CASE STUDIES IN ANATOMY

THIRD EDITION

ERNEST LACHMAN, M.D.

CASE STUDIES IN ANATOMY

THIRD EDITION

ERNEST LACHMAN, M.D.

Late Regents Professor Emeritus of Anatomical and Radiological Sciences University of Oklahoma College of Medicine

In collaboration with

KENNETH K. FAULKNER, Ph.D.

Professor of Anatomical Sciences University of Oklahoma Colleges of Medicine and Dentistry

NEW YORK OXFORD OXFORD UNIVERSITY PRESS 1981

Copyright @ 1965, 1971, 1981 by Oxford University Press, Inc.

Library of Congress Cataloging in Publication Data Lachman, Ernest, 1901–1979

Case studies in anatomy.

Bibliography: p. Includes index.

1. Anatomy, Pathological-Case studies.

I. Faulkner, Kenneth Keith, joint author.

II. Title. [DNLM: 1. Anatomy, Regional.

2. Pathology. QZ4 L138c]

RB31.L3 1981 616′.09 80-18696

ISBN 0-19-502813-9 (pbk.)

Printing (last digit): 9 8 7 6 5 4 3 2 1

Preface to the Third Edition

Late in 1978, Dr. Ernest Lachman asked me to collaborate with him in preparing this third edition of *Case Studies in Anatomy*. Much of the revision work had been completed when, on September 21, 1979, Dr. Lachman passed away, leaving the completion of this task in my hands. I hope I have accomplished this in a manner that is in keeping with his memory. Dr. Lachman was a great teacher and educator, always tireless in his efforts to further scholarship and knowledge in his chosen areas.

This third edition serves, in general, the same purpose as the first and second. The educational philosophy that was expressed in the preface to the first edition continues to be vindicated. Beginning students in American medical schools are confronted with the problems of clinical medicine almost from their first formal classes. There is a need to synthesize factual information obtained in basic science courses and to demonstrate its application to those clinical problems.

It was our intention to enlarge the scope of the book and present a more complete coverage of the anatomy of the body by adding eight new case stadies to the text. These were inserted in the sections on head and neck, abdominal and pelvic viscera, upper extremity, and lower extremity. In addition to these case studies, an appendix on the peripheral anatomy of selected visceral reflexes is new to this edition.

We have also sought to improve the existing case studies by making corrections and additions where deemed necessary.

Several of the new case studies have been published in journals; for inclusion in the book, however, they have been revised,

amplified, and often changed in format. The case studies concerned with the anatomy of female gonorrhea and the anatomy of hanging have been modified from articles appearing in Resident and Staff Physician, published by The Resident, Inc. The case study on the anatomy of male gonorrhea was published by Romaine Pierson Publishers, Inc., in Medical Times. The case study dealing with bipartite patella and fabella appeared in The New Physician. Much of the substance of the appendix was presented in an editorial in the Oklahoma State Medical Association Journal. For permission to utilize this material in this edition, I am most appreciative to the respective editors and publishers of these journals.

I am grateful to Martin Jensen, M.D., who is responsible for the preparation of most of the figures used in the added case studies. He was a fourth-year medical student at the time and spent many valuable hours in order to complete the artwork. I am especially appreciative to Mrs. Patricia Friedel-White, Mrs. Paula Moser, and Mrs. Irene Miles whose assistance was essential in the preparation of this manuscript.

Many thanks to my colleagues in the Department of Anatomical Sciences at the University of Oklahoma Health Sciences Center for their many ideas, suggestions, and genuine interest.

My wife and family deserve special mention for their tolerance, understanding, and assistance.

A special acknowledgment should go to Oxford University Press and their editors who did so much in getting this edition to press.

Kenneth K. Faulkner, Ph.D.

Oklahoma City July 1980

Preface to First Edition

In the teaching program in Gross Anatomy we face the wellknown dilemma that at the time the student has to master a large body of anatomical information he is not aware of its application to clinical medicine. On the other hand when he is ready to utilize his knowledge at the bedside, he has forgotten a substantial part of this material. Yet the importance of anatomical reasoning and the application of anatomical principles in the explanation of clinical signs and events and in the design of therapeutic procedures can be exemplified almost from the first week of the basic course. This will strengthen the student's motivation for learning and satisfy his thirst for information relating to clinical medicine. We cannot afford to stifle this basic interest which has brought a large proportion of our students to medicine. The student can be made to realize from the beginning that his day-by-day learning is meaningful in terms of his future work as a physician.

Thus, the case studies presented in this book are directed specifically to the first and second year medical student for collateral reading either in the basic anatomy course or in advanced courses in the field. Elective courses in the clinical years can readily be based on the exercises presented here, particularly if they are supplemented by pertinent and specialized dissections, executed by the student himself. Residents may find these case reports useful in their review studies, particularly for board examinations.

In each case a short history, physical findings, diagnosis, therapy, and further course are given. This is followed by a discussion of the material from the anatomical viewpoint, generally in the form of questions posed and answers given. The underlying anatomy is illustrated by drawings. This presentation lends itself to self-study since all questions formulated are answered in detail and in a comprehensive discussion of the subject matter.

The individual exercises are based on case histories chosen from the literature and from the author's experience and present a composite picture that exemplifies the characteristic anatomical features of the problem under discussion. In a few instances the history is taken from one of the classical collections of masterfully composed case studies available in the literature, such as the works of Hertzler, Cabot, or Kanavel. This type of presentation should call the student's attention to a stimulating form of medical instruction. In this connection it may be worth noting that in law classes in American universities the case method has been utilized for many years, even in the freshman year, whereby principles of law are illustrated by actual cases and real-life legal problems.

All case histories contained in this book have appeared previously in *The New Physician*, but in many instances their format has been changed, a large number have been revised and amplified.

Grateful acknowledgement is made to *The New Physician* and its editorial staff for permission to utilize these case histories.

Permission was also granted by the C. V. Mosby Company to use part of two case histories from Hertzler's Clinical Surgery by Case Histories; by the W. B. Saunders Company to utilize a portion of one case history published in Volume I of Cabot's Differential Diagnosis; by Lea and Febiger to use a case history from Kanavel's Infections of the Hands; by Doctors R. D. Duncan and M. E. Myers, Dr. L. B. Rose, and Dr. D. H. O'Donoghue to utilize material from individual case histories published by them. To these publishers and authors I express my grateful appreciation.

Thanks are due to the artists Mr. E. F. Hiser and Dr. J. E. Allison for their fine co-operation in executing the drawings, and to Frank Romano.

I am greatly indebted to Doctors G. H. Daron and K. K. Faulkner for many thoughtful suggestions.

Especially warm thanks are rendered to Mrs. Pat Friedel, our department secretary, whose tireless efforts were so helpful in bringing the work to speedy completion.

E. L. Oklahoma City October 1964

Contents

HEAD AND NECK, 1

- 1 Facial Paralysis (Bell's Palsy), 3
- 2 Trigeminal Neuralgia, 8
- 3 Cavernous Sinus Thrombosis, 20
- 4 Cancer of Lip with Lymphatic Spread, 29
- 5 Bilateral Dislocation of Mandible, 39
- 6 Rheumatoid Arthritis of Temporomandibular Joint, 47
- 7 Fracture of Mandible, 52
- 8 Tracheostomy, 59
- 9 Anatomy of Hanging, 68
- 10 Torticollis, 76
- 11 Whiplash Injury of Neck, 83

BODY WALL AND BACK, 91

- 12 Cancer of Breast, 93
- 13 Indirect Inguinal Hernia, 101
- 14 Prolapse of Intervertebral Disk, 109
- 15 Lumbar Puncture, 114
- 16 Psoas Abscess, 123

THORACIC VISCERA, 131

- 17 Segmental Abscess of Lung, 133
- 18 Middle Lobe Syndrome, 137
- 19 Lung Cancer with Metastasis, 144
- 20 Mediastinal Pleurisy, 153

- 21 Angina Pectoris, 158
- 22 Cardiac Infarction, 163
- 23 Coarctation of Aorta, 171
- 24 Obstruction of Superior Vena Cava, 180
- 25 Cancer of Esophagus, 187

ABDOMINAL AND PELVIC VISCERA, 195

- 26 Biliary Colic-Cholecystectomy, 197
- 27 "Dropped" Stomach, 205
- 28 Perforated Ulcer of Stomach, 213
- 29 Arteriomesenteric Occlusion of Duodenum, 220
- 30 Liver Abscess with Rupture into Lung, 230
- 31 Appendicitis, 238
- 32 Hydronephrosis due to Aberrant Renal Vessel, 244
- 33 Prolapse of Uterus, 249
- 34 Vasectomy, 256
- 35 Vaginismus, 263
- 36 Anatomy of Male Gonorrhea, 270
- 37 Anatomy of Female Gonorrhea, 283

UPPER EXTREMITY, 295

- 38 Fracture of Clavicle, 297
- 39 Cervical Rib Syndrome, 303
- 40 Subclavian Steal Syndrome, 308
- 41 Embolism of Brachial Artery, 315
- 42 Intravenous Injection, 320
- 43 Tendon Sheath and Thenar Space Infections, 328
- 44 Carpal Tunnel Syndrome, 334

LOWER EXTREMITY, 345

- 45 Intragluteal Injection, 347
- 46 Intracapsular Hip Fracture, 354
- 47 "Unhappy Triad" of Knee Joint, 365
- 48 Bipartite Patella and Fabella, 372
- 49 Anterior Tibial Syndrome, 378
- 50 Flatfoot, 383

Appendix: Peripheral Anatomy of Selected Visceral Reflexes, 393

Head and Neck

此为试读,需要完整PDF请访问: www.ertongbook.com

1 Facial Paralysis (Bell's Palsy)

A 36-year-old woman librarian slept close to an open window on a cold drafty night. She woke up in the morning with some aching pain in and around the ear; the right side of her face felt numb and swollen. On arising she noticed that her face was distorted and deformed. She could not close her right eye. She had some difficulty in speaking, eating, and drinking. Food seemed to collect between her teeth and her right cheek. Saliva and liquids that she tried to drink ran out of the right corner of her mouth, although she had no difficulty swallowing. She became quite apprehensive and consulted a physician.

EXAMINATION

On examination the right side of the face of the patient appears immobile and without expression. All wrinkles have disappeared from her right forehead; the right nasolabial fold is less distinct than the left. The right eyebrow droops and there is sagging of the right lower eyelid. There is some flow of tears down the side of her face. The nose and mouth seem deviated toward the unaffected side and the right corner of her mouth is sagging

On further examination the following facial movements prove to be affected. The patient cannot frown on the right side when asked to do so. When patient attempts to shut her eyes, the right eye does not close completely. She cannot purse her lips tightly, whistle, or puff out her cheeks. Asked to show her teeth, she uncovers them only on her unaffected left side and her lips seem to be drawn to the left side. On attempt at laughing, the distortion of her face becomes considerably more noticeable and disfiguring:

DIAGNOSIS

All signs and symptoms of this patient point to a disease known as facial paralysis or Bell's palsy, the latter term perpetuating the name and memory of Charles Bell, a British anatomist and surgeon who first described the disease in 1821.

THERAPY AND FURTHER COURSE

Under treatment with analgesics, corticosteroids, and local heat, the pain disappeared. Later, electric stimulation of the involved muscles, massage, and active exercises were resorted to. After five weeks the patient was almost completely recovered and only traces of the previous paralysis, particularly around the mouth, could be demonstrated.

DISCUSSION

The exact cause of the condition is not known, but the paralysis of the facial nerve is presumed to be due to an inflammation of the facial nerve in the facial canal. Remember the course of the facial nerve in the petrous portion of the temporal bone where even slight swelling of the nerve within its tight-fitting bony surroundings would subject the nerve to destructive pressure. What is the name of the foramen at the lower end of the canal, through which the nerve emerges from the skull? Is this also the site of entrance of an artery that supplies the facial nerve within the canal? Some authors ascribe the damage of the nerve to constriction of this artery caused by chilling. Of what artery is it most commonly a branch? The facial nerve emerges from the skull through the stylomastoid foramen. This is the site of entrance of the stylomastoid artery, a branch of the posterior auricular artery of the external carotid artery.

Effects of paralysis on facial muscles

The motor deficiencies present in this case exemplify the actions of the facial muscles innervated by the seventh nerve.

These muscles are conventionally grouped under the heading of mimetic muscles or muscles of facial expression and are responsible for voluntary movements of the face and emotional expression. How do you explain the disappearance of wrinkles of the forehead and certain facial folds in facial paralysis? It must be realized that some folds of the skin are brought about by habitual wrinkling of the skin, as on the forehead in frowning and around the eyes in squinting. With paralysis of the facial muscles, such as the frontalis which normally is responsible for transverse folds on the forehead, or the orbicularis oculi which causes "crow's feet," the tonus of these muscles which attach in part to the skin, is lost and the folds disappear.

Paralysis of what muscle explains the inability to close the right eye and causes the sagging of the lower eyelid? The former deficiency leads to the most serious complication of facial paralysis, that is, inflammation of the conjunctiva and cornea, and possible corneal ulceration. The sagging of the lower lid results in its eversion and the spilling of tears, as in this case. Where do tears normally drain? What muscle opens the eye? Is it affected in facial palsy? The muscle whose paralysis is responsible for sometimes serious eye complications is the orbicularis oculi which when functioning acts like a windshield wiper to keep the cornea moist and clean. Tears normally drain through the lacrimal puncta and canaliculi into the lacrimal sac and from there via the nasolacrimal duct into the nose. The muscle responsible for opening the eye is the levator palpebrae superioris, which in its somatic portion is supplied by the unaffected oculomotor nerve while its visceral (smooth muscle) component is innervated by sympathetic fibers from the superior cervical ganglion.

Paralysis of which important muscle causes food to collect between cheek and teeth and is responsible for the inability to whistle? The buccinator has the essential function of maintaining tension of the cheek and to keep food from passing between cheek and teeth. It also prevents the mucous membrane of the cheek from being caught between the teeth in the act of mastication.

The inability to purse the lips and show the teeth of the affected side is due to paralysis of the orbicularis oris, which

through its action as a whole or in parts can either protrude the lips as in pouting or draw them against the teeth.

The absence of the mimic expressions of smile and laughter is the result of the dysfunction of numerous small facial muscles, such as the zygomaticus, the risorius, the nasalis, and the levator labii superioris, all of which have in common a superficial subcutaneous location, the absence of a muscle fascia, and insertion into the skin. Their paralysis is responsible for the previously mentioned characteristic feature of Bell's palsy: loss, on the paralyzed side, of expression of emotion, such as surprise and attention, joy and sorrow.

Involvement of the scalp muscles (frontalis and occipitalis) and the extrinsic muscles of the ear as well as of the stylohyoid and posterior belly of the digastric muscle is difficult to demonstrate.

Ear symptoms and taste deficiency in facial paralysis

Occasionally, the stapedius muscle also is paralyzed. Would that give any clue as to the site of attack of the noxious agent? This paralysis can only occur if the facial nerve is affected in the facial canal, central to the origin of its branch to the stapedius muscle. Since it is the function of the stapedius muscle to dampen the vibrations of the ossicles by tilting the footplate of the stapes, what is the result of paralysis of this muscle? The consequent increased acuity of the sense of hearing (hyperacusis) may be quite annoying to the patient.

How do you account for the aching pain in and around the ear and the sensation of numbness in the face? Does the facial nerve contain any general somatic afferent fibers? In which ganglion are their cell bodies located? The geniculate ganglion contains the cell bodies of these somatic sensory fibers. The peripheral course of the fibers mediating pain from the ear in facial palsy is somewhat uncertain. Two pathways are possible: one by way of a communication from the facial nerve in the lowest part of the facial canal to the auricular branch of the vagus and with it to the external ear; the other by way of sensory fibers accompanying the motor fibers of the posterior auricular branch of the facial nerve. Whether the facial nerve contains fibers mediating deep sensibility of the face including deep-seated pain is disputed.

Absence of clinically demonstrable signs of involvement of the chorda tympani does not help in locating the site of the lesion below the origin of this nerve since visceral efferent impulses as well as visceral afferent impulses, which ordinarily utilize the chorda tympani, may also follow other pathways. This makes it frequently impossible to demonstrate deficiencies in case of damage of the chorda tympani. Where would absence of taste be noticeable if the chorda tympani were involved and no alternate path for taste impulses were available? Taste would be absent in the anterior two-thirds of the tongue on the affected side.