to a neuritis, the diagnosis of neuralgia being apparently no longer justified ; the boundary between the two is, however, often very difficult to draw. Hyperæsthesia of the skin is often present, and diminution of sensibility also in the later stages. Marked diminution of the sensibility is, however, unusual in pure neuralgia, and points to neuritis or to a central disease.

Among the causes of brachial neuralgia, the *neuropathic* disposition occupies the first place. The great majority of individuals affected are hysterical and neurasthenic. In *hysteria* there occur not only vague pains of indefinite localisation in the arms, which almost always radiate to the neighbouring areas of the trunk and head, associated with paræsthesia and unilateral diminution of sensibility, but in exceptional cases there is also an apparently true neuralgia in the region of the brachial nerves. *Anæmia* and *cachexia* also create a predisposition for brachial neuralgia. In three of my cases *diabetes* was the cause, and in one of these the neuralgia passed off as the sugar disappeared. *Injuries* are very often responsible, and the most severe forms of nerve pain may be of traumatic origin. In such cases, however, there is generally a neuritis, due either to direct injury of the nerve or to its irritation by a foreign body, a cicatrix, a splinter of bone, or a callus. In former times venesection sometimes gave rise to a neuralgia due to the nerve lesion. In not a few cases in which the neuralgia developed after an injury of the peripheral branches of the nerve at the fingers (bruising, bites, etc.) it was mostly of a reflex origin, and could be clearly differentiated from an ascending neuritis. But *traumatic reflex neuralgias*[1] are often merely local symptoms of a general traumatic neurosis.

Brachial neuralgia may be produced by *rheumatic* influences. Amongst the *infective diseases*, malaria, typhoid, and influenza give rise in rare cases to this trouble. *Gout* is a comparatively common cause (Gowers). In one of my patients I attributed the brachial neuralgia to a gouty diathesis on account of a long-past renal colic. The family physician doubted the correctness of this opinion. A few days after the consultation a typical attack of gout occurred, and as it disappeared so did the neuralgia.

Of the toxic causes, we may mention alcoholism, lead-poisoning—which, however, more often gives rise to arthralgia and myalgia—and carbonic-oxide poisoning (Bernhardt). Diseases of the heart, aneurisms of the aorta and subclavian artery, are sometimes accompanied by neuralgic pains in the arm, usually the left (whilst in disease of the liver the pain appears in the right arm). Angina pectoris may be associated with ulnar neuralgia or may alternate with it (Löwenfeld). In one case,

[1] In a few cases (Bell, Hesse) a decayed tooth was said to be the cause of brachial neuralgia, which recovered when the tooth was extracted.

which had been diagnosed as brachial neuralgia, I found aneurismal dilatation of the subclavian; the area of the musculo-spiral nerve was specially affected, and an atrophy of the triceps showed that neuritis or degenerative changes had already taken place in the nerve. The same cause was discovered in other cases. Arterio-sclerosis is not infrequently the origin of brachial neuralgia, as a case of Loewenfeld's shows. The so-called "intermittent claudication" (see below) has in the same way been observed in the arm. Cervical ribs may also give rise to this neuralgia by pressure.

With regard to the *differential diagnosis*, we must be particularly careful not to confuse this disease with affections of the spinal cord and its roots. Hypertrophic cervical pachymeningitis, spinal caries, tumours growing from the meninges, etc., may for a considerable time be masked by the symptoms of a brachial neuralgia. In these cases, however, the symptoms usually appear in both arms. *Tumours* alone tend to simulate a unilateral brachial neuralgia, but the further course always shows that the disease is a destructive one, as paralysis, atrophy, and anæsthesia supervene. Pressure-points on the nerve tract are also generally absent, whilst the spinal column is the site of spontaneous pain, and single vertebræ are often extremely tender to percussion. The pain in cervical tabes is also bilateral, and it gives rise to other characteristic symptoms. In one case in which the neuralgia was ascribed to cervical ribs, I found symptoms of spinal gliosis. Radiography may be of use in establishing the diagnosis. In muscular rheumatism the pain does not follow certain nerve tracts, nor is it the nerves, but the muscles themselves, and especially their insertions, which are tender to pressure; and further, the pain has not the neuralgic character. As to the occupation neuroses, to which "tennis-arm" certainly belongs (Clado),[1] the corresponding chapter should be consulted.

Taking all in all, I regard a true, pure brachial neuralgia as a rare affection; there is usually a background of hysteria, neurasthenia, of an organic disease, or a constitutional illness (diabetes, etc.). Since the publication of the first edition of this text-book, I[2] have found more and more that brachial neuralgia is as a rule a *brachalgia*, a brachial psychalgia, *i.e.* a pain in the arm of indefinite localisation, which is a symptom of a neuropathic or psychopathic general condition and is therefore generally associated with other nervous troubles, especially with depression, sleeplessness, excitement, etc. This brachalgia often conceals the neurosis or psychosis. Pressure-points are also frequently discovered in this form, and are not always due to a hyperæsthesia of mental origin, to a suggested sensibility to pressure, but also to a real exaggeration of the mechanical excitability of the corresponding sensory nerves. I have observed this condition in both men and women. It is usually not difficult to discover the psychogenic origin. I should not like, however, to suggest by this statement that I entirely deny the existence of brachial neuralgia. I myself have seen a number of cases in which the trouble could be interpreted neither as a psychalgia nor as due to an organic process.

The *prognosis* and *treatment* are those of neuralgia in general. In recent cases, diaphoretic measures and blistering should be employed, the latter also in advanced cases. The application of fly-blisters over

[1] *Progrès méd.*, 1902.

[2] *B. k. W.*, 1898.

the nerve trunks, the use of the button cautery, etc., are specially beneficial. Quinine, arsenic, salicylate of soda, ol. terebinthinæ, phenacetin, pyramidon, aspirin, etc., have been tried. Belladonna is included among the remedies. The electric current is often exceedingly successful.

According to what has been said as to the nature of this disease, treatment should bear special relation to the psychogenic origin of the pain. From this point of view we would refer to the treatment in the chapter on hysteria and neurasthenia. In the most intractable cases, *nerve-stretching* may be the most suitable method. Other surgical operations (extirpation of a tumour, etc.) may be indicated. Section of the corresponding posterior root should only be undertaken in the most severe cases, when all other methods have failed.

Jacoby has lately published a further contribution to this question in the *New York Med. Journ.*, 1907 (see literature here).

Nägeli describes and advocates bloodless stretching of the nerve. In one of my cases of aneurismal dilatation of the subclavian artery due to arteriosclerosis, in which the diagnosis could at first only be that of neuralgia, later of compression neuritis, recovery was brought about by the simultaneous use of iodide of potassium, electricity, and the local application of an ice-bag. Kader [1] saw neuralgia of the right cervical and brachial plexus of many years' duration, due to contracture of the right cervical muscles from left-sided torticollis, disappear after tenotomy of the left sterno-mastoid.

Intercostal Neuralgia

Neuralgia of the nerves arising from the dorsal roots of the spinal cord affects chiefly the anterior branches, viz., the intercostal nerves. Those of the left side are more often attacked. It is seldom confined to one, but usually extends to the area supplied by several neighbouring intercostal nerves. The pain sometimes passes to the inner side of the arm. It is generally constant, but with acute exacerbations, and in some cases it is intensely violent. It is usually situated in the anterior and lateral parts of the thorax. It follows the course of the intercostal spaces and may also occupy a circumscribed area. Some patients say that along with the pain running in the intercostal spaces, a stabbing pain is felt through the breast from in front backwards. It comes on spontaneously, is aggravated and often elicited by shaking the head, by coughing, sneezing, deep breathing. The *pressure-points* are of diagnostic importance. There are usually three, one close beside the spinal column at the level of the origin of the affected intercostal nerve (vertebral point), one in the axillary line, corresponding to the lateral perforating branch (lateral point), and one beside the anterior middle line of the body, just where the anterior perforating branches emerge, therefore at the sternum and the rectus abdominis. It is not usual for the intercostal nerve to be tender to pressure throughout its whole course.

The cutaneous area corresponding to the affected intercostal nerve is sometimes hyperæsthetic ; even the lightest touch or pressure of the clothes may be painful. Anæsthesia is rare and makes the diagnosis

[1] *Mitt. a. d. Grenzgeb.*, ii. Some remarks on therapeutics are to be found also in Harburn *Brit. Med. Journ.*, 1905).

of a pure neuralgia improbable, but the sensibility may be diminished. Intercostal neuralgia is the form which is most frequently accompanied by *herpes*, but the relation is not constant (compare following chapter).

The pain may radiate into the back, the arm, or the shoulder of the same side. Intercostal neuralgia is occasionally associated with angina pectoris. In two of my patients the bilateral intercostal neuralgia came on regularly only during sleep, so that they were awakened by the pain.[1] There was no syphilis.

This neuralgia chiefly affects women in youth and middle age, but it also frequently occurs in men and old people.

Hysterical individuals very often complain of neuralgia and pain in the side. This may be true neuralgia, but is more frequently an hysterical pseudo-neuralgia. The preponderance of intercostal neuralgia in the left side, and especially in the area of the fifth to ninth intercostal nerves, has been attributed to circulatory disturbances, particularly to the obstruction of the flow of blood from the veins of this region due to anatomical conditions (Henle). It is more probable that the cause is to be found in the relation of intercostal neuralgia to hysteria, in which the sensory symptoms all tend to be located in the left side. Moreover, morbid introspection directed specially to the heart often excites the onset of left-sided intercostal neuralgia. It may also be associated with disturbances of the innervation of the heart (*bradycardia*, *tachycardia*), but it more frequently happens that both these and the neuralgia itself are co-ordinated symptoms of a heart disease.

Anæmia, cachexia, and conditions of exhaustion after lactation, the puerperium, feverish illnesses, and loss of blood create a favourable basis for this disease. Sexual excesses, onanism, disease of the uterus and ovaries may all be of etiological importance. Bilateral intercostal neuralgia of syphilitic origin, with nightly exacerbations of the pain, has been described. Injuries and fractures of the ribs may bring on the development of the neuralgia, but in such cases there is usually a traumatic neuritis due to direct nerve lesion or to compression of the nerve by a callus. *Scoliosis* and deformities of the spine tend to produce intercostal neuralgia. The intercostal pain occurring in disease of the spine and the spinal cord has usually a neuralgic character, but is to be distinguished from intercostal neuralgia. This is true also of real inflammation of the roots (radiculitis), whether of primary or of meningopathic origin in Chipault's sense.[2]

Some points in the differential diagnosis may be found in the Thesis of Boutin (Bordeaux, 1904-1905, ref. *R. n.*, 1906).

Nothing is definitely known as to the origin of the form of this affection which occasionally occurs in diseases of the lungs and pleura. It may usually be regarded as a tubercular neuritis. Aortic aneurisms may cause neuralgic pain by direct compression of the intercostal nerve. It may also, however, be produced in a reflex manner by diseases of the heart, aorta, lungs, etc., as explained by Head (p. 108). Feuillet treats of the gastro-intestinal origin of intercostal neuralgia in his Thesis. An epidemic onset of intercostal neuralgia has occasionally been noted (see following section).

The *prognosis* is doubtful. If the trouble is not merely symptomatic it

[1] *B. k. W.*, 1899.

[2] *R. n.*, 1902.

is usually of long duration, although it does not endanger life. Thorough examination of the spinal column, the heart and vascular system, the lungs, and the functions of the spinal cord, will prevent confusion with symptomatic neuralgia. But spinal tumours in particular may for years simulate a true neuralgia. It is not difficult to diagnose neuralgia from rheumatism of the pectoral muscles or from pleurodynia.

Treatment.—Treatment should be directed towards the cause, if that can be discovered. Conditions of weakness and constitutional anomalies must especially be dealt with in a rational manner. If there is a callus, a badly healed fracture, or a tumour in the ribs to which the pain can be traced, then the formation which is keeping up the irritation should be removed. If there is chronic atrophy of the spinal column, then suitable orthopædic treatment should be instituted. If there is an insufficiently compensated cardiac defect, the use of digitalis may also have a good effect upon the neuralgia, as I have found in several cases. For the rest, we may refer to the measures already laid down for the treatment of neuralgia. Subcutaneous injections of osmic acid (1 per cent.), hydrochloric acid (2 per cent.), ether, alcohol, etc., are recommended. Subarachnoidal or epidural injection of a solution of cocaine, etc., is said to have had good results (Widal, etc.).

For the most severe cases there remains surgical treatment. Nerve-stretching has frequently cured the neuralgia. Schede induced recovery in one case by resection. I have seen a case in which nerve-stretching was a failure, but recovery followed the use of the galvanic current, and another in which several intercostal nerves were widely resected without curing the neuralgia.

As to section of the roots, see the remarks on p. 561.

Appendix: Herpes Zoster

This name is applied to an eruption of vesicles which follows the distribution of one or more nerves, coincides in its extent with the areas of innervation of the posterior roots, or, according to another theory, with the sensory cutaneous areas of the spinal-cord segments. The investigations of Sherrington,[1] and especially those of Head [2]—with which those of Blaschko,[3] Seiffer,[4] Armand-Delille, Camus, Fröhlich-Grosser,[5] and others coincide—have, it is true, thrown some light upon this question, but as yet there is no entire unanimity in the physiological conception of herpetic zones and their relation to the sensory innervation of the skin. Herpes most frequently develops on the trunk in the region of the dorsal roots, and it was for this pectoral herpes which extends in the form of a half girdle that the name of herpes zoster was specially chosen. It has since then been also conferred on all the forms of herpes which follow the course of nerve tracts. The trigeminal region is very often affected, chiefly that of its first branch: zoster frontalis ophthalmicus. There is also a zoster occipito-collaris, cervico-brachialis, dorso-abdominalis, lumbo-inguinalis, sacro-ischiad., etc., but the terms chosen by Head and corresponding to its area are prefer-

[1] "Phil. Trans. Roy. Soc. of London," Vol. clxxxiv. and cxc.

[2] *Br.*, xvi. and xvii; also, "The Sensory Disturbances of the Skin," etc., Head-Campbell; "The Pathology of Herpes Zoster," *Br.*, xxiii.

[3] *A. f. mikr. Anat.*, xxx., and *A. f. Derm.*, Bd. xliii. and xliv.

[4] *A. f. P.*, xxxiv.

[5] *Z. f. N.*, xxiii.; see also Grosser, résumé in *C. f. Gr.*, 1904.

able : herpes sterno-nuchalis, cervico-subclavicularis, dorso-ulnaris, etc. Blaschko has also given precise data as to the topography.

The following is the typical picture of herpes zoster : Groups of regularly arranged vesicles appear on a reddened ground ; their contents are at first a clear serum, which later becomes opaque and purulent, less often hæmorrhagic. The vesicles dry up and there appear brownish crusts or scabs, which fall off and leave brown spots. These also finally disappear. The number of vesicles and groups varies in each case. When the eruption is thick, they may become confluent and form blisters as large as a pigeon's egg (herpes bullosus). *Hæmorrhagic* herpes zoster occupies a special place as it heals with the formation of cicatrices. This is true in a greater degree of *gangrenous* herpes zoster, in which from the commencement there is necrosis and formation of scabs, deep scars being left after recovery.

Whilst the eruption of the individual groups of vesicles tends to heal in a few days, the whole process may extend over three or four weeks, and the more severe forms may last for about two months. The zoster usually extends on the thorax over several intercostal spaces in the form of a semi-girdle, but it may be confined to a small area.

Fabre (*Prog. méd.*, 1903) describes unusual forms of distribution (bifid, forked).

Neuralgia is one of the most frequent, indeed it is an almost constant accessory symptom. It sometimes accompanies, sometimes precedes the eruption. It tends to disappear as the herpes heals, but it may long outlast it, and may, especially in old people, be very distressing from its persistence and intensity. Among the inconstant symptoms are sensory disorders, viz., hyperæsthesia and the somewhat more common anæsthesia, disturbances of the sweat secretion, pruritus (Bettmann), canites or loss of hair in the corresponding nerve area. Gerhardt describes a " sensory reaction of degeneration."

In ophthalmic zoster there is occasionally anæsthesia of the conjunctiva and cornea, neuroparalytic keratitis, less often panophthalmitis, oculo-motor paralysis, and optic neuritis. Some of these symptoms are not directly dependent upon the herpes, but are to be explained by the fact that the disease to which the herpes is due has extended to other nerves. Cases of this kind have of late years been published by Ahlström,[1] Désirat,[2] etc. It is also not unusual for a facial paralysis to develop during or after a facial zoster or a cervical herpes (Ebstein,[3] Fraser, Cousot,[4] etc.). Extension of the vesicular eruption to the corresponding mucous membrane has been seen in both ophthalmic and facial zoster.

A treatise by Körner (*M. m. W.*, 1904) deals with otic herpes and its accessory symptoms.

Sacral herpes zoster (gluteal, etc.) may be preceded by transient paralysis of the bladder and intestine, as described by Davidsohn (*B. k. W.*, 1890) and noted once by myself.

Herpes is frequently a *febrile* illness. The eruption is often accompanied by fever and corresponding disturbances of the general condition, or a rise in the temperature may precede the appearance of the vesicles. Swelling of the lymphatic glands is very common in this febrile form (Kaposi, Barthélemy, Blaschko).

In the great majority of cases the disease is limited to one side of

[1] *Hygiea*, 1904. [2] *Thèse de Bordeaux*, 1902-1903. [3] *V. A.*, Bd. cxxxix.

[4] Ref. in *R. n.*, 1904 ; the author treats the question of zoster paralysis in general. Paralysis of the extremities associated with zoster is described by Magnus (*Norsk. Mag.*, 1903) and others.

the body, but a bilateral distribution has been observed in a few cases, by Moers, Kaposi, Thomas, Weis, Hartzell, Reckzeh, etc. I have treated a young man with a bilateral zoster in the ulnar region, which was accompanied by severe swelling of the glands. The fact should also be noted that the disease occurs as a rule only once during life, relapses being very rare; they are described by Kaposi, Behrend, Düring, Beathy, Bewley, Laveran-Matignon, Hirtz-Salomon,[1] Vörner [2] and others.

Among the *causes*, *poisoning* should be mentioned first. Poisoning with arsenic (Hutchinson, Gerhardt, Hebra) and with carbonic oxide (Leudet) may produce the herpes. Re-vaccination was blamed in one case (?). It has occasionally been noted in the course of diseases of the internal organs (pleurisy, etc). It has also been associated with gastric disorders. A few cases, such as that of Reusz,[3] point to gout as a cause. Injuries affecting the nerve or its root and diseases of the spinal column and cord which extend to the root or spinal ganglia, may also give rise to it. Under these circumstances, herpes is rather secondary and symptomatic, but many published cases, and especially the observations and investigations of Head and Campbell already mentioned, show that there can be no doubt that it is frequently an independent affection, a *primary acute infective disease*. Some of the factors mentioned—the febrile character, the swelling of the glands, the general *immunising* influence of the disease—point in themselves to its infective nature. Micro-organisms have also been found in the cerebro-spinal fluid (Achard and Loeper, Widal, Brissaud-Sicard). It has been repeatedly noted that the affection may appear in an epidemic form (Rohé, Kaposi, Weis, Reilly, Dopter). O. Sachs [4] reports an epidemic of herpes in Breslau. House epidemics have also been described (Dopter, Baudoin). The disease may further develop during the course of other infective diseases, *e.g.* pneumonia (Biehl).

As regards the site and pathological anatomy of herpes, Bärensprung [5] was the first to show by his investigations that it may arise from a disease of the spinal ganglia. On the other hand Curschmann and Eisenlohr [6] proved that a peripheral neuritis (a neuritis nodosa) may also produce herpes zoster, and Dubler [7] was able to demonstrate not only the neuritic nature and the peripheral site of the disease in some cases, but also the fact that the spinal ganglia were intact. The neuritic origin was also accepted by Friedreich, Charcot, and Cotard. Others distinguish between a peripheral neuritic origin—*e.g.* from slight contusion of the skin (Gaucher-Bernard)—and a central one. Brissaud [8] was a strong supporter of the spinal origin of the disease. But recently Head, working partly independently, partly in collaboration with Campbell,[9] has shown by his exhaustive investigations of a very large number of cases, that underlying a *primary*, *spontaneous herpes*, there is often an *acute*, frequently a *hæmorrhagic inflammation of the spinal ganglia* (or of the homologous Gasserian ganglion), a process which he regards as acute posterior poliomyelitis, analogous therefore to acute anterior poliomyelitis. The affection, which tends to be limited to one ganglion, may in slight cases disappear without leaving any definite alteration

[1] *Bull. de la Soc. méd. de Paris*, 1902.
[2] *M. m. W.*, 1904.
[3] *Orvos. hetil.*, 1904.
[4] *Z. f. H.*, xxv. (Literature).
[5] *Charité-Annalen*, ix., x., xi.
[6] *A. f. kl. M.*, xxxiv.
[7] *V. A.*, Bd. xcvi.
[8] *Bull. méd.*, 1896.
[9] *Br.*, 1900.

in it. In severe cases the ganglion may become sclerosed. The disease of the ganglion causes a secondary degeneration in the posterior roots and peripheral nerves.

Head does not exclude the possibility that the process may arise from any other site of the viscero-sympathetic-spino-radicular tract, *i.e.* from the reflex arc which extends from the viscera through the corresponding sympathetic fibres into the spinal cord, and thence through the posterior roots, ganglion, and peripheral nerve to the skin. Spitz has of late specially advocated this view. Head's view is supported by Howard (*Amer. Journ. Med. Sc.*, 1905), Hedinger (*Z. f. N.*, xxiv.), Magnus, and Dejerine-Thomas (*R. n.*, 1907). Changes in the spinal cord and notably hæmorrhagic foci in the grey matter of the corresponding lateral horn and similar degeneration of the communicating rami of the sympathetic were found in one case by Thomas-Laminière (*R. n.*, 1907). See also Bruce's case (*R. of N.*, 1907). The presence of lymphocytosis in the cerebro-spinal fluid is also regarded by Chauffard (*Méd. moderne*, 1903), Brandéis (*R. n.*, 1904), as a confirmation of Head's views. In urticaria with a herpes-like distribution, Dopter (*Gaz. des hôp.*, 1904) brought on a fresh attack by lumbar puncture.

In opposition to the neural theory, Peiffer suggests a *vascular* one; he thinks that the distribution of the herpes follows that of the vessels.

The *prognosis* of primary herpes is favourable. Complications may, of course, make the prognosis more grave, and may even endanger life. In symptomatic herpes the prognosis depends upon the character of the primary disease. It has already been mentioned that the neuralgia may last considerably longer than the herpes. This is chiefly to be feared in old and cachectic patients. Hæmorrhagic and gangrenous herpes zoster is also less favourable from this point of view. Pétren and Bergmark [1] conclude from their observations that persistent neuralgia is to be expected when the sensory disorders are marked.

Treatment.—Rubbing and irritation of the vesicles should be prevented by bandaging the parts in cotton-wool, and powdering with zinc amyl, etc. Care must be taken not to open the blisters in applying the ointment, as this tends to the formation of scars. Bleuler recommends very highly the use of cocaine ointment (1 per cent. with lanoline and vaseline), applied carefully by means of strips of linen. The neuralgia is treated according to the principles already described. Of late years epidural injection of soothing drugs has been employed in herpes zoster also. Jarisch specially recommends the combination of antipyrin and salicylate of soda (aa. 0·5).

In some cases observed by Abadie lumbar puncture and the removal of about 20 c.cm. of fluid have had a curative effect (*R. n.*, 1903). This statement, however, requires further corroboration.

Neuralgia of the Mammary Gland. Mastodynia. Irritable Breast (Astley-Cooper)

There is a form of neuralgia which is limited to the nerves of the mammary glands. These arise from the intercostal nerves. The skin over the gland is innervated by the anterior and lateral perforating branches of the 2-6, the glandular substance by the lateral perforating branches of the 4-6 intercostal nerves.

The pain comes on in spasms, and may be extremely severe. It is usually associated with *hyperæsthesia* of the skin, and in particular

[1] *Z. f. k. M.*, Bd. lxiii.

with excessive sensibility of the nipples. There may be circumscribed *redness* and *swelling*, or even local *swelling* of the gland substance, which may simulate a tumour. In a few cases it is said to have been regarded as a neuroma or fibroma (Cooper). Erb and Lesser (*V. A.*, Bd. lxxxvi.) have observed a milk-like fluid appear, after or during the attacks. In a few cases vomiting occurred at the height of the attack of pain. Herpes is seldom associated with this neuralgia. Pressure-points are not usually to be found on the nerves of the mammary gland, but over the spinous processes of the second to the sixth thoracic vertebræ. Tubercula dolorosa is sometimes present on the nerves of the mammary gland.

The disease occurs almost exclusively in middle age. It is seldom observed in men. Anæmia and hysteria are frequent underlying causes. Pregnancy, lactation, fissures at the nipples, trauma or emotional excitement may produce the neuralgia. Pendulous breast tends to bring it on. In a few cases floating kidney was regarded as the cause (Fischer). Exacerbations of the pain usually occur during menstruation.

The *diagnosis* is not difficult. Differentiation from malignant tumour is only difficult when there is circumscribed induration in the substance of the gland. Such swellings are, however, inconstant in neuralgia; they do not show any gradual increase, nor do they lead to swelling of the lymphatic glands.

The *prognosis* as to recovery is not favourable, as this neuralgia is persistent and yields but little to treatment. Duchenne recommends the use of the faradic brush. The constant current may also have an excellent effect, as I have seen in one case. The pain is eased by supporting the breast. A light compression bandage, warm covering, narcotic ointments, etc., may also alleviate the pain. Painting the nipple with a strong solution of cocaine has sometimes a palliative effect. There is nothing new to add as regards treatment by drugs. Nägeli recommends a kind of stretching of the gland.

Lumbar Neuralgia

We usually distinguish between neuralgia of the short and of the long lumbar nerves. The former are the nerves which innervate the lumbar, sacral, lower abdominal, inguinal, and part of the genital regions, namely, the ileo-hypogastric, ileo-inguinal, the genito-crural with its two branches, the external spermatic, and the lumbo-inguinal. Neuralgia in these nerves is known as *lumbo-abdominal neuralgia*. The long lumbar nerves are the external cutaneous, the anterior crural, and the obturator. Each of these nerves may be affected by neuralgia, especially the crural anterior.

Lumbo-abdominal neuralgia is characterised by pain which radiates from the lumbar towards the sacral and lower abdominal region, into the flexure of the groin, the scrotum, and eventually into the spermatic cord. The highest part on the anterior surface of the thigh may also be affected. Sometimes one nerve branch is chiefly involved, sometimes another. Pressure-points are found beside the lumbar spine, above the crest of the iliac bone, near the linea alba, at the inguinal region, the scrotum, etc. Cutaneous hyperæsthesia is occasionally present, and herpes zoster is one of the ordinary accessory symptoms. Spasmodic contraction of the abdominal muscles or spasm of the cremaster and

vomiting are occasionally observed at the height of the paroxysm. As the pain is aggravated by walking, the patient avoids this altogether, or moves very cautiously, slowly, and with the least possible excursion of movement. Sexual excitement, ejaculation of semen, and increased micturition are uncommon symptoms.

Crural neuralgia may be limited to the external cutaneous nerve; the pain is then localised on the exterior surface of the thigh as far as the knee-joint, and a pressure-point is found at the anterior superior spine of the ilium. I have twice seen this neuralgia after influenza and once after gonorrhœa and acute articular rheumatism. (See also paræsthetic meralgia, p. 459.)

If the *anterior crural nerve* is affected, the pain follows the course of this nerve and its branches, especially the middle and internal cutaneous and the long saphenous, and therefore passes down the inner side of the leg as far as the internal margin of the foot. Tender points are found on the anterior crural nerve in the flexure of the groin, sometimes also in the course of the long saphenous. The pain is increased by movement of the leg, especially by extension of the hip-joint, which the patient, therefore, usually keeps flexed. *Vasomotor* symptoms, *hyperidrosis* and *herpes* are not uncommon in this neuralgia. The presence of paræsthesia, and in particular of anæsthesia, and the absence of reflexes, point to the probability that the neuralgia is due to an organic disease.

The *obturator* nerve is least often affected. The pain follows the course of the obturator nerve from the obturator foramen along the inner surface of the thigh down into the region of the knee.

All these forms of neuralgia must be diagnosed with care. In the majority of cases they are merely *symptomatic*. Tumours of the lumbar vertebræ and in the pelvis are specially apt to be masked for a considerable time by lumbo-sacral or crural neuralgia. The most thorough examination, comprising not merely palpation through the abdominal wall, but also per anum et vaginam, and illumination by the X-rays, are necessary to prevent grave errors in the diagnosis. The diagnosis of neuralgia cannot be maintained if marked symptoms of paralysis appear, such as atrophic paralysis, Westphal's sign, anæsthesia, etc. I have, however, lately seen a case in which I was at first inclined to diagnose a neuralgia.

Among the causes of the idiopathic forms, chill, exhaustion, trauma, constipation, masturbation, and affections of the genital organs have been mentioned. Diabetic neuralgia is not infrequently localised in the anterior crural and obturator nerves, but neuritis is usually the cause of it (see p. 457; also the remarks upon so-called intermittent claudication in next chapter). The neuralgia may follow disease of the testicle or urethra, but whether reflexly or by means of an ascending neuritis is not yet clear. Injury or dislocation of the ankle-joint occasionally causes neuralgia in the region of the long saphenous nerve. Neuralgia in the obturator nerve is usually due to pressure and traction upon the nerve by an obturator hernia.

The thesis of Roux (Paris, 1907) deals with ileo-lumbar neuralgia in aneurism of the descending aorta.

The *prognosis* of idiopathic lumbar neuralgia is not unfavourable.

Complete recovery may be anticipated when the patient is young and robust. With regard to treatment, we would refer to the general part and the following chapter.

Sciatic Neuralgia, Sciatica. (*Malum Cotunnii*)

Sciatica is the name applied to neuralgia which affects the nerves of the sacral plexus, the sciatic nerve and its branches, and not infrequently also the small sciatic nerve, and which in rare cases radiates to other roots of the lumbo-sacral plexus.

The disease is very common. Men, especially in middle life, are much more liable to it than women. It is not rare in old age, but is practically unknown in childhood.

It should be stated here that although it is difficult in general to draw a sharp distinction between neuralgia and neuritis, this is very specially the case with regard to the form under consideration. There is no doubt that symptoms of sciatica are frequently due to a *slight* neuritis of the sciatic nerve,[1] which occasionally becomes so severe that it produces clinical symptoms of the neuritic process. In the great majority of cases these signs are absent, but there is no doubt that all the *stages of transition between neuralgia and typical neuritis* do occur. We cannot, therefore, make a practical differentiation as regards the etiology, but must include among the causes of sciatica many factors which, as we imagine, would produce a mechanical lesion and an inflammatory process in the nerve.

The *neuropathic* disposition certainly exaggerates the tendency to this disease, but it does not play so prominent a part in this as in the other neuralgias. Sciatica attacks individuals who have been hitherto quite healthy, strong, and well nourished, not less frequently than thin persons. It may develop from *gout* (Gowers, Hyde [2]—according to whom it is the most common cause) and *diabetes mellitus* (Worms,[3] Ziemssen,[4] Lagardère [5]). Bilateral sciatica in particular may be of diabetic origin. The relation to *syphilis* is less certain, apart from the fact that gumma are sometimes found in the nerves, but syphilitic sciatica is often mentioned in recent literature. The tubercular etiology which is assumed by some French writers, seems to us doubtful in true sciatica. Chronic poisoning (from alcohol, lead, and other metallic poisons) may give rise to this neuralgia. It sometimes follows acute infective diseases, and may even be a result of *gonorrhœa* (Fournier, Lesser). In many cases it is undoubtedly due to *muscular rheumatism* arising in the lumbar muscles. *True myositic affections* may also cause inflammatory processes in the neighbouring tissue, which extend to the sciatic nerve. Chill is one of the most important causes of sciatica. The symptoms may appear directly after a chill; sitting upon cold damp ground is particularly dangerous in this respect. Trauma is also of great importance. In addition to direct injury to the nerve, we may mention bruising of the gluteal region, falls on the hips, buttocks, etc. Sitting on a hard seat may be sufficient

[1] Pathological investigations have been made in a few cases, such as those of Gubler-Robin, Leudet, Hunt (*Amer. Med.*, 1905), Thomas (*R. n.*, 1905). Surgical treatment has sometimes supplied an opportunity for study of the uncovered nerve. The result was either negative, or swelling and sometimes also thickening of the epineurium were detected. Microscopic results were, in the few cases examined, either negative or insignificant, or, as in Thomas's case, they hardly gave any assistance in making a diagnosis of pure typical sciatica.

[2] *Lancet*, 1896. [3] *Gaz. méd.*, 1880. [4] *M. m. W.*, 1885. [5] *Thèse de Paris*, 1902.

to produce sciatica in thin persons. Professional over-strain of the legs may also be responsible (Seeligmüller).

Sciatica is often the first indication of intrapelvic pressure on the nerve or its roots. Pelvic tumours, the pregnant uterus, the head of the child, and in rare cases retroflexion of the uterus (Offergeld [1]), etc. etc., may have this effect. Mechanical lesions of the sacral plexus *during labour*, notably application of the forceps, may give rise to sciatica. It has even been traced to the compressing effect of hard fæcal masses. *Venous congestion* in the pelvis, in the venous plexus accompanying the sacral nerve, or in the sheath of the sciatic nerve itself, may, according to Quenu [2] and others, produce sciatica—"varicose sciatica." I could find no other explanation of some cases. Inflammatory processes in the pelvis, especially *perimetritis*, may involve the sciatic region.

Sciatica is usually unilateral. *Bilateral* sciatica is as a rule symptomatic, *i.e.* it is due to diseases of the spinal cord, tumours of the spinal column, of the sacrum and pelvis, or to constitutional diseases (especially diabetes); but an idiopathic sciatica may affect both sides.

Symptoms.—The pain does not usually commence immediately in its full severity, but increases for some days or weeks. The patient has at first only an unpleasant sensation of dragging at the back of the thigh or only in the sacral and lumbar region, which soon, however, becomes severe, stabbing, irritating, boring, or even fulminating pain, passing through the extremity as a rule from above downwards. This pain follows the course of the sciatic nerve. It may commence in the sacral region at the point where it emerges from the sciatic foramen or higher up, passes down the middle of the back of the thigh, and then into the peroneal nerve and its branches or into the tibialis posticus. It may involve the whole leg and foot—with the exception of the inner parts innervated by the crural or saphenous nerve [3]—or it may be confined to one segment of the extremity. It generally holds to the course of one nerve, so that the patient can trace *the whole pain tract with his finger*. The cut. fem. post. is often and sometimes alone affected. It is very exceptional for the plantar nerves to be the only ones that are affected. This plantar neuralgia may cause intense pain (Romberg).

The pain is felt in the skin or in the deep parts. It comes on either only in paroxysms or it is continuous with exacerbations, especially during the night. It is brought on and increased by pressure, movement, and an uncomfortable position. As a rule, walking increases it. The patient endeavours to ease the affected leg as much as possible, and specially avoids movements which cause any dragging on the sciatic nerve, such as flexion of the thigh with the knee extended, or extension of the leg with the hip flexed. As a rule the limb is kept slightly flexed at the hip and knee and abducted at the hip, whilst the foot touches the ground lightly.

According to the observations of Largelette, Albert,[4] Nicoladoni,[5] etc., scoliosis of the lumbar spine (*scoliotic sciatica, neuropathic scoliosis*)

[1] *D. m. W.*, 1906.

[2] *Arch. de Neurol.*, xxxiii.; *Traité de Chir.*, ii., and *Gaz. des hôp.*, 1892.

[3] I cannot in any way confirm Ehret's theory that the process underlying sciatica usually involves the crural nerve, although it is to be admitted that this nerve is occasionally affected. Thomayer (*Casop. lek.*, 1905) goes still further than Ehret.

[4] *Wien. med. Presse*, 1886. [5] *Ibid.*

often develops, the spine showing a concave curve towards the healthy side (Fig. 240). The origin of this crossed scoliosis has been explained in various ways. It has been thought that it is due to the endeavour of the patient to ease the affected leg, which displaced the centre of gravity towards the healthy side. Others (Schüdel,[1] Kocher, Gussenbauer[2]) believed that the erector spinæ muscle of the affected side was relaxed, in order to protect the affected sensory branches which pass through it, or that the patient instinctively dilated the intravertebral foramina and thus lessened the pressure upon the emerging roots (Nicoladoni). Ehret[3] regards the scoliosis as a result of the alteration of position which the leg undergoes along with the pelvis. From the instinctive effort of the patient to relax the affected nerve, the leg of that side is flexed at the hip-joint, abducted and rotated outwards, so that the so-called lumbar space—the distance between the crest of the ilium and the false rib—is enlarged at this point. Lorenz[4] takes a similar view. This explanation is rejected by Mann,[5] who considers that the cause of the scoliosis is a paresis of the erector spinæ of the affected side. It has also been suggested that the patient endeavours by this attitude to lessen the pressure of the inferior margin of the pyriformis muscle upon the sciatic nerve (Lesser), etc., Seiffer,[6] Kreche[7]), and other writers practically agree with Ehret.

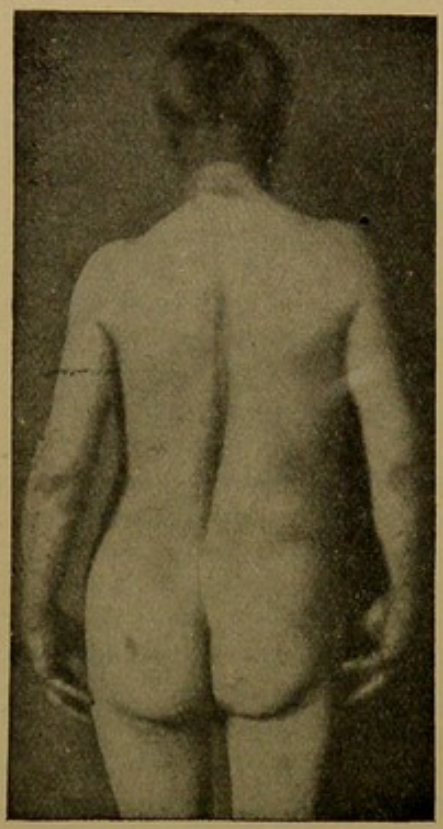

Fig. 240.—Scoliosis in left sciatica. (Oppenheim.)

Moreover, there may be a scoliosis towards the affected side (Brissaud's homologous scoliosis), which is attributed to a contracture or reflex-spasm in the erector spinæ of the same side. The contracture of this muscle is sometimes distinctly apparent and should not be confused with a simple protrusion of the belly of the muscle which is caused by torsion of the spinal column in crossed scoliosis. Remak[8] describes an alternating scoliosis which has also been seen by Seiffer and Meige. Kyphosis very seldom develops.

Ehret further points out that in consequence of the depression of the affected side of the pelvis the gluteal fold lies much lower down and the anal fold (the perpendicular fold between the two buttocks) runs obliquely, *i.e.* with its upper part inclined towards the affected side. In sitting also, the patient's attitude is peculiar; he sits on the tuberosity of the ischium of the healthy side in order to protect the tender sciatic nerve from pressure.

[1] Langenbeck's *Arch.*, Bd. xxxviii. [2] *Prag. med. Woch.*, 1890.
[3] *Mitt. a. d. Grenzgeb.*, iv. and xiii., and "Ischias scoliotica," eine krit. Studie, Wien and Leipzig, 1897.
[4] *D. m. W.*, 1905. [5] *A. f. kl. M.*, Bd. li. [6] *Charité-Annalen*, 1900.
[7] *M. m. W.*, 1900. [8] *D. m. W.*, 1892.

In severe cases the patient sits on the edge of the chair, and bends the spine very much backwards so as to bring the thigh and the trunk as much as possible into a line. In rising from the chair he bends as far forward as he can, places the feet, especially the foot of the affected side, as far back as possible, and then slowly raises himself (Ehret). In bending he places the affected leg as far back as he can, the flexion movement being carried out only by the sound leg. It is particularly difficult for a patient with sciatica to sit upon the ground. He does not like a normal person sit upon the tuberosity of the ischium, but on the sacrum. In lying also the above-mentioned peculiarities of position of the leg, viz., abduction, flexion, and external rotation, become evident. It must be remembered, however, that these special attitudes, which Ehret has very carefully studied, are only markedly apparent in severe cases, and are not always equally pronounced even in these. On the subject of scoliotic sciatica, see the review of Oberndörfer (*C. f. Gr.*, 1906).

The patient cannot usually sit or lie down for any considerable time. Coughing, sneezing, and straining are very apt to excite the pain, and are therefore anxiously avoided.

The sciatic nerve is seldom painful to pressure throughout its whole course. There are usually a number of *tender points*, of which the following are the most constant: one near the spine of the sup. post. ilium, one over the site of emergence of the nerve from the foramen ischiad. majus, one on the lower margin of the gluteus maximus, between the trochanter and the tuberosity of the ischium, one in the middle of the popliteal space, another below the head of the fibula, and often one upon the malleolus, etc. Tenderness of the sacral nerve to pressure is detected sometimes per anum or vaginam. In other cases there are no tender points whatever. Gara lays stress upon the tenderness to pressure of the spinous process of the fifth lumbar vertebra. The sensitiveness of the nerve may be demonstrated in another way: whilst the patient is lying on his back, the leg which is extended at the knee is flexed in the hip-joint; this is usually followed by pain on the posterior side of the thigh or in the gluteal region, when the leg is raised 1 to 1½ feet; but the pain often does not occur until the leg forms an angle of 90-100° with the pelvis. This pain immediately disappears when the leg is flexed against the thigh; it is therefore due to the stretching of the sciatic nerve. This *sciatic-phenomenon* (Laségue) [1] is of great diagnostic value. It may be elicited when the patient is sitting down by bringing the knee into the extreme position of extension. It may occasionally be induced in the leg of the unaffected side (Fajersztain).

Sensibility is not usually affected; in exceptional cases sensation is slightly diminished in the region of the posterior tibial or the peroneal.

This subject has been exhaustively studied by Dubarry (*Thèse de Bordeaux*, 1902-1903), Gauckler-Roussy (*R. n.*, 1904), and in particular by Lortat-Jacob and Sabareanu (*Rev. de méd.*, 1906). The last-named writers describe a number of cases in which the subjective complaints in sciatica corresponded to a sensory disturbance mostly in the region of the fifth lumbar, and the first and second sacral roots. They speak of a root sciatica, the pathological basis of which is not yet clear; apparently a syphilitic process in the region of origin of the roots is the most common cause.

The tendon reflexes are usually exaggerated on the affected side, sometimes on the other also, but the Achilles jerk may disappear on the side of the sciatica (Sternberg, Babinski,[2] Sarbó [3]). In many cases I have

[1] *Arch. gén. de Med.*, 1864. Also Beurmann, *Arch. de Physiol.*, 1884.
[2] *Gaz. des hôp.*, 1896.
[3] "Der Achillessehnenreflex," Berlin, 1903.

found that the Achilles tendon was flaccid and could be displaced with abnormal ease. It seemed to be sunken and reduced in size (Fig. 241).

Fibrillary tremors are occasionally noted, and in longstanding cases slight atrophy of the muscles. If, however, there is a qualitative change of electrical excitability (partial R. D.), a neuritis is present. Vasomotor disturbances and herpes are rare. Erben [1] mentions coldness of the skin in spots as a frequent symptom. At the height of the paroxysm the pain may radiate into other nerve tracts, even into those of the lumbar plexus, and muscular contractions and even clonic spasms of the legs and other symptoms, such as paresis of the gluteus maximus and the abdominal muscles, which point to an extension of the process to the plexus and beyond it, may appear. But this is very unusual.

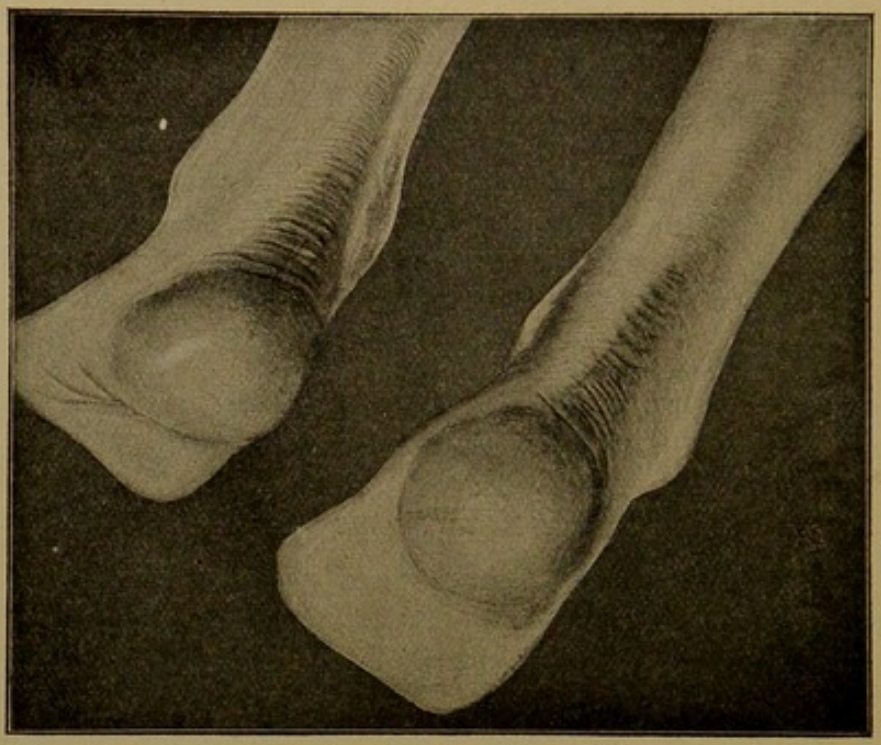

FIG. 241—Relaxation and diminution in size of the right Achilles tendon in right-sided sciatica.

Glycosuria has been found in a few cases of sciatica (Braun, Schiff). Polyuria may also occur (Debove).

Gonorrhœal sciatica is, according to Fournier and Lesser, characterised by the *fulminating* onset of the pain, the rapid course, and occasionally by intermittent fever.

I should like to draw special attention to the following complication, which may develop in the course of sciatica, not in hysterical women only, but also in previously healthy men, viz., a *reflex neurosis*, characterised by weakness, tremor, paræsthesia, and sensory disturbances of the type of hysterical hemianæsthesia in the whole half of the body on the affected side.

Differential Diagnosis.—Sciatica is a disease which is decidedly too often diagnosed. Many physicians go so far as to give the diagnosis of sciatica to any pain which is confined to one leg. The characteristic signs, viz., the pain along the course of the sciatic nerve, the tender

[1] *W. kl. W.*, 1894.

points, the pain on stretching the nerve, the motor disturbances to which the latter gives rise, etc. etc., have been mentioned above. On the negative side we would lay special emphasis upon the absence of paralysis, of marked anæsthesia, of degenerative atrophy, etc. Marked diminution of sensibility, especially when accompanied by reaction of degeneration, such as described by Nonne,[1] Guinon-Parmentier,[2] and Charcot, point to a neuritis which, when it is recognised, should be distinguished from neuralgia. Absence of the Achilles jerk on the corresponding side also points to a neuritis, but I would not reject the diagnosis of sciatica on account of this sign alone.

Pain in the sciatic nerve, when due to *affections of the spinal cord*, is hardly ever limited to one side. These affections also give rise to other symptoms, such as paralysis of the muscles of the extremities, weakness of the bladder, etc. etc.

Diseases of the *hip-joint* give rise to pains in the hip- and knee-joints which do not follow the line of the nerve. Movements in the joint are also painful, especially that of pushing the head into the socket, etc. Hoffa points out that in arthritis deformans the patient cannot separate the legs, whilst in sciatica abduction is unaffected.

For the symptoms of *nervous coxalgia*, see the chapter on hysteria. In *rheumatism of the lumbar muscles* (lumbago, etc.) the pain is diffuse, and there are no tender points, whilst kneading the muscles and pressure on their insertions is painful.

Minor (*D. m. W.*, 1898) has drawn attention to another point of distinction which is brought out by the way in which the patient raises himself from the ground. In lumbago he puts his hands out in front of him, supports himself on the ground by them, and then raises himself, somewhat like a person suffering from dystrophy (p. 239). Minor contrasts this with the backward pose of the sciatic patient, who always puts his hands behind him, and then pushes the pelvis slowly backwards, whilst he flexes the knee, brings the soles of the feet underneath the gluteal region, and thus passes the flexed lower extremities into the space between the hands; he then pushes himself up from the ground with one hand, balancing himself in the air with the other. Our experience is that this description is true as a whole, but departures from it are frequent, and we should not lay too much weight upon it.

Plantar hyperæsthesia should not be confused with sciatica. It is due to neuritis of the nerves of the sole of the foot, *e.g.* in alcoholism or gout, or it may be an hysterical affection.

Flat-foot may also cause a sensation of pain in the thigh and hip (Ehrmann), but this is to be differentiated from sciatica. Pain of indefinite nature and distribution occurs in the legs in persons suffering from *hæmorrhoids* or varicose veins. This is due to reflex irritation of the sensory nerves, and perhaps also to dilatation of the veins in the spinal canal and pressure of the varicose veins on the emerging roots within the intravertebral or sacral foramina. The symptoms may, however, entirely correspond to those of sciatica (see above). Tender points are usually absent in the sciatic pain caused by compression of the nerve. In the later course also, signs of interruption of the continuity in the nerve may appear. A thorough examination of the pelvic organs (per vaginam et per rectum), which should never be neglected, will give the diagnosis.

Hysterical sciatica will be recognised by the indefinite character of the pain and its localisation, as well as by the influence of mental factors.

[1] *B. k. W.*, 1887. [2] *Arch. de. Neurol.*, 1890.

In one of our cases of this kind the intractable pain disappeared after a fit of weeping.

From a diagnostic point of view we must refer also to *intermittent claudication*. The condition was first observed in horses (Boulay). Charcot [1] then described it in man. It consists in a sensation of numbness, fatigue, and pain, which in walking comes on in one or both legs and increases with exhaustion, so that walking is at first difficult and then—often after a quarter of an hour or even a few minutes—quite impossible. The symptoms disappear during rest, and the patient can walk again after an interval. It is only in the later stages that an attack of pain may come on even during rest, or that the pain may be persistent. The sensation is sometimes felt in the whole leg, sometimes only in single segments, such as the calf, the thigh, less frequently along some particular nerve. One of my patients repeatedly stated that the symptoms came on only when walking outside, and not in the house (see below). *Vasomotor* symptoms, cyanosis, pallor, feeling of cold, are almost always present. They tend to come on chiefly in walking. I recommend the patient to walk for a time and then to look quickly at the sole of his foot ; it is found as a rule to be waxy-white on the affected side. I have only once seen œdema as an accessory symptom. The local bloodlessness produced by pressure of the finger disappears with unusual slowness (Zoege-Manteuffel). Slight hypæsthesia is sometimes found on the toes or the sole of the foot. The tendon reflexes are as a rule quite normal, but the knee-jerk may be exaggerated, whilst the Achilles jerk is sometimes absent, as in three of my patients. There is usually either *arterio-sclerosis*, calcification of the arteries (Saenger, Holzknecht [2]), or *obliterating endarteritis*, and the weakness or absence of pulsation at the arteries of the affected extremities is very striking (Charcot, Bieganski). Whilst these authors laid most stress upon the absence of pulsation at the *great* arteries, Erb has shown by careful investigations that one of the most important and constant symptoms is *absence of pulsation in the arteries of the foot*, viz., in the dorsalis pedis and posterior tibial arteries. In healthy individuals the pulse can almost always be felt in these vessels, although some skill and care may be required to detect it. I would advise that it should always first be localised by sight, as I have often found it easier to recognise the pulse in this way than by touch.

Erb regards chill (forced cold-water treatment by Kneipp's method), syphilis, *chronic nicotine poisoning*, alcoholism, and gout as the chief causes of the condition. His patients have almost exclusively been men, but I have treated women also for it. According to Erb's latest statistics, 120 of his patients were men and only seven women. The *neuropathic* diathesis has already been considered by Erb and Brissaud, and I have also been able to show the pre-eminent part played by this factor in the etiology. I have found the disease very frequently combined with neuroses or psychopathic conditions, and occuring in persons in whom the stigmata of degeneration, *e.g.* medullated nerve fibres in the discs,

[1] *Comptes rendus et Mém. de la Soc. de Biol.*, 1858 ; *Gaz. méd. de Paris*, 1859 ; also *Prog. méd.*, 1887. Further literature : Erb, *Z. f. N.*, xiii. ; Goldflam, *D. m. W.*, 1895, *N. C.*, 1901 and 1903 ; Oppenheim, *Z. f. N.*, xvii. ; Erb, *Verhandl. d. Kongr., f. inn. Med.*, 1904 ; *ibid.*, *M. m. W.*, 1904, *Z. f. N.*, xxix. and xxx., *Mitt. aus. den Grenzg.*, iv., *D. m. W.*, 06 ; Higier, *Z. f. N.*, xix. ; Hagelstam, *Z. f. N.*, xx. ; Saenger, *N. C.*, 1901 ; Idelsohn, *Z. f. N.*, xxiv. and xxxii. ; Zoege-Manteuffel, *Mitt. aus. Grenzg.*, x.

[2] *W. kl. R.*, 1903.

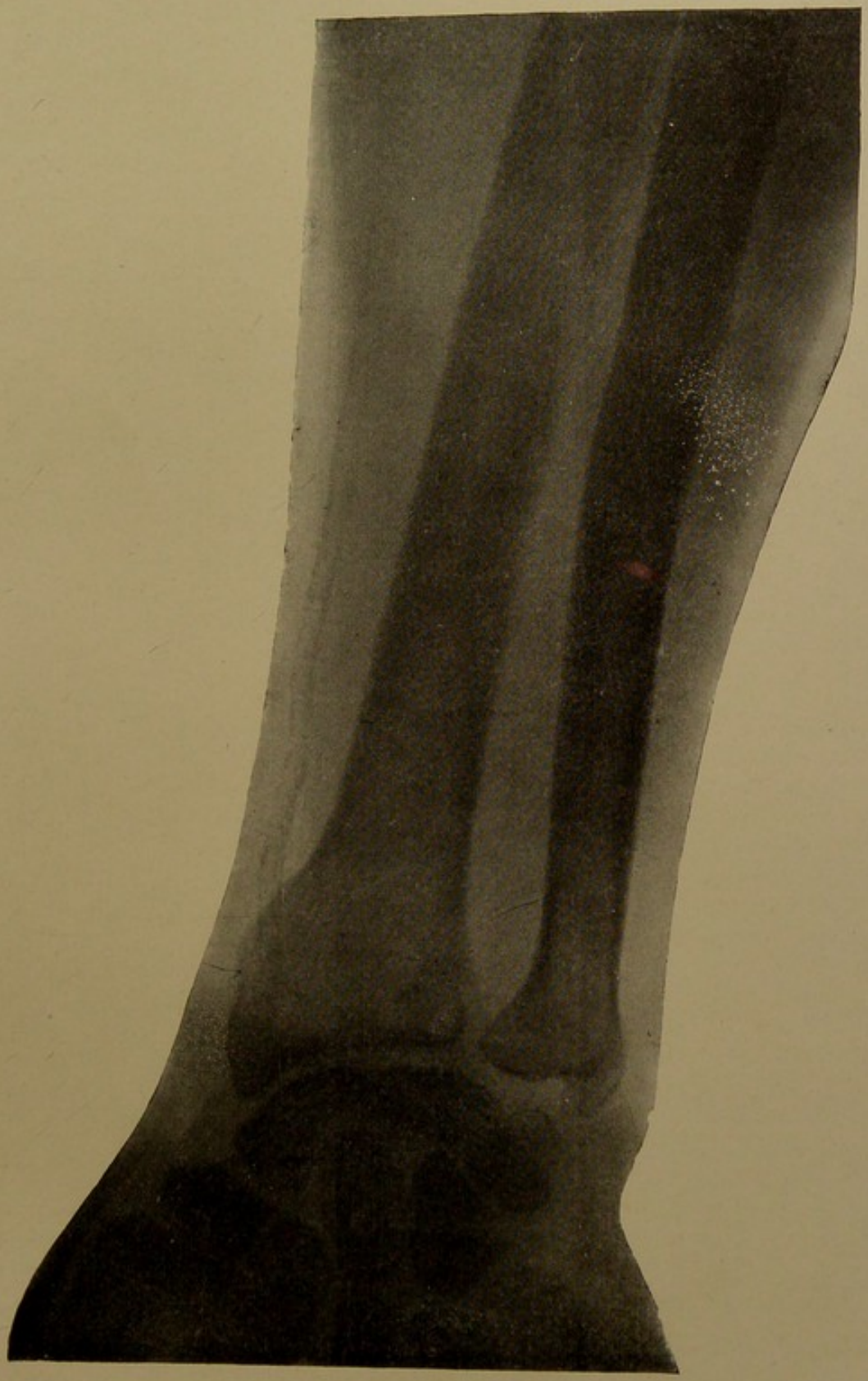

Fig. 242. (After Beck.)

Otto Schulze & Co., Edinburgh.

pigmented discs, malformation of the fingers, etc., pointed to the congenital disposition. My experience has been confirmed by Goldflam, Higier, Idelsohn, and others. This also explains the facts that the Jewish race shows a special tendency to this disease, and that it has sometimes a family character. Incongruity between constitution and climate, *e.g.* the weakly constitution of the Jewish race and the harsh climate and severe cold of the Russian winter, may also play a part. Saenger and others think that overstrain of the legs may be of etiological importance. Pal, Idelsohn, and others regard flat-foot as a predisposing factor. I have once seen the condition brought on in a patient with a gouty diathesis by Müller's gymnastic exercises. Charcot ascribed the condition to narrowing of the great arteries (iliac, crural) caused by arterio-sclerosis or obliterating arteritis, which gave rise to defective muscular nutrition. Erb has confirmed this. Cases of a similar kind are reported by Barth, Zoege-Manteuffel, Hagelstam, etc. I usually, though by no means always, find symptoms of general arterio-sclerosis. I have seen the disease occur in a man whom I had treated a year previously for symptoms of an encephalomalacia of the medulla oblongata, due to arterio-sclerosis. I have also observed a combination with hemiplegia.

In another of my patients this connection was revealed by the fact that a paralysis of the fourth nerve developed during the illness. Marinesco found marked changes in the muscles due to obliterating arteritis. The disease often forms a prodroma of gangrene.[1] It is sometimes, although rarely, related to diabetes. Whether an arterial spasm caused by vasomotor disorders may give rise to intermittent claudication is doubtful. I expressed my opinion in the following words in the second edition of this work, upon a question which, I think, I have by my later experiences been able to determine definitely: There is a *benign* form of intermittent claudication due to *spastic vascular conditions*, and not to an organic disease of the wall of the vessel. The fact that the majority of my patients have a neuropathic or psychopathic diathesis, and that gangrene remained absent although the illness had lasted for years and decades, compelled me to this conclusion. The remarkable influence of mental processes in producing the attacks of pain also supports this view. I further suggested that *congenital narrowness of the arteries* or congenital weakness of the vascular system, which led to its premature deterioration, might tend to produce the disease. The experience which I have acquired since then favours this view. I do not, of course, thereby exclude the possibility that the *functional angio-spastic form* may sooner or later develop into the *organic* form, in accordance with our experience that an arterio-sclerotic process or an obliterating arteritis may develop from vasomotor disturbances. Thus in one of the cases upon which I base my opinion, gangrene developed after fifteen years, and in two others, in which X-ray examination at first showed normal conditions, I found calcification of the arteries two and five years later. This is a typical example of the diseases which I term neuro-vascular.

Although the importance of the neuropathic diathesis had already been recognised by Erb, Brissaud, and Goldflam, the theory of the benign, angiospastic form of intermittent claudication

[1] Zoege-Manteuffel states the remarkable fact that he has observed this termination almost only in men, and Idelsohn notes that he has seen it almost exclusively in Jews.

and the hypothesis of congenital narrowness of the vessels was my own (*Z. f. N.* xvii.). I mention this because the facts have several times been misrepresented in the literature.

There are also atypical cases in which the patient succeeds, after the first pain is over, in walking for a considerable time without any pain (Erb); I have also learned this from a few patients.

It should be mentioned that in addition to the gangrene, a benign ulcer may appear.

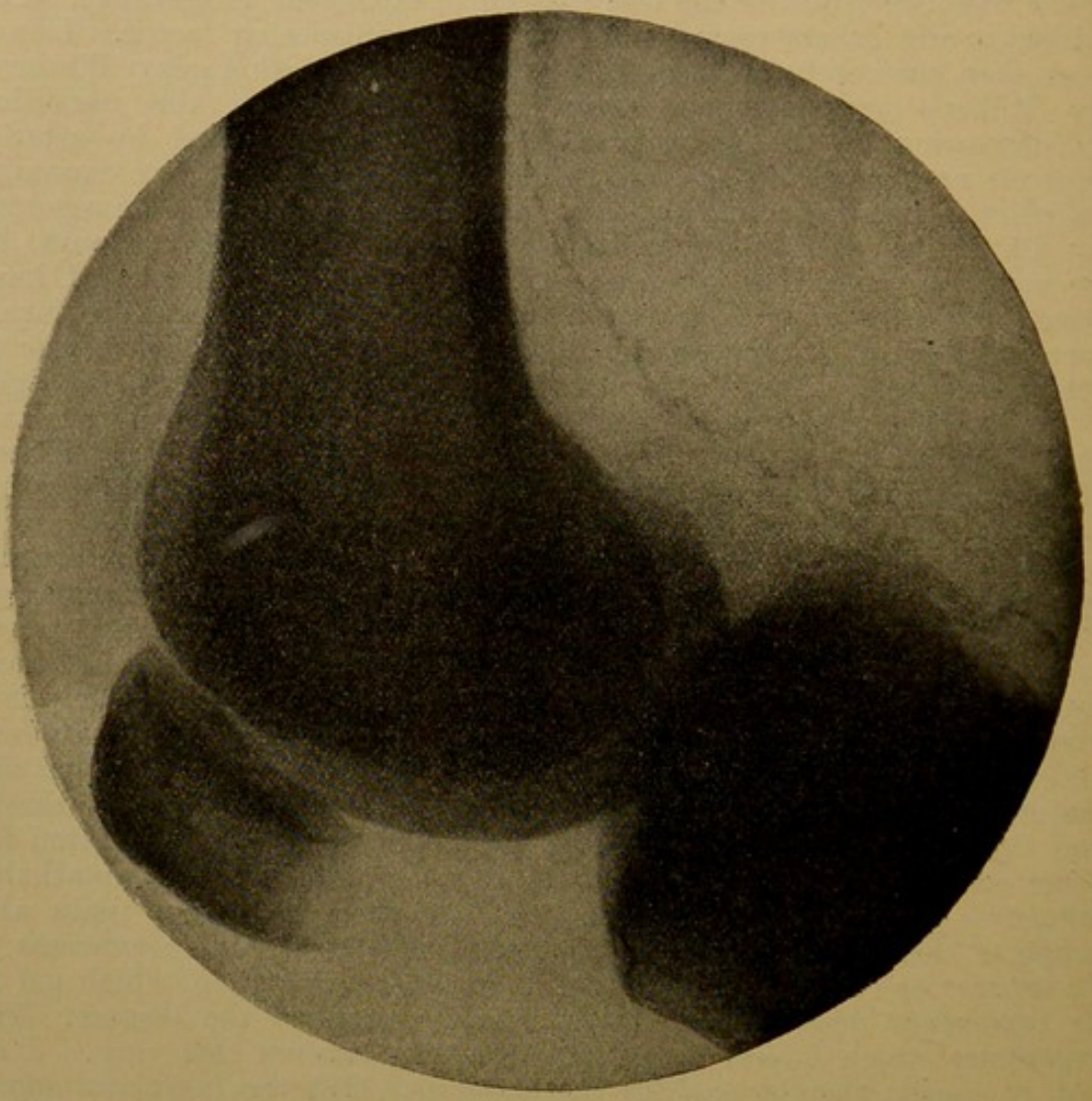

FIG. 243.—Calcification of the arteries in intermittent claudication. X-ray photograph. (Oppenheim.)

Radiography has been used to demonstrate the vascular disease by Beck, Saenger, Holzknecht,[1] Hunt,[2] and others. See Figs. 242, 243.

These writers regard the even deposit of lime salts, by which the vessels appear to be uniformly covered in their whole extent, as a distinction from arterio-sclerosis. My experience has also been that many things show that this process is not identical with the common form of arterio-sclerosis.

Unfortunately this grave disease is still often unrecognised; I have seen only a few cases in which the diagnosis had been rightly made. Two of my cases had been diagnosed by a cele-

[1] Fuchs und Holzknecht, *W. kl .R.*, 1903.

[2] *Med. Rec.*, 1905; also Freund, *Wien. med. Presse*, 1904; Bernert, *W. kl. R.*, 1904.

brated orthopædist as flat-foot, which was present in a slight degree, but which did not cause the troubles. The patient was allowed to drink and smoke, and the condition was thus steadily aggravated. What may not be covered by the good old flat-foot!

The application of Esmarch's bandage, recommended by Moskowicz (*Mitt. aus d. Grenzgeb.*, xvii.) for the detection of occlusion of the arteries, may be also used with care for the diagnosis of intermittent claudication, if the criteria given above are not sufficient.

The term of dysbasis intermittens arteriosclerotica, proposed by Erb, does not seem to me very happily chosen. This is even truer of the name selected by Walton and Paul—angina cruris (in analogy to angina pectoris).

We should also remember that arterio-sclerosis may give rise to local pain (Nothnagel, Laache, Lang, Edgren, Markwald, Lapinsky).

It is definitely proved that a syndrome of a similar kind may appear in other parts of the body, caused by corresponding vascular processes in other regions.

There is an intermittent claudication *of the arm*, of which I have seen several instances; in one of my cases (see the *Dissertation* of S. Kahn, Berlin, 1905) the radial pulse was completely absent on the affected side. As both legs were also affected, a kind of triplegia appeared when the limbs were used.

Cases of this kind have been reported by Nothnagel, Goldflam, Massaut, Wedenski, Erb, Determann (*Z. f. N.*, xxix.). Pässler (*N. C.*, 1906) describes an interesting case. There are forms of angina pectoris which in their origin and symptomatology are quite analogous to this disease.

Similar conditions also occur in the *intestinal arteries*; their symptomatology has been specially described by Ortner.

The fact that the *cerebral arteries* may also be the seat of this process is of great interest. The first case of this kind which I met, has been described under another diagnosis by Determann, to whom I handed the patient over. The correctness of my interpretation was confirmed by the subsequent course. Grasset[1] and Dejerine[2] have specially described this condition, but the former extends the conception much too widely. Rosenfeld[3] reports symptoms of this origin in the optic nerve.

Achillodynia (Albert,[4] Schüller[5]) can hardly confuse the diagnosis. Here intense pain occurs at the point of insertion of the Achilles tendon in walking and standing. Swelling often appears at that point. The condition has been attributed to a bursitis. It may develop after gonorrhœa, or from malaria, gout, or trauma. Schanz describes a typical form of the latter kind caused by pressure of the upper edge of the shoe. Similar symptoms also appear in the heel (talalgia, tarsalgia), partly from localised morbid processes, partly without any objective condition. Myositis of the calf muscles, occurring particularly after overstrain, may occasionally give rise to a wrong diagnosis.

The nature of so-called *Morton's*[6] *metatarsalgia*—intense pain in the region of the fourth metatarso-phalangeal joint—is still unexplained. Women are specially affected. It has been usually attributed to wearing narrow boots and shoes, which press on the nerves of the foot, *i.e.* on the periarticular nerves, overstrain from prolonged standing, the gouty diathesis, general nervousness, etc. It was then shown that the fifth metatarsal bone is peculiarly mobile and that its head does not extend so far forward as that of the fourth, so that in a lateral compression of the foot the former is curved over the latter and thus exerts pressure upon it.

[1] *R. n.*, 1906 ("La Claudication intermittente des Centres nerveux). [2] *R. n.*, 1906.
[3] *D. m. W.*, 1906. [4] *W. m. Pr.*, 1893. [5] *Ibid.*
[6] *Med. Surg. Rep.*, 1896, and *Intern. Med. Mag.*, 1896.

Morton and Peraire-Mally have shown this subluxation by skiascopic examinations. In another case (Pantolini) a rarefying ostitis of the metatarsal head was demonstrated by this means. According to John and Tubby,[1] the head of the fourth metatarsal lies lower on the transverse section than the other metatarsal bones, and moreover a nerve twig runs obliquely over it, which may therefore be easily compressed and affected by neuritis. The condition has also been regarded as true neuralgia of the external plantar nerve, or neuralgia of the joint (Fuchs). Neurofibroma, varicose veins, etc., have also been blamed. The condition seldom occurs at the joints of the other toes.

The various forms of pain in the sole of the foot, their causes and treatment, have been exhaustively dealt with by F. Francke (*D. m. W.*, 1904), and Idelsohn (*Pet. med. Woch.*, 1905) studies the same question. From the diagnostic point of view we should remember also the chronic rheumatic lymphangitis described by Wilms (*Beitr. z. kl. Chir.*, Bd. l.).

Course and Prognosis.—Sciatica in many cases assumes a rapid and favourable course and recovers completely in a few weeks or months. In others, however, it proves very obstinate, lasts for a year or more, has many fluctuations in its course, and leaves, even after recovery, a marked tendency to relapses. The prospects are best in recent rheumatic and slight traumatic cases. The general condition and the situation of the patient are also of special influence. If he can at once do what is requisite for his recovery, and can spare himself in every way, a rapid cure may be looked for. The prognosis is grave in longstanding cases, and in senility, and if there is no primary cause which can be removed.

Treatment.—Rest and care are urgently indicated; a comfortable position must be found in which the nerve is protected, and hard seats must be avoided.

In recent cases diaphoretic measures are advisable—a vapour *bath* followed by packing. *Blood-letting* (leech, cupping) over the chief tender point may have a wonderful effect. Cold cannot usually be borne, whilst *hot* fomentations are often soothing—hot-water applications, as by Siegrist's [2] method, hot sand-bags, peat fomentations, local and general fango baths, electric-light baths, hot-air treatment after Tallermann, Greville, Lindemann, Neumann, etc., or with the apparatus of Kiefer-Kornfeld, Hilzinger-Reiner, etc. All these methods are, it is true, uncertain in their results, but they have often a wonderful and even a curative effect. Preissnitz's pack, the Scottish douche, and a hot steam-spray of one to three minutes, alternating with a cold spray of five to twenty seconds' duration, have been used by myself and others. Local spraying with methyl chloride or ethyl chloride and external application of bisulphide of carbon and condensed carbonic acid over the painful parts have also been recommended.

Counter-irritants are greatly to be commended, especially fly-blisters, which may do good even in cases of long-standing, and large fly-blisters applied successively or simultaneously over the tender points.

[1] *Lancet*, 1896, and *Ann. of Surg.*, 1898.

[2] A towel folded lengthways so that it forms a compress 10 cm. broad, is immersed in hot water of 40° to 50° R., wrung out, and laid along the affected nerve; over this is placed a broad strip of flannel, which covers the towel, and above this again several layers of paper. The fomentation is renewed after 10 to 15 minutes, and this process is kept up for 1 to 2 hours, several times a day. For other such methods see E. Sommer, *Z. f. physik. und diät. Th.*, ix., and *Würzburger Anhandlungen*, vi.

Ehret states that he applies these blisters over an area 20 to 30 cm. long and 4 cm. broad, along the nerve, with very satisfactory results.

Cauterisation with concentrated hydrochloric acid, and with nitrate of silver, are recommended for producing cutaneous irritation. The button cautery produces a stronger effect ; superficial blisters are raised with the hot iron above the sciatic nerve, and especially over the tender points. It is not advisable to keep up the suppuration too long. Cauterisation at distant sites, even on the lobe of the ear, was among the methods of the old school. Empl. oxycroceum (Pharm. Belg.), which produces an eczema on the skin, is an old remedy which should not be discarded. Williams' porous plaster may also render good service.

If the diaphoretic methods have failed, we may at once, or after trying counter-irritation, resort to *electricity*, which is almost unanimously regarded by the profession as a valuable method in these cases. Galvanic electricity is the most useful form. One large electrode is placed upon the nerve at its point of emergence, the other upon one of the peripheral tender points, in the popliteal space, the calf, etc. Currents of medium intensity are usually required, although some writers recommend weak, others strong applications. It is well to commence with the weaker currents and to vary the strength according to the result. One may modify the treatment by moving the electrode gradually along the nerve so that a portion of it is always directly under the current. It is also advisable to apply one electrode to the gluteus maximus above the great sciatic foramen, and the other opposite to it in the lumbar region, sending a strong current through them. Stanowski has achieved great success by using large electrodes and prolonging the application for a half to a whole hour.

The faradic brush is not more efficacious than the other modes of cutaneous irritation, but it permits of the stimulation being renewed daily, and it causes no discomfort. Very strong currents are necessary for any effect. Energetic faradisation of the muscles has been successful in longstanding cases. Bernhardt and others have employed Franklinisation with advantage. Cataphoric treatment and constant wearing of a galvanic element may also be suggested. Krefft approves of the magneto-electrical method.

Massage deserves special commendation. It is particularly good for chronic, slowly yielding forms. It is often also of great use in recent cases, although it is too frequently forced, and the nerve which is already injured becomes further irritated. It is advisable to begin with gentle rubbing and kneading, and to limit this as much as possible to the neighbourhood of the nerve. In the rheumatic and gouty forms massage is often splendidly successful. Negro recommends treatment by means of compression of the tender points, especially of the one on the sciatic notch, and Arullani has constructed a special apparatus for the employment of this method.

Cramer (*Z. f. ortho. Chir.*, xiv.) recommends that the extremity be fixed by means of plaster of Paris bandages in the flexed, abducted, and externally rotated position. In obstinate cases Schmidt (*M. f. U.*, 1906) has obtained good results from extension by weight.

As to treatment by drugs, we may mention the anti-neuralgics : salicylate of soda, ol. terebinth. (best given in capsules of 1·0, three times a day), arsenic, quinine, antipyrin, and phenacetin. In some cases (even

where syphilis is not detected) *iodide of potassium* had a good effect. Glorieux has been successful with copaiba balsam (40-60 drops in capsules per day), and Klemperer with methylene blue (0·1, 3 to 6 times a day). Harburn recommends the following combination: aspirin 0·4, phenacetin 0·3, salicylate of quinine 0·1, and codeine 0·015-0·03. Subcutaneous injection of osmic acid, carbolic acid, antipyrin, and methylene blue (0·01-0·08, after Ehrlich and Leppmann) are advocated. The osmic-acid solution must be freshly prepared. In these injections the drug must be brought as nearly as possible into direct contact with the nerve. But this method has produced severe neuritis (Kühn, Dopter, Fischler, etc.).

During late years the method of injection of large quantities of fluid into the nerve or its immediate neighbourhood, described on p. 559, has come greatly into favour. Its value has been specially extolled by Lange, who is supported by Kellermann, Umber, Schlösser, Alexander, Krause, Strümpell-Müller, etc. Lange injects 70-80 c.cm. of a 1‰ β-eucaine + 8‰ chloride of sodium solution; others use, instead of the eucaine, tropococaine, or a simple solution of chloride of sodium. Strümpell and Müller and P. Krause use the following mixture: stovain 0·1-0·2, suprarenin. hydrochlor. (sol. 1 : 1000) gutt. x (-xx) aq. dest. ad. 100·0. F. solut. steril. per ½ h. The fluid is then injected by means of a syringe containing 10 c.cm. and a needle of 8 to 10 cm. in length at the chief pressure-point, *i.e.* at the site of emergence of the nerve from the great sciatic foramen, between the trochanter and the tuberosity of the ischium. This is done after the skin is disinfected and anæsthetised by Scleich's method, by placing the point of the needle perpendicularly upon the skin, and whilst gently ejecting a few drops of the fluid pressing it deeper down until the patient winces and thus shows that the nerve has been reached. The contents of the syringe are then emptied (see Fig. 244), and the patient is kept lying down for a few hours.

For the details of the technique, see also Bum, *W. m. Pr.*, 1907, Summary in *N. C.*, 1907.

Schlösser has cured thirty-six out of thirty-eight cases by this method. F. Schultze also reports success. The rise of temperature which sometimes follows the injection is transient and of slight importance. Unfortunately, however, a peroneal paralysis has followed in a few cases.

The investigations of Finkelnburg and of Brissaud, Sicard, and Tanon, have shown the dangers of alcohol injections.

I have had occasion to treat three cases in which perineural injections had been carried out elsewhere without any result, but in these the disease was of a severe form. In any case Lange's method of using osmic acid, carbolic acid, and other chemical substances is preferable.

For violent pain we have to use *narcotics*, but it is most advisable to abstain as far as possible from subcutaneous injections of morphia. If this is unavoidable, the injection should be made at the site of the pain. Atropin may also be tried. Nitroglycerine (1-5 drops of a 1 per cent. solution) is commended by Krauss. The methods of introducing soothing drugs into the subarachnoid, or better into the epidural space of the spinal cord, described on p. 174, have frequently been used with success in sciatica (Widal, Sicard, Maria-Guillain, Souques, Achard, Suffit-Delille, Broca, Magri, Durand, etc.), but they have recently been supplanted by Lange's method.

If there is constipation with hard masses of fæces in the intestine, it is advisable to give an aperient. A sufficient dose of castor oil may immediately cure the sciatica, but a single evacuation of the bowel is not generally enough to effect a cure. The constipation must be permanently overcome by suitable aperients or by a treatment at a watering-place.

To meet the causal indications it may be necessary to replace the uterus, remove a tumour, prescribe an anti-arthritic diet, or a rest-cure, etc. etc. Bruns saw recovery immediately follow straightening of a lateral twisting of the uterus.

Longstanding cases are often cured by the use of *indifferent springs*, or a course of treatment at Teplitz, Gastein, Wiesbaden, Wildbad, Warmbrunn, Baden-Baden, Pistyán, and Ragatz. Mud-peat baths and

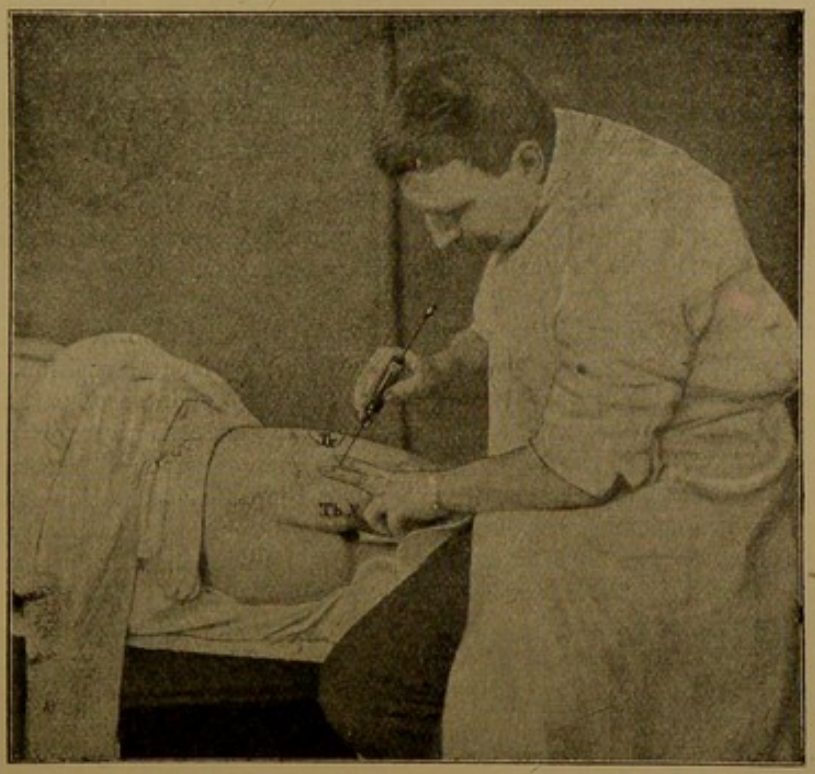

Fig. 244.—Perineural infiltration of the sciatic nerve. (After Strümpell-Müller.)

hot sand baths (of Köstritz) deserve to be mentioned. Hydrotherapy is also recommended. I have found it of special value in neurasthenic pseudo-sciatica.

Nerve-stretching or neurolysis may be tried as a last resort. This is specially suitable in certain forms of perineuritis, or in cases where the nerve is adherent to or embedded in cicatricial tissue. Good results are reported by Seeligmüller, Crawford-Renton,[1] Schede,[2] König, Barger, Hiltbrunner,[3] Halley,[4] Pers, and Bardenhauer.[5] The last named exposed the sciatic roots by partial resection of the sacrum or the sacro-iliac synchondrosis and embedded them in the muscles, with the idea that the compression was effected within the narrow bone canals. He recommends this method, which he names neurinsarcoclesis, as being very efficacious,

[1] *Brit. Med. Journ.*, 1898. [2] Handbuch by Penzoldt-Stintzing.
[3] "Die Ischias und ihre Behandlung," *Inaug. Diss.*, Bern, 1898.
[4] *Scot. Med. Journ.*, 1902. [5] *M. m. W.*, 1903, and *N. C.*, 1906 (ref.); *Z. f. Chir.*, Bd. lxvii.

Hölscher advises that the exposed nerve be sprayed with a 5 per cent. carbolic solution. I have been consulted in a case in which this method had caused a severe and apparently irreparable paralysis of the peroneal nerve. The surgeon was said to have used a concentrated carbolic solution. The case was all the more sad in that it had not been one of true sciatica, but merely of an hysterical ischalgia.

A kind of surgical laceration of the nerve (" hersage ") is recommended by French authors (Marty [1]).

The descriptions of the nature and the reason of this treatment are not clear. Some writers have been successful with suspension and bloodless stretching of the sciatic nerve. Bonuzzi's method (p. 172) may be used for this purpose. Another method is described and recommended by A. Lewandowski.

The advice of Lazarus (*Z. f. phys.* and *diät. Ther.*, x.) to stretch the extremity after it has been made insensitive by lumbar anæsthesia can hardly be followed so long as the method is combined with danger. Alexander advises that the leg should be stretched after it has been relaxed by means of Schleich's injection.

The scoliosis requires no special treatment. I have often noticed that the deformity disappears when the sciatica is cured.

In Morton's metatarsalgia, absolute rest with freedom from any pressure by the shoe is the first indication. A special shoe (Gibney) is also recommended, and in the most intractable cases the head of the metatarsal bone should be removed by operation. Péraire (*R. n.*, 1906) reports surgical cure.

As regards treatment of *intermittent claudication*, Erb has laid down the main indications as rest, care of the affected limb, which may be kept in a horizontal position, a diet suitable for vascular disease, avoidance of cold-water measures and also of intense heat, keeping the feet warm, and local application of galvanic foot-baths, etc. The latter is given by placing each foot in a different vessel containing warm or salt water, into each of which an electrode is dropped. A current of 5 to 15 MA. is then turned on in alternating directions—duration of 5 to 15 minutes. In one case treated with special perseverance and energy by an experienced layman, who used strong currents also, there was complete recovery. The galvanic foot-bath may also be used as a monopolar measure. The strength of the heart should be increased by tonics or strophanthus. The vascular process indicates the use of preparations of iodine (iodide of potassium, iodipin). Warm (but not hot) foot-baths should also be tried. Zoege-Manteuffel have lately advocated the careful use of massage, limited entirely to the thigh and not touching the foot. Idelsohn has tried Bier's congestive treatment.

According to Erb's experience and my own, the disease may be arrested or considerably improved.

Neuralgia of the Pudendo-Hæmorrhoidal Nerve

Neuralgia in the region of the pudendo-hæmorrhoidal plexus is rare, but very persistent. It is most frequently localised in the region of the spermatic cord and testicle. It is characterised by extremely intense pain, which runs along the spermatic cord to the testicle and epididymis, or from these along the spermatic cord into the lumbar region. These structures, and sometimes also the skin covering them, are so sensitive that even a light touch gives rise to severe pain (irritable testis). During the paroxysms there may be spasmodic contraction of the cremaster, contractions in the legs, and vomiting. Priapism and ejaculations may also occur. Periodic swelling of the testicle has been observed. This very intractable (and almost always unilateral) disease is the cause of marked depression and has even led to suicide.

[1] *Thèse de Paris*, 1897.

It cannot be said for certain whether the neuralgia is situated in the external spermatic nerve or in the sympathetic branches.

A suspender should be worn and electrical treatment and the well-known anti-neuralgic drugs should be prescribed. Firm compression of the spermatic cord by means of a pledget of cotton wool has also been tried. In desperate cases castration has been resorted to. In one case resection or neurectomy of the testicular nerve led to recovery. Chipault [1] has recently reported a case cured in this way. Patureau dealt with this question in his Thesis (Paris, 1901). In a case treated in this manner by Donath and Hüls the result was not permanent.

As regards the diagnosis we must guard against confusion with tumours (tuberculosis, etc.); the lightning pains of tabes may also chiefly affect the testicle. In many cases, such as one which I have recently had occasion to see, the disease was undoubtedly a mental one—a neurasthenic, hysterical, hypochondriacal pseudo-neuralgia.

An *ano-perineal neuralgia* (W. Mitchell [2]) has been observed in masturbators, but it also occurs in individuals who have never masturbated. In one case I induced recovery by means of suppositories of cocaine, in another every method failed: the patient acquired the morphia habit and wandered fron one institution to another.

Neuralgia of the rectum is the name given to a prolonged feeling of pain following evacuation of the bowels. Cases of this kind must be very rare. I have, however, treated a few persons who complained of an excessive feeling of weakness after each defæcation. Neuralgia of the bladder, urethra, prostate, and penis are also mentioned. These localised neuralgias occur in cyclists. Neuralgia in the region of the pudendo-hæmorrhoidal plexus is often accompanied by herpes.

Of the newer contributions to this question, see that by Albu, *B. k. W.*, 1907.

Neuralgia of the Coccygeal Plexus, Coccygodynia

The name of coccygodynia is given to a severe neuralgiform pain in the region of the coccyx, which occurs almost exclusively in women. The pain comes on spontaneously or in the act of sitting down, of walking, or of emptying the bladder and intestine, or it is increased by any factor which is associated with contraction of the muscles which are inserted at the coccyx. The coccyx is usually sensitive to pressure and to movement. The disease follows a severe confinement and trauma, but it may have a spontaneous onset. It has even been observed in children. It may develop without any exciting cause in hysterical persons.

In many cases it is a true neuralgia; in others it is due to an *inflammatory process* in the muscles inserted at the coccyx, or in the surrounding soft parts or bones. These conditions can usually be distinguished from neuralgia by careful bimanual examination. I have seen slight cases recover in a few days or weeks, *e.g.* under the influence of *opium suppositories*, others in which the refrigerating double current sound was helpful, and severe cases which defied all treatment and necessitated an operation (separation of all the soft parts from the coccyx, or its removal) (Simpson, König, etc.). Bryant,[3] Kidd,[4] and Swiecicki [5] were successful with this treatment.

[1] *R. n.*, 1900. [2] *Phil. Med. Times*, 1873. [3] *Dublin Journ.*, 1867.
[4] *Ibid.* [5] *Wien. med. Presse*, 1888.

When hysteria is present, psychotherapy must be employed. Seeligmüller[1] reports that within a short time he rapidly cured a coccygodynia of twelve years' standing by *faradisation* (one electrode in the vagina, the other on the coccyx). Others have used electrical treatment, especially the faradic current, with success.

All the cases that I have hitherto seen have been associated with hysteria or psychasthenia.

Visceral Neuralgias

Severe pain of neuralgiform character is often felt in the internal organs. It accompanies diseases of these organs themselves, or is due to a nervous disease. Thus neuralgic pain in the abdominal nerve is one of the symptoms of gastric crises. *Gastralgia, i.e.* attacks of intense pain in the epigastrium, radiating towards the back and sometimes associated with vomiting, may be one of the symptoms of hysteria, or may form an equivalent of hemicrania. But a true neuralgia of the abdominal nerves does apparently occur, even in healthy persons, although it is extremely rare. It must be treated by narcotics. In some cases belladonna gives a successful result, in others cocaine (0·003-0·01), codeine (0·01-0·06), and morphia. Bismuth subnitrate and nitrate of silver have sometimes a good effect. I have seen cases of this kind in which a malignant disease had been diagnosed and laparotomy had been decided upon, and which I cured by suggestive treatment or cataphoric electricity.

A pure neuralgia of the nerves of the intestine, liver, spleen, or kidneys has hardly ever been definitely observed, but these organs are not seldom the seat of severe pain in the functional neuroses, particularly in hysteria. Gowers reports a case in which periodic attacks of pain occurred in the kidneys during forty years, without any evidence of the presence of renal calculus. Cases of *neuralgia of the liver*, with symptoms of gallstone colic, have been described several times in the last few years. Subphrenic pain, which he localises in the cœliac plexus, is reported by F. A. Hoffmann.

Rénon mentions that pseudo-neuralgia of this kind may be produced by syphilitic aortitis.

According to Head the cutaneous pain which accompanies visceral diseases, and which is usually associated with hypersensibility of definitely localised areas of the skin, affects the areas of innervation of the corresponding segments of the spinal cord. These cutaneous areas derive their nerve fibres from the same roots or spinal segments from which the sympathetic nerves of the affected viscera also arise. The visceral pain is reflected into the tract of this cutaneous nerve. In herpes zoster the same areas are affected (see p. 109).

Tumours of the Nerves

For literature on the subject see: Recklinghausen, "Über die multiplen Fibrome der Haut," etc., Virchow-Festschrift, Berlin, 1882; Thomson, "On Neuroma and Neurofibromatosis," Edinburgh, 1900; Adrian, *Bruns Beitr. z. klin. Chir.*, xxxi., and Review, *C. f. Gr.*, 1903; Bruns, "Die Geschwülste des Nervensystems," 1st ed., 1897, 2nd ed., 1908; Hulst, *V. A.*, Bd. clxxv.; Fraenkel and Hunt, *Pub. of Cornell Univ. Med.*, 1904. Strauss discusses plexiform neuroma (*Z. f. Chir.*, Bd. lxxxiii.).

Various kinds of new growths may arise from the nerves or be localised in their region: *neuroma*, *fibroma*, *sarcoma*, *glioma*, *myxoma*, and *syphiloma*. Carcinomatous and syphilitic infiltration of the peripheral nerve is almost always secondary to extension of the tumour to the adjoining nerve. On Virchow's advice, we make a distinction between *true* and *false* neuroma, according as the nervous tissue (medullated and non-medullated

[1] *Neuropath. Beobachtungen.*, Festschrift, Halle, 1873.

fibres) plays an essential part in the construction of the tumour or not. Billroth, Ziegler, and others oppose this distinction; the latter in particular entirely denies the active participation of the nerve fibres in the formation of the tumour. According to this view there is no real neuroma, but merely *neurofibroma* (or fibroma, etc.), *neurosarcoma*, *neuromyxoma*, originating from the peri- and epi-neurium. More recent investigations (Petrén, Whitfield), however, and particularly the discovery of ganglion cells in one group of these tumours (Knauss, Busse, Beneke), confirm the existence, rare though it be, of true neuroma. Knauss[1] and also Adrian ascribe these to the sympathetic nervous system. Neurolipoma also appears.

Neuroma may be *solitary* or may have a *multiple* distribution. It develops on all the nerves, but mainly on the spinal. Multiple neuroma may be limited to the area of one nerve, *e.g.* to the brachial plexus, the cauda equina, the pelvic nerves, or it may extend to the nerves of the whole body. As many as 3000 have been found in one person. It sometimes shows a moniliform arrangement on the nerve (Fig. 245). The size of the neuroma varies from that of a pea to that of a child's or a man's head; on an average it is the size of a pea, a nut, or a pigeon's egg.

Fig. 245. — General neurofibromatosis. Bead-like arrangement of the neuroma along the cutaneous nerves of the right arm. The small nodules can be seen distinctly under the skin. (After Robert Smith; reproduced by Marie.)

It varies greatly in consistency; it usually feels soft, or like a cyst, or, as in one of my cases, like an empty sac. The tumour may indeed shrivel up, leaving behind an empty cutaneous pouch (Adrian). But in other cases I have found it almost as hard as cartilage, so that at first I suspected a cysticercus. Cystic formations and myxomatous degeneration take place in the tumours. Cysts with hæmorrhagic contents have occasionally been found.

The name of *tubercula dolorosa* is given to small tumours, the size of a millet seed or a pea, which are found either singly or in great numbers on the sensory cutaneous nerves. These can be distinctly palpated and are usually very painful. They are almost always *fibroma*, sometimes angioma. *Plexiform neuroma* (Rankenneurom, congenital elephantiasis) represents a congenital tumour which gives rise to a string and knot-like thickening and plexus-like disintegration and twisting of the nerve (Fig. 247). It occurs mainly on the *trigeminus*, but appears also on other nerves; it is extremely rare and does not necessarily give rise to any symptoms. The columns can be distinctly felt below the skin. We cannot here discuss the relation of this disease to pachydermatocele and elephantiasis mollis.

General neurofibromatosis (fibroma molluscum multiplex) or *Recklinghausen's disease* is a condition in which numerous fibromata are found

[1] *V. A.*, Bd. cliii.

on the branches of the cutaneous nerves, along with *neuromata* (also plexiform) on the nerve stems and *patches of pigment* (nævi) on the skin (see Figs. 248, 249, and 250). There is also diffuse pigmentation in addi-

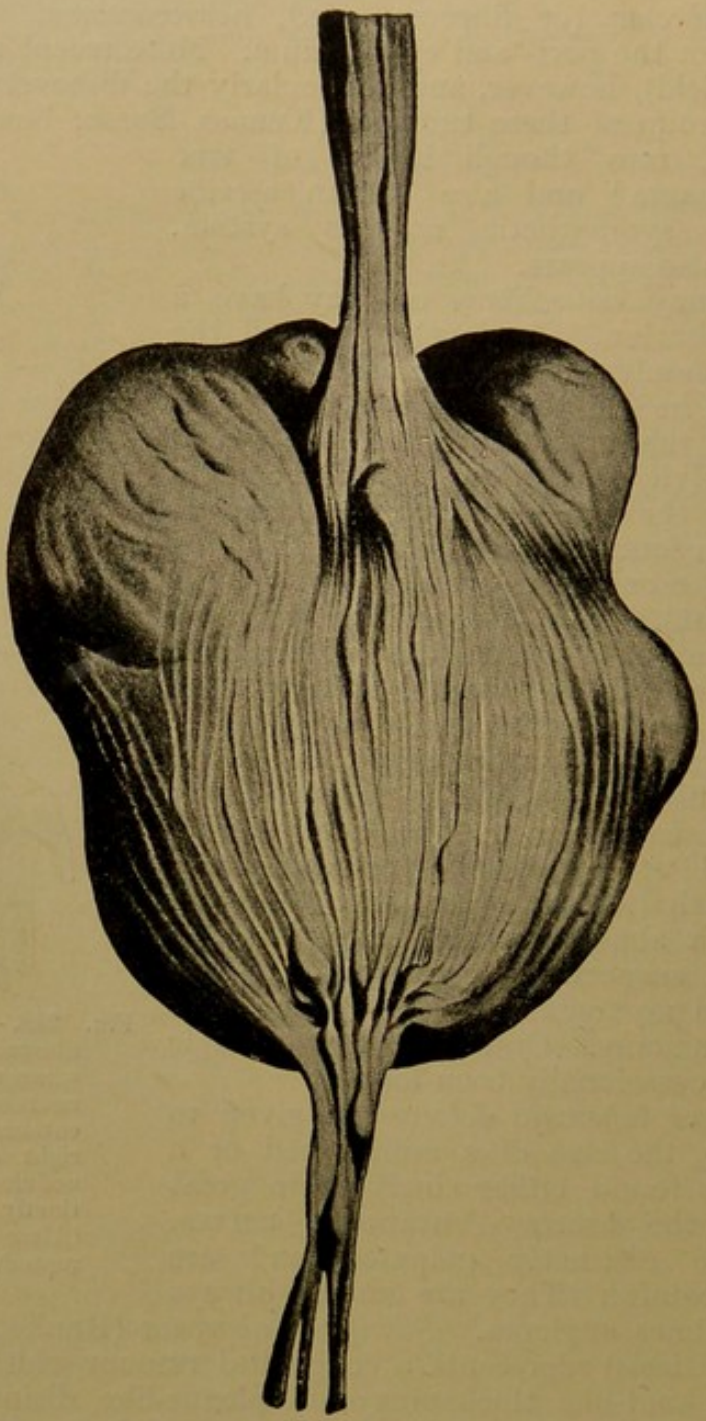

FIG. 246.—Neuroma of the sciatic nerve. (After Smith, reproduced by Bruns.)

tion to the pigmentation spots of various sizes. It extends less frequently to the mucous membranes, *e.g.* to that of the mouth, as described by Oddo (*R. n.*, 1905). The patches of pigment are sometimes accompanied by nævi vasculosi and angiomata. The neuromata are also sometimes found on the sympathetic and visceral nerves, especially in the mesen-

tery, peritoneum, in the intestinal wall, etc., and also on the tongue and mucous membrane (Büngner,[1] Knauss, Knoblauch, Gerhardt,[2] Berggrün,[3] etc.). The relation of the tumours to the cutaneous nerves has been shown by Recklinghausen. Incompletely developed forms of

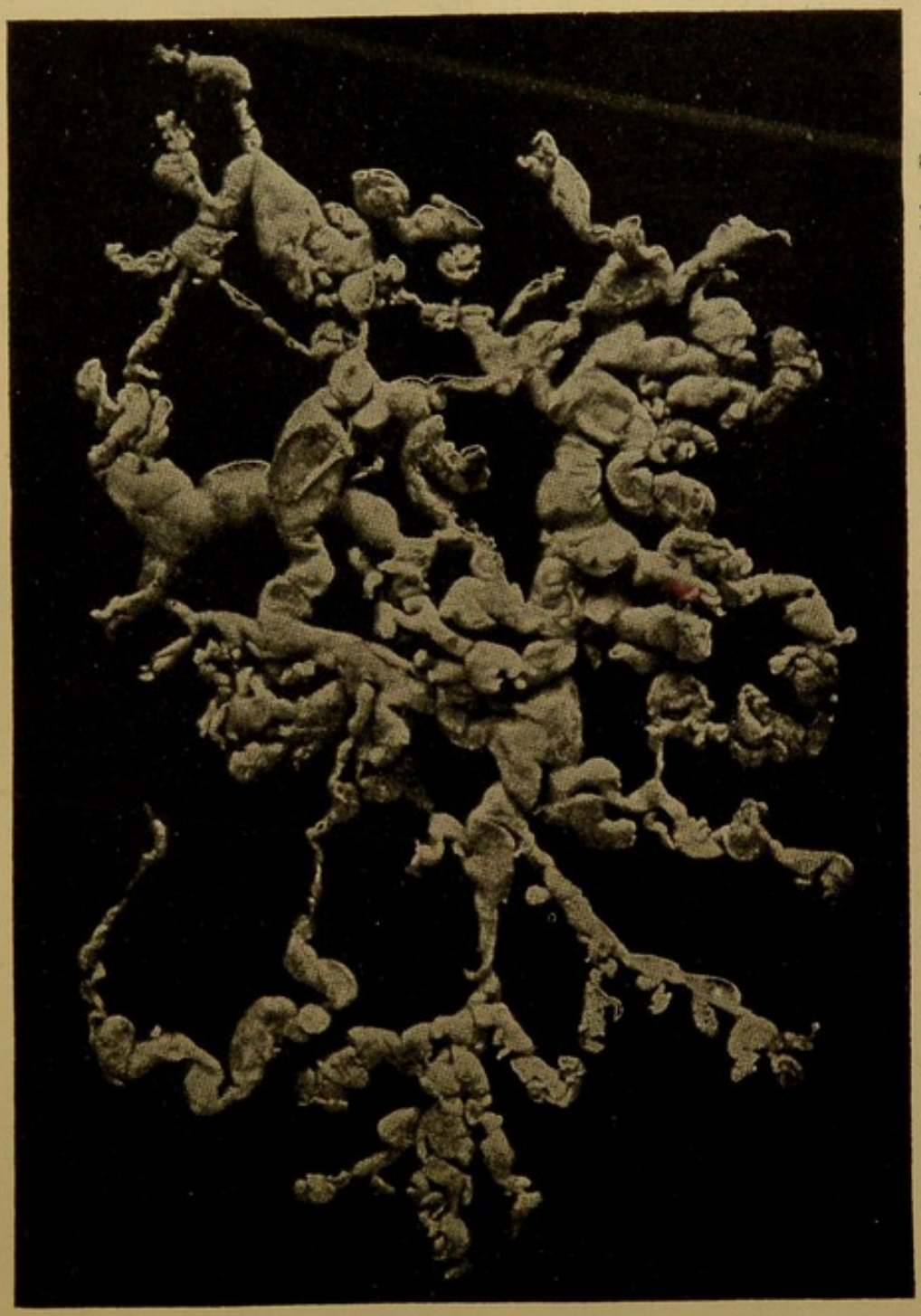

Fig. 247.—Plexiform neurofibroma of the median nerve. (After Thomson, reproduced by Bruns.)

Recklinghausen's disease also occur (Thibierge, Feindel-Oppenheim,[4] etc.). These include cases in which the spots of pigment are only accompanied by a tumour the size of an egg, others in which there is no pigmentation, or in which there is pigmentation associated with a mental disorder (see below). Alexis Thomson a short time ago published an exhaustive

[1] *A. f. kl. Chir.*, 1897.
[2] *A. f. kl. M.*, xxi.
[3] *A. f. Kind.*, xxi.
[4] *Arch. gén. de. méd.*, 1898.

Fig. 249.—Multiple neurofibroma in the left arm. (Oppenheim.)

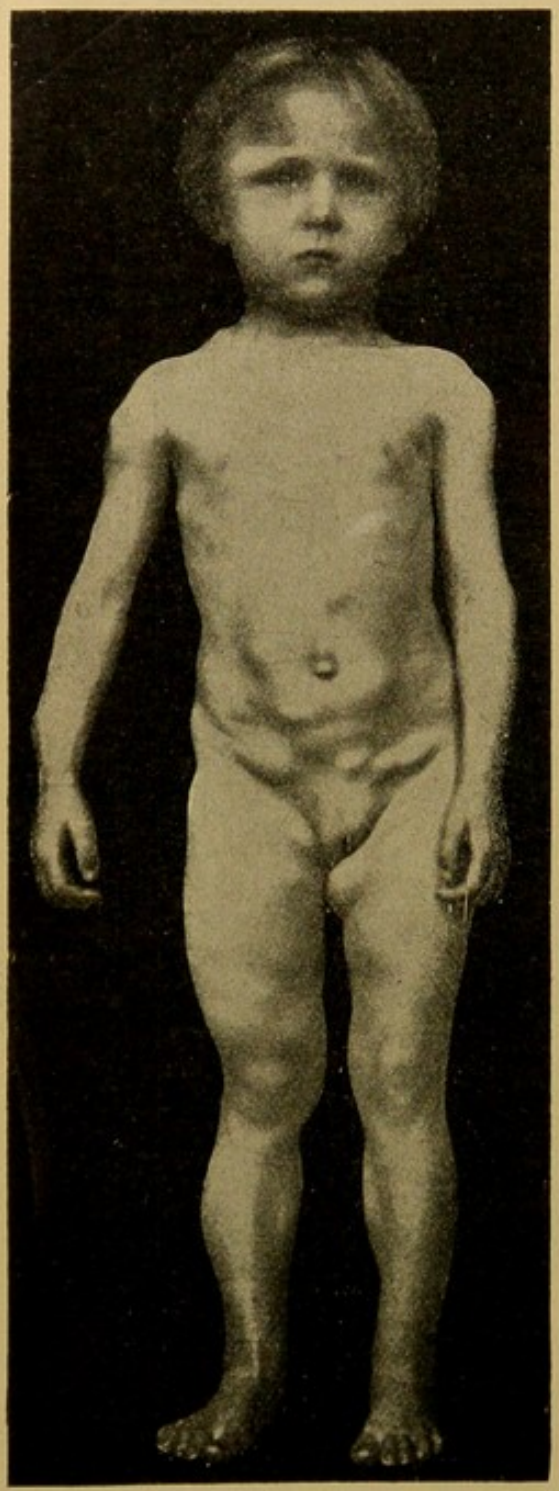

Fig. 248.—True multiple neuroma. (After Bruns.)

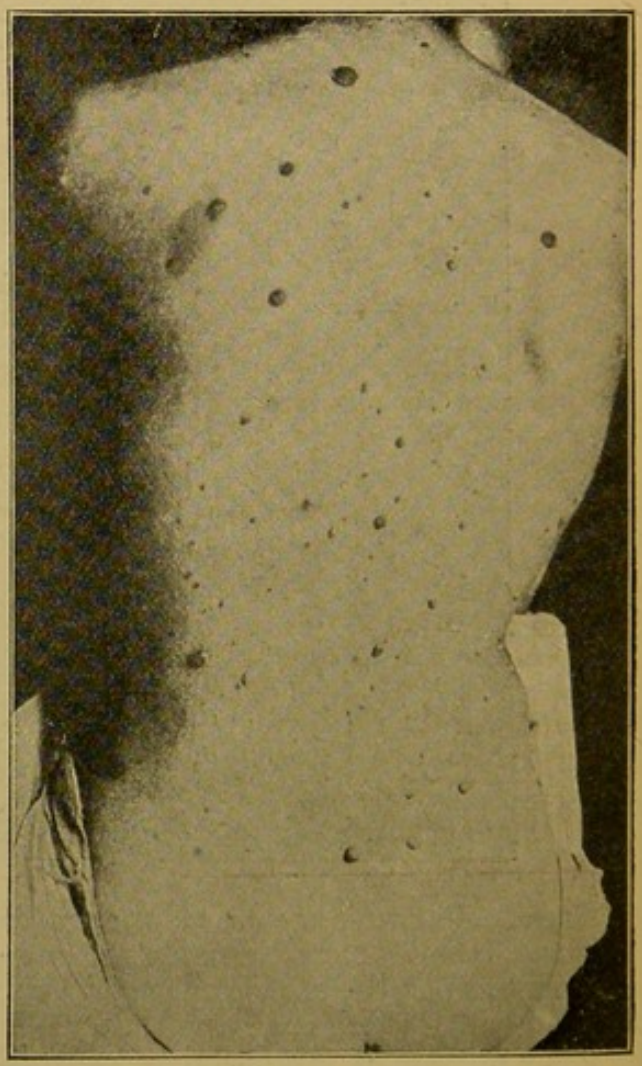

Fig. 250.—Generalised neuro-fibromatosis. (Oppenheim.)

exposition of our knowledge of neuroma and neurofibromatosis. We own to Adrian a summary and careful review of the whole subject.

We know little for certain as to the etiology of neuroma. Tuberculosis creates a predisposition to the condition, and trauma may give rise to it.

Multiple neuromata are as a rule of *congenital* origin. The *neuropathic diathesis* also plays a part in the etiology of these growths. The disease is thus often hereditary and familial, but it may develop during later life. Marie and Convelaire [1] saw it appear in the fifty-second year. It is frequently associated with a congenital mental defect.

Symptoms are either entirely absent, or they correspond more or less to those of *neuralgia* or *neuritis*. The neuromata may remain latent and be accidentally discovered during an examination or after death. They are frequently confused with lipomata. Even when the tumour presses the fibres of the nerve apart, there may be no signs of any interruption in conduction. In other cases these tumours manifest themselves by *intense pain* which arises at certain sites and radiates towards the periphery. There is also great *tenderness to pressure*, and when the growth is superficially situated one can feel that the nerve is swollen or has a mobile tumour upon it. If it arises from the nerve itself, it cannot, in contrast to paraneural tumours, be displaced in the direction of its longitudinal axis. This test frequently gives rise to pain. As a rule there is paræsthesia, sometimes hypæsthesia in the area of distribution of the affected nerve, but rarely paralysis and atrophy. Reflex local muscular contractions may also develop, and even general convulsions of the type of reflex epilepsy. I have once observed a combination of the disease with epilepsy, and more frequently with hysteria, hypochondria and neurasthenia. In the case of a gentleman who developed a tic in his seventieth year, I was struck with the absence of any etiology, until I discovered that he was suffering from a family neurofibromatosis. In another of my cases a kind of snoring spasm was present. Schüle [2] described a combination with universal alopecia. According to Thomson and Adrian, inhibitions of development of the most various kinds (idiocy, macroglossia, epispadia, cryptorchismus, etc.) have been observed in this disease.

The general condition is usually unaffected if the growth does not give rise to pain, or if the pain is slight. When there is severe neuralgic pain, marasmus may gradually develop. General neurofibromatosis is specially apt to bring on marasmus in the later stages. Mental disorders, sensory disturbances, and convulsions also occur in this disease (Marie). Alterations of the skeleton, especially deformities of the spinal column and thorax, have been noted by Jeanselme, Marie-Convelaire, Haushalter,[3] Lion-Gasne,[4] Sahut,[5] and myself, and have been traced to osteomalacic processes. Jeanselme [6] describes partial defects in the bones. Articular changes of indefinite genesis have occasionally been mentioned.

In one case of this kind I was struck by the fact that from time to time there was spontaneous swelling of the tumours (from vasomotor disturbances ?), which made them more distinctly visible and caused great discomfort. I learn from Adrian's work that this symptom has already been observed by Bazin and Tilenius, Hecker, and others.

[1] *Nouv. Icon.*, 1900.
[2] *A. f. P.*, xxxvi.
[3] *Nouv. Icon.*, 1900.
[4] *R. n.*, 1905.
[5] *These de Paris*, 1902.
[6] *R. n.*, 1905.

The symptomatology may be further extended by the simultaneous *onset of neuroma in the central nervous organs, or the roots of the cranial and spinal nerves*, especially of the cauda equina. Thus I have occasionally found the signs of a general neurofibromatosis in patients who came under my treatment with symptoms of a spinal tumour. The formation of tumours in or upon the central nervous system has been found by Soyka, Mossé-Cavalié, Hesselbart, Heller, Berggrün, Sorgo,[1] Oppenheim, Cestan,[2] etc.[3] Our experience in this respect has been considerably amplified during the last few years. The excellent observations of Henneberg and Koch [4] in particular have taught us that so-called neurofibroma of the auditory nerves (see chapter on cerebral tumours) not infrequently accompanies a general neurofibromatosis. I myself have seen eight cases in which the general neurofibromatosis was masked behind symptoms of brain tumour. It would appear from Adrian's account that these tumours may develop on any of the cranial nerves. They occur most frequently on the vagus, most rarely on the optic nerve. Functional disorders corresponding to the involvement of this nerve have not always been recorded, but B. Rubesch,[5] for instance, reports a case in which a large fibroma of the vagus caused tracheal stenosis and recurrent paralysis.

The symptoms may be produced chiefly by tumours of the internal organs, signs of a gastro-intestinal affection, of a compression of the vena cava, etc., being thus most evident. These and other symptoms, such as cachexia, have been attributed to involvement of the sympathetic. Askanasy and others describe multiple development of neuroma on the sympathetic plexus of the intestinal walls.

Although the formation of neuroma does not directly threaten life, these complications, in particular tumour of the brain or spinal cord, and the marasmus which frequently develops later in Rechlinghausen's disease, may lead to a fatal termination. It must also be remembered that neuroma not infrequently develops into sacroma. Multiple neuro-fibro-sarcomatosis is described by Cestan,[6] Raymond,[7] Hulst,[8] etc. The latter always insists upon its relation to multiple neurofibromatosis. Myxomatous degeneration may also occur.

The disease is as a rule a *slowly progressive* one, but it may be arrested, and the tumour has even been observed (Michel) and assumed (Henschen [9]) to disappear. On the other hand it creates a certain predisposition for the development of malignant tumours at other sites.

If the tumour is isolated and accessible, its removal is indicated, in case it may give rise to grave symptoms but it should be remembered also that malignant degeneration has frequently followed excision of a single nodule (Garrè). Where it is feasible, the nerve stumps should be united directly or after the methods described on pp. 415 *et seq.* In a few cases excision of a large portion of the nerve has not given rise to symptoms of paralysis or these have rapidly disappeared. The tumour is sometimes so slightly adherent with the nerve that it can be separated from it without interrupting its continuity.

[1] *V. A.*, Bd. clxx. [2] *R. n.*, 1900.

[3] We leave out of the question here, the so-called true neuroma of the spinal cord (small tumours from the medullated nerve fibres in the posterior horn), such as are described by H. Schlesinger, Raymond, and others.

[4] *A. f. P.*, xxxvi. [5] *Prag. med. Wochnschr.*, 1903. [6] *R. n.*, 1903.

[7] *Semaine méd.*, 1903. [8] *V. A.*, 1904. [9] *Mitt. a. d. Grenzgeb.*, xi.

We would refer to Adrian for an account of the differential diagnosis from other multiple cutaneous tumours, leprosy, etc. As regards its symptomatological relationship with hypertrophic interstitial neuritis, see p. 255.

If we are dealing with multiple tumours, it is always advisable to remove those which cause the most serious trouble. In malignant tumours, however, these symptoms usually re-appear. Apart from operation we must confine ourselves to *symptomatic* treatment, and to alleviation of the pain. We should, however, point out that according to the observations of some physicians (M. Meyer), the *galvanic current*, used percutaneously, has effected the resolution of these tumours (?).

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