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THE SHOULDER

RUPTURE OF THE SUPRASPINATUS TENDON AND OTHER LESIONS IN OR ABOUT THE SUBACROMIAL BURSA

E. A. CODMAN, M.D.

Consulting Surgeon

Massachusetts General Hospital, Boston, Massachusetts

BRITISH MEDICAL
-4 OCT 1938
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of the

Members of the

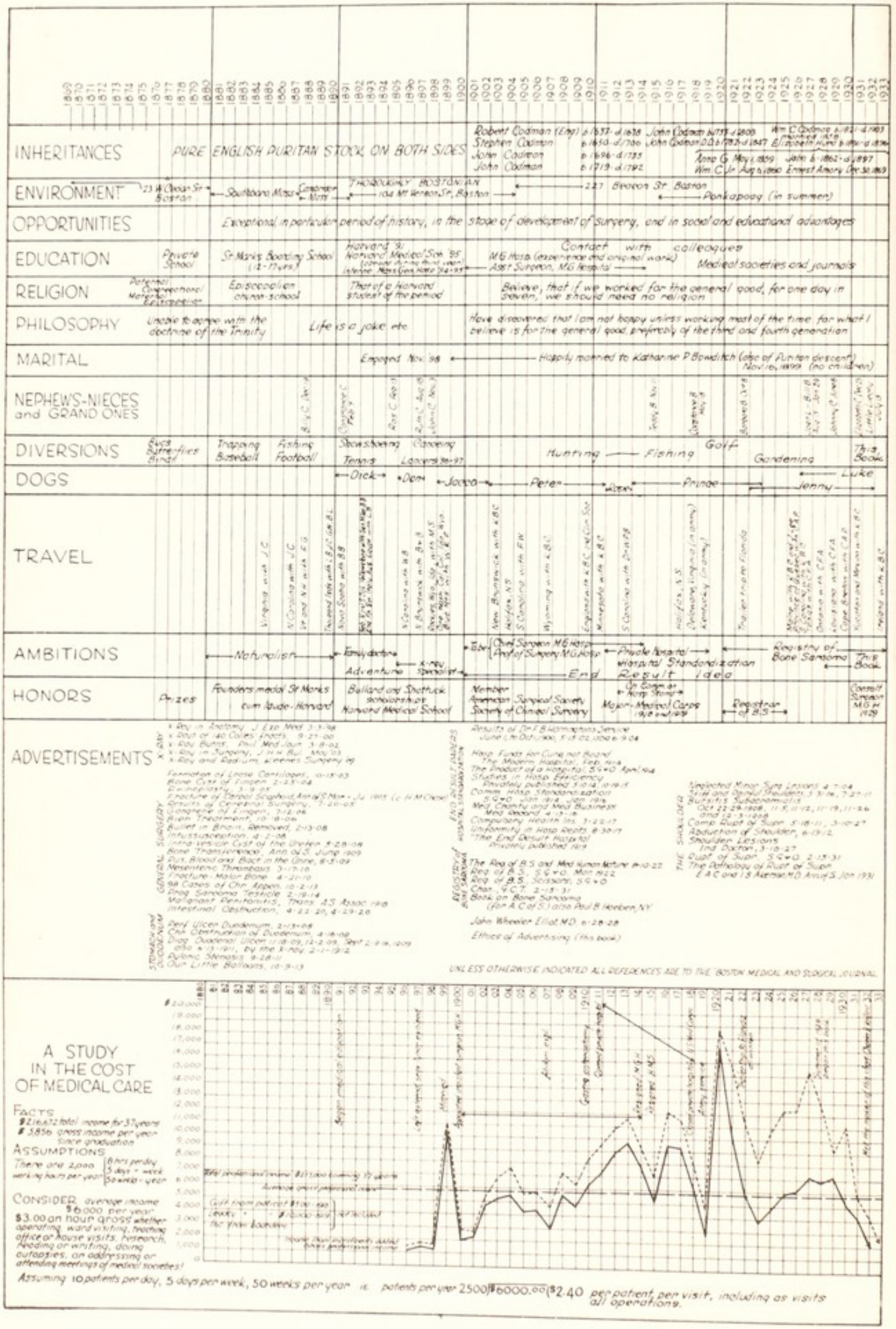
American College of Surgeons

AN AUTOBIOGRAPHIC PREFACE

The prefaces in medical books, particularly in those that concern new fields, are often too brief and impersonal. If an author has conscientiously labored to present his material in clear English, properly punctuated and painstakingly illustrated for the benefit of the reader, surely he deserves to be allowed to indulge himself in his preface. Let him try his sense of humor, however heavy it may be, let him ride his hobbies, relate his favorite anecdotes, tell his life history or otherwise endeavor to please himself. Despise these amusements if you must, but do not forget that they are the normal pleasures of the average man, especially if he is over sixty. No one is obliged to read a preface, but in it the author should introduce himself to the reader and give him a glimpse of his own personality, amusements and intellectual processes. Both author and reader, before they begin any serious study, should enjoy themselves after the example of a pair of interlocking directors beginning their business with some passable golf, a shower, a rubdown, a cocktail, dinner and coffee, before they go to work. These good business men may have heard each other's stories before and may thoroughly distrust one another, but the exercise, the glow, the comfortable, warm, satiated abdominal sensations, predispose not only to digestion but to reasonableness. A preface should produce a similar mutually-forgiving human state of mind, and give the reader and author a certain percentage of trust in one another—of course not complete.

Another advantage of this type of biographic, conversational, light, perhaps flippant preface, might be a chance that the book would be of permanent value, for most medical books are scarcely more enduring than shooting stars, in fact many are obsolete by the time they are published. Hence a preface, serenely, frankly, kindly and even egotistically written by a happy, understanding soul, might preserve a volume through the centuries, although the subject matter of the book might last only a year.

I want egotism in my author or teacher. I want to know what life he has led, what were his aspirations and what are his regrets. Let him be as unconventional as he wishes, but let him refrain from even customary lies and give freely of himself. Things which have become conventionalized like prefaces, funeral services, wedding vows, and legal preambles are to be suspected of evading responsibility. Give me something that is different, for there is a chance of its being better. Voluntarily to be different is to take responsibility and every sentence in an original book might begin with I, for the author only is to be judged.


1859 1860 1861 1862 1863 1864 1865 1866 1867 1868 1869 1870 1871 1872 1873 1874 1875 1876 1877 1878 1879 1880 1881 1882 1883 1884 1885 1886 1887 1888 1889 1890 1891 1892 1893 1894 1895 1896 1897 1898 1899 1900 1901 1902 1903 1904 1905 1906 1907 1908 1909 1910 1911 1912 1913 1914 1915 1916 1917 1918 1919 1920 1921 1922 1923 1924 1925 1926 1927 1928 1929 1930 1931 1932 1933
INHERITANCES
PURE ENGLISH PURITAN STOCK ON BOTH SIDES
Robert Codman (Eng) b 1637- d 1678
Stephen Codman b 1650- d 1736
John Codman b 1696- d 1735
John Codman b 1719- d 1792
John Codman M 1757 d 1800
John Codman D.D. b 1783- d 1847
Wm C Codman b 1821- d 1903
Elizabeth Hurd b Mar d 1894
Anne G May 1859
John b 1862- d 1897
Wm C Jr Aug 1860
Ernest Amory Oct 30 1863
ENVIRONMENT
23 Hancock St Boston
Southboro Mass
Cambridge Mass
"THOROUGHLY BOSTONIAN"
108 Mt Vernon St, Boston
227 Beacon St Boston
Penikapoog (in summer)
OPPORTUNITIES
Exceptional in particular period of history, in the stage of development of surgery, and in social and educational advantages
EDUCATION
Private School
St Mark's Boarding School (12-17 yrs)
Harvard '91
Harvard Medical Sch '95
(Summer during these years)
(Winter Mass Gen Hosp 94-95)
Contact with colleagues
M.G. Hosp (assistantships and original work)
Asst Surgeon, M.G. Hospital
Medical societies and journals
RELIGION
Paternal Congregational Maternal Episcopalian
Episcopalian on non-school
That of a Harvard student of the period
Believe, that if we worked for the general good, for one day in seven, we should need no religion
PHILOSOPHY
Unable to agree with the doctrine of the Trinity
Life is a joke, etc
Have discovered that I am not happy unless working most of the time for what I believe is for the general good, primarily of the third and fourth generation
MARITAL
Engaged Nov '98
Happily married to Katharine P. Bowditch (also of Puritan descent) Nov 16, 1899 (no children)
NEPHEWS-NIECES and GRAND ONES
DIVERSIONS
Bugs Butterflies Birds
Trapping Baseball
Fishing Football
Skating Boating Canoeing
Tennis
Lancers 96-97
Hunting — Fishing
Golf
Gardening
This Book
DOGS
Dick
Dan
Jocca
Pete
Wisp
Prince
Luke
Jenny
TRAVEL
Virginia with J.C.
N Carolina with J.C.
Va and N.C. with E.C.
Halifax N.S. with M.B.
Nova Scotia with M.B.
Bermuda with M.B.
S. Carolina with M.B.
S. Dakota with M.B.
Colorado with M.B.
Utah with M.B.
New Brunswick with M.B.C.
Halifax N.S.
S. Carolina with F.W.
Wyoming with M.B.C.
England with M.B.C. and Con Soc
Minneapolis with M.B.C.
S. Carolina with D.H.B.
Halifax, N.S.
Delaware, Virginia (in honeymoon)
Kentucky (in honeymoon)
Trip to Hot Springs
Florida
Maine with M.B.C. and Con Soc
Bermuda with M.B.C.
Ontario with C.F.A.
Adirondacks with C.F.A.
Cape Breton with C.F.A.
Yosemite and Mexico with M.B.C.
Europe with M.B.C.
AMBITIONS
Naturalist
Family doctor
Adventure
X-ray specialist
Chief Surgeon M.G. Hosp
Prof of Surgery M.G. Hosp
Private hospital
Hospital Standardization
End Result Idea
Registry of Bone Sarcoma
This Book
HONORS
Prizes
Founders medal St Mark's
cum laude-Harvard
Ballard and Shattuck scholarships at Harvard Medical School
Member American Society of Clinical Surgery
Surgical Society
Dr Corps at Hosp Stores
Major-Medical Corps 1918 and 1919
Registrar of B.S.
Consult Surgeon M.G.H. 1929
ADVERTISEMENTS
X-RAY
X Ray in Anatomy J.E.P. Med 3-3-06
X Rays of 140 Colles' Fracts. 9-27-00
X-Ray Burns, Phil. Med Jour 3-8-02
X-Ray in Surgery, J.H.W. Bul. May '03
X-Ray and Radium, Keenes Surgery 19
GENERAL SURGERY
Formation of Loose Cartilages, 10-18-03
Bone Cyst of Finger, 2-23-04
Elephantiasis, 3-9-05
Fracture of Carpal Scaphoid, Am J.S. Mar-Ju 1905 (C.H.M. Clerk)
Results of Cerebral Surgery, 7-20-05
Gangrene of Finger, 3-12-06
Rein Treatment, 10-18-06
Bullet in Brain, Removed, 2-13-08
Intussusception, 4-2-08
Infra-vesical Cyst of the Ureter 5-28-08
Bone Transplantation, Ann. of S. June 1909
Dys. Blood and Blood in the Urine, 8-5-09
Mesenteric Thrombosis 3-17-10
Fracture: Malar Bone 4-21-10
98 Cases of Chr. Appen. 10-2-13
Drug Sarcoma Testicle 2-19-14
Malignant Peritonitis, Trans. A.S. Assoc 1918
Intestinal Obstruction, 4-22-20, 4-29-20
STOMACH AND DUODENUM
Perf. Ulcer Duodenum, 2-13-08
Chr. Obstruction of Duodenum, 4-18-08
Drug Duodenal Ulcer 11-8-09, 12-2-09, Sept 2-9-10, 1909
Ulcer 4-13-1911, by the X-Ray, 2-11-1912
Pyloric Stenosis, 9-28-11
Our Little Bedfasts, 10-9-13
END RESULT IDEAS
Results of Dr F.B. Hornsby's Service June (to Oct.) 1900, 5-13-02, 1906 & 1904
Hosp. Funds for Cure not Taxed
The Modern Hospital, Feb. 1914
The Product of a Hospital, 5-6-40 April 1914
Studies in Hosp. Efficiency
Privately published 5-10-14, 10-18-15
Comm. Hosp. Standardization
5-6-40 Jan 1918, Jan 1919
Med. Charity and Med. Business
Med. Record 4-17-15
Compulsory Health Ins. 3-22-17
Uniformity in Hosp. Reports 8-30-17
"The End Result Hospital"
Privately published 1919
REGISTRY OF BONE SARCOMA
The Reg of B.S. and Med. Human Nature 9-10-22
Reg. of B.S., 5-6-40, Mar 1922
Reg. of B.S., Scissors, 5-6-40
Chair, 9-C.T. 2-15-31
Book on Bone Sarcoma
(for A.C. of S.) also Paul B. Hoeber, N.Y.
John Wheeler Elliot M.D. 6-28-28
Effects of Advertising (this book)
THE SHOULDER
Unjected Minor Surg Lesions 4-7-04
Stiff and Painful Shoulders, 5-30-06, 7-27-11
Bursitis Subdeltoidea
Oct 23-24-1908, 11-5, 11-12, 11-19, 11-26
and 12-3-1908
Comp Rupt of Supr. 5-18-11, 3-10-27
Abduction of Shoulder, 6-13-12
Shoulder Lesions
Ind. Doctor, 3-10-27
Rupt of Supr. 5-6-40, 2-15-31
The Pathology of Rupt of Supr.
E.A.C. and I.B. Mumford M.D. Ann of S. Jan 1931
UNLESS OTHERWISE INDICATED ALL REFERENCES ARE TO THE BOSTON MEDICAL AND SURGICAL JOURNAL
A STUDY IN THE COST OF MEDICAL CARE
FACTS
$216,372 total income for 37 years
$ 5,856 gross income per year since graduation
ASSUMPTIONS
There are 2,000 working hours per year (8 hrs per day, 5 days = week, 50 weeks = year)
CONSIDER average income $6,000 per year
$3.00 an hour gross whether operating, ward visiting, treating office or house visits, research, reading or writing, doing autopsies, or addressing or attending meetings of medical societies!
$20,000 19,000 18,000 17,000 16,000 15,000 14,000 13,000 12,000 11,000 10,000 9,000 8,000 7,000 6,000 5,000 4,000 3,000 2,000 1,000 0
Assuming 10 patients per day, 5 days per week, 50 weeks per year ie. patients per year 2500 $6000.00 ($2.40 per patient, per visit, including all visits all operations.

Necessity for economy compels me to reduce my life history to the accompanying chart, and to refrain from describing many incidents which have been exciting and amusing or of absorbing interest to me, personally. It is my intention to comment mainly on those events which concern my work on the shoulder and on what I term the End Result System of Hospital Organization, on propaganda for which I have spent most of my energy, including that expended during the last five years on this book. Meanwhile I have had to earn most of my living and therefore present income curves on the chart to permit me to introduce a necessary part of my thesis, the cost of medical care, a subject that has much to do with Hospital Organization.

I was a conventional enough Boston-Harvard boy, with relatives and acquaintances among the well-to-do, and took two years in the Harvard Medical School with success, and in the third winter had the opportunity to travel in Europe and Egypt with a friend, on the understanding that I could spend as much time as I wished at the Clinics in the various cities we visited, London, Paris, Berlin, Vienna, Cairo and others. This experience and some study on the way enabled me to pass my third-year examinations and to get my degree on my return.

It was in Vienna that my attention was first attracted to the subdeltoid bursa, because it was mentioned in a little book by Dr. E. Albert ("Diagnostik der Chirugischer Krankheiten." Alfred Holder, Wien, 1893). I had never heard this bursa spoken of at home by my teachers, nor do I think it was mentioned in American medical literature at that time. Soon after my return I served two years as surgical interne at the Massachusetts General Hospital in Boston, and during this period, sometimes made diagnoses of subdeltoid bursitis, which were ignored by my seniors. Starting practice in 1895, I became Assistant in Anatomy at the Harvard Medical School, and, for several years, having many opportunities to dissect the bursa and to study its pathology, I gradually came to appreciate its clinical importance. Appointed Surgeon to Out-Patients at the Massachusetts General Hospital (hereinafter M. G. H.) in 1899, I began to have great clinical opportunity, and treated many patients on the diagnosis of bursitis. My first paper mentioning the subject was in April, 1904, although previously at the request of Dr. Mumford, then Chairman of the Staff Meetings, I had presented a *résumé* of my work, demonstrating many anatomic specimens and some patients. This attracted the attention of Dr. George Crile of Cleveland, who invited me to read a paper on the subject before the Medical

Society of that city. Flattered by this invitation, I wrote a paper with great care and it was well received. During the discussion Dr. Carl A. Hamann of Cleveland mentioned a paper by Küster published in 1902. I had, at that time, never seen this article, and, though I am frequently quoted as having been the first to describe subdeltoid bursitis, this paper shows clearly that I was not. After seeing Küster's paper, I adopted his name of subacromial bursitis as better than the term subdeltoid. My work was original so far as I knew at the time, and it was pleasant rather than the reverse, to find that the great surgeon, Küster, had also thought it worth while to write even a short paper on the subject.

Through much of my life I have suffered somewhat from a sense of isolation, because I have always been thinking, or saying, one thing or another, with which other doctors did not agree. This, in my early years, made me suspect myself of being peculiar, so that, from time to time, I would conform again to general opinions which I knew to be irrational. Even now I have this sense of isolation, although I have become more and more content to wait for acceptance of my views. My regrets are for wasting so much time on the opinions of a previous generation and not realizing that it was the approval of my pupils, rather than of my masters, that was desirable.

When I have given a great deal of study to a subject, I am apt to think I know more about it than those who have not studied it. This confidence probably came because I often stood at the head of my school and yet I have never been able to form very strong opinions on the probable results of races, elections or in futures in the Stock Market, as do many of my friends who were not good scholars. They know whether or not a certain man should be President, whether we should or should not go to war, or even what church to attend, whereas I am in doubt about such things. I am inclined to be impatient with others who do not accept my views on subjects to which I have given much attention, but if any one else has also given such study and does not agree with me, my confidence in my opinion is readily shaken. Thus, I was greatly pleased to find that Küster had agreed with me, or I with him, for I was no longer isolated in my opinions. The fact that he had priority was of no significance to me then. I did not realize that it was Küster who should be pleased because I had sustained his ideas, for now my pleasure comes from having younger men agree to ideas which my contemporaries rejected, or accepted very reluctantly.

It so happened that Röntgen made his announcement of the discovery of the X-ray in December, 1895, at just the time when I

started private practice, with the intention of becoming a surgeon. Believing in its importance to surgery, I at once started to learn the technique and sought the help of Professor Trowbridge of Harvard, and also that of Professor Elihu Thomson of the General Electric Company at Lynn. Perhaps it is not generally known that apparatus similar to that with which Röntgen worked had existed for a number of years in many other laboratories. Experiments had been performed to study the effect of electric currents in a vacuum tube invented by Professor Crookes of England. The apparatus had been of great theoretic interest to physicists and was used to produce cathode rays whose paths were chiefly inside the tube. It is said that Röntgen's discovery was accidental because he noticed that a piece of barium-platino-cyanide paper, which happened to be lying on his laboratory table near a Crookes tube, became luminous while the tube was in operation, and that the luminosity stopped immediately when the electric current, passing through the tube, was turned off.

This observation led to his finding that rays, other than those from the cathode, were generated and were projected far beyond the glass walls of the tube. Having noted this essential fact, he went on with a carefully thought-out series of experiments, which demonstrated most of the important points known today in regard to the X-ray. His first paper was a masterpiece. Since every well-equipped physical laboratory already had the apparatus, his experiments were immediately repeated in many places and his conclusions were at once corroborated. Trowbridge and Thomson were among the first in this country to do this, and I had their most kindly, personal, instruction. Having learned the essential points, I found at the laboratory of the Harvard Medical School similar apparatus and began clinical work early in 1896. For five years I devoted most of my time to the X-ray, although still continuing to work in the Surgical Out-Patient Department of the M. G. H. and to assist the late Dr. F. B. Harrington in the practice of surgery. At the end of five years, having written a number of articles on X-ray subjects, including one on X-ray burns, which is still quoted, I saw that I must choose between surgery and röntgenology. An appointment as Out-Patient Surgeon, a title in those days equivalent to that of Assistant Visiting Surgeon, was given me and thereafter my time was devoted chiefly to surgery.

Meantime, 1896 to 1899, experience with the shoulder continued, and in the Anatomic Department I studied the joints and bursæ injected with non-radiable material. In 1898, after two years of these anatomic studies, I presented to the Warren Museum an album, which contained standard X-ray anatomic pictures of each joint of

the body in flexion, extension, etc. It was a tremendous piece of work, and for me at that time, a very expensive one. Recently, in poking round the Museum, I came across this album covered with dust. It probably had not been opened since left there. However, the experience had been valuable, for, after completing this study of the normal joints, I became interested in their pathology, especially in that of the wrist, knee and shoulder. Furthermore, the fact that my atlas of the normal joints was not used by my colleagues, was a good lesson to my personal sensitiveness and taught me, to some extent, to postpone hope of recognition of labor. A by-product of these anatomic X-ray studies was the light they threw on the normal motions of the wrist joint. These I described in a paper in the *Journal of Experimental Medicine.* I do not think they had been accurately described before, or have been studied much since. This work led to an interest in fractures of the carpal scaphoid and resulted later in a monograph on the wrist, which has since been recognized.

It would be impossible to give the reader an idea of the thrill experienced by those of us who did the early X-ray work. We each made weekly discoveries, only to find that our fellow workers in the same city and in all other cities had made the same ones at the same time. Announcements of new uses of the X-ray, which are now familiar, came with every issue of the Medical Journals. Each of us had the self-importance to think that we were the first to show fractures of various types, to diagnose bone tumors or to locate foreign bodies in new parts of the anatomy. I remember that an early contribution of mine in the *Boston Medical and Surgical Journal* was to show that the X-ray was likely to help us in studying the epiphyseal lines! My plate, which was made with a tube which did not focus, after an exposure of over fifteen minutes, showed the epiphyses in the arm of a dead baby. Yet, what I wrote was then unknown to the great majority of readers. We almost forgot that it was all because Röntgen had noticed something which many others might have observed. Probably other things of great importance are showing themselves to us daily, and we look but do not see.

Let any modern Röntgenologist look up my old paper on the wrist, and he will see excellent pictures, taken in 1896, with an old Ruhmkorff coil which Professor Bowditch occasionally used for his physiologic experiments. I remember the Professor's delight when I showed with it, an old bullet in his ulna, which he had carried, without knowing it, for thirty years since the Civil War. Even more delighted was an old lady, who had insisted for sixteen years that she had a needle in her foot, when, after I had located and removed it, she shook it in the

face of her doubting family. There were many amusing, exciting and tragic episodes in those days, for we all had burns and some of us gave them. Many of my old friends are now dead from X-ray cancer. It was fortunate for me that my interest in surgery was greater than in Röntgen's discovery.

Side by side with other work, I managed to submit a very extensive monograph for the Gross Prize, which was then given every five years in Philadelphia. The subject was "The Use of the X-ray in the Diagnosis of Bone Diseases." I had collected the histories, pathological reports, X-ray films and end results of cases of bone disease, just as the Registry of Bone Sarcoma does now. There were even examples of such rare diseases as ainhum, chondrodystrophia fœtalis, osteogenesis imperfecta and osteoarthropathy pneumonique. With all this material was a *résumé* of the diagnostic points in which the X-ray was of help. The Committee was composed of prominent Philadelphia surgeons, among whom were Dr. W. W. Keen and Dr. J. W. White, and the prize for that year was awarded for an essay on ligation of the carotids in cases of malignant disease of the face! The author claimed that thus starving the growth of tumors by stopping their blood supply was of great help in controlling the disease. The method is now seldom if ever used, yet today, practically everything my paper contained is common knowledge among röntgenologists. It is hardly possible to realize now, that at that time (1905), busy surgeons had no idea of the practical value of the X-ray in the diagnosis of bone diseases, and that the pictures which I presented to this Committee were to them *unintelligible!*

It was not until five years later, when Dr. Keen asked me to write a chapter on "The Use of the X-ray in Surgery," that I could make up my mind to the shock it gave me to feel that my essay had been discarded, for I felt absolutely sure that it was worthy of the prize. This was my second severe lesson in not being in a hurry about having one's ideas confirmed. However, I had great satisfaction in pulling out from a closet the unpublished paper submitted to Dr. Keen five years before, and, with practically no changes, presenting it to him for his book. Not only most of the illustrations, but the descriptions of the X-ray characteristics of various bone diseases, were from the material submitted for the Gross Prize. The chapter still appears in Keen's Surgery without change.

In 1905-1908 I again worked intensively on the shoulder and published my chief paper which has been so much quoted in the American literature.

We younger surgeons at that time did most of the night emer-

gency operations, and in one such case I was able to make a preoperative diagnosis of perforated duodenal ulcer and to successfully operate on the patient. This took my mind from the shoulder to the duodenum, as it was the first case thus diagnosed and operated on at that hospital. Within a short time afterward, I had several others which were also successful. This led me to study chronic duodenal ulcer, and the shoulder remained displaced behind the duodenum and stomach for the next two years, although my clinical experience with shoulder lesions continued, and unfortunately also, my personal experience as a patient with duodenal ulcer. My chief interest during the next few years was in the surgery of the duodenum. I think I was among the first to appreciate its importance, for I wrote a paper in 1909, when the diagnosis was made so seldom, that I was only able to collect fifty proved instances from our surgical, medical and postmortem records. Of these, eleven were my own cases. I have recently reviewed this paper and was agreeably surprised by the accuracy of the statements as shown by the confirmation of my predictions. However, although working chiefly on the surgery of the stomach and duodenum during this period, I added a point of importance to our practical knowledge of shoulder lesions, namely the demonstration, by actual successful suture of two cases, that ruptured supraspinatus tendons may be repaired. I also drew attention to the importance of examining the patient in a stooping position as an aid to diagnosis in these cases.

Thus, in the year 1910, at the age of forty, I was deeply interested in the surgery of the upper abdomen, still studying lesions of the shoulder, steadfast to my general surgery at the M. G. H., and successful enough to be making a reasonable living in private practice. Then began the great and still unsuccessful interest of my life, over which I have toiled harder and suppressed more regrets, than over any other star-gazing period of my career. Already in 1900 I had become interested in what I have called the End Result Idea, which was merely the common-sense notion that every hospital should follow *every* patient it treats, long enough to determine whether or not the treatment has been successful, and then to inquire "if not, why not?" with a view to preventing similar failures in future. My chief, Dr. F. B. Harrington, and I had been applying this plan practically to our service since 1900. We had found that this routine tracing of every case, interesting or uninteresting, had brought to our notice many things in which our knowledge, our technique, our organization, our own skill or wisdom, and perhaps even our care and our consciences, needed attention.

From the day in the summer of 1910 on which Dr. Edward Martin of Philadelphia and I drove back to London in a hansom cab from the Tuberculosis Sanatorium at Frimly, this End Result Idea has taken the major share of my intellectual efforts. Martin at once recognized that the idea was practical, and took advantage of my monomania to make me the servant of his own ideas about Hospital Standardization. We were visiting the British Surgeons as members of a small American Association called the Society of Clinical Surgery.

We had been entertained at a grand dinner by the Royal College of Surgeons, and clinics had been given for us by its various members at the London hospitals. Our little society was composed of very active-minded members, most of whose names are under the accompanying photograph. In this traditional environment, talk of an American College was inevitable among such a group. As far as I know the visit to our British confreres was not arranged for this purpose, but it certainly led to the organization of our American College. If any one, with that idea in mind, arranged that trip, he was certainly the founder, for such men would be sure to go home and found a bigger, if not a better, College. I think that it may have been J. G. Mumford's plan, and that he probably at once talked it over with Cushing, for they were the original founders of the Society of Clinical Surgery. Cushing tells me that he had talked of the question with Ochsner even before this time. I do not know how much had been planned beforehand, but I do know that by the end of that meeting, the American College of Surgeons was under way, even if the method of procedure was not definitely laid out.

Edward Martin, after hearing me discourse on my End Result Idea in the hansom cab, caught at it as the catalyst to crystallize the College Idea. An American College would be a fine thing if it could be the instrument with which to introduce the End Result Idea into the hospitals; in other words to standardize them on a basis of service to the individual patient, as demonstrated by available records. As Martin remarked, "the tail is more important than the dog, but we shall have to have the dog to wag the tail." An American College of Surgeons should be formed to standardize the hospitals, and, when that was done, to continue other good works.

The association with the members of this party, who were not only successful as practical surgeons, but most of whom were connected with teaching or surgical research in our American institutions, was very stimulating to me, although I was only an assistant at home, had never had a hospital service entirely my own, and, owing to the seniority system then in vogue, apparently very unlikely ever

(A guest) G. Davis E. A. Codman J. Munro
J. Hutchinson John Gibbon R. H. Harte R. Le Conte Ellsworth Elliot Willard Bartlett C. A. Porter
J. G. Clarke S. Alexander J. M. T. Finney G. Woolsey C. Frazier M. L. Harris E. Rixford F. B. Lund C. H. Peck
G. E. Brewer A. D. Bevan G. W. Crile T. Pridgin Teale J. B. Murphy Ed. Martin Chas. H. Mayo L. L. McArthur

SOCIETY OF CLINICAL SURGERY (1910)

to have one. It was also a tremendous stimulus to see the work of the English surgeons, especially that of Mr. Moynihan, at whose clinic I spent several weeks after our official meeting was over. Part of this time I was a patient, and thanks to his skill and a gastro-jejunostomy, I was greatly relieved physically, and my zeal for surgery was greatly stimulated mentally. I returned in September, 1910, full of enthusiasm and determined to undertake the following things:

First: To proceed with my work on the shoulder, because it was very clear to me that the English surgeons had not yet become interested in subacromial bursitis or in rupture of the supraspinatus tendon. I felt that here was a thing in which, although I could never approximate the contributions of the men with whom I had been associating, I might do my bit, if I could sometime prove in this small matter, that I had added a little to surgical science.

Second: Coming fresh from Mr. Moynihan's Clinic and immensely impressed with his ability to make diagnoses of lesions of the upper abdomen with almost uncanny detail, I wanted the opportunity to demonstrate what he had taught me, and if possible, to progress still further in this kind of work.

Third: More than either of these strictly surgical plans, my talk with Edward Martin and the discussions I had listened to about an American College of Surgeons, took the dominant part in my mind.

I determined that, as any increased opportunity at the M. G. H. was most unlikely since the tradition of a seniority system was so firmly fixed, I would start a small hospital where I would be my own master and could work out my own ideas. I especially wished to make it an example of the End Result Idea. There would be no trustees to consult or other members of the staff to placate, if I wished to state publicly the actual results of the treatment which the patients received. In other words, I would make this small hospital an example of the advantage of an organization based on actual efficiency analyses of the results of treatment.

I set about these plans at once with the result that within a year my hospital was running, and I had two assignments at the M. G. H., one to study shoulder cases, and the other to treat 100 successive cases of ulcers of the stomach and duodenum. I did not succeed in obtaining these assignments without a great deal of trouble, as may be imagined. It was necessary first to convince the rest of the staff that intensive studies of special series of cases were essential for progress, if we were to compete with the large clinics in the neighboring cities where individual surgeons dominated a great deal of ma-

terial. We had seen the grand old London hospitals being overshadowed by their provincial competitors as an example of such a lack of coöperation. Provided that we each had the spirit to do intensive work on series of cases sufficiently large to make our papers of real value at national meetings, we might still keep our hospital among the foremost in the country and also furnish our community with eminent specialists. Necessarily, if all of our eighteen operators, who shared a service of only 180 beds, were to have a sufficient number of relatively rare cases, each of us must agree to give his major attention to one field.

In search of a convincing argument, I took great pains to look up the end results of our cases of stomach surgery, a total of about 600 in the previous ten years. I tabulated these, not only according to the lesions, but according to the results of each individual operator. These tables offered overwhelming evidence that good results had not been obtained by the eighteen surgeons. There was no chance for discussion, once the facts were reviewed. The rest of the staff, although I was only a junior member, were fair-minded enough to accept the argument and the system of "assignments" was then inaugurated, and still persists at the M. G. H. with the result that some of our operators do have national reputations on the particular lines they have chosen, and yet remain excellent general surgeons. However, I naturally earned by this campaign a certain amount of hard feeling. My colleagues were very glad to have me attend to the shoulder cases, for nobody else was interested in them, nor has anybody selected that specialty since, although it is now twenty-three years since I studied my series of one hundred.

Meantime, I was stressing the End Result Idea and urging the staff and, through them, the trustees, to make our clinic the pioneer in the movement. Through private donations, I obtained the money to provide an "End Result Clerk," whose duty it was to endeavor to trace each patient a year from the date of discharge from the hospital and to enter the result, as noted by the doctor, who had operated, on an End Result Card. I hoped that the M. G. H. might become the example to which to point as a demonstration of the practicability of using this system. It did to a great extent and I was able to use it after the campaign began. (Report of Committee on Standardization of Hospitals. *Surg. Gyn. and Obst.*, Jan., 1914. Not in main volume, but in a supplement on Clinical Congress of Surgeons in back of bound volume, page 7. Another Report of the same Committee appears in the same Journal, 1916, 22: 119.)

I must now digress a little and speak of the Society of Clinical

Surgery, which had been in existence a few years before the London meeting in 1910, and was the first of the peripatetic societies organized to meet in different cities to have the members actually demonstrate to one another their operative work and methods of teaching and research. It was undoubtedly the example of this Society which led to the formation by Dr. Franklin Martin of the great meeting of surgeons which came to be known as the Clinical Congress of Surgeons of North America, and which had its first informal meeting in October, 1910, in Chicago. This Congress did not really take shape until the next year in Philadelphia, when Professor Edward Martin was elected President. It met in New York the following year (1912) and Dr. Edward Martin's first act was to appoint two committees; one to organize an American College of Surgeons, with Dr. Franklin Martin as its Chairman; the second, a Committee on Standardization of Hospitals of which I was appointed Chairman, with Dr. W. W. Chipman of Montreal, Dr. J. G. Clarke of Philadelphia, Dr. Allen B. Kanavel of Chicago, and Dr. W. J. Mayo of Rochester, Minn., as the other members of the Committee.

If the subject of the origin of the College ever interests historians, they may be confused as to the parts played by the two Martins. It is at least certain that the then President of the Congress, Dr. Edward Martin of Philadelphia, appointed Dr. Franklin Martin of Chicago to organize the College. Whether Martin of Chicago had previously appointed Martin of Philadelphia as President of the Congress so that he of Philadelphia could appoint him of Chicago to organize the College, I do not know. I do know that the idea of founding the College, to standardize the hospitals, was in embryo in the brain of the Philadelphia Martin two years before this, between Frimly and London. I clearly remember my own feelings, on arriving at this New York meeting in 1912, on being told that I had been appointed Chairman of the Committee on Hospital Standardization, and was to be responsible for the tail to be wagged. This was proof enough for me of the result of my talk in England two years before, with Edward Martin, about the End Result Idea. He had recognized the zealot in me, and had taken this opportunity to thrust on my Puritan conscience the duty to preach the doctrine I had expounded to him. Certainly he also recognized Franklin Martin's ability as an executive, and whoever made the suggestion, chose wisely in appointing him to head the Committee to form the College. Naturally this appointment interrupted my shoulder work, and for eight years my chief thought was to spread the End Result Idea among the surgeons and hospitals of this country. There was, as yet, no authority and little money

with which our Committee could work and propaganda was our only means. Since the other members of the Committee were busy men, the chairman had to take the responsibility and get the approval of his Committee afterward. The opening gun was fired at Philadelphia on May 14, 1913. Edward Martin, by adroit advertising, gathered an enormous audience in the great hall of the Academy of Medicine and I spoke on "The Product of a Hospital." (*Surg. Gyn. and Obst.*, April, 1914, pp. 491-496.) There was much that seemed very radical in this address, and the audience showed itself not only interested, but stirred. I asked and discussed such questions as these:

"For whose *primary interest* is it to have the hospital efficient?

For (1) The patient who seeks relief.

(2) The public who support the hospital and in return expect a high standard of knowledge on the part of their own private physician or surgeon.

(3) The hospital itself which, as an institution, has an individuality of its own.

Who represents or acts for these interests?

Strangely enough the answer is: No one; it is for the *interest* of no one. It is the *duty* of no one.

For instance: For whose *interest* is it to investigate what is the *actual result* to the patient operated on?

For whose interest is it to insist on the resignation of incompetent old Doctor So-and-So, who is one of the best fellows that ever lived?

Who will warn the largest contributor that his agreeable classmate, Doctor So-and-So, is totally unfitted to remove his stomach?"

"There is a difference between interest and duty. You do your duty if the work comes to you, but you do not go out of your way to get the work unless it is for your interest.

Let us make attention to the medical and surgical efficiency of the hospital the *duty* of some one."

I closed the address with the following suggestions:

"That each prominent hospital in this city appoint an efficiency committee consisting of a trustee, a member of the staff, and a superintendent.

That these committees inquire into the efficiency of their own hospitals with a view to answering the questions which are sure to come from the Carnegie Foundation" (which had just then agreed to help with the movement).

"That an example of this kind set by the Philadelphia hospitals would lead to the establishment of similar committees in other cities, and eventually lead to a national organization representing the patient, the public and the individual institutions."

The surgeons of Pennsylvania rose to the occasion and, under the leadership of Dr. Edward Martin, set a grand example. Dr. Baldy did heroic work, and, for a time, Pennsylvania was the shining light of this new form of hospital housecleaning.

The first report of our Committee was read at the next meeting of the Clinical Congress of Surgeons held in Chicago, November 11, 1913, as was also the report by Dr. Franklin Martin's committee which, thanks to most energetic work on his own part, had organized and incorporated the American College of Surgeons on May 5th of that year. Not until four years after this did the Committee on the Standardization of Hospitals become a committee of the College itself, and hence much more potent than had been our first merely suggestive Committee, whose only authority was the informal Clinical Congress. By 1917, Dr. Franklin Martin had produced a very strong, lusty dog to wag the tail of Hospital Standardization. Moreover, in the sixteen years which have passed since then he has kept that dog in a healthy, hearty condition, in spite of the fact that the incessant wagging of the tail has disturbed many slumbering hospital trustees and indolent or inefficient hospital staffs. He has given a most interesting account of the formation of the College in *Surg. Gyn. and Obst.* (1925, 40: 129), but, perhaps owing to lack of space, he does not mention the work which was done by our Committee during the period prior to 1917, when the cause was unpopular and the College was not strong enough to undertake the expensive practical program.

I think we deserve some credit not only for preparing the minds of those interested in hospitals, but of those who later furnished much of the money with which the work was eventually carried out. I have the greatest admiration for the way in which Dr. Franklin Martin has managed this organization and carried it thus far on its successful career. Nevertheless, although he has actually done the work, it is my opinion that members of the Society of Clinical Surgery, and especially Edward Martin of Philadelphia, helped plan the project and the methods by which it was launched, and have constantly and consistently helped Dr. Franklin Martin by putting their shoulders to the wheel whenever they were wanted. No further proof of this is needed than the public lists of those who have served the College as officers, the great majority of whom have been the members of this small Society.

Dr. Edward Martin is a person who dreads praise more than blame. He has shirked the public responsibility of receiving any kind of praise for the altruistic work he did in the period from 1910 to 1917, until the success of the College became assured. Dr. Franklin Martin has done the hard work and done it well, but the part that Edward Martin took should not be allowed to pass without public mention. I bear witness in this book, because I have given much of my energy through all these years to do what I could for the College

according to the ideas which were talked over with Edward Martin on the occasion above mentioned. Perhaps other memoirs than mine may prove whether the Philadelphia Martin, as I think, maneuvered the Chicago Martin into his part in the play, or that the reverse was the case. I am sure that the Philadelphia Martin, taking advantage of my dominant idea and my hereditary Puritan characteristics, made me the servant of his own plans. This is acknowledged in the dedication of the first report of my own hospital. Furthermore, if I had not been working on this plan between 1910 and 1912, I could not have produced the paper entitled "The Product of a Hospital," read in May, 1913, nor could I have produced my "Study on Hospital Efficiency," which was presented in May, 1914, at a meeting of the American Gynecological Society, where it was well received and published in their transactions (Vol. 39) of that year. In this study I was able to use the cases which had been at my own hospital from its opening, August 25, 1911, to July 30, 1913, as a practical example of the operation of the End Result Idea. The correspondence which I conducted during these years has been stored in the Boston Medical Library, in case it may be of interest to some future student of this era. Whatever historians may ultimately conclude, I am personally satisfied that the End Result Idea took an important part in the founding of the College and that this is proved by the first report of our committee in 1913, although the basic suggestions contained in the report have been obscured in the complexity of the record systems later recommended by the College.

During all the time that I was trying to saddle our medical community with the End Result Idea, I do not recall ever hurting any of my colleagues or trying anything more unfair than harmless ridicule. I may have hurt their feelings. If I picked at all on individuals, they were men in high positions such as President Lowell, Dean Bradford, Dr. Washburn and Richard Cabot. I doubt if their feelings were hurt or even their self-esteem. I talked to trustees only through their staffs, the press and my pamphlets. I did not even go behind the backs of the members of the staff of the M. G. H., although a number of the trustees were relatives, personal friends, or members of social clubs to which I belonged. There was one exception — a cousin who was a trustee of another hospital, to whom I used to vent my ideas, but he thought little of them, as cousins usually do of those of their younger cousins, whom they remember as little, freckled-faced boys.

In order to attract the attention of the trustees of the M. G. H., I resigned from the staff in 1914 "as a protest against the seniority system of promotion," which was obviously incompatible with the

End Result Idea. On the day on which I received the acceptance of my resignation, I wrote again, asking to be appointed Surgeon-in-Chief on the ground that the results of my treatment of patients at their hospital during the last ten years, had been better than those of other surgeons. I had tabulated my results in case they should ask to see them, but as no one had ever inquired into the results of other surgeons, there was of course nothing with which to compare mine. Thus, as I had planned, this fact was brought to the notice of the trustees, although at some personal sacrifice on my part. Naturally, my letter was ignored, and I was not appointed Surgeon-in-Chief. However, it was not long before the seniority system was dropped, and a portion of their budget became devoted to a Follow-up System.

It became apparent to me that the medical profession of Boston, its great hospitals and the Harvard Medical School, must be made to pull together with real strength of will, if Boston was to set the example in this movement. Only three great cities in the country were, in my opinion, fitted to take the lead — Boston, Philadelphia and Baltimore — for in each of these cities the majority of the profession were graduates of their respective medical schools and, therefore, there existed in each a certain *esprit de corps.* Philadelphia, at that time, was a little ahead. It seemed to me that Boston had the best opportunity, for the Harvard influence extended not only through the medical schools and hospitals, but into the banks and into every branch of business, philanthropy or social endeavor. There were two ways open to unite the wills of the various branches of our community, leadership on my part, or a defence-reaction on theirs. Had I the qualities of leadership, I might inspire a band of hard-working lieutenants, and in time succeed in uniting the required number of wills to change any precedent either at Harvard or at its affiliated hospitals. This would be a matter of many years, and I was only a junior surgeon who must also earn his living. I had, on the other hand, observed that the defence reactions of our social forces were fairly prompt and forceful. Harvard is sensitive to ridicule, and also, I sincerely believe, to presentation of facts. If I could awake the steam roller of Harvard public opinion, either by a clear presentation of facts, or by well-advertised ridicule, I felt sure I could get at least a united defence-reaction and some inquiry into existing conditions. I was confident that the End Result Idea would become an intellectual landmark of which any university would be proud, and which, in time, Harvard would claim as a jewel in her crown, and set it with the diamonds of ether anæsthesia and social service.

I was so much influenced by the End Result Idea that I even con-

sulted two friends who were distinguished alienists and put the plain question to them: Am I a victim of a dominant idea because I am willing to make the main object of my life the demonstration of the importance of the simple plan that hospitals should constantly inquire into the results of the treatment of their patients, and modify their organizations when necessary to obtain better results? I received the answer from both alienists that the degree of mental pathology varied with the value of the idea and with the degree of success in making it appreciated. This comforted me somewhat and made the experiment more interesting, for there was some criterion to look forward to as to my own sanity. However, I was a little in the position of the child who prayed, "Now I lay me down to sleep in my little bed; if I die before I wake, how will I know I am dead?"

I admit now that I should have done better to choose either one or the other path and have become a leader or a satirist. I tried to do both and probably lost some time thereby. I tried to run a hospital of my own with an organization to set an example, and at the same time by publishing aggressive reports from this hospital, to apply a little ridicule to Harvard and its affiliated institutions.

As I was Chairman of our local Medical Society in 1914, I took the opportunity to arrange a meeting with the following announcement:

A Meeting for the Discussion of Hospital Efficiency
AT THE BOSTON MEDICAL LIBRARY,
WEDNESDAY, JANUARY 6TH, 1915, AT 8.15 P.M.
UNDER THE AUSPICES OF
THE SURGICAL SECTION OF THE SUFFOLK
DISTRICT MEDICAL SOCIETY.

Up to the present time the public and the medical profession have regarded Hospitals as places for the treatment of the sick, but not necessarily for their *efficient* treatment. Attention has been paid to the cleanliness of institutions, to the architectural arrangement of the buildings, to the kindliness of the staff and nurses, etc., but no attempt has ever been systematically made to determine whether the treatment so freely given has been efficient — that is, as successful as possible.

In most hospitals there has been no official or department whose duty it has been to ascertain the results of treatment at

all, much less to compare the results attained by different members of the staff in any one institution, or even to make a collective comparison of the results attained by the whole staff, with those of another similar institution.

Evidently, Trustees, as a rule, have felt that the best they could do was to appoint respectable men on their staffs and then to leave the degree of efficiency of the treatment given the patients to the individual conscience and ability of the physician or surgeon on duty. The terms of duty have been arranged by the calendar or by seniority.

Obviously, if there is any difference in the value of the services of one surgeon or physician and another — which the public seems to admit by its willingness to pay large fees — this difference must be capable of demonstration by some comparative test, so that the distribution of the cases may be made more rationally than by the calendar or by seniority. No physician or surgeon nowadays can be expected to be proficient in all the branches of even a single specialty.

Has the time come when hospital organization can be based on the idea of giving the patients successful and effective treatment as well as care and kindness? Is it possible to compare therapeutic results in medicine and surgery, or must we admit that no matter how much we read, study, practice and take pains, when it comes to a show-down of the results of our treatment, no one could tell the difference between what we have accomplished and results of some genial charlatan or some less painstaking and energetic colleague?

Comparisons are odious, but comparison is necessary in science. Until we freely make therapeutic comparisons, we cannot claim that a given hospital is efficient, for efficiency implies that the results have been looked into. Hospital efficiency is mainly therapeutic efficiency.

The meeting on January 6th is to stimulate thought on these questions. Has it occurred to you that no person or department in a charitable hospital is responsible for the medical and surgical efficiency?

The speakers will discuss the question of who should be responsible.

The following is the provisional Programme:

Hospital Efficiency from the standpoint of an efficiency expert.
MR. FRANK B. GILBRETH, of Providence, R. I.

Hospital Efficiency from the standpoint of a hospital surgeon.
DR. ROBERT L. DICKINSON, Brooklyn, N. Y., Surgeon to the Brooklyn Hospital (Gynæcology).

Hospital Efficiency from the standpoint of a hospital superintendent.
DR. HERBERT B. HOWARD, superintendent of Peter Bent Brigham Hospital.

Hospital Efficiency from the standpoint of hospital trustees.
DR. WALTER WESSELHOEFT, trustee of the
Mass. Homeopathic Hospital.
DR. JOEL E. GOLDTHWAIT, trustee of the
Robert B. Brigham Hospital.

Hospital Efficiency from the standpoint of a public servant.
His Honor Mayor JAMES M. CURLEY.

General discussion.

E. A. CODMAN, M.D., *Chairman.*

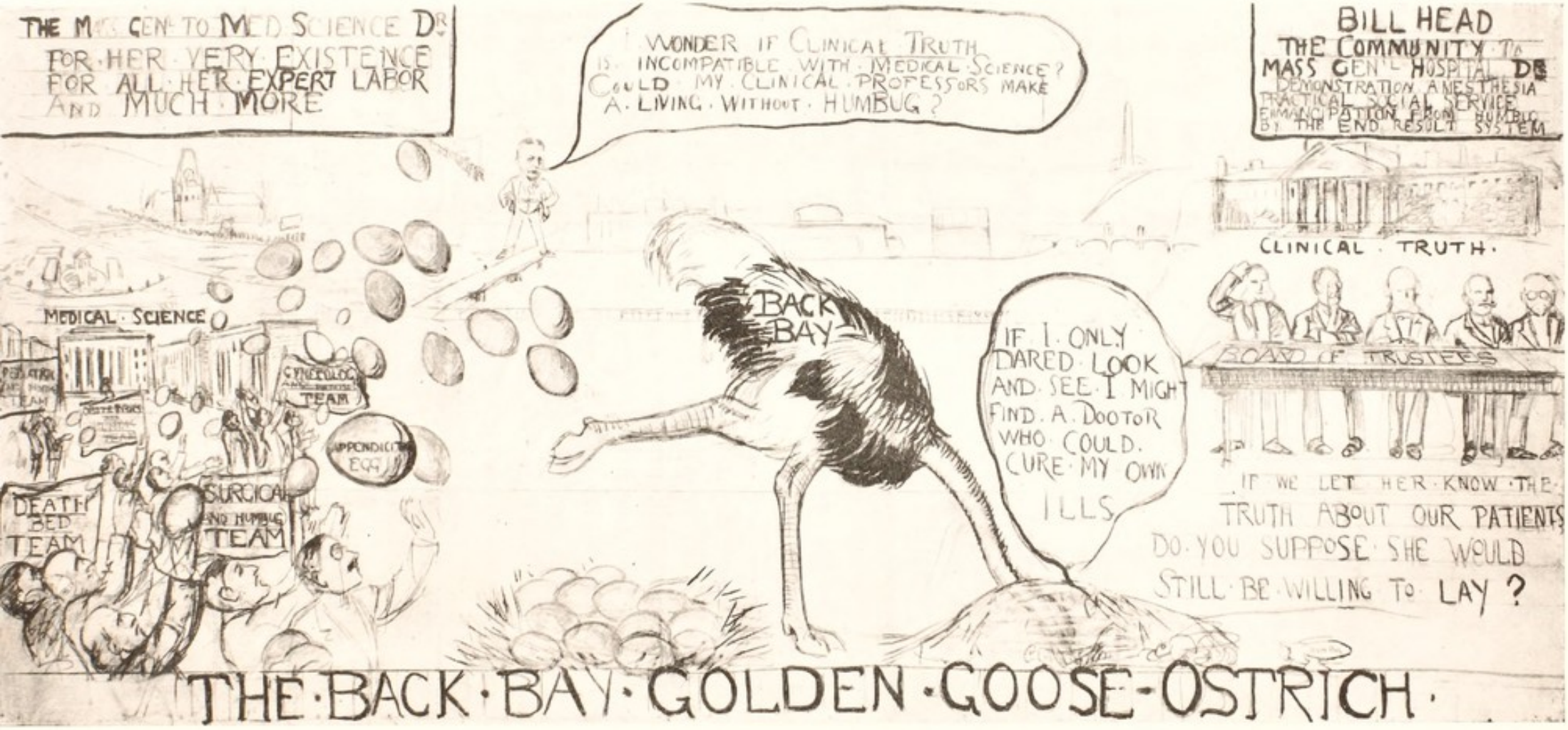
BETH VINCENT, M.D., *Secretary,*
295 Beacon Street, Boston.

It was not possible for me to get the proper speakers for this program. I should have had the President of the University, the Dean of the Medical School and the Chairmen of the Trustees of the larger Boston Hospitals. Even some of the leaders of the local profession or representative successful practitioners would have helped. Unfortunately, the President of the University had an engagement for a small social dinner far in advance of the date of the meeting, and so on down the line. Nobody in any position of authority in our medical school cared to take the responsibility of answering these simple direct questions. They all knew that the answer was, that nobody was responsible for examining the results of treatment at hospitals, and that the reason was MONEY; in other words, that the staffs are not paid, and therefore cannot be held accountable. Furthermore, I knew that even the speakers whom I did succeed in obtaining, could not, as guests of our Society, be as frank as perhaps they would like to be, and would not suggest the reason for it, although they might admit the fact that there is no analysis of results in most hospitals.

To make sure that the questions should be answered, I had prepared beforehand, secretly, a cartoon about eight feet long and concealed this under a cover at the back of the stage, ready for use at the end of the discussion, in case no one on the platform or from the audience should dare to suggest the almighty dollar. The large hall in the Medical Library was packed. There was hardly standing room, and the size of the audience was so unusual as to indicate a real desire to hear the questions answered. The presence of the Mayor insured publicity. Moreover, the speakers were interesting and succeeded in sustaining a certain degree of excitement. As my ideas were already known to the majority of the audience, there was an expectant silence, when I rose to close the meeting.

I told of my efforts to get responsible local speakers, how one had a dinner engagement, another was too busy, etc. Then I said that I would present my own answers to the questions in the form of a painting which had kindly been made for me by a friend — the late Philip L. Hale, the artist. No one but Mr. Hale and myself had seen this cartoon, not even the officers of the Society, the secretary of the meeting, or my own wife, who was in the audience. I did not wish any one to share the responsibility for the shock I knew it would give that audience, and I so stated before it was unveiled, with a great flourish, by Mr. Galbraith. The audience held its mouth open while I explained the meaning of the picture, and even after I had finished, continued to be aghast for a minute or two. Then there was as near an uproar as ever I have seen at a Medical Meeting. Some fine old men who had loyally worked for the university, and whose careers I respected, got up and walked out with bowed heads. Other younger ones of the same type rose together to seek the floor, with anger but with nothing practical to say. The great majority, however, were amused more than they were shocked, and a few even risked their reputations by coming publicly forward and shaking hands. For weeks some of my friends did not speak to me, and if I entered a room where other doctors gathered, the party broke up from embarrassment or changed their subject. I was asked to resign as Chairman of the local Medical Society.

For some months I was in disgrace, but the publicity obtained, which spread not only in our local papers but in those of all the other large cities, fulfilled my expectations. My wife and friends had to explain the whole matter daily to other friends, and everybody had to say that what I was after was all right, but my *methods* were abominable. As nobody else was doing anything about what all admitted was true and important, I had no methods to compare with mine, which did not seem to me either dishonorable or cruel to any one in particular. In a newspaper or in the *Lampoon*, the cartoon might hardly have been noticed, but at a "scientific" meeting of a Medical Society, attended by the Mayor (although he left after his own speech and did not see the cartoon), it was sure to create the reaction I desired. Soon after, I was dropped from the position of "Instructor in Surgery," which I then held at Harvard. The chart shows the personal financial depression which followed the cartoon, but this did not last long. I had already resigned from the M. G. H. as explained above, and thereafter had only my own hospital for clinical opportunity.



THE BACK BAY GOLDEN GOOSE OSTRICH

However, I continued my campaign from my hospital, and in 1916 published my third "Study in Hospital Efficiency." This was quite a volume, and since it covered the End Result Reports of five years' work, and also an analysis of all the cases which had died after my operations in fifteen years at the Massachusetts General, it presents ample evidence of the value of efficiency analysis. I sent a copy of this report to every member of the Massachusetts Medical Society and of the American College of Surgeons who would receive one, at a personal expense of about $3,000.00. I still have many copies of this study, one of which I can send to any one who wishes to exchange two dollars for it. I could barely induce people to receive it as a gift at that time, but now I feel that it has a money value. It pleased me greatly that later I had many requests for copies from hospital trustees. In the case of the Woman's Hospital in New York, almost everything that I recommended has been adopted, and I am glad to say improved on, in many details. Several other New York hospitals also accepted the suggestions to some extent. This is evidence that with my sauce of ridicule I served some very solid food for thought. Moreover, in all this campaign I have stood in a glass house while I threw my stones at others, whether they were doctors or trustees. As I did fight from a glass house, I am grateful to them for not destroying me altogether, for, since then, the Harvard Medical School gave me a room for five years from which to conduct the Registry of Bone Sarcoma, and the Massachusetts General Hospital has, since 1929, honored me with the appointment of Consulting Surgeon, which enables me to operate on private patients whom I have referred to its various departments. Still more remarkable than this, the cartoon has been mounted on cloth, arranged like a folding map, bound and placed for safe keeping in the Boston Medical Library. I publish this cartoon now, because, having been condemned by a previous generation on its account, I hope that I may be judged by a future one to whom the subject will appear less serious. It depicts President Lowell standing on the Cambridge Bridge, wondering whether it would be possible for the professors of the Medical School to support themselves on their salaries, if they had no opportunity to practice among the rich people of the Back Bay (the residential portion of Boston). The Back Bay is represented as an ostrich with her head in a pile of sand, devouring humbugs and kicking out her golden eggs blindly to the professors, who show more interest in the golden eggs than they do in Medical Science. On the right is the Massachusetts General Hospital with its board of trustees deliberating as to whether, if they really used the End Result System, and

let the Back Bay know how many mistakes were made on the hospital patients, she would still be willing to give her golden eggs to support the hospital, and would still employ the members of their staff and thus save the expense of salaries. Across the river and over the hill are seen armies of medical students coming to Harvard because they have heard that the End Result System will be installed in her affiliated hospitals.

A few of my contemporaries have credited me with "moral courage," but I deserve no such credit, for I have merely reacted to stimuli. I may relate, as a parallel, an incident which occurred in 1897 in a part of the country called the Big Horn Basin, then a wild and desolate region. I was traveling with a "pack outfit" when, one day, we met a lone rider with a huge, ugly-looking dog, more like a mastiff than one of any other breed. The rider was a typical "bad man" who "toted" two guns and was quite as unprepossessing as one of the western villains of the modern movie. He rode along with us for a time and presently we came across a small herd of cattle, loose on the range. The dog made for them and rushing into the herd, "cut out" a cow and a calf, and proceeded to grab the calf by the ear. The bellowing cow made repeated dashes at the dog, but at every rush the dog would swing the calf around so adroitly that the mother butted the calf instead of its enemy. What with the bellowing of the agonized mother, the squealing of the frightened calf and the growling of the dog, it was a noisy and most unpleasant scene. As the owner of the dog did nothing, I dashed up, dismounted, mixed up in the rumpus and kicked the dog with all my might. He crumpled up with a gasp and lay still, so that I thought he was dead until presently he pulled himself up and whimpered off with his tail between his legs. I got away with it, but I was probably in the presence of the greatest danger of my life, not only from the two-gun man and the dog, but especially from the cow. The act was from impulse and was not a product of thought or intention. I simply could not stand the sight. It was folly, not courage, and the other youth in my party had more sense than I. Our old guide later informed me that the dog was very valuable to its owner, who was a "rustler" and had trained the animal to "cut out" calves and hold them to be branded; but the point I wish to make clear is that my real danger was from the cow, whose cause was also mine.

Whatever credit I may deserve for my tirades should not be for moral courage, but it seems to me that I deserve some credit for restraining myself as well as I have done, having once started on the campaign. To see our hospitals turn their faces away from evident

facts is as repulsive to me as to see a calf bullied by a dog, while I stand by and do nothing. What straight-thinking surgeon will deny that on any day at any public hospital one may see suffering, greater than that calf's, caused by incompetence or by neglect of well-known principles of diagnosis or treatment, on the parts of either the physicians who treated the patient before his entrance into the hospital, or those in charge at the hospital. It is not these physicians I attack, but the lack of logical organization in our profession where, as students, we get instruction by example. I am sure I have suffered in greater degree by refraining from saying more about our medical and surgical customs than I have from the professional and social slights received in consequence of saying as much as I have said. If the western scene had been in the center of our Harvard Stadium, and the owner of the dog had been a favorite of the university, imagine what a brute I should have been called! The university, like the poor cow, regarded me as an enemy for trying to remove humbug from its grip on her offspring's ear. However, social position protected me in both instances. On the plains my companions carried guns and our old guide had his rifle across his lap. When I attacked our system of medical education, argument was impossible, for every man in the audience knew that I was right. I had held it naked in the glare of publicity, and the only thing that could be done was to ignore the episode, and to proceed to dress it up a little in case of another flash of lightning. There is now an improvement in the salaries of our professors, who are able to confine themselves more strictly to their teaching and their records, but a thoroughgoing comparative analysis of the results at their affiliated hospitals is not, as yet, a conspicuous feature in their prospectus. They are still obliged by the sentiment of our community to give more publicity to the art than to the science of medicine, while they continue to perform their individual experiments.

When the War broke out I was contentedly carrying out my numerous plans. My friends in the Society of Clinical Surgery, although perhaps also shocked at my methods, were loyal. Even my former comrades at the M. G. H. showed some forbearance and were doing what they could to help. The fellow members of the Committee on Hospital Standardization had signed my reports, although at their own hospitals they were unable to fully exemplify the high ideals which we were recommending to others. By 1917 the American College of Surgeons had taken over the Hospital Standardization work, and it has since flourished under the able leadership of Dr. MacEachern. I was enlarging my little hospital as fast as I could make,

or borrow money. The M. G. H. was maintaining its End Result cards; the follow-up system and the special assignment policy were flourishing. Other hospitals were following suit. My operations at hospitals other than my own were supporting me and slowly helping me to expand mine. I heard more and more signs of appreciation of what I was trying to do. I often think that had it not been for the War, my plans would have reached a real fruition, but when War came, the thoughts of men, my own included, left their jobs. My appeals for improvement fell on deaf ears. Most of us worked in the line of our habits, with minds on the War and intellectual processes wandering even from hobbies. No one was interested in avoidable improvements in hospitals or in ideals. Even with my head full of the latter, and although intellectually I could take Germany's point of view, I, too, wanted to volunteer and be with other friends who were joining British units. However, my hospital was holding me fast, and so did my conscience, for I felt I could do more for my country by making a demonstration of the End Result Idea than by doing what other surgeons could do as well in the Army. Conscience is a queer thing in war time. It appears in the hard-boiled and in the ne'er-do-well who have never manifested it before; it makes moral heroes out of some physical cowards and turns some honest men into daring spies or into chivalric aerial murderers. As in peace, we use it to excuse our doings. I regard my own as a troublesome inheritance from my Puritan ancestors which must to some extent be appeased like any other appetite. It is as much a part of me as my foot, although under excitement both may be forgotten for a time.

Then came the great disaster at Halifax. I telegraphed my good friend, Dr. Thomas Walker of St. John, New Brunswick, offering the help of my hospital staff and a few hours later we were on our way. Dr. H. V. Andrews of Boston fortunately came with us, for he soon demonstrated how a surgeon, who has earned his living in general practice during his early years, can be of more help in such an emergency than any single specialist, for our patients, although for the most part needing surgical care, also presented problems in the whole range of specialties, from obstetrics onwards. We took part in the organization of an emergency hospital, which, when we left two weeks later, was running smoothly with an End Result Card for every patient. Although these cards may now be as scattered as the leaves of the Cumæan sibyl, at the time, they served to keep my finger on every pulse in the hospital and to illustrate the simplicity of installing the plan, even in a city paralyzed by a calamity. One physician told me that in half an hour he had used all his stock of drugs, and found

himself of more use as a man in helping steadily for two days to rescue those still alive who were buried or crushed in wrecks of houses than in his rôle as a physician.

Having left, with regret, the work in Halifax, where, as Major in the Medical Corps of the Canadian Army, I could have been of real service, and having returned to my hospital purely to try to save money, an indescribable restlessness came over me, until in September, I found myself in our own Medical Corps, wrestling, as Senior Surgeon of the Coast Defences of the Delaware, with the impossible "paper work" of our Army, in the midst of the influenza. Presently, what with patients and enlisted men assigned as orderlies to care for them, I had more soldiers under me than had the general in command of the three old forts. At the end of my endurance, I stood one night in the upper ward of the old hospital in which had been concentrated those whom I judged to be hopeless. My other medical officers were sick abed — even my tireless and capable junior, Captain Ellis, was on that night exhausted. The floor was slippery with bloody sputum; there were no nurses; no petticoats of any kind; no bedpans; no gauze and few medicines; in fact, there was no medical or nursing care. Those that were able lurched to a toilet with the aid of some other soldier who had yesterday been a recruit and now found himself an orderly in this death house, mopping up bloody slime from the floor or cleaning the bed of another boy after he had helped to dump the body and the soiled blankets in a box. I turned and said good-night to those boys who were facing their dangerous duty as bravely as those who fought in the trenches. After a few hours I was able to get up and go on with the "paper work," reporting the numbers of sick and dead, filling out the death certificates, making applications for transfer of insane recruits and otherwise obeying the orders of my competent subordinate, a Sergeant of the Medical Corps. There was no time to test the simplicity of my system of records, but there was necessity for me to learn the use and deficiencies of the Army System.

In November, as Regimental Surgeon in the Artillery, I had a card for every one of 1,800 men, and enjoyed the new duty of studying how to keep men well, and of getting rid of them when sick. Even the old army sergeant was surprised when I would send for a certain list of men to see whether they still had scabies, or some other minor ailment, and no orders had come for an inspection. He gave me to understand that it was customary to wait until a disease had spread sufficiently to attract some attention from a mysterious medical headquarters, which then issued orders to inspect and report. There was

much that was interesting to me about this experience, but as it does not bear on my subject I will merely add that, at this camp in Virginia, after the Armistice, I received an honorary appointment as Fish and Game Officer for the General in Command, and my Christmas leave was spent in camp with daily expeditions after pike and bass, duck, quail, and wild turkey, with my agreeable superior.

In January, 1919, not having applied for discharge, because the need for medical officers seemed greater than before, I was transferred to be Surgeon-in-Chief to the Base Hospital at Camp Taylor, and again had a chance to test the working of my record system. Five hundred hospital beds and some 300 convalescent soldiers in barracks gave an excellent opportunity. An orderly carrying my box of cards attended all visits or operations. The cards were not substituted for the regular records, but served to keep in touch with them when desirable. The senior surgeon of a hospital of 500 beds, if he worked eight hours a day, could give less than one minute to each patient, even if he did no operating or executive work (60 minutes $\times$ 8 hours $=$ 480 patients), yet, with the aid of my catalogue and of a good orderly, who has since become Dr. Fraasch, I kept a certain amount of supervision over every patient, operated on many, dressed difficult wounds, and made personal notes on the condition of nearly every soldier at entrance, and again at discharge. At least once a week I inspected each serious wound and often had consultations in the Medical Wards as well. My cards were of the greatest help, for I could talk over his cases with each ward officer as often as seemed desirable. However, I must admit that my day was often longer than eight hours.

In June, 1919, I returned to my closed hospital, in debt, with no borrowing capacity, and somewhat disillusioned as to the possibility of altering the ways of human nature by my intellectual efforts. I had patched up too many fine young men to feel much enthusiasm about keeping the aged and infirm alive, or to listen with any pretense of sympathy to even the nicest lady's description of the daily behavior of her digestion. I determined to be a money-maker, at least until I had paid off my debts, and for two years charged most of my patients three times as much as formerly. My hospital reverted to an apartment house, and for nearly a year I steadfastly abstained from embarking on any new adventures for the benefit of coming generations. Nevertheless, I subtly drifted into the organization of the Registry of Bone Sarcoma, because one of my best patients had a bone tumor. My dream was that this one disease could be used as an example of the inadequacy of our present methods, and that some

day the records would serve to demonstrate the value of the End Result System in hospital organization. So far as establishing an undeniable record of our present inefficiency goes, I have succeeded, but even now there is little organized effort to prevent these unfortunate patients from being treated for "rheumatism," until it is too late to save them. I have probably spent more time over this Registry, during the last thirteen years, than the average medical student requires to get his degree, yet, in all this time, I have had the actual care of not more than a half dozen patients with this disease, perhaps less than if I had not written at all on the subject. Many consultations, of course, but the patients are not turned over to me, although one may live next door. What is the reason?

The hardest thing in my Quixotic career to explain to my colleagues is my plan of free consultation in cases of bone sarcoma. Why spend years studying a subject and then refuse consultation fees? In the first place my object was not to make money, but to illustrate a principle. In 1920 we had, in this country, four eminent authorities on this subject, Bloodgood, Coley, Ewing and Mallory, who, by years of study, had amply earned more than they would ever be paid. I doubt if any of them have received three dollars an hour for the time they have spent studying, reading and writing about, lecturing and operating upon, or caring for patients with diseases of the bones. I did not wish them to consider me a competitor, but as a helper, and furthermore I needed their authoritative opinions to establish the Registry. These men became authorities because they had been earnest students and teachers. I believe all of them regard the large fees that may occasionally be paid them by the well-to-do, as merely necessary incidents in their work — a vicarious payment at hourly rates for labor and expense. I have received their coöperation, especially that of Dr. Ewing, who has, even more constantly than the others, been willing to undergo that extreme test of authoritative opinion, a written diagnosis before treatment is undertaken and the eventual result known. This is a real test, not only of knowledge, but of integrity, for necessarily these authorities have voluntarily signed their names to mistakes in diagnosis which may cost life or limb, and, in future, be regarded as evidence of ignorance and even of stupidity, after the results are known and better diagnostic methods have become established. Enough years have already passed for most of us to feel humble.

The second reason was to illustrate how little recompense comes to the true student of difficult medical and surgical fields, except the honors accorded them, if they point a way which others may follow

with profit. The public pays the prominent surgeon who amputates the leg, not the scholar who first describes the disease, the practitioner who makes the early diagnosis, nor the pathologist who takes the real responsibility at the exploratory operation and gives the final decision to amputate. The surgeon can do a satisfactory amputation before he graduates as interne, and from that time until the day when his hand is tremulous, bifocals are necessary and his own interne keeps him in hand. One surgeon can amputate practically as well as another, but in 1920 there were not a dozen surgeons in America who had an adequate knowledge of the different varieties of bone tumor, to tell, with reasonable certainty, whether, in a given case, amputation was indicated. Even now, when what knowledge we have has been put in an available form, there are not many who have studied it, and yet any surgeon will consider himself justified in making this decision for a patient, perhaps in consultation with some more prominent surgeon who has little real knowledge but much authority.

I wished to make my knowledge of Bone Sarcoma so conspicuous that my opinion would be acknowledged to be of real value, and by making no charge for consultation, clear myself forever from the imputation that I was advertising for that purpose. On the other hand, if a patient, rich or poor, were referred to me to be treated, I would accept the responsibility and operate or not, as I thought wise, making a moderate charge for the conduct of the case, just as I would in one of appendicitis or of some other condition requiring routine surgical technique and less expert knowledge. Furthermore, I was still somewhat in disgrace at that time, and had no charitable hospital appointment and therefore could care for no really poor patients. I wanted to make this obvious, for the paradox showed that the trustees of hospitals do not appoint their surgeons because of their knowledge. In other words, my attitude would constantly bring up to those surgeons, who happened to have a case of bone sarcoma, the questions: "Have I a right to operate on this case merely to get the fee for amputation when there is another available surgeon who can not only amputate as well, but has also spent years in the study of similar difficult cases and whose advice I may ask with no cost to the patient? Would not this patient prefer the care of the other surgeon if he knew the facts? Are we doing what is fair by our patients if we let them think that skill in operating or general reputation are more important than knowledge about their diseases?" This was not a policy likely to increase my popularity. I did not wish to become known as a surgeon who had special skill, but I did wish to induce the

above train of thought in the minds of my colleagues in order that they might talk about these questions at their clubs.

Bone sarcoma was a particularly good illustration, not only because I had no claim to special success in treatment, but because it is a rare and usually fatal disease in which accurate diagnosis and wise advice as to choice of treatment are far more important than the slight superiority in operative dexterity which any one surgeon may possess above the average. In 1920, blunders in the diagnosis or in the choice of treatment were very common in these cases, but could always be pardoned because the most eminent surgeons made similar ones. I had no claim to preëminence as an operator, but the amount of time I had devoted to the study of the pathology and to tracing the results of treatment, would, if the patients themselves could have known its extent relative to that of the surgeons into whose hands they came, have given them some misgiving. My chief interest in all this work was to show, in epitome, an example of the End Result Idea. Could any hospital, which really aimed to do its best for its cases, permit patients with a rare disease to be cared for by members of its staff, no matter how dexterous, who were not conversant with all attainable knowledge about that disease? What incentive would there be to thoughtful young men to spend years in the study of obscure conditions, if patients with these conditions were to be assigned, by the ward or by the calendar, to other less studious surgeons who were too busy making money even to read the literature of the subjects?

Of course these ideas of mine are unpopular with the majority of our profession who have spent their lives in the practice of the art of medicine rather than in that of the science, and, being financially successful, are able to influence the trustees of hospitals against an analysis of results. For years they have deceived themselves into thinking that they were giving their services to the hospitals, and comparison of achievements would be, to them, as odious as a comparison of incomes. They know our results are not as brilliant as the public thinks. They cannot understand my attitude of beseeching them, the country over, to make a "show-down" in at least the few cases who have bone tumors. They vaguely, and I think correctly, fear that if we succeeded in collecting complete records of every case of bone sarcoma, the evidence would lead to radical changes in our hospital methods. At present there is only a minority which desires such reforms. All honor to the men who have registered the 1,500 cases so far received; they have truly contributed to science.

And now this book is presented to you as a final illustration of my life work, rather than as a monograph on the shoulder. It offers you an instance of how an *apparently* trivial injury may fall through the mesh of the loose net of our present system, and how the cost of this leakage is paid by the community in the end. The epilogue will use the same "trivial" lesion to illustrate the need of some form of advertising, in order that patients with rare or little known diseases may promptly reach those doctors who are best qualified to care for them. I propose to show that, twenty-three years ago this lesion had already been accurately described, its symptom-complex clearly recorded and appropriate treatment pointed out, yet, in the hospitals of the world today it will be found that it is often unknown, seldom recognized and rarely successfully treated. This is a state of affairs which could not exist under the End Result System, for no "uninteresting" condition which causes prolonged pain and disability, could be thus neglected. Furthermore, I hope to convince you that the cost to the community of only 100 neglected injuries of this kind would have paid for my own schooling and medical education, added to all the money I have ever inherited or received as professional fees. Hence the little study in the cost of medical care which is shown on my chart.

I have given you an account of efforts rather than of achievements, for much has been attempted that has not been accomplished; even the standardization of hospitals on the basis of End Result analysis and the Registry of Bone Sarcoma are not yet successes. Although my effort on every interest has been largely futile, I have at least worked hard while my wagon was hitched to each star. It is difficult to measure the degree of success one attains in anything, but there may be a law of the Conservation of Human Work, just as there is one of the Conservation of Energy, viz., that if a man labors earnestly for some object, while he may not attain that object, his work will count in some way. The by-products may be more important than the product for which an industry was established. I like to think, for instance, that my End Result Idea had a part in the origin of the American College of Surgeons, and is the basic principle of any rational hospital standardization; that the Registry has helped to diffuse the knowledge of bone sarcoma which Ewing possessed, and that it has also afforded opportunity to the brilliant mind of Kolodny; that the introduction of the policy of Special Assignments has made the Massachusetts General an example to other hospitals, has led to many contributions in difficult fields of surgery, and furnished our community with some true specialists.

And now perhaps this book, in which I advocate a plan that all hospitals make, for a time, a combined study of the shoulder, may lead to some unexpected transformation of my labor. If work is accurate and earnest, I do not believe the amount need be large. Momentum is the product of mass and velocity, and there should be a parallel law that the momentum of one's labor should equal the degree of intellectual accuracy multiplied by the quantity of energy given to it. Röntgen wrote his masterpiece in a few months and the small quantity of work was multiplied by a superlative accuracy, yet his contribution would have been impossible without the momentum of the labor of Crookes and Lenard, who produced the X-rays without observing that they had done so. Only the apex of the projectile was defined by Röntgen, but the momentum of all the work on electricity since that of Galvin and Faraday, sent it in a flash around the world. I would like to make this book so accurate and truthful in every way, that it will greatly multiply the labor it has cost. Its intellectual momentum should be of use somewhere, even if not in the way I plan. It may add to that of a book by Veresaeff, "The Confessions of a Physician," published in 1904 by Frederick A. Stokes Co., New York, and Grant Richards, London.

Have you the impression that you have been listening to a hard-working student who has spent his days and nights, months and years, in the belief that he was anointed to reform his brethren? If so, it is false, for I am no reformer on any principle, but merely because I am naturally disgusted with humbug, self-deception, hypocrisy, smugness, cupidity, and injustice, just as many another may be, who has not been in a position to indulge his prejudices. I believe Don Quixote had a good time, and I have had mine, too. What if we have occasionally attacked windmills? There was the joy of battle. I really have no moral principle beyond trying "to do as I would be done by," "honesty is the best policy," and a few other human maxims.

Although seeing no clear reasons for belief in, or worship of, a deity, and having no expectation of an after-life, I find little intellectual difficulty in explaining to myself a desire to take what is usually called a moral point of view. I am satisfied with recognition of the fact that a happy and satisfactory life is impossible, unless one has the sense of being of service to others. The normal individual cannot evade this conclusion, for heredity from countless generations has given him a tribal instinct to appease.

It is so instinctive to wish to be popular with, rather than despised by one's own generation, that great philosophic effort must be made

to satisfy this tribal urge in substituting appreciation after death for present wealth or honor. But if the prophet is confident of the value of his service, he may keep his equanimity in spite of the jeers of his contemporaries. Although the End Result Idea may not achieve its entire fulfillment for several generations, I hope to be as content when dying as any soldier on the battlefield, who, although he may have fought for quite the wrong side, feels the glow of patriotism, or as many an old financial baron, breathing his last in his four-poster, convinced that he has left his children protected from a wicked world. Honors, except those I have thrust on myself, are conspicuously absent on my chart, but I am able to enjoy the hypothesis that I may receive some from a more receptive generation.

Agnosticism has advantages over most creeds. It accepts without thanks, from generation upon generation of ancestors who have painfully perfected it, the wonderful psycho-physical machine we call mind and body. They did not voluntarily perfect this machine on our account, although they may have prayed for our souls as well as their own, during a small time-fraction of their racial existence. We need not thank them, but now that we have come to realize that we shall bequeath similar machines to our progeny, we might do something more and leave them instruction books describing the detail of parts, oiling methods and general care. To my mind, morality is almost synonymous with the degree of wisdom we use in caring for and enjoying the use of these delicate but resistant mechanisms and of the ambitions and desires which came with them. Complete enjoyment of our seventy years of allotted use implies the exercise of all the virtues. We must be moderate and proportionate in driving it, although it has been built with self-regenerating mechanisms to stand the vilest abuse. Its desire for a place in the community is as real as its mechanical wants. For full enjoyment all such needs must be acknowledged. If we become disgusted with the folly of our own generation, we can always employ our machine to do something which we think will make life more enjoyable for a future one. My contribution will be instructions as to how to avoid delay on the road when a certain tendon is broken. But appreciation for such work must be postulated, and this is not easy, although to my mind preferable to the embarrassment of accepting the keys of a great city.

Perhaps most creeds have erred in pointing to the past instead of to the future. Men have sanctified and deified their ancestors, and have even sacrificed themselves and each other to them, but no religion of which I know has pointed to the heaven which might exist on earth,

if, neglecting our own souls, we sacrificed for the normal lives of the generations yet to come. Many individuals have done this, but it has never been a creed. Why not deify the supermen we may take part in making? There would be more logic in being burned at the stake as a part of a carefully-conducted series of experiments which would eventually render humanity fireproof, than merely to save our own souls. Even if without going so far as to be martyrs, we all gave up accumulating property, and constantly worked for all the children we shall never see, our own heirs would stand nearly as good a chance as they do at present, so far as I may judge from my own chart. Yet I am not a believer in socialism but in aristocracy. The best are none too good to govern us, and even our wise men, individually, are often gullible in medical or spiritual matters. The world will still be pagan while wealth and energy are devoted to the saving of past and present souls, instead of to the benefit of the race, which is the after-life.

At school I gave more thought to collecting birds' eggs, trapping rabbits, muskrat, mink and skunks, than I did to religious instruction; and since I have practiced surgery, my attention has been riveted on so managing my life that I could get "days off," during the spring for trout fishing and a month in the fall for partridge and woodcock, that I have given little thought to morals and have substituted reasonable habits. If you are to know me, I may as well admit that I have averaged at least thirty days a year in hunting and fishing. I have tried these things in thirty-six States of the Union, in England, Scotland and Ireland; in Ontario, New Brunswick, Nova Scotia, Quebec, Cape Breton; in Egypt and in Yucatan; and in the case of at least two New England states, in nearly every township. Yet, I have never hit ten ruffed grouse in succession. A few years ago I got six in sequence on different days, and am still hopeful, but as in my professional work, the thing I try to do consummates in something else. In this case, in many friendships. My playmates have varied in character from a godly minister, a great philanthropist, a noble general practitioner, to an outlaw who was a confessed murderer, and several town ne'er-do-wells who were "born in rubber boots." Yet most of them have been men I trusted even in their fish stories.

Perhaps I have sacrificed my success as a distinguished surgeon to these pursuits. I have loved them better than teaching dozing medical students, the pride of amphitheatre dexterity, or the hushed dignity of the consultant at the bedsides of important persons. On many a bright October day I have been glad that my talents as a teacher were not in demand. In the spring when I dig up the first

worm in my garden, I say with Hambone: "That old red worm he looks up in my face and say, 'Whar yo' fishing pole?'" Then I get my reward for not being an overworked "Chief of the Surgical Service." In summer as I drift about on some out-of-way pond in my portable boat, watching the cotton wool in the clouds, and momentarily expecting a strike from "a big one," I am grateful that I am not in demand at the bankers' bedsides.

But speaking about that portable boat which has accompanied me to many states and has shared some very happy days, you must hear of the day it was first unpacked. The directions which accompanied it were very detailed and specific; just how to lay the floorboard, the ribs, seats, etc. The last one was something like this: "And now get in, and row out into the pond in the nicest little boat you ever sat in." So I say to the reader: "Now start in and read the best book there is on the human shoulder (it is the only one) and do not fail to note that it shows that only ten neglected cases of the injury, prominently mentioned in its title, may cost our community more than would the distribution of 3,000 copies of this book at $10.00 a copy, preface, epilogue, cartoons and all!"

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INTRODUCTION

THIS BOOK is confessedly "somewhat unusual," as it was heralded to be, three years ago, in my circular letter to the members of the American College of Surgeons. For example, the preface is illustrated, the epilogue offers resolutions, the index is a chart of differential diagnosis, and every chapter in the central portion presents original ideas which may, or may not, be of value. The introduction should be in keeping, and it shall be, for I invite each reader to write his own introduction and provide for him several blank pages on which to do so *in ink*. If his opinions on the frequency and importance of rupture of the supraspinatus or on professional advertising differ from mine, let him record them now, so that in ten years he may look back and see which of us was right. Let him attack my views as sharply as he likes, but let him not in the intervening years excise and burn the pages bearing his handwriting! I am on record as long as copies of the book exist; let him be fair and commit himself also.

Introductions are usually written by some distinguished friend or patron of the author. The reader may ask why some one of the surgeons in the group on page xiv has not written this one. There are several reasons. One is that I feel quite sure that not a single one of them would take the time to read the book through. Those who are still living are all older than I am, and I know that, young as I am, I could not read one of their books through, without falling asleep in my chair, time after time! I think that any of them would probably write for me a conventional introduction in spite of my cartoons, but I do not propose to subject them to such responsibility. However, since they know me, they may *all* wish to express their opinions, and here are blank pages provided for them!

After indulging in a luxurious preface and speaking frankly of various taboos usually mentioned by doctors only in hushed voices—income, results, motives, religion, advertisements, dependence on bankers, personal poverty—I must have made plenty of enemies! Let *them* write introductions. An introduction from an enemy who has read the book should be more enlightening than from a friend who only dozed through it.

If you are inclined to avoid controversy, you may use these blank pages for references to future articles on shoulder injuries. If you do, begin with one which has appeared since the central portion of

the book was completed. Dr. Keyes has already confirmed Dr. Akerson's work, and some of my ancient prophecies.

KEYES, E. LAWRENCE—Observations on Rupture of the Supraspinatus Tendon. *Annals of Surgery*, Vol. 93, pp. 849-856. June, 1933.

But please do not avoid controversy. Study the book; write an introduction; send one copy of it to the president of the American College of Surgeons, and another to me; then pass your book to another member of the college, or better still get him to send $5.00 for one for himself, so that he also may write an introduction. For the present, although each copy will cost me nearly twice this amount, I am glad to sell one to any member of the college, in order that the President and Board of Regents may be informed of the opinions of as many other members as possible, in case I may be encouraged to present resolutions, similar to those in the epilogue, at the next meeting of the Board of Governors.

Provided for Your Comments Before You Pass This Volume to Another Fellow of the American College of Surgeons

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ACKNOWLEDGMENTS

The custom of printing acknowledgments in books which purport in any way to be scientific, seems to me an excellent one, since it obliges the author to reflect on his relatively small share in the *opus*, which, at this stage, he may be inclined to think *magnum*. He is always, I presume, highly elated at having finished something of which he has become intolerably weary, and it must ease his descent to a normal state of mind to list those who have labored for him, although his superiority complex may wilt too rapidly when he realizes the number of co-workers to whom he is indebted. He must also admit to himself the assistance which has come from various impersonal sources, such as the era in which he lives, his inheritances, environment, opportunities, etc., and then try to estimate the degree of excellence which might well be expected in his achievement. Consideration of my own exceptional advantages makes me feel quite humble, but a look at the list of those who have thought it worth while to help me, has just the contrary effect.

The writer of a medical book, when he thinks that *he* has just completed it, should also reflect on his own education and on the trouble and annoyance he has caused his parents, teachers and fellow students. It is to be hoped that he may answer at least one new question in return for the innumerable painstaking answers he has received. His book will seem a poor return, through science, for the patient efforts of those who taught him the fundamentals of his subject or to those who, bit by bit, built up the basic sciences on which his branch was founded. Not *he*, but these hosts of individuals prepared most of the book: in fact, all but the doubtful parts which he has presented for possible confirmation.

In my case there is also a great debt to be acknowledged to those whose enthusiasm has built up the *esprit de corps* of the three institutions so often mentioned in these pages. In spite of my gibes I take great pride in having been a product of the Harvard Medical School and of the Massachusetts General Hospital, and in being a member of the American College of Surgeons, which I have seen arise and grow in strength. The latter has already developed a sense of loyalty among its members, which will, some day, take on the indestructible qualities which the spirits of the two former institutions have long possessed. Woe to the writer who permanently offends the sensibilities of such spirits, but temporary opposition is to be expected from them

when improvements are suggested. Such conservatism is wholesome, until a demonstrated truth remains unrecognized.

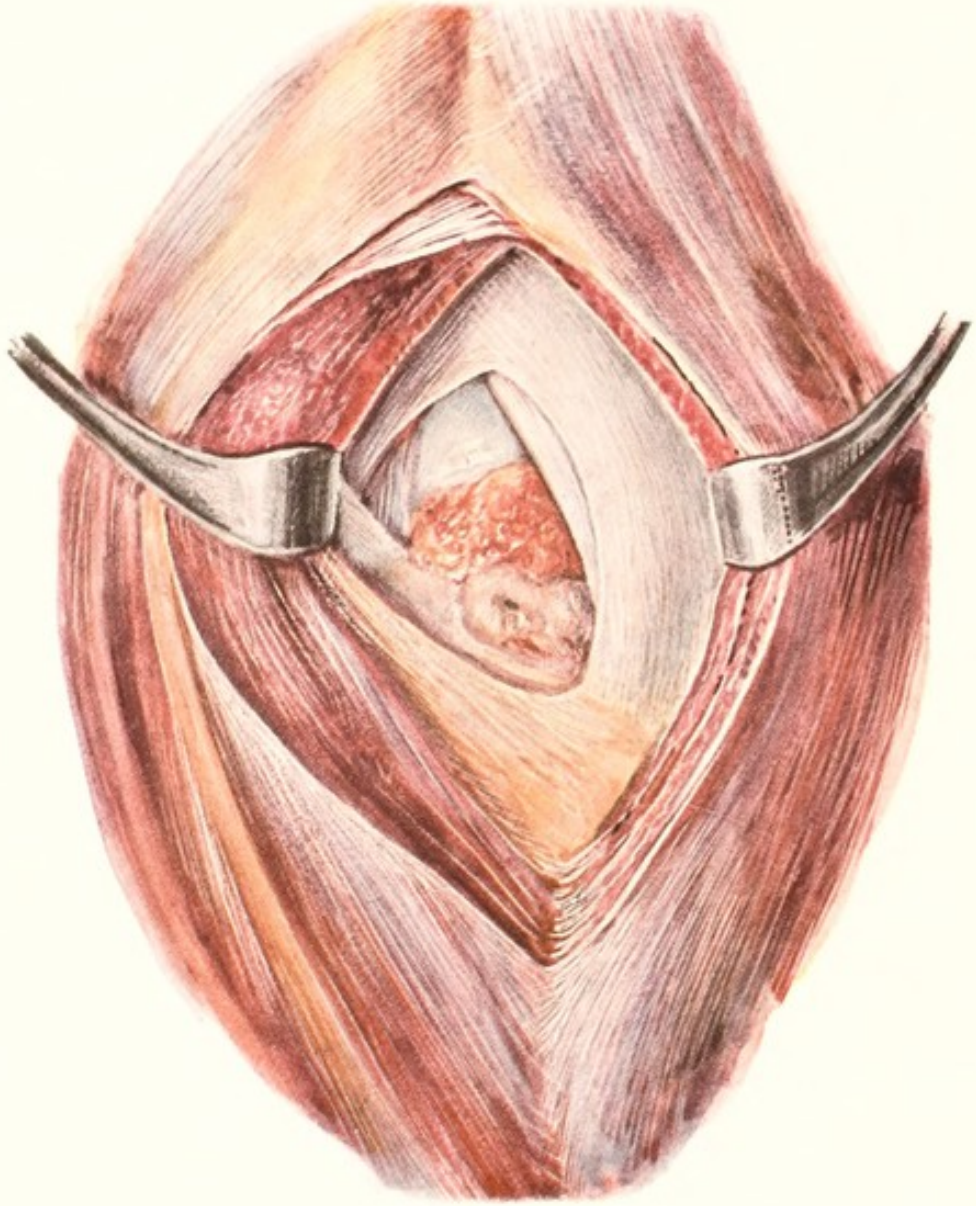
An author usually admits that there are individuals "without-whose-help-this-book-would-or-could-not-have-been-written." This does not refer to those who discovered the printing press, the microscope, the X-ray, or the dictionary, but to living friends, who by timely flattery, by adverse criticism, by the loaning of talents or money, or, harder still, by faithful drudgery, have contributed to his achievement. I have a long list of such helpers. For instance, I know that I should not have attempted this task had it not been for the blarney of Dr. Francis D. Donoghue, the wise medical director of our Industrial Accident Board. Dr. Henry C. Marble, director of the medical department of the American Mutual Liability Insurance Company, is hardly less responsible than Dr. Donoghue, for he made me convince him by actual demonstration on individual cases, that my essential claims were correct. As for the talents and money and hours I have borrowed, other paragraphs are required.

My cousin, Lady Carter, with a twinkle in her eye, made for me the little sketches in chapter two; Philip Hale, hurriedly but effectively, did the first cartoon on some brown wrapping paper; Miss Piotti and Mr. Aitken have used their recognized talents; Charles D. Vaillant has done most of the other drawings, including not only the second cartoon, but the marvelous lettering beneath it. Dr. Akerson is among my artists as well as among my "without-whom's," for he has illustrated, as well as contributed, essential and enduring evidence for my argument. Dr. Fordyce Coburn gave his experience to a review of my manuscripts and has diminished my literary blunders, and so has Professor Lewis in the case of two chapters which relate to his field of anatomy. Professors Ewing, Mallory and Wolbach have helped me to study the pathology, especially that of bone tumors. I also register my thanks to two assistants who contributed many hours of drudgery, although I am sure it did them no harm. Dr. William M. Stevenson reviewed for me the literature on fractures and dislocations about the shoulder, and Dr. Roy E. Mabrey prepared much of the chapter on Rare Lesions. And think of the hours which Dr. Stevens must have spent on his chapter!

A dog may bark up a tree a long time before any one comes to see what is up in the branches. For twenty years I bayed, though not continuously, about the frequency and importance of rupture of the supraspinatus, and I owe a debt to Dr. Philip L. Wilson, the first prominent surgeon to take time enough to study the evidence that there was something at which to bay. His paper, three years ago,

definitely put this lesion on the list of those which industrial and orthopedic surgeons should recognize and treat. Who will now put it on lists in order that the family doctor may not only promptly recognize it, but may know who, in his locality, has studied the subject enough to be qualified to suture the tendon?

There is a firm of printers in Boston, old and respected, and noted for its reliable work. Thomas Todd and Company are not publishers, although they have printed many books, usually for private circulation. They have not interfered with what I have written, but have painstakingly, graciously and cheerfully aided me in every way. Their staff and employees have shown the greatest consideration for my foibles and fussiness, and have let me superintend, in every detail, the arrangement of the text, charts, tables and illustrations. They are not to be censured for any of the offences herein displayed, against conventional book structure or content, and are only responsible for the printing, and for loaning the money to have it done. I hereby record my gratitude, and hope to return the money.



Rupture of the Supraspinatus Tendon

FRONTISPIECE

Thanks to Dr. F. B. Mallory I was able to obtain the autopsy specimen of a case of a completely ruptured supraspinatus, from which this painting was made by Mr. Aitkin.

The skin and subcutaneous tissues were removed; then the fibers of the deltoid separated and held apart by retractors as in the usual routine incision. The diamond-shaped area between the two retractors is the floor of a rather large bursa. Nearly the whole right half of this floor retains its normal, smooth, whitish appearance, but in the left-hand portion of the base or floor is a roughly triangular area which

represents the gap formed by the retracted supraspinatus tendon. At the right of this triangular gap, the long head of the biceps appears just beneath the falciform edge of the portion of the musculo-tendinous cuff formed by the subscapularis. In the left angle of the triangular area is seen a falciform edge formed by some of the superficial fibers of the infraspinatus. Just superior to this are a few vertical fibers of the deep posterior part of the supraspinatus which have not been evulsed. This was a very thin, tenuous bit of tissue. The remaining central portion is roughly divided into three parts. The upper, bluish third is the exposed cartilage of the true joint. On its shiny surface near the very edge of the true joint cartilage, we see the high light of the reflection of the window. The lower third of this central space shows a typical "volcano" on the tip of the tuberosity, such as those depicted in Plate V, Figure 1, and in Figures 36 and 40. Between this "volcano" and the cartilage, and also occupying about one-third of the central area and bounded on the right by the margin of the biceps tendon, and on the left by the film-like, untorn edges of the infraspinatus and supraspinatus, we see a red, granulation-like irregular surface. This is the pathologically changed facet of insertion of the supraspinatus tendon and of a portion of that of the infraspinatus from which the tendons have been torn. Compare Figure 40, which is the Röntgen picture of the same specimen.

It must be understood that this picture represents the result of an injury experienced, in all probability, many years before; the tuberosity is in the recessing stage, and the edges of the torn tendons have become smooth by becoming falciform. The distal stub of the supraspinatus tendon, which was probably present in the first few months after the injury, being functionless, has disappeared. The proximal end of the tendon has retracted upward and could only be demonstrated if the newly formed falciform edge of the whole rent were removed. Even in this old case it could be isolated, pulled down and attached to the tuberosity, although with difficulty. One can readily imagine the pain which this patient endured during the first few years after his injury from the mere mechanical irritation from the tuberosity striking on the edge of the acromion during efforts at elevation of the arm, although nature has gradually nearly smoothed off the former prominent tuberosity, and, by partial healing of the edges of the torn structures, has made a new base of approximately spherical surface to pass under the acromion. The writer's operative efforts have mostly been concerned with relieving the results of such conditions. When the general practitioner has learned to recognize the symptoms of these lesions within a few days of their occurrence, suture of such torn tendons will be easily and successfully accomplished.

Chapter I

THE ANATOMY OF THE HUMAN SHOULDER

It would be very convenient for the reader if all that Gray has to say about the structures composing the shoulder could be reprinted here for ready reference. It is not necessary to specify which Gray or even to state the title of his book, although I may note that the date of my own copy is 1887. This young man, for he died when he was only thirty-five, bequeathed a real legacy to almost every English-speaking doctor who has studied medicine since his time. A doctor may throw away his Bible, but he always keeps his Gray. In spite of the convenience it would be to have the standard facts of anatomy at hand, most purchasers of this book would probably prefer to have it published at less expense and to let them refer when necessary to the "Gray's" which they themselves have already thumb-marked. Yet I know that when I read about a subject in which I am only half interested, I do not bother to use reference books as I should, and probably do the author an injustice by not being sure of the fundamental facts which he assumes I know, but which I have in truth forgotten.

The other extreme would be to assume that every doctor who may read this book already knows the standard anatomic structures of the shoulder, so that the whole chapter could be omitted. This alternative cannot be accepted because it is highly probable that even the graduating classes of the best medical schools could hardly qualify in this respect. Later, as year by year passes after graduation, our anatomic memories are crowded out by other more vital ones, so that it is very unlikely that half the fund of knowledge acquired by early anatomic instruction persists a decade. After two decades of practice, any surgeon will admit that he has forgotten such matters as the branches of the brachial plexus or the origin and insertion of the levator anguli scapulæ.

Even if my readers know all the well-recognized facts about anatomy, I should still have to write a chapter on the subject to accentuate the proportionate values of certain points which appeal to me. Moreover, there are a few undescribed facts which I need to present. Obviously compromise is necessary for I can neither assume that my readers know their anatomy nor wish me to repeat every detail which they may turn to in their text books. Nevertheless, to do justice to me they should refresh their memories, and for

this purpose a list is presented of all the terms which concern the shoulder used by Gray in my own worn edition. By running his eye over this list the reader may detect the weak spots in his own memory and, if he pleases, can turn to his Gray and revivify them. I beseech him to do so. Moreover, since this book is written quite as much for the coming generation as for my own and the present one, this list is presented in three columns so that it may serve as a glossary to all three generations. It appears that the professors of anatomy intend that future medical students in all countries shall be taught the international Basle Anatomic Nomenclature.

BONES OF THE SHOULDER

International or Basle Anatomic Nomenclature	English Term in Use in Harvard Medical School in 1929	Term Used in Harvard Medical School in 1895
B. N. A. Term	*Common Term*	*Old Term*
1. Clavicula	Clavicle	———
2. Extremitas acromialis	Acromial end	———
3. Caput humeri	Head of humerus	———
4. Collum anatomicum	Anatomical neck	———
5. Collum chirurgicum	Surgical neck	———
6. Articulatio humeri	Shoulder joint	———
7. Tuberculum majus	Greater tubercle	Greater tuberosity
8. Tuberculum minus	Lesser tubercle	Lesser tuberosity
9. Corpus humeri	Shaft	———
10. Tuberositas deltoidea	Deltoid tuberosity	Deltoid tubercle
11. Sulcus intertubercularis	Intertubercular groove	Bicipital groove
12. Vagina mucosa intertubercularis	Tendon sheath of biceps	———
13. Sulcus nervus radialis	Groove for radial nerve	Musculospiral groove
14. Scapula	———	———
15. Processus coracoideus	Coracoid process	Coracoid
16. Acromion	———	———
17. Cavitas glenoidalis	Glenoid cavity	Glenoid cavity (scapula)
18. Collum scapulæ	Neck of scapula	———
19. Spina scapulæ	Spine of scapula	———
20. Tuberositas supraglenoidalis	Supraglenoid tuberosity	Supraglenoid tubercle
21. Tuberositas infraglenoidalis	Infraglenoid tuberosity	Infraglenoid tubercle
22. Fossa supraspinata	Supraspinous fossa	———
23. Fossa infraspinata	Infraspinous fossa	———
24. Fossa subscapularis	Subscapular fossa	———
25. Incisura scapulæ	Small scapular notch	Suprascapular notch
26. Facies articularis acromii	Articular facet of acromion	———
27. Articulatio acromioclavicularis	Acromioclavicular articulation	Acromioclavicular joint
28. Plica axillaris anterior	Anterior axillary fold	———
29. Plica axillaris posterior	Posterior axillary fold	———

B. N. A. Term	*Common Term*	*Old Term*
30. Margo axillaris	Axillary border	———
31. Margo vertebralis	Vertebral border	———
32. Margo superior	Superior border	———
33. Facies costalis	Costal surface	Anterior surface
34. Facies dorsalis	Dorsal surface	Posterior surface
35. Angulus lateralis	Lateral angle	External angle
36. Angulus inferior	Inferior angle	———
37. Angulus medialis	Medial angle	Superior angle

LIGAMENTS OF THE SHOULDER

B. N. A. Term	*Common Term*	*Old Term*
1. Stratum fibrosum capsulæ articularis	Fibrous stratum of capsule	Capsule of a joint
2. Ligamentum acromioclaviculare	Acromioclavicular ligament	———
3. Discus articularis	Articular disc	Interarticular fibrocartilage
4. Lig. coracoclaviculare	Coracoclavicular ligament	———
5. Lig. trapezoideum	Trapezoid ligament	———
6. Lig. conoideum	Conoid ligament	———
7. Lig. coracoacromiale	Coracoacromial ligament	———
8. Lig. transversum scapulæ	Superior transverse ligament	Transverse or coracoid ligament
9. Lig. transversum scapulæ inferior	Inferior transverse ligament	
10. Lig. coraco-humerale	Coraco-humeral ligament	———
11. Not listed	Gleno-humeral	
12. Not listed	Transverse humeral	Humeral
13. Labrum glenoidale	Glenoidal labrum	Cotyloid or glenoidal ligament

MUSCLES ABOUT THE SHOULDER

B. N. A. Term	*Common Term*	*Old Term*
1. M. Trapezius	Trapezius	———
2. M. Latissimus dorsi	Latissimus	———
3. M. Levator scapulæ	Levator scapulæ	Levator anguli scapulæ
4. M. Rhomboideus minor	Rhomboideus minor	———
5. M. Rhomboideus major	Rhomboideus major	———
6. M. Pectoralis major	Pectoralis major	———
7. M. Pectoralis minor	Pectoralis minor	———
8. M. Subclavius	Subclavius	———
9. M. Serratus anterior	Serratus anterior	Serratus magnus
10. M. Deltoideus	Deltoid	Deltoid
11. M. Supraspinatus	Supraspinatus	———
12. M. Infraspinatus	Infraspinatus	———
13. M. Teres minor	Teres minor	———
14. M. Teres major	Teres major	———
15. M. Subscapularis	Subscapularis	———
16. M. Coracobrachialis	Coracobrachialis	———
17. M. Biceps brachii	Biceps brachii	———
18. M. Brachialis	Brachialis	Brachialis anticus
19. M. Triceps brachii	Triceps brachii	Triceps extensor
20. M. Omohyoideus	Omohyoideus	Omohyoid

ARTERIES ABOUT THE SHOULDER

B. N. A. Term	*Common Term*	*Old Term*
1. Arteria thoracalis suprema	Highest thoracic	Superior thoracic
2. Arteria thoracoacromialis	Acromiothoracic	Acromial thoracic
3. Arteria thoracalis lateralis	Lateral thoracic	Long thoracic
4. Arteria subscapularis	Subscapular	———
5. Arteria circumflexa humeri anterior	Anterior humeral circumflex	Anterior circumflex
6. Arteria circumflexa humeri posterior	Posterior humeral circumflex	Posterior circumflex
7. Arteria circumflexa scapulæ	Circumflex scapular	Dorsalis scapulæ
8. Arteria transverse scapulæ	Transverse scapular	Suprascapular
9. Arteria axillaris	Axillary	———

NERVES ABOUT THE SHOULDER

B. N. A. Term	*Common Term*	*Old Term*
1. Nervus thoracalis anterior lateralis	Lateral anterior thoracic	Ext. ant. thoracic
2. Nervus thoracalis anterior medialis	Med. ant. thoracic	Int. ant. thoracic
3. Nervus musculocutaneus	Musculocutaneous	
4. Nervus thoracalis longus	Long thoracic	Long thoracic or Nerve of Bell
5. Nervus axillaris	Axillary	Circumflex
6. Nervus ulnaris	Ulnar	———
7. Nervus dorsalis scapulæ	Dorsal scapula	Muscular branch
8. Nervus subclavius	Subclavius	N. to subclavius
9. Nervus suprascapularis	Suprascapular	Suprascapular
10. Nervus radialis	Radial	Musculospiral
11. Nervus thoracodorsalis	Thoracodorsal	Long subscapular
12. Nervus subscapulares	Lower subscapular	Middle subscapular
	Upper subscapular	Short subscapular
13. Nervus medianus	Median	———
14. Nervus cutaneus antibrachii medialis	Medial antibrachial cutaneous	Internal cutaneous
15. Nervus cutaneus brachii medialis	Medial brachial cutaneous	Lesser cutaneous
16. Funiculus lateralis	Lateral cord	Outer cord
17. Funiculus medialis	Medial cord	Inner cord
18. Funiculus posterior	Posterior cord	———

BURSAE

B. N. A. Term	*Common Term*	*Old Term*
1. B. Subcutanea acromialis	B. Subcutanea acromialis	Not named and only superficially described.
2. B. Subacromialis	Subacromial bursa	
3. B. Subdeltoidea	Subdeltoid bursa	
4. B. M. Coracobrachialis	Bursa of coracobrachial muscle	

B. N. A. Term	*Common Term*	*Old Term*
5. B. M. Infraspinati	Bursa of infraspinatus muscle	
6. B. M. Subscapularis	Subscapular bursa	
7. B. M. Latissimi dorsi	Bursa of latissimus dorsi	
8. B. M. Teretis majoris	Bursa of teres major	

The writer believes that numbers 2, 3, and 4 of the above list are essentially one bursa, although they are often separated by thin, movable films of tissue.

He believes numbers 5 and 6 to be really extensions of the joint as is also the synovial lining of the bicipital canal, number 13 in Fig. 18, p. 29.

The bursæ numbered 9, 10, 11, 12, in the same diagram, are inconstant and have no official anatomic names.

Even if you have satisfied your mind that each term in the B. N. A. list carries its appropriate picture, let me give you my personal way of thinking of some of the parts of the human shoulder.

The shoulder. In descriptive anatomy there are no more sharp lines of distinction separating the regions called the shoulder, the neck, the back, the chest, the upper arm and the armpit, than we indicate in our ordinary uses of these words. Even the bones which compose its skeletal support cannot be sharply defined. Does the upper end of the humerus belong to the shoulder? Anatomists are accustomed to say no, and describe it with the arm. Surgeons certainly think of the upper end as a part of the shoulder, but would have no distinct lower limit. We shall include the upper end of the humerus above the deltoid tubercle, and leave the other limits still somewhat vague.

The shoulder girdle is not a complete girdle because the clavicles are separated by the manubrium (top of the sternum) in front, and the scapulæ do not quite meet behind. In birds the clavicles do meet and form the wish bone. In fact, "girdle" means the two bones of one side only, so it takes two shoulder girdles to not quite girdle the body. It means for man the two bones which form a saddle for the arm to ride on the body, *i.e.*, the scapula and the clavicle, but in birds and some other creatures it includes the coracoid as a separate bone, which is more massive than the scapula.

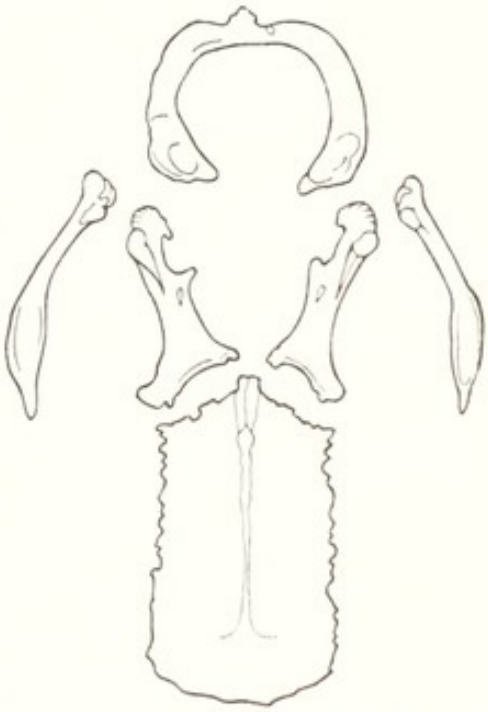


FIGURE 1. THE EAGLE

Did time and space permit, it would be instructive to discuss the comparative anatomy of the shoulder in the lower animals, for in different species there is great variation in the relative sizes and shapes of the muscles and bones and even in the proportions of the three component parts of the scapula itself. Most animals have only a trace of the acromion process which in man and in the monkey is extremely well developed, probably to stabilize the joint for its many newly developed uses.

So far as I know, no one has yet attempted to study the reasons which have led to the development of the variations of the different parts of the human scapula. Possibly I was the first to draw attention to a fundamental difference between the human and the quadruped mechanism in the use of the forelimb; *i.e.*, the quadruped uses his supraspinatus to accelerate a pendulum, while in man, in raising the arm, this muscle acts at a disadvantage against gravity and under great strain.

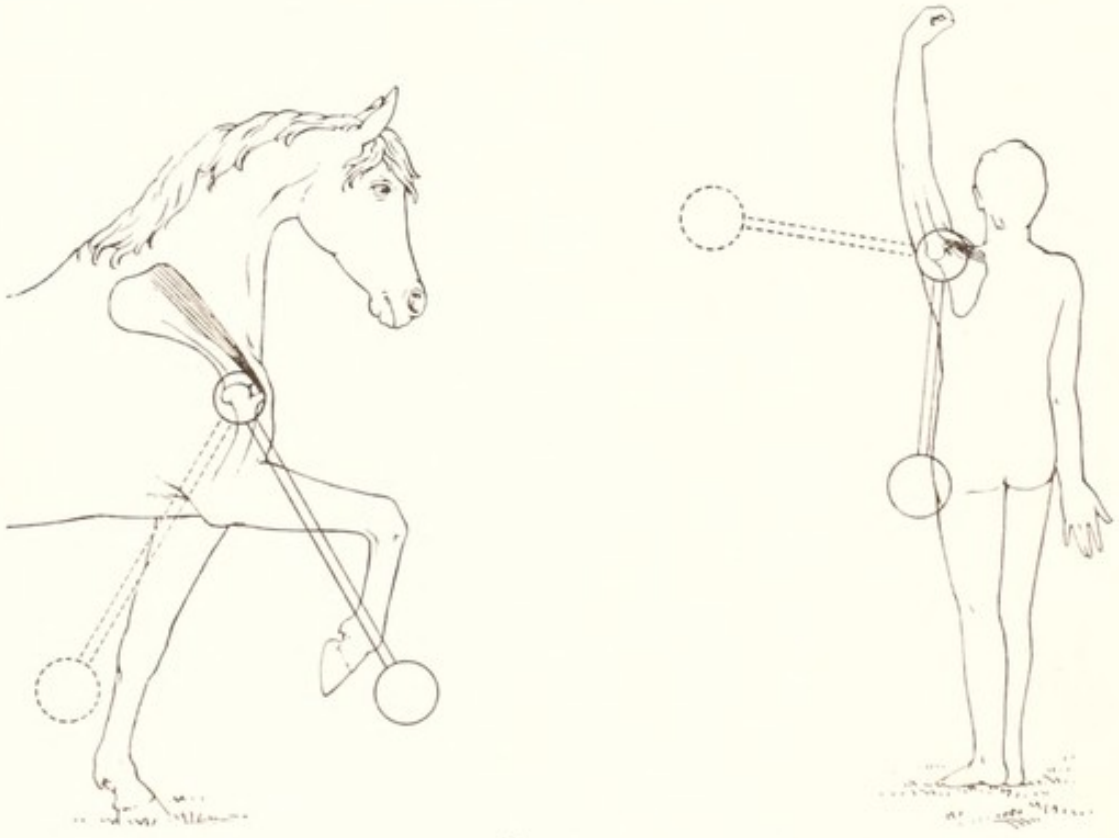


FIGURE 2.
The horse swings a pendulum. Man lifts weight with a long lever.

I hope to convince the reader in later chapters, that rupture of this muscle in man is almost equivalent to dividing the hamstring in an animal. If man walked on his forelegs, patients with this lesion would limp pitiably and get much more sympathy than they do now when they can only allege that they cannot raise their arms. At

any rate, from a mechanical point of view, man's ambitious change to the upright position resulted in a new form of shoulder joint. He has a relatively large and powerful acromion process to act as the mast of a derrick, to which his enormously developed deltoid is attached, and under which a relatively small supraspinatus is chiefly useful in holding the boom (humerus) on the fulcrum (the glenoid).

We are proud that our brains are more developed than those of animals; we might also boast of our clavicles. It seems to me that the clavicle is one of man's greatest skeletal inheritances, for he depends to a greater extent than most animals except the apes and monkeys, on the uses of his hands and arms. The clavicle holds the shoulder away from the body and therefore permits us to use our arms

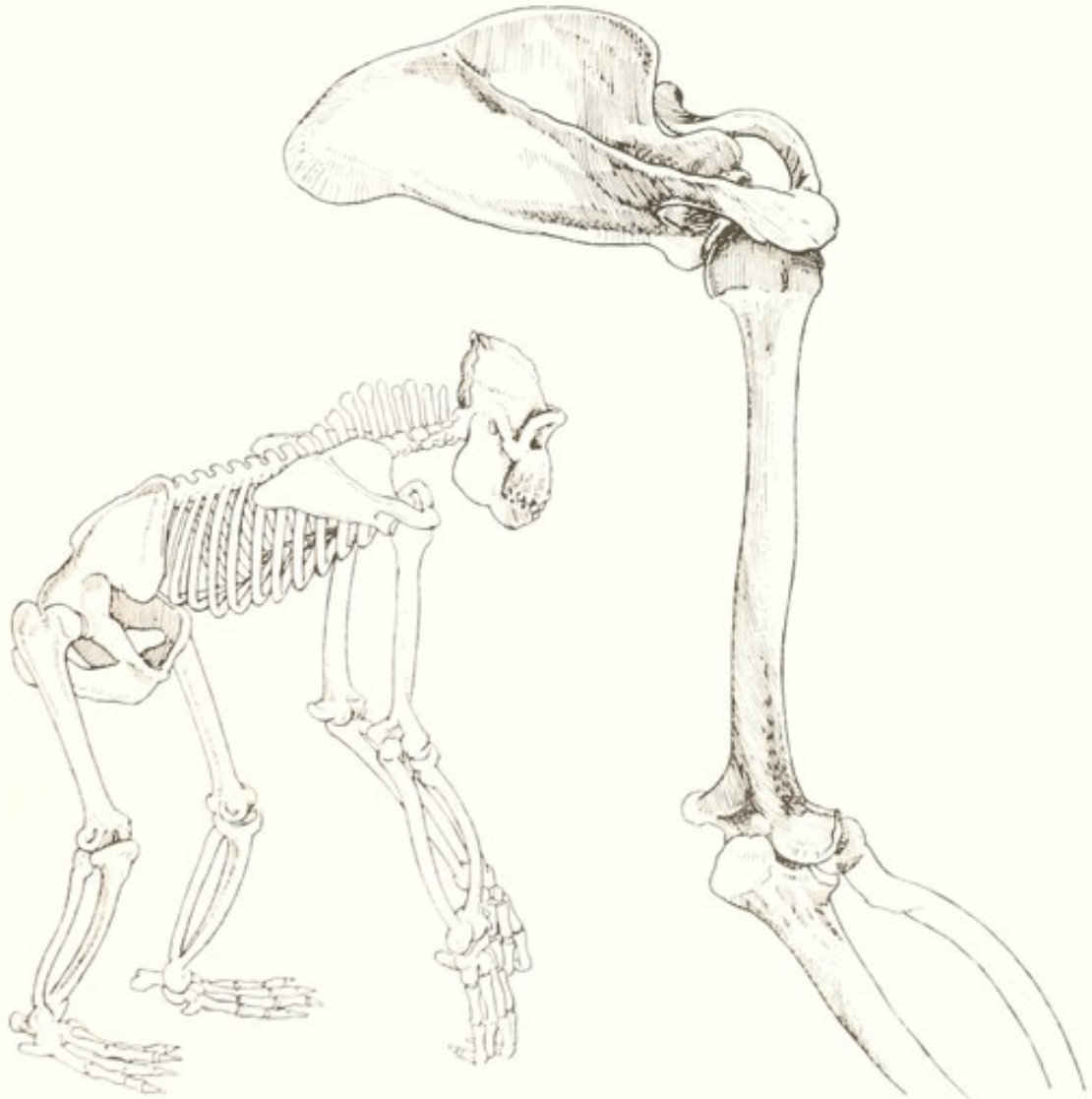


FIGURE 3. THE SCAPULA OF A GORILLA

The gorilla's scapula is even more highly specialized than man's. I have never had the opportunity to dissect a gorilla, but he must have a well-developed subacromial bursa. Our relative also has a good clavicle, which seems small in this figure because it is foreshortened. Like a human clavicle it is S-shaped.

with power and skill in abduction and adduction to a degree which few animals except the monkeys can approach. The gorilla's shoulder girdle is as well if not better developed than man's. Some of the lower monkeys have less developed ones resembling those of the quadrupeds, but all have clavicles.

Mammals that specialize in swimming (*e.g.*, seals and whales) or in running (dogs, foxes, wolves) or in grazing (horses, cows, deer, pigs and other hoofed animals) have no clavicles. Even the carnivorous cat tribe (lions, tigers, leopards and the domestic cat) have only rudimentary or very small collar bones. In the bats, the clavicles are long and curved like those of birds. Moles have short, almost cuboid bones which are homologous with the clavicle, combined with the coracoid. Those species which do have clavicles appear to be flyers or climbers. Some of the rodents which climb trees—for instance, squirrels, have them. Other rodents have none whatever. Curiously enough, some of the more ancient but waning species, such as the duck-bill platypus, kangaroo, opossum and armadillo, have well-developed clavicles. Furthermore, the armadillo has the longest known acromion process and very few other animals have any to speak of. It appears that the acromion is developed in this animal to aid in carrying his shell.

The beaver has the most highly developed clavicle of any four-footed animal that I know of. His skeleton is also notable because he has a well-developed acromion process and a large deltoid tuberosity. His shoulder girdle is the best instance I can find for my theory that animals with strong clavicles use their arms in adduction and abduction, for the beaver's mechanical skill in building his huts and constructing his dams is well known. Bears are my worst examples, for bears are clever in climbing, and yet have no clavicles.

FIGURE 4. BEAVER

The sterno-clavicular joint. Any one can easily feel the joint where his clavicle touches the top of his sternum and realize that this is the only point at which the weight or power of his shoulder can get direct support from the rest of his skeleton when he pushes with his arm. And yet after removal of the clavicle, a man has pretty good use of his shoulder just as do horses and dogs who have no clavicles.

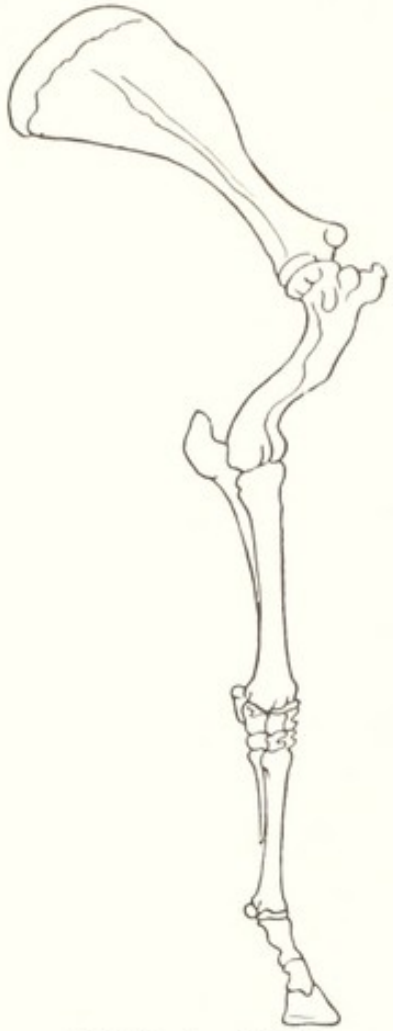


FIGURE 5. HORSE

The acromio-clavicular joint. One can feel this at the other end of the clavicle and realize that this, too, must through its small surface transmit power or weight. One can also feel that the clavicular side is higher than the acromial side of the joint. It is an important fact. When one looks at the clavicle from above, one sees no joint surface, but when one looks at it from below one sees a joint surface at each end. This provides for upward displacement at either extremity, and therefore we never find downward dislocation of either end of the clavicle.

The coracoid process is in shape and size much like a crooked forefinger projecting forward from the neck of the scapula just as the acromion process projects back of the joint. The outer end of the clavicle crosses it transversely and is very firmly united to it by the coraco-clavicular ligaments which one cannot palpate because they are wholly underneath it. One can just feel the end of this process below the outer end of the clavicle. It seems to be a part of the head of the humerus, but by rotating the latter, one may note motion between the two.

The acromion process. This forms the whole of the posterior part of the top of the shoulder, while the head of the humerus makes the rounded forward outline. The acromion extends well behind the articular head and the plane of its broad end is obliquely downward and backward and outward. Notice on your own shoulder how you can put your finger under the back of it. Notice that you cannot, with a club, hit a man from above or from behind on the top of his humerus, for the acromion is always in the way. If the arm is raised, flexed forward or abducted; *i.e.*, when he is raising his arms to fight or crawling away on hands and knees, the whole humeral head is protected by the acromion and you could not hit the top of it from any direction. If his elbow is drawn backward, you could, from in front, strike on the front of the top of the tuberosities but not on the articular cartilage. This is a very important anatomic point for the surgeon, for only when the arm is in dorsal flexion can one feel

the gap where a supraspinatus tendon has been ruptured. It also leads to the conclusion that histories are unreliable which claim an injury to the top of a humerus from a fall or a blow. One cannot fall on the top of one's humerus, or strike any one else on the top of his humerus.

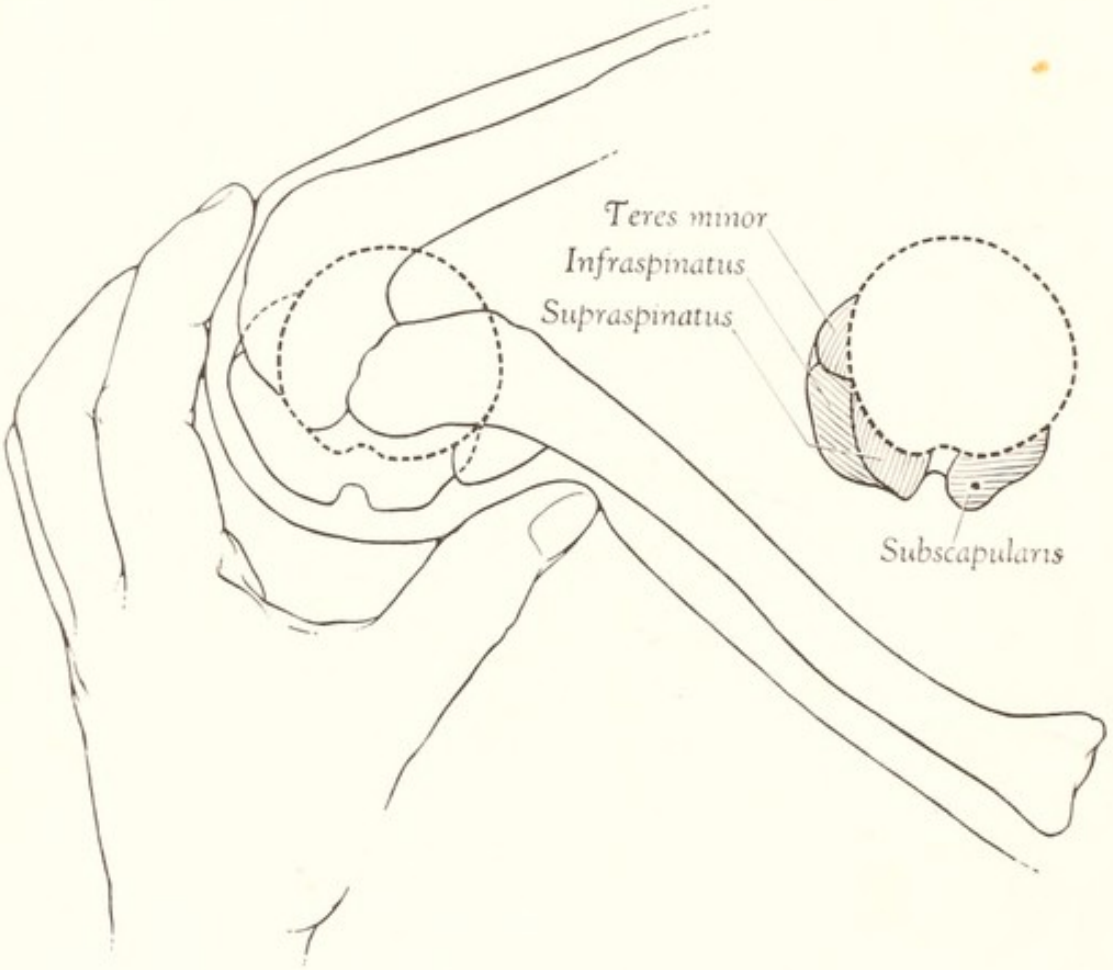


FIGURE 6. THE SHOULDER SEEN FROM ABOVE

The head of the humerus is completely protected from above and behind, and the contour in front is formed by the tuberosities.

The inner contour on which the thumb naturally comes when the shoulder is grasped is formed by the tip of the coracoid.

The forefinger sinks under the edge of the acromion. Notice that part of the facet for the infraspinatus lies in front of that of the supraspinatus, and compare with Fig. 9.

The position of the bicipital groove varies with the degree of rotation of the arm. Try this grasp on your own shoulder and rotate the humerus with the elbow flexed to a right angle. Bear in mind that the bicipital groove lies just outside of the median line of the upper arm in the anatomic position.

The spine of the scapula is the long base of the acromion and there are two little bony lumps on its lower edge which one can always feel and which are very useful as measuring points. The dorsal surfaces of the acromion and of the spine of the scapula are subcutaneous; *i.e.*, there are no muscles between them and the skin. Evidently the ridge which they form was made to be hit; *i.e.*, to protect

the shoulder joint and the brachial plexus from blows from above and behind. Consider the mechanics of its structure and notice how it, with the clavicle, forms a movable arch over the vessels and nerves

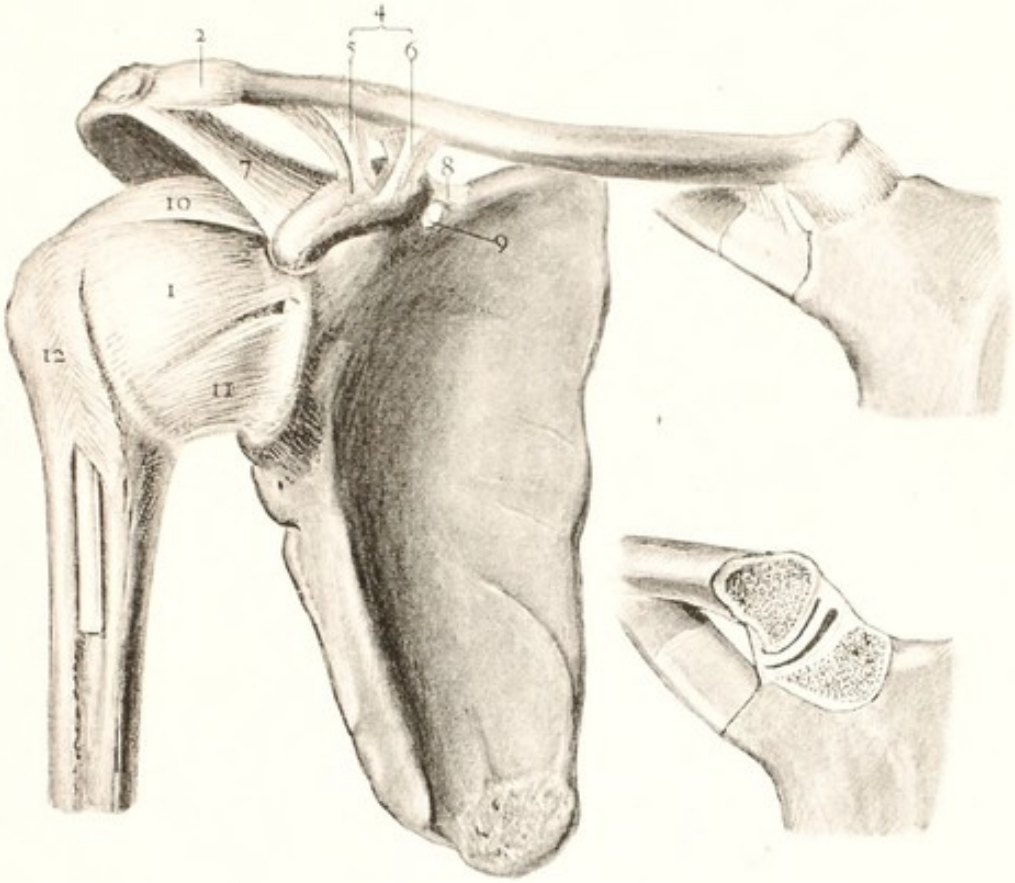


FIGURE 7. THE LIGAMENTS OF THE SHOULDER

The numbers refer to the list of ligaments on page 3.

The arch formed by the coracoid, the coraco-acromial ligament and the under side of the acromion is a hemispheric dome under which the hemisphere formed by the musculo-tendinous cuff and by the tuberosities exactly fits. Under this arch the head of the humerus can move many degrees in all directions by the aid of the subacromial bursa.

The reader must understand that the dissection pictured above is quite artificial. Such a capsule does not exist unless made at the expense of the musculo-tendinous cuff, a section of which is shown in Fig. 9. One should try to visualize Fig. 8 in combination with this figure, for the supraspinatus fills the gap shown between the numerals 7 and 10 in this figure.

of the arm. This arch flaps like a wing as you hunch your shoulder and lower it, or abduct your arm and lower it. Observe also that the spine of the scapula rises between the supra- and infraspinatus to give attachment to the superficial layer of great muscles, *i.e.*, the

trapezius and the deltoid. The two sets of muscles would be separated by sawing through the spine of the scapula. (See Fig. 8.)

The glenoid is the shallow cartilage-covered surface where the head of the humerus obtains its fulcrum as the arm is raised. Notice that the plane of its surface is at no particular angle with the rest of the scapula, for it faces somewhat forward and upward, and outward. Notice its narrow superior portion.

The blade. The upper portion of the blade of the scapula from the lateral view forms in most people an obtuse angle with the rest of the blade so that it can fit over the curve of the upper ribs at the base of the neck. Thus the thin blade of the scapula when pressed forward from behind fits nicely over the back of the upper seven ribs. Furthermore, the bony structure of the scapula is beautifully arranged to distribute force applied from behind.

The clavicle is a strut which connects the rest of the shoulder with the skeleton. It prevents the upper portion of the shoulder from jamming up against the rest of the body. In other words, the clavicle acts as does the iron rod a man uses to hold a bull who has a ring in the end of his nose. The clavicle is capable of a limited degree of circumduction, which is facilitated by the double joint at the sternal end. Circumduct your whole arm with the forefinger of the opposite hand on the outer tip of the clavicle and you will find that the latter passes through an irregular circle about three inches in diameter. The clavicle is the boom of a derrick (the neck) and enables the trapezius to raise the whole shoulder.

The ligaments. It is well to distinguish four kinds of ligaments which occur about the shoulder.

(1) Those that pass between two different bones about a joint, such as the capsular ligaments of the sterno-clavicular, the acromio-clavicular and the scapulo-humeral joints, or strengthening bands in these capsules such as the superior acromio-clavicular ligament or coraco-humeral and gleno-humeral ligaments which are merely slightly firmer portions of the capsular ligaments.

(2) Those that bind two bones together without a joint but permit a very limited amount of motion. The coraco-clavicular ligament composed of conoid and trapezoid portions is a very typical example.

(3) The interarticular fibro-cartilages are usually classed as ligaments. There are three of these related to the shoulder; a very typical one in the sterno-clavicular joint; a less typical, often rudimentary one in the acromio-clavicular joint; and the glenoidal labrum which encircles the glenoid cavity and makes it slightly deeper.

(4) Those that pass between two processes of one bone in a static manner, apparently with a purpose of restraining the mobility of other structures. In the shoulder there is such a ligament crossing the suprascapular notch through which the nerve and vessel pass. The transverse humeral ligament which makes the bicipital groove into a canal to restrain the long tendon of the biceps, is another. The coraco-acromial ligament is one of this kind, and we must speak of it in more detail.

The coraco-acromial ligament will be mentioned again and again in this volume. It is wholly a scapular ligament, passing between the two processes from which it takes its name which are parts of one bone. Its under surface forms most of the posterior part of the roof of the subacromial bursa, and the tuberosities of the humerus pass upward underneath it when the arm is elevated. It lies between the bursa and the acromio-clavicular joint. Its function appears to be largely to restrain the head of the humerus from gaining a fulcrum on this joint or on the under side of the end of the clavicle. It is more elastic than bone but quite firm. Evidently the coraco-acromial ligament has an important duty and should not be thoughtlessly divided at any operation. One always finds it if the bursal incision is carried upward. The bursa, with this ligament and the two bones to which it is attached, really forms a secondary shoulder joint. (See Fig. 15.)

The other ligaments with simple but confusing names—the *acromio-clavicular* and the *sterno-clavicular*—surround their respective joints and pass between two different bones and have the functions usual in other joints. We have already spoken of the *coraco-clavicular* ligaments (p. 9) which are not connected with any joint and are the mainstay in binding the shoulder blade by the coracoid process to the collar bone. The *coraco-humeral* and *gleno-humeral* ligaments should never have been described as entities. They are merely somewhat variable parts of the joint capsule.

The muscles. I have few particular comments to make about the muscles which seem to be easier to remember from our student

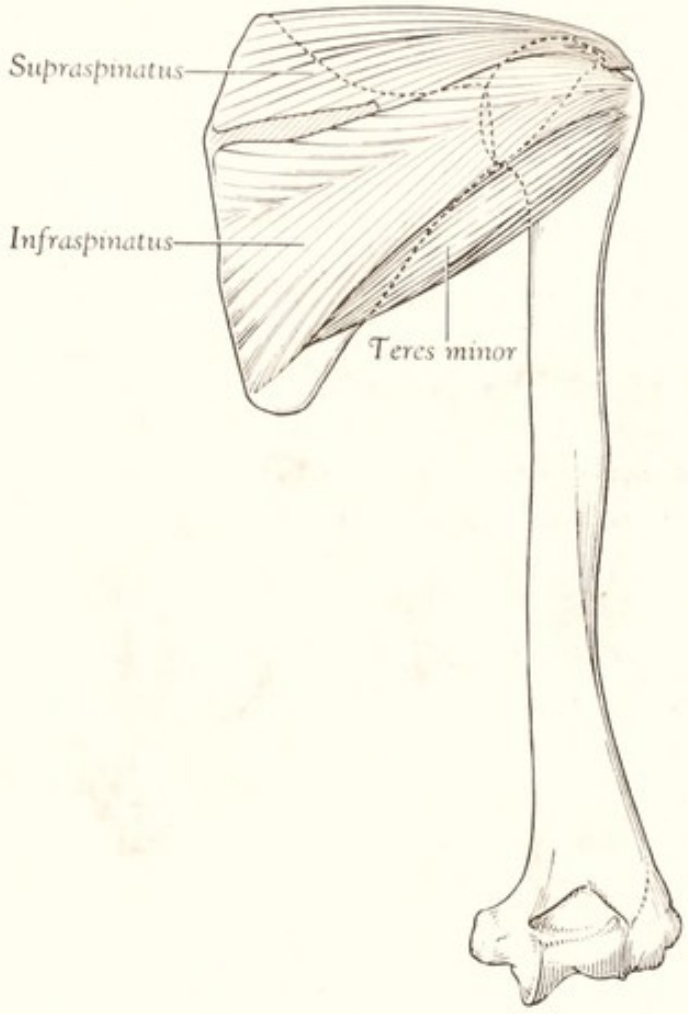


FIGURE 8. INNER MUSCULAR UNIT

The compact entity made up by the short rotators as a unit, changes its shape very little during rotation of the humerus.

days. I would like to call attention to the compact entity formed by the short rotators when the deltoid and trapezius have been removed. When one thinks of the shape of each of these individual muscles, one must remember that the joint is mobile and that the position of the humeral head on the glenoid greatly alters the shape of each muscle attached to its tuberosities.

Taking the group as a unit they form an entity which remains of about the same shape whether the arm is rotated in or out, but each of these muscles becomes hooked around in the direction toward which the humeral head happens to be rotated. Not only is this true, but their shapes are much altered by whether the humerus is pointing downward or upward. I should like to make a plea for teaching the student that in thinking of the shapes of muscles, he should be able to visualize their positions in the extremes of motion of the adjacent joint.

The subclavius muscle is seldom alluded to in practical surgery. Its function seems to be to draw the clavicle down toward the ribs when the former has been raised by the trapezius, either in hunching the shoulder or in abducting the arm. Possibly it has some importance when division of the nerve supply causes contracture, just as the clavicular portion of the pectoral may limit the motion of the arm by contracture when its nerve supply has been destroyed by careless dissection of the axilla. The subclavius is mentioned chiefly because no one has yet reported any clinical condition in which it is an important factor. Here is a chance for original work.

The head of the humerus is very much larger than the glenoid cavity of the scapula on which it rests and on which it has to gain its fulcrum whatever the position of the arm when in use. There is no

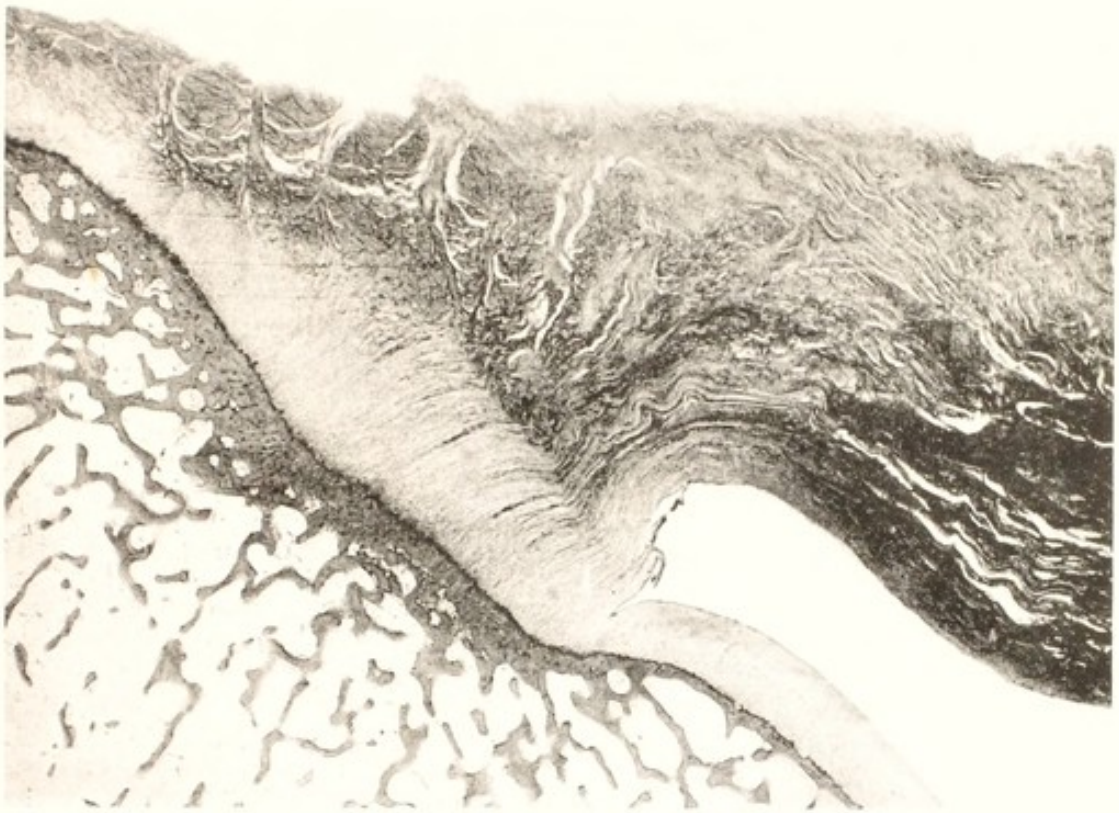


FIGURE 9. INSERTION OF SUPRASPINATUS TENDON

Notice the transverse fibers in the upper portion of the tendon. These are probably some of those of the infraspinatus. See in relation to this Fig. 6, which shows that the insertion of the infraspinatus overlaps that of the supraspinatus to some extent. Each of the other tendons also interlaces its fibers to some extent with its neighbor's tendons. Notice that even with the microscope no distinction can be made between capsule and tendon in this region.

The synovial layer of cells beneath the tendon and the finger-like processes which attach the tendon to the facet cannot be seen without more magnification.

Notice the dense bone on the surface of the facet. This is increased in density in cases of inflammation and is shown in the X-ray.

Notice the palisade-like structure of the fibro-cartilage which often remains on the bone when the tendon is ruptured. Refer to page 89 for a description of the usual points of rupture.

Notice how close to the rim of the articular cartilage the fibers are attached and that a few of them in this specimen have given way at the very edge.

Notice the lamellated structure of the tendon. It is crinkly because it was detached from the scapula before fixation.

fulcrum in the standing position with the arm at rest at the side, or in any other position in which the arm is at rest without any of the muscles in use. In such positions of rest, the head of the humerus is held in contact with the glenoid surface by atmospheric pressure.

The capsule of the joint is quite unlike the capsule of other joints, for it must admit of motion in any direction, and hence it cannot have any of its parts in a state of tension when it is in a mid-position; therefore, the capsule is approximately twice as big as the size of the anatomic head of the bone, and any part of its circumference will only be tense when the extreme of motion is reached in the opposite direction. (Fig. 33.)

The short rotators. Another peculiarity of the joint is the fact that the tendons of the short rotators, viz., the supraspinatus, the infraspinatus, teres minor and subscapularis, are closely incorporated with the capsule through almost their whole extent. The tendons are broad and flat and only about an inch in length. This matter is spoken of more at length on page 74. It is impossible either to dissect these tendons from the capsule on the one hand, or from the synovial base of the subacromial bursa on the other. Within a half inch of the sulcus which surrounds the cartilaginous head of the bone, even a microscopic section in this region shows no distinction between the tendon substance and the joint capsule. Anatomists have concluded that this arrangement of the tendons prevents the redundant portions of the capsule from getting caught between the articular surfaces as the joint moves. Fig. 9 shows a section through the supraspinatus tendon at the sulcus and illustrates the manner in which the tendon is inserted into the bone.

The student who is taught his anatomy from the dried bones, may get a false impression from having the facets of insertion of the short rotators pointed out to him as specifically the places where these muscles are attached to the bone. As a matter of fact the attachment takes place throughout most of the upper half of the sulcus, which is called the anatomic neck, and which separates the cartilaginous edge from the tuberosities. Doubtless the heavier portions of these tendons are inserted in the smooth facets which anatomists point out on the tuberosities. However, if one excises the head of the humerus with these short rotators attached to it, one finds that the insertions of all four muscles are so intimately incorporated with the capsule, and with each other, that one cannot, even with careful dissection, separate any one tendon from the neighboring tendon. This fused structure will be alluded to as the musculo-tendinous cuff.

This *musculo-tendinous cuff* does not extend completely around the head of the bone. It occupies approximately the upper half of

the circular depression, called the anatomic neck. There are no tendons inserted on the lower axillary side, and in this region the capsule is reflected farther away from the rim of the cartilage than it is in the portion where the tendons are inserted. There is no very definite mark on the bone on this axillary side to show where the capsule was attached.

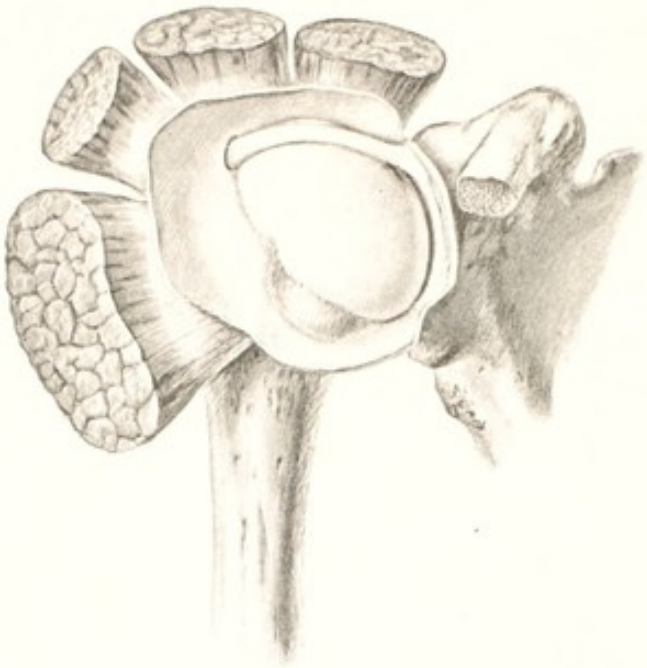


FIGURE 10. MUSCULO-TENDINOUS CUFF

The inside of the joint is shown from the anterior aspect especially to indicate that there is no sulcus in the upper half of the anatomic neck and that in the lower half of the joint the insertion of the capsule is some distance from the articular rim. Here the bone is covered only by a thin layer of fibrous tissue and synovia.

The capsule has been cut just proximal to the line where it becomes welded into the musculo-tendinous cuff. The author wishes to emphasize the fact that in autopsy specimens one frequently finds bare bone between the attachment of the tendons and the articular cartilage and that such conditions are the end results of unrepaired ruptures of the tendon fibers. (Plate V.)

The sulcus and anatomic neck. Looking from the inside of the joint of a fresh cadaver there is no sulcus in the upper portion opposite the attachments of the supraspinatus and infraspinatus, or opposite most of the adjacent parts of the subscapularis and teres minor. About opposite the middle of the insertion of the teres minor on the one side, and the middle of that of the subscapularis on the other side, the line between the cartilage edge and the insertion of the cuff begins to get broader; *i.e.*, the lower portions of the insertions of the subscapularis and of the teres minor are slightly farther from the articular rim than are the insertions of the other muscles. On both sides, as we pass downward, the line gets broader until at its broadest point, it is nearly a half inch wide on the axillary side of the capsule. (Fig. 10.) The reader must understand that all this refers to the sulcus before the tendons are removed and while the membrane is still intact. After maceration, the bare bone sulcus (the

anatomic neck) is quite the reverse, and is broad where it was formerly narrow. When looking at the bare bone, one sees a deep sulcus (the anatomic neck) between the tuberosities and the rim of the articular surface and one must realize that in life this sulcus is filled by the musculo-tendinous cuff. (See Figs. 9 and 10.) In the bare bone one sees that there are many vascular foramina in this part of the sulcus. The reader should see for himself the points described in this paragraph, because many shoulder symptoms may be accounted for by the evulsion of fibers of insertion at the articular margin of this sulcus. In cases of complete rupture of the tendons the bony sulcus is palpable even through the thick deltoid. (See Fig. 42.) I believe that when at autopsy one finds any sulcus present between the cartilage and the attachment of the supraspinatus, or a broadening of the sulcus opposite the attachments of the other muscles so as to expose bare bone, we have proof that a pathologic condition has existed. I have never seen any mention of one of these rim rents in any book on anatomy, pathology or surgery, yet on examination of these joints at autopsy it is a very common finding. In fact, in aged people it is hard to obtain a perfectly normal shoulder joint, just as it is to find a perfectly normal aorta.

Bursæ about the shoulder. It is very difficult to find any accurate description of the *subacromial or subdeltoid bursa*, and I feel that even if I could give a clear description that I could not in any words convey as much as I could by one demonstration. At the time I first wrote about this bursa its importance was not recognized, and even its anatomic outlines were very poorly described. The main reason for the inaccuracy of our knowledge was that no one had studied it from inside. In dissecting, anatomists removed the deltoid muscle and then described the bursa, the upper portion of which, together with some of the periphery, was necessarily removed with the deltoid. All that was left of the bursa was an inconspicuous portion about the size of a half dollar which remained attached to the greater tuberosity. The portion of the roof, which was attached to the under side of the acromion, would not be noticed. Furthermore, in the average dissecting room specimen, the tissues are badly discolored and the limits of the synovial membrane, which is very thin indeed, are not clearly marked.

The functions and structure of bursæ in general should be considered before going any further. Nature provides bursæ in many parts of the body where a considerable degree of motion between

parts of the anatomy is necessary, and yet no cartilaginous joint is required. Especially is this true where two muscles cross each other in opposite directions, or where a muscle or a tendon and a bone move past each other without actual articular contact.

Some of the best known bursæ occur between some portion of the skeleton and the skin over it. The skin is more or less movable over most parts of the body, and the mobility is permitted by the fatty or areolar tissue which lies between it and the deep fascia, but occasionally, as over the patella and over the elbow, the skin must move to a considerable extent, so that spaces are formed in the areolar tissue which allow it to glide over the bony prominences. The prepatellar bursa, for instance, has a base firmly fixed to the upper surface of the patella and a roof firmly fixed to the under surface of the skin. The same is true of the olecranon bursa at the elbow. It is the mobility of the periphery which permits motion rather than the attached parts of the roof and base. In other parts of the body bursæ lie between bony prominences and fascia or between tendons and muscles, but the principle of attached portions and movable peripheries holds good in all. In the shoulder we have examples of the several kinds; *e.g.*, there is a small subcutaneous bursa over the acromion, and a triangular bursa between the tendinous fibers of the trapezius and the dorsum of the scapula at the base of its spine.

FIGURE 11. BURSÆ

Referring to Fig. 11, A-B is the fixed roof of a bursa and C-D is the fixed base. The periphery A-C and B-D is movable.

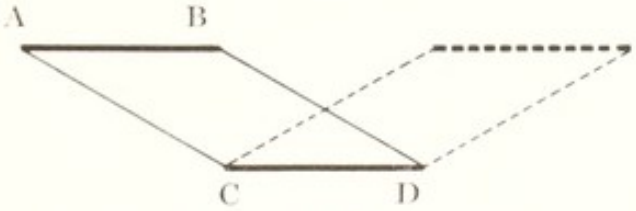


FIGURE 12. BURSÆ

A-B may move away from C-D or *vice versa* in any direction even in rotation, but probably not in separation vertically on account of atmospheric pressure.

FIGURE 13. BURSÆ

In Figs. 11 and 12 the surfaces have been depicted as if separated, but of course in the normal living tissue these surfaces are in contact and their motion is linear as suggested in Fig. 13. One must realize that the thin, movable periphery sometimes has to fold on itself as base and roof move about. The nictitating folds seen on the inside of a bursa are double layers of the periphery, so very thin that they readily roll on one another as do the membranes which come across a bird's eyes.

In the subacromial bursa the section of the space in most positions is a curved line (Fig. 14) which represents a section through the segment of an almost perfect hemisphere.

FIGURE 14.

This hemisphere is almost a counterpart in size and curvature of the articular surface of the true joint. (Fig. 15.)

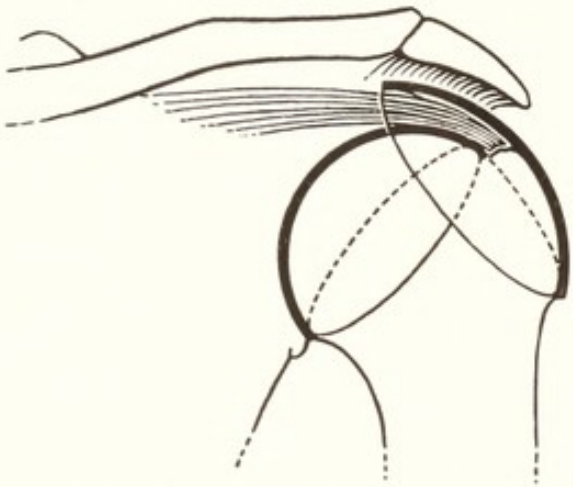


FIGURE 15. SYMMETRY OF BURSA AND JOINT

The hemisphere of the joint surface is slightly smaller than the hemisphere of the base of the bursa, which functions as an auxiliary joint.

It seems to me that it is very doubtful whether it is best to apply the term bursa to extensions of the joint cavities, such as that beneath the tendon of the quadriceps at the knee. This synovial space is chiefly useful to enable the tendon of the quadriceps to ride over the anterior portions of the condyles of the femur. In a fully flexed knee, practically the whole of this surface has passed downward over the cartilage. It is simpler to regard this so-called quadriceps bursa as part of the synovial lining of the joint, where it does not need a true capsule, because reinforced by muscle. In the shoulder joint

there are two so-called bursæ which the anatomists describe (Bursa M. subscapularis and B. M. infraspinati), which to my mind are simply extensions of the joint for the same purpose as that of the quadriceps bursa at the knee. It requires little imagination to see that when the humerus is rotated inward, the infraspinatus bursa would be largely straightened out with the stretched infraspinatus

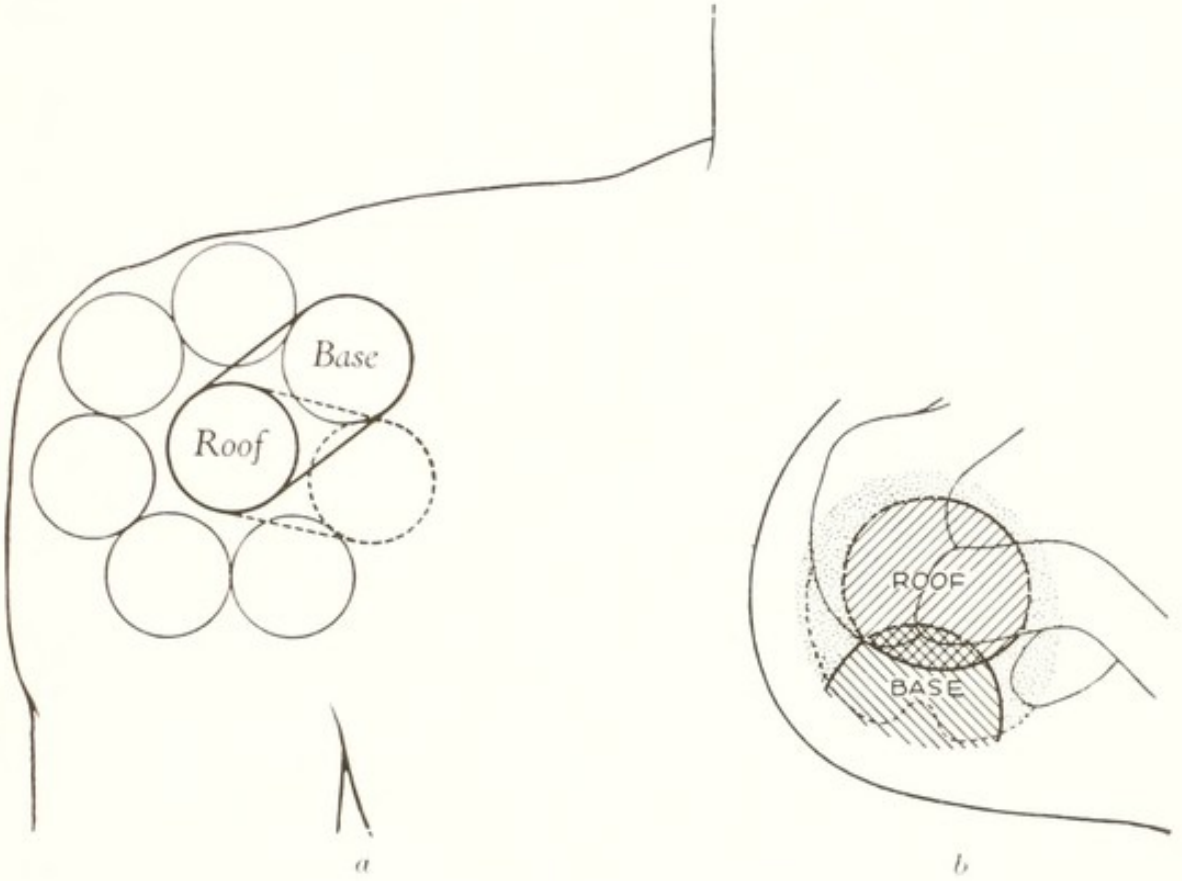


FIGURE 16. RANGE OF MOVEMENT OF BURSA

The base and roof of the subacromial bursa are somewhat larger than any two circles depicted in this figure, which expresses diagrammatically the extent of motion of the bursa in varying positions of the joint.

To understand this diagram (*a*) one must visualize the circle labeled "roof" as firmly fixed to the under side of the acromion and the acromio-clavicular ligament, while the circle labeled "base" is firmly fixed to the top and anterior aspect of the tuberosities and to the adjacent half inch of the tendons of the short rotators; an area, in fact, about as large as a silver half dollar. The base, in varying positions of the arm, moves away from the roof; the movable periphery of the bursa tends to straighten out as depicted in the two ellipses shown in the diagram. One may imagine such ellipses in any position of the arm.

Since the bursa is concavo-convex in shape, and since it lies obliquely, it cannot be depicted in a diagram either from the strictly anterior aspect or from above. (See Fig. 44-b.)

b depicts the actual possible extent of motion of the base in relation to the roof, projected as seen from above. The dotted area represents the possible positions to which the base may move. Notice the indentation of the bicipital groove which, during rotation, can pass to any point on the anterior dashed line. The base moves posteriorly as the arm is elevated, and is farthest back in relation to the acromion when the arm is in the pivotal position (cf. Figs. 25 and 26).

muscle, and *vice versa*, the subscapularis bursa would likewise become a part of the capsule of the joint in extreme external rotation. (Fig. 33.)

On examining many joints from *inside the capsule*, one does find occasionally that the openings from these bursæ, or extensions of the joint, may be considerably smaller than the width of the extensions a little further back than the edge of the glenoid. This gives them the appearance of diverticuli. There is great variation in the size of these openings.

Gray, after describing these two bursal sacs related to the shoulder joint, says, "A third bursal sac, which does not communicate with the joint, is placed between the under surface of the deltoid and the outer surface of the capsule." This description of the subdeltoid or subacromial bursa, which is the only one he gives, is good so far as it goes, but Gray could not have appreciated its extent or its surgical importance or he would have written much more about it. Part of it is placed "between the under surface of the deltoid and the outer surface of the capsule," but that is far from describing its full extent, as will be seen later.

Piersol goes into the description of the bursa in slightly more detail, but it evidently did not greatly interest him and he was not very accurate in regard to it. For instance, he says, "The large subacromial bursa and the subdeltoid bursa have been described as to their possible enlargement;" *i.e.*, he considers that there are two bursæ, whereas there is really only one. The fact that he says "as to their enlargement" shows that he did not appreciate that they constituted essentially a secondary joint which needed pages of description on account of its clinical importance. He also says, "Bursæ are sacs filled with fluid found in various places where friction occurs between different layers or structures." To describe them as "sacs filled with fluid," gives a wrong impression. They are really spaceless spaces, not filled with fluid, but supplied with a most wonderful self-oiling mechanism, so that their walls glide on one another with their surfaces no farther apart than the thickness of the thinnest sheet of paper. In consequence of this spaceless structure, any method of demonstrating bursæ by filling them with opaque materials, as wax, or even with air, causes distention of their cavities and contraction of the periphery. In other words, the circumference of a bursa, when not distended, is very much larger than when it is blown up in the form of a sphere.

Piersol states, "The subdeltoid bursa does not usually communi-

cate with the joint," leaving one to think that it occasionally does communicate. As a matter of fact, when I began my work on the bursa, Dr. Thomas Dwight, who was then Professor of Anatomy at Harvard, disputed my finding ruptures of the supraspinatus which permitted the joint fluid to flow into the bursa. He explained these communications as semi-normal openings between the joint and bursa, similar in structure to the communications of the bursæ under the infraspinatus and subscapularis. So positive was he on this point, that it took me a long time to convince him that the instances of communication which we found in his dissecting room were pathologic. So frequent are these ruptures in the ordinary anatomic material, that it is quite natural that the anatomists have thought they were merely instances of persistent congenital anomalies and should have ignored them as the results of a common form of trauma.

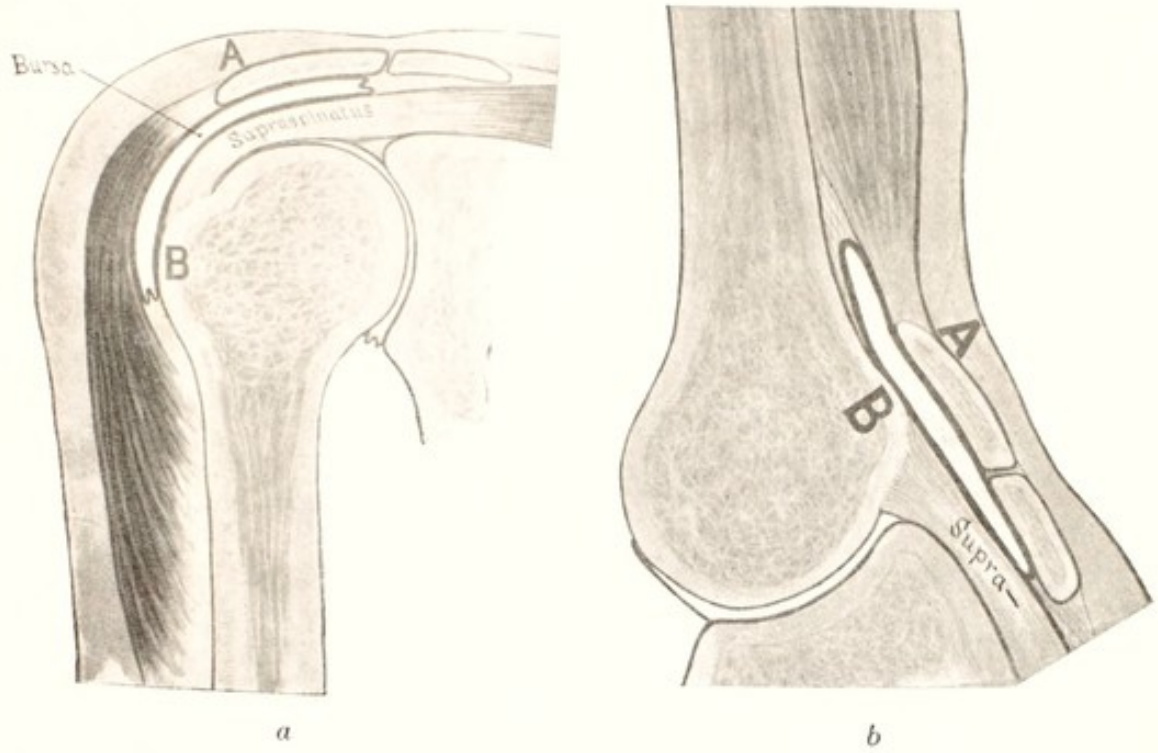
The subacromial bursa itself is the largest in the body and the most complicated in structure and in its component parts. It is in fact a secondary scapulo-humeral joint, although no part of its surface is cartilage. Instead of being between bone and skin, it is between bone and tendon, muscle and bone. It is firmly attached on its base to the upper and outer three-fourths inch of the greater tuberosity, as well as to about a three-fourths inch of the tendons of the four short rotators where they are attached to the tuberosities. Part of its base covers the bicipital groove. Its roof is firmly attached to the under side of the acromion and to the under side of the coraco-acromial ligament, and to the fibers of origin of the deltoid from the edge of the acromion. Its periphery extends loosely downward under the deltoid, backward and outward under the acromion, and inward under the coracoid, between it and the subscapularis, and under the common origin of the short head of the biceps and the coraco-brachialis. The roof and base are in intimate contact and it is lined by synovial membrane, which is almost as thin as a cobweb. Nevertheless this layer has the property of secreting just enough synovial fluid to render the movement between the two surfaces practically frictionless. Beneath this membrane is always a network of fine blood vessels, so that the secretion can be increased or diminished on demand. This arrangement also permits it to become congested in a short time, just as the peritoneum may.

The subacromial bursa is an absolutely necessary part of the shoulder joint. When its surfaces are inflamed so that they cause painful friction, the arm cannot be rotated or abducted. Their complete adhesion has the same effect.

Let us return now to the incision to demonstrate the bursa. One who intends to read this book would save himself much trouble if he could perform on a cadaver the following experiment. Make an incision two inches long from the acromio-clavicular joint downward between the deltoid fibers. The fascia under the deltoid is thin and beneath it will be the roof of the bursa. Pick this up between forceps and incise it as if it were the peritoneum. Enlarge the opening upward until it meets the fibers of the coraco-acromial ligament. Enlarge the incision downward until you arrive at the lower edge of the incised synovial membrane; then pass a probe into the bursa and determine its limits in all directions. You will find that it goes upward beneath the acromion more than three-quarters of an inch; it also extends outward and inward for about the same distance, and you have already opened it an equal extent downward. It is therefore a concavo-convex circular cleft about 1½ inches in diameter. Its base is a hemisphere which fits inside its roof, which is the inside of another hemisphere. Its periphery is movable to almost as great an extent as shown in Fig. 16. With the forearm flexed at a right angle, rotate the humerus inward and outward, and the base of the bursa will pass in review across the bottom of your incision. With the arm in extreme internal rotation, mark the most external point on the base which you can see through the incision, then put the arm in extreme external rotation and mark the point on the base which is nearest the inside of the incision. Mark with a knife down to the bone the upper and lower points of the base when the arm is carried into extreme dorsal flexion and extreme frontal flexion. Later on, removing the deltoid, you will be surprised to find that these marks are on the periphery of a circle nearly two and one-half inches in diameter. Next, with retractors in the wound, pull the arm downward so as to allow air to enter the subacromial portion of the bursa. You will find a cavity large enough to put your forefinger in, and you can sweep the finger around to the inner side so that with the arm in internal rotation your finger will pass between the tendon of the subscapularis and the tip of the coracoid process. When you retract the inner edge of the incision and the arm is in internal rotation, you can pass the handle of the knife into the subcoracoid portion of the bursa, which some anatomists have described as a separate bursa, but which in reality is not, although sometimes there is a film of synovial membrane crossing the bursa so as to partially separate this portion from the remainder of the bursa.

If you will repeat this experiment on cadaver after cadaver, you will become convinced that the *subacromial bursa*, the *subdeltoid*

bursa and the *subcoracoid bursa* are one and the same thing, although films of tissue may separate them.



a *b*

FIGURE 17. ELEVATION OF SCAPULO-HUMERAL JOINT

Coronal sections of right shoulder in anatomic position and in elevation. These two figures were made for me by Mr. Aitkin in 1908, and have been used as lantern slides on many occasions since. The fact that I publish them again, after twenty-five years of further study of this region, indicates my belief in their essential accuracy. Although drawn from frozen sections they are confessedly semi-diagrammatic. Notice the deltoid and its origin from the edge of the acromion. Notice the subdeltoid or subacromial bursa with its roof made by the under surface of the acromion and by the fascia beneath the upper portion of the deltoid. Its base is on the greater tuberosity and on the tendon of the supraspinatus, which separates it like an interarticular fibrocartilage from the true joint. *b* illustrates the condition which would be found in elevation, the tuberosity having passed under the acromion and the point B having passed the point A. It is obvious that the convex floor of the bursa, as it lies on the tendon of the supraspinatus and on the tuberosity, should have a smooth, even, rounded surface. As a matter of fact, the first time one cuts into a normal bursa one is startled to find how much the base looks like the joint itself. One cannot distinguish by sight the line between the portions which are on bone or on tendon beneath the shiny base.

It is obvious that if the surfaces of the bursa between the points A and B in *a* were adherent, it would be impossible for the joint to pass into the position shown in *b*.

Note for the reader who likes puzzles.

Notice in Fig. *b* the changes that the axes of the letters A and B have undergone in relation to the bottom of the page. Each letter has rotated twice. The letter A has rotated upward 45° by elevation of the clavicle, but since the clavicle has rotated backward, the artist has had to rotate the vertical axis of the letter in order to make it legible. The letter B has twice rotated, each time in an opposite direction. Once because the artist rotated it on the humerus and once by elevation of the humerus itself. You may not understand this now, but after you have read the next chapter and have mastered Figs. 30 and 31, return to this puzzle and agree with me, only to disagree again, for the B has been inverting as it rotates and should be upside down and right side out, like the contour of the articular surface.

When the arm is elevated, even the subdeltoid portion becomes subacromial. When the arm is rotated inward some of the subdeltoid portion becomes subcoracoid.

Now if you please, remove the deltoid, being careful to leave the roof of the bursa which is underneath it. When you have examined it again without the deltoid, clear away the portion which was formerly attached to the deltoid and examine the base to see how much overlies the tendons of the short rotators, and how much overlies the tuberosity. You will find that the upper half of the hemisphere lies over the tendons while the lower half lies over the tuberosity, and yet the whole base presents such an even convex surface that it is almost a perfect hemisphere, showing very little sign of the different structures underneath it. By careful palpation, however, you can determine the edge of the tuberosity and the bicipital sulcus. Next, remove the acromion process at its base, and observe the arrangement of the short rotators in relation to the bursa. This is best done by cutting off the muscles of the short rotators from their origins on the scapula, and reflecting them over the head of the humerus. As you reflect them outward over the head of the humerus, you will find that the dissection readily reveals the glenoid portion of the capsule, but when you come within an inch of the insertion of the tendons into the tuberosities you can only separate the tendons from the capsule by sharp division. Leave the short rotators thus inverted over the head of the bone as a boy pulls his sweater halfway over his head, and remove the glenoid attachment of the capsule. Inside the capsule you will find the long head of the biceps free except for its attachment to the upper edge of the glenoid. Now look inside the capsule from the glenoid end and observe the condition of the sulcus which was described on page 17. Note the canal where the biceps tendon follows into the groove through the intertubercular notch. Slit open the groove and follow the tendon down to see where the synovial membrane is reflected. Observe that in doing this you cut across the tendon of the pectoralis major and you pass between the attachments of the pectoralis major on the outer side and those of the teres major and latissimus dorsi on the ridge which forms the inner lip of the groove. Do not forget to examine from the inside of the joint the extensions under the infraspinatus and subscapularis which have been called bursæ and were alluded to on page 20.

The *subcoracoid portion of the bursa* (Bursa M. coraco-brachialis) has been described by some anatomists as a separate bursa. Goldthwait (*Am. Jour. Orth. Surg.*, May, 1909) has attempted

to account for certain clinical symptoms as arising from inflammation in this bursa. I regret that I cannot subscribe to Goldthwait's anatomic description, nor to his interpretation of the clinical symptoms caused by it. I have great admiration for the work which he has done in regard to the conditions in the lumbosacral and sacroiliac regions. He has been a great pathfinder in the treatment of these conditions, but I feel that his articles on the shoulder are less accurate and sometimes misleading. I think that most of the cases which Goldthwait attributes to subcoracoid bursitis are essentially subacromial, for the subcoracoid bursa is only a part of the subacromial bursa. I have already explained that the subacromial bursa in the anatomic position becomes largely subdeltoid, and *vice versa*, in abduction, becomes largely subacromial. When the arm is behind the back the subdeltoid bursa becomes largely subcoracoid as the humerus is rotated internally. In external rotation the subcoracoid portion is nearly effaced as the movable periphery is stretched outward from beneath the coracoid process. Even in cases in which there may be a synovial fold between the subcoracoid and subdeltoid portions of the bursa, the two portions are essentially one. I have found this partition complete, although not infrequently I find a nictitating fold of the finest synovial membrane which forms only a partial partition between the two. These nictitating folds are always found in the subacromial bursa, not only in this situation, but in other parts. They are really parts of the movable periphery. They fold on themselves when not stretched by some extreme motion, and because they are almost as thin as cobwebs, the double layer has little thickness and is quite transparent. Even when double, they are thinner than the normal peritoneum. After trauma or operation in this region, these film-like partitions in various parts of the bursa may become adherent and cause it to be split up into a number of small sacs. Even when the subdeltoid portion of the bursa has been removed, a new subdivided sac takes its place.

The reader should thoroughly understand that there is a difference between the subcoracoid portion of the subacromial bursa (Bursa M. coracobrachialis) and the bursa connecting with the joint spoken of on page 20 which lies under the subscapularis, between it and the edge of the glenoid (Bursa M. subscapularis). This extension of the joint has nothing to do with the subcoracoid bursa about which we are speaking, which lies anterior to the subscapularis (Fig. 17). The subcoracoid bursa or, as I prefer to say, the subcoracoid portion of the subacromial bursa, lies between the subscapularis muscle, the coracoid process and the combined origins of the tendons

of the short head of the biceps and coracobrachialis, which arise from the coracoid process. It is therefore separated by the whole thickness of the subscapularis muscle from the extension of the joint beneath it. However, bursæ, even more than muscles or tendons, vary in form in different individuals. For instance, since both bursæ approach each other on the superior surface of the subscapularis, we sometimes find them united over it, so that a horseshoe shaped arch is formed beneath the coracoid process and the tendons arising from it. Under this arch the subscapularis may run with much freedom. I think such communications are not uncommon, particularly where the bursæ have become dilated from the presence of fluid.

Since the movement of the subscapularis muscle is at right angles with the tendons of the coracobrachialis, etc., it is quite necessary for nature to supply a bursa in this region. The arc of motion of any point on the lesser tuberosity is somewhere about an inch and a half between the position of internal rotation and the position of external rotation, and this would be a long stretch for simple areolar tissue. When I first wrote about these bursæ, I did not fully realize that the subcoracoid and subacromial bursa usually communicated, and the old figures were inaccurate for that reason. (Fig. 33.)

Other bursæ about the shoulder have been claimed. Piersol says:—

"*An infraserratus bursa* has been described (Terrilon) situated between the inferior scapula angle and the chest wall. Its enlargement gives rise to friction-like crepitation or creaking, which has been mistaken for fracture of ribs or scapula, or for an arthritis of the shoulder." He also quotes Nancrede as discussing these symptoms. Goldthwait has also written on a subscapular bursa which he claims may give similar symptoms. This bursa is said to be between the upper and anterior portion of the blade and the back of the first three ribs. It also would necessarily be "infraserratus."

We must agree that in some people, there are peculiar cracklings when the shoulder blades are moved, and that pain sometimes occurs with the cracklings. Goldthwait is inclined to attribute these peculiar cases, in which there is a painful crepitation or creaking, to this subscapular bursa, and it is quite possible that Terrilon and Nancrede and others have attributed the same symptoms to the above-mentioned infraserratus bursa at the lower angle. I have sometimes thought that such noises were due to unusual friction in the small inconstant subtrapezoid bursa which lies over the triangular surface at the base of the spine of the scapula under the trapezius. This

particular bursa is very apt to become sore in cases where there are scapulo-humeral adhesions and the shoulder blade is obliged to do double work. I am apt to attribute such symptoms to this subtrapezoid bursa and am not prepared to take a positive stand as to the existence of either the infraserratus bursa at the lower angle of the scapula, nor of the subscapular bursa at the upper angle of the scapula. I recognize cases which have painful crepitation in these regions, but practically I do not see cases with severe enough symptoms to make me feel like operating on these patients. Goldthwait's article is worth reading, and he has undoubtedly cured patients with these symptoms by removing the upper portion of the scapula. It seems to me that this is a rather radical operation for a doubtful condition, but the operation is not dangerous and the symptoms sometimes very severe. One should consider the question carefully. The shoulder is prone to reverberate crackling noises which oftentimes are perfectly painless. I can move my own scapula so as to make it sound about the room without the slightest pain, and yet I cannot even locate the exact spot where the primary vibration takes place. Creaking shoulders are as common as creaking knees and are usually painless. One must be on one's guard not to attribute symptoms to conditions which cause creaking noises, for the symptoms may be due to something which accompanies rather than causes the creaking.

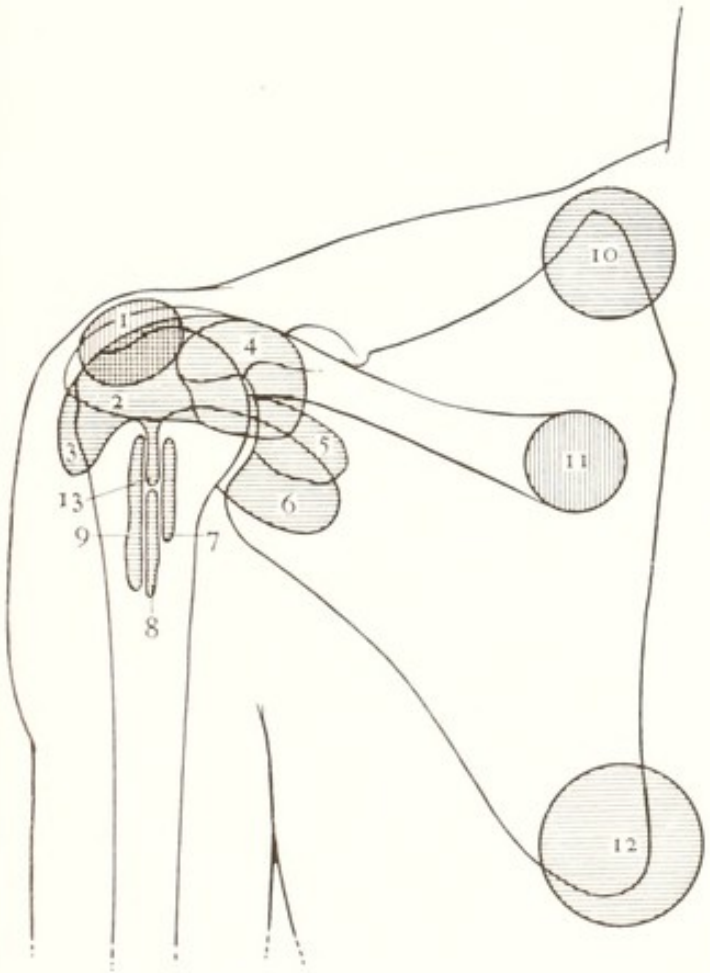


FIGURE 18. NORMAL BURSÆ ABOUT SHOULDER

Diagrammatic projection of all bursæ with numbers corresponding to list on p. 4.

Small bursæ are described beneath some of the tendons near their attachments to the humerus on the ridges on each side of the bicipital groove. These bursæ probably exist to permit the bone to turn away from the dense tendons when the latter exert their power to ro-

tate the bone on its long axis. The teres major, latissimus dorsi and pectoralis major are said to have such bursæ. I have never seen lesions in any of them, although sometimes I have found local tenderness over their locations in living patients. Such cases have usually promptly recovered; I have never known of any chronic condition in one of these bursæ.

I have also seen a case where there was a very distinct bursa superior to the coracoid process, between it and an anomalous insertion of the pectoralis minor to the outer side of the process. This anomaly, if found to be a frequent condition, might explain a few cases which are now clinically obscure.

The synovial lining of the bicipital canal has sometimes been described as a bursa. To my mind it is more properly called a sheath, but it is in fact merely an extension of the joint cavity with which it freely communicates.

An anatomic point which is readily forgotten in thinking of the shoulder is that the upward motion called *hunching* is performed by the upper part of the trapezius muscle, and this muscle is supplied by the spinal accessory. Not long ago, a patient was sent to me for shoulder symptoms which had not been attributed to a recent operation on the neck!

An interesting, if not important point, is that the *platysma extends down into the skin over the shoulder*, and Gray says that it actually has an attachment to the edge of the acromion. In cases where the trapezius has been paralyzed for a long time, one may see the shoulder feebly lifted by the platysma.

It does not seem to me necessary in this book to review the situations of the *arteries and veins*. Surgeons have come to think lightly of arteries because it is so easy to tie them and find no apparent immediate sign of harm following the ligation. As a rule one can safely rely on the anastomoses to supply the region with sufficient blood. Personally I try to save arteries and even large veins when I can. In plastic operations about the shoulder joint, it is doubly necessary, for if one ties too freely, one may tie not only the main artery of the part, but its collaterals, and healing may be delayed.

The *nerves* which supply the muscles about the shoulder will be taken up with the discussion of the various forms of paralysis, but the reader should at least know well the upper portion of the brachial plexus.

There are *three free bony surfaces* in the shoulder which one should bear in mind, for they are of practical importance beyond

the slight attention called to them in anatomic text books. One of these is on the back of the upper end of the humerus, in the region of the surgical neck, internal to the origin of the external head of the triceps. This bare portion of course is not, strictly speaking, bare, for it is covered with periosteum and areolar tissue, but it has no muscular origin or insertion on it, and is opposite the glenoid head of the triceps. The other two similar surfaces are on the inner and outer side of the neck of the glenoid on the body of the scapula. The anterior is beneath the subscapularis and the posterior beneath the infraspinatus. These surfaces should be borne in mind when one is visualizing fractures or dislocations of this neighborhood. The extensions of the joints (B. M. subscapularis and M. infraspinati) under these muscles lie in the spaces over these smooth areas.

The following chapter discusses the normal motions which the structures spoken of in this chapter may perform in relation to one another. The reader should endeavor to visualize the parts taken by the supraspinatus and by the subacromial bursa as he follows the text.

REFERENCES

Comparative Anatomy

FLOWER, WM. H., An Introduction to the Osteology of the Mammalia, third edition, 1885.

PARKER, W. K., The Structure and Development of the Shoulder Girdle and Sternum, Ray Society, 1868.

I have found no mention of the subacromial bursa in any anatomic description of lower animals, although such bursæ certainly must exist in the primates and possibly in beavers.

Human Anatomy. I know of no good descriptions of the anatomy of the supraspinatus tendon or of the subacromial bursa, but I have not had access to the following articles to which reference is made in the literature. Possibly these, although written long ago, describe these structures adequately.

JARJAVAY, J. F., Die Anatomie und Pathologie der Schleimbeutel und Sehnenscheiden, Erlangen, 1863.

GRUBER, W., Die Oberschulterhakenschleimbeutel (Bursæ mucosæ, supracoracoideæ). Eine Monographie mit Vorbemerkungen enthaltend: Beiträge zur Anatomie der Regio infraclavicularis und deltoidea, 4°, St. Petersburg, 1861, Repr. from: Med. Acad. imp. d. sc. de St. Petersburg, 7, x, iii, no. 11.

Superficial descriptions of the bursa are given in a few text books and in some of the many articles referred to in the bibliography of Chapter VI, but it does not seem necessary to go into detail in regard to the points in which they differ from mine. I feel quite confident that the reader will find the structures which I have described in this chapter, if he will carefully study the best book there is on the subject of anatomy — the human body — but he must be painstaking in following the instructions. More detail in regard to the microscopic anatomy of the insertion of the tendon will be found on pages 97 to 107.

Chapter II

NORMAL MOTIONS OF THE SHOULDER JOINT

There are certain anatomic terms customarily applied to the motions of the joints in general which are well understood except when applied to the shoulder. For instance, will the reader kindly flex his shoulder, *i.e.*, his scapulo-humeral joint, to its utmost. Please pose in turn in extreme abduction, extreme adduction, and in extreme extension. I plead with you to try these positions before you read any further, for it probably will be necessary for you to re-educate yourself so far as the meanings of these terms are concerned. Having determined what you think they mean, answer the following questions:

What motion occurs in the scapulo-humeral joint when you raise your finger tips as high as you possibly can toward the ceiling?

In what position is the joint when your body is in the "anatomic" position?

Picture to yourself a horse taking a jump. Does he flex his scapulo-humeral joint before he takes the jump or after?

Figure 19. Horse Taking a Jump

Before reading further please name to yourself the position in which this figure shows the scapulo-humeral joint. In the man? In the horse?

There is some room for a difference of opinion on these questions, because we may be guided by the point of view of comparative anatomy or accept the terms which have become customary for the biped human. In man, the conventional anatomic position adopted for teaching purposes is a standing one with the arms at the side and the palms forward. The anatomic position for a horse is the position in which we usually see him standing, with his forelegs at a right angle with his spine. Man would have to raise his arms to a right angle in front of his body to approach the horse's position.

Sisson in "Comparative Anatomy of the Domestic Animals" (1921 edition) gives the following definitions:

"Motion which diminishes the angle included by the segments forming the joint is termed *flexion*, while that which tends to bring the segments into line with each other is called *extension*.

Circumduction. This designates movements in which the distal end of the limb describes a circle or a segment of one. In man such movement is easily performed, but in quadrupeds it is possible to a limited degree only, and is to be regarded usually as an indication of disease.

Adduction and abduction designate respectively movement of a limb toward and away from the median plane."

The horse clearly flexes his scapulo-humeral joint as he takes a jump and extends it as he lands on the other side. From a quadruped point of view we might say that the anatomic position of man is one of flexion of the shoulder; complete elevation of the arm (the Statue of Liberty or the diving position) is extension.

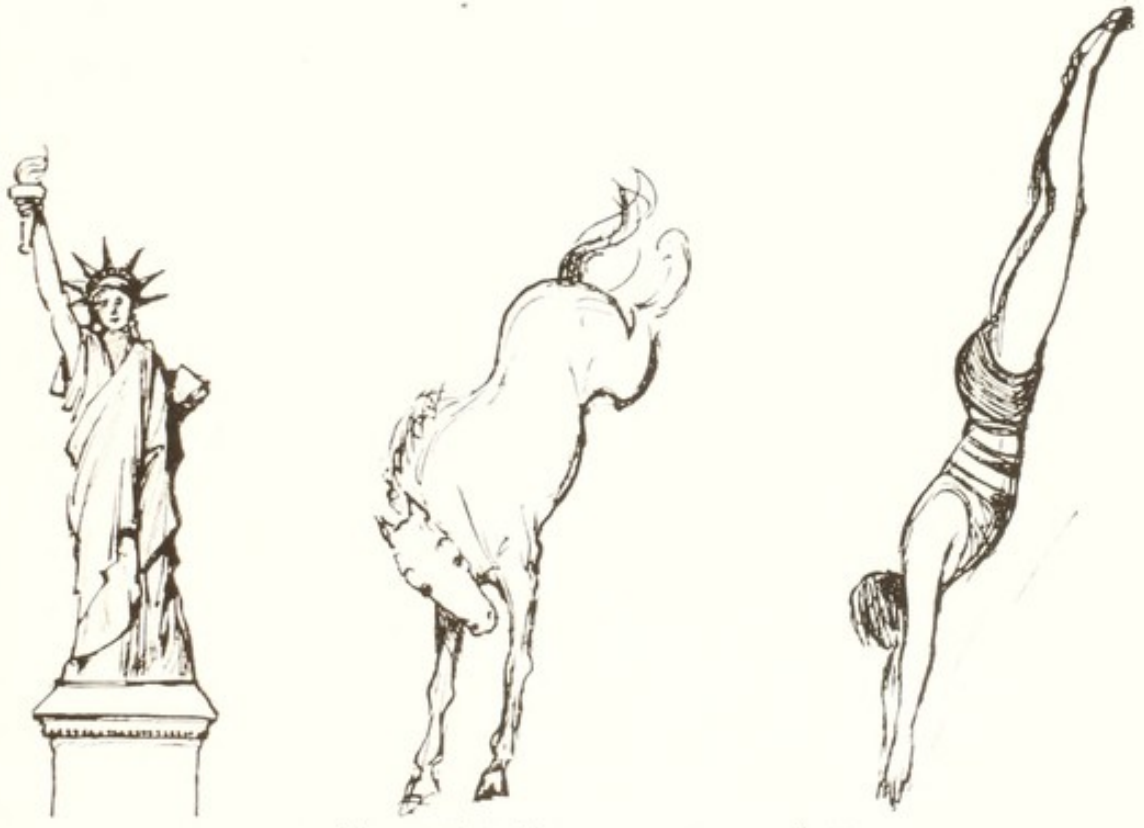


FIGURE 20. ELEVATION OF THE ARM

The term "elevation" clearly applies to the Statue of Liberty, but the arms seem to be "depressed" in the case of the diver, and it would seem absurd to use it to describe the position of these joints in the case of the horse, yet the relations in his shoulder bones are the same as in those of his human companions. A comparative anatomist would hold that all these figures represent "extension" of the scapulo-humeral joint. In this book the term "elevation" will be used for this relation of the humerus and scapula whatever the position of the body, but occasionally, to make the meaning clear, the term "extension," usually qualified by "quadruped," will be used.

From general usage in medical works, on the other hand, we think of the anatomic position as the starting point. Abduction to most surgeons probably means raising the arm to the completely elevated position pointing to the sky, and they would not specify whether this should be done in internal or external rotation, nor in what plane. Extension would probably mean to them the position assumed in enthusiastic greeting with both arms thrust forward (*i.e.*, the anatomic position for the quadruped). When one falls in this position one is said to fall on the extended hands or arms. If you ask a surgeon to put his shoulder joint in extreme flexion he will usually flex his elbow and bring his arm forward and upward. On the principles of comparative anatomy, he should bring his elbow behind his back, or shrug his shoulder. I have several times asked audiences of doctors to rise and place their arms in flexion, extension, adduction or abduction; the results were amusing enough to convince all of us that these terms have very vague meanings, so far as the shoulder is concerned.

The fact is that the members of the medical profession have taken very little interest in this wonderful joint and do not think of its mechanism as precisely as they do of that of their favorite automobile. For the very reason that there are no standard definitions I must be somewhat arbitrary in defining the terms I shall use in this book.

When in doubt it is well to follow Gray in anatomic matters, but in this instance he avoids the issue with great skill. Gray says: "The shoulder joint is capable of movement in every direction, forward, backward, abduction, adduction, circumduction, and rotation. The humerus is drawn *forward* by the pectoralis major, anterior fibers of the deltoid, coracobrachialis, and by the biceps when the elbow is fixed; *backward*, by the latissimus dorsi, teres major, posterior fibers of the deltoid, and by the triceps when the elbow is fixed; it is *abducted* (elevated) by the deltoid and supraspinatus; it is *adducted* (depressed) by the subscapularis, pectoralis major, latissimus dorsi, and teres major; it is *rotated outward* by the infraspinatus and teres minor; and it is *rotated inward* by the subscapularis, latissimus dorsi, and teres major."

Observe that he has not used *flexion* or *extension* and thus avoids controversy. He qualifies "abducted" by a bracketed (elevated) and "adducted" by (depressed). Are the arms in diving depressed, elevated, abducted or extended?

Piersol does not help us, for according to him the diver's arms are flexed. "A motion bringing the distal end of a limb bone nearer

the head is called flexion; the opposite movement, extension." If we accept this definition for the scapulo-humeral joint, flexion would occur as we raise the arm from the anatomic position to bring the elbow up beside the head, a motion which in the horse would be extension.

FIGURE 21. ABDUCTION

The comparative anatomist would admit that the scarecrow holds its arms abducted, but he would differ with the anatomist as to the degree of abduction, and as to the plane in which the humerus had arrived at this position. The polo pony, he would contend, is in the same position as is the scarecrow; *i.e.*, the forelimbs are carried outward from the median line as far as nature permits. The anatomist would say that the scarecrow had performed abduction for about 90° from the anatomic position through the vertical coronal plane; the comparative anatomist would insist that the motion had occurred in the horizontal transverse plane. Abduction for the comparative anatomist has reached its limit, but the ordinary surgeon would say that the arms could be still further abducted until they pointed vertically upward at the side of the head; yet the anatomist might call this portion of the motion, flexion. Both would agree that the scarecrow's arms are abducted, and I shall call this position *abduction* irrespective of the degree of rotation of the humerus, or of the plane through which it is attained.

A horse or a dog may appear to abduct one leg somewhat as in the lower cut (*c*), but careful observation will show that most of this abduction is obtained by flexion of the elbow joint plus a little rotation of the scapula; little or no outward motion has occurred in the scapulo-humeral joint itself.

Piersol says that *adduction* is bringing the limb "towards the median plane of the body," yet most surgeons would, I think, consider the arm adducted when in the anatomic position at the side, as Gray says, "depressed." Suppose that Piersol had placed a student in the scarecrow position and then asked him to adduct his scapulo-humeral joint. Would he mean to have the student move it upward to a position by the head; "towards the median plane of the body" or

downward (depressed) to the anatomic position "towards the median plane of the body"? Certainly he could not find fault with the student who had studied his pre-medical biology and carried his arm in across his chest.

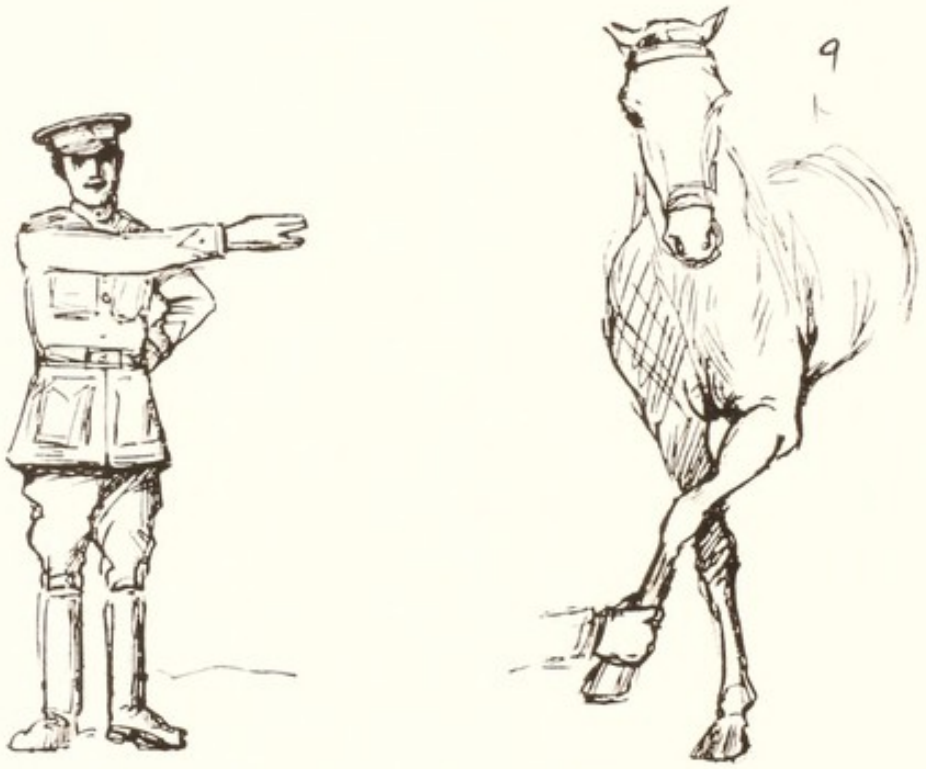


FIGURE 22. ADDUCTION

The horse, even when coaxed by a kind master, can adduct his foreleg only to a very slight extent; man himself has much less ability to adduct than he has to abduct. If the reader will try the position shown in this drawing of a traffic officer, he will be surprised to find how little true scapulo-humeral adduction occurs. Be sure not to move your scapula.

Will the reader undertake to analyze the positions of the scapulo-humeral joints in the well-known group of Laocoön and his sons? Which of Laocoön's arms is flexed?

FIGURE 23. LAOCOÖN ILLUSTRATES DORSAL FLEXION

The position of Laocoön's *left* arm is not defined anatomically in English, but it has been called "retroversion" by Braus, the German anatomist, and I have christened it "dorsal flexion."

Laocoön's *right* arm is not in a complete position, either from the point of view of elevation or from that of rotation.

After giving much thought to these matters, I have concluded to adopt the wise policy of Gray and avoid using the terms *flexion* and *extension* so far as I may. But what shall I call the position of Laocoön's left arm as he struggles to drag the head of the serpent from his hip? I shall want to speak of this position again and again, for it is the very position in which it is easiest to feel a ruptured supraspinatus and much the most favorable position in which to operate on such cases. Yet neither anatomists nor comparative anatomists have a definite name for it. I will christen it "dorsal flexion," although it is merely extreme flexion to a horse as he takes a jump.

What of the opposite motion? Shall we call it forward flexion or extension, or abduction, or elevation in the sagittal plane? I shall use the first when necessary but usually the last.

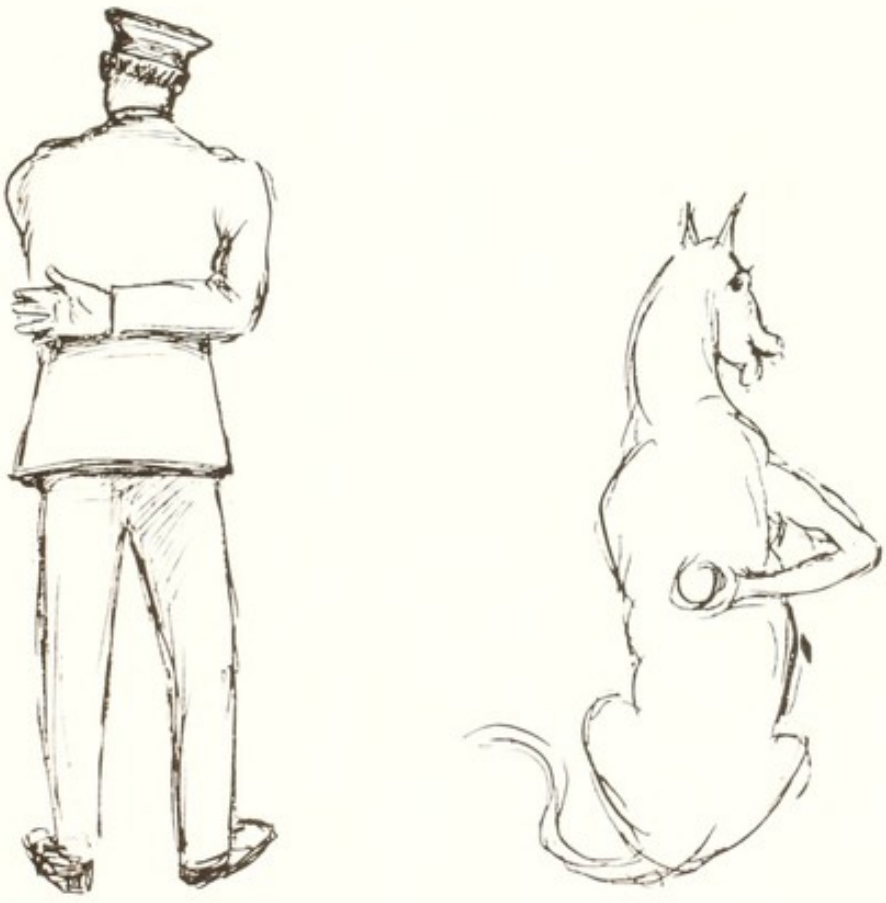
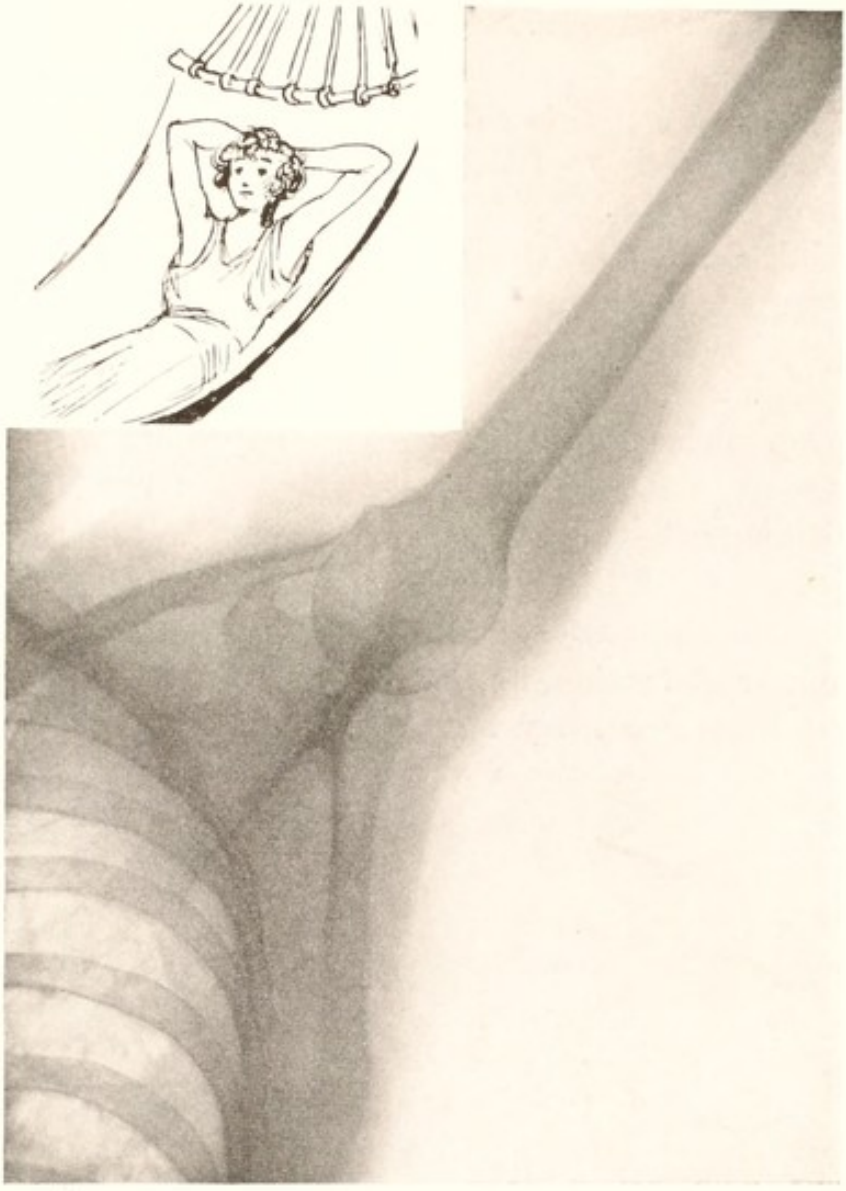


FIGURE 24. INTERNAL ROTATION

When the hand is placed behind the back the humerus is rotated inward to its full extent and yet lies in the same long axis which it assumes in the anatomic position; therefore, so far as the shoulder joint is concerned it may be described as in the anatomic position, but the degree of rotation should also be mentioned. The horse cannot permit his foreleg to assume this position, even with the help of the imagination of the artist, for the horse cannot rotate his humerus on its long axis.



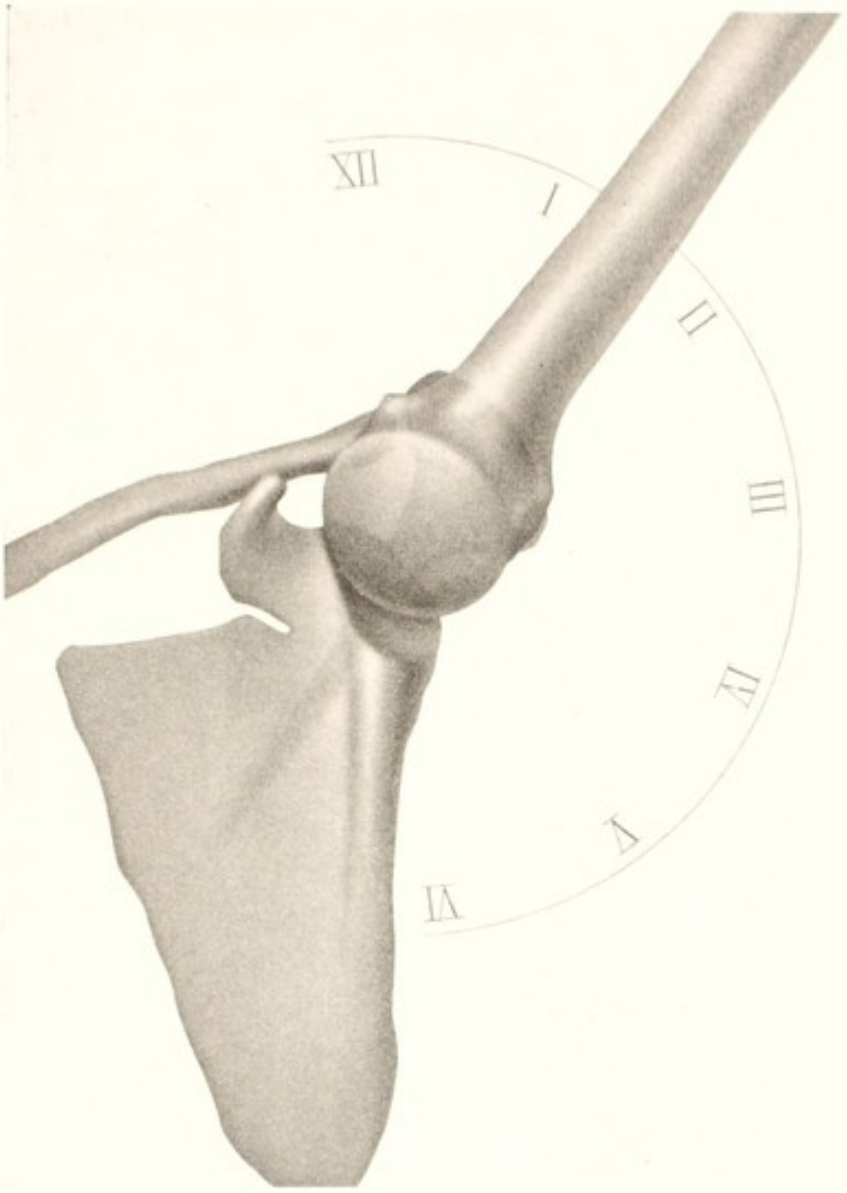
FIGURES 25 AND 26. ABDUCTION AND EXTERNAL ROTATION

This is a very natural position for the human arm when the body is recumbent. Many people sleep with one or both arms held in this manner. The scapulo-humeral joint itself has almost reached the pivotal position but the clavicle is not fully elevated. Since the deltoid, the supraspinatus and the infraspinatus are relaxed, it is the most favorable position to encourage physiological repair of lesions in and about the tuberosity.

Antero-posterior view in hammock position. Notice the symmetrical relation of the acromion to the head of the humerus. Notice that the axis of the humerus is in line with the axis of the spine of the scapula, and that changes in the relations of these axes would make different parts of the periphery of the acromion act as fulcrums. Notice that in this position the axis of the head and neck of the humerus is in the same plane as both of these axes; in other words, it points toward you at about 45° from the long axis of the shaft. If this patient raised her arm to complete elevation, with the axis of the humerus vertical, the rounded contour of the articular surface would point outward and forward, and the axis of the neck would make an obvious angle with that of the shaft.

This shoulder joint is locked, so far as posterior motion is concerned, but it still might have a number of degrees more lateral motion in the coronal plane, provided it rotated a little before it reached the true pivotal position where it would become locked. Such a position as that shown in this picture I call a subordinate pivotal position; *i.e.*, a position in which the joint is locked posteriorly so far as dorsal motion is concerned, but lateral motion is still possible if the humerus be rotated.

Subordinate pivotal positions occur all the way from the anatomic position to that of complete elevation. They are conjunctions of the two bones in quadruped abduction with different degrees of humeral rotation. They are expressed graphically by the triangle in Fig. 29, or by the face of the clock in this diagram.



For example when the arms are akimbo, the humerus and acromion lock, so far as motion in the coronal plane is concerned, when the axis of the humerus has reached an angle a very little over 45° from the vertical, as the patient stands. If, when in this position, the humerus be rotated outward 90° more, further elevation may be made until locking occurs at about the horizontal plane and the humerus then will be in extreme internal rotation; and although the humerus has just been rotated outward, you cannot, now that it has been raised, rotate it much inwardly! However, it will easily rotate outward 90° more, and in the coronal plane ascend to the pivotal position, where it will be almost completely locked, and the possibility of rotation in either direction will be very slight. On the way to this complete locking it will pass this neutral position, which I have called the hammock position, and in which the joint and its adjacent structures are very nearly at rest. In this position the X-ray shows the facets of the teres minor and of the subscapularis in profile, and the axis of the humerus points at one-thirty on the dial of the acromion.

All of this goes to show that the scapulo-humeral motions cannot be determined by experiments on the cadaver with the scapula held in a vise, for the humerus moves in different ways according to the position of the scapula. Furthermore, not only the subtle changes due to rotation of the humerus on its long axis, and the still more subtle changes in the shape of the coraco-acromio-glenoidal cup while the clavicle rises, but changes in the position of the spine, must be considered to attain a true understanding for clinical purposes. Tell me how many degrees of elevation of the humerus the scapulo-humeral joint permits; then put your hand behind your back and demonstrate it to me! You are in a subordinate pivotal position at about six o'clock on the dial of the acromion. Contrast this with the motion when you raise your arm in external rotation in the coronal plane. If you are young and limber, the axis of your humerus, when in superior posterior adduction, may precede noon on the dial of the acromion, and when in inferior posterior adduction it may pass six o'clock.

Beware of the fact that X-ray pictures are projections and angles are deceptive. A metal W could appear in an X-ray picture as either a transverse or a vertical line.

Elevation and rotation seem to be somewhat interchangeable, like wattage and voltage. For instance, when the arm is rotated inwardly 100 per cent, elevation in the coronal plane is practically nil; when it is elevated 100 per cent, internal rotation is practically nil.

Gray was wise in putting (*elevation*) in brackets after his definition of abduction. Elevation is a good term for our special needs. When we raise an arm to point directly to the sky we certainly elevate it, whether we lift it in the coronal plane in abduction and external rotation or extend it according to the comparative anatomist, or flex it in a sagittal plane to please the anatomist, or divide our attention and elevate it as we may, anywhere in the 90 degrees between the two planes. We may even keep it adducted as we raise it. As will be seen later it will reach in complete elevation the same ultimate position no matter whether it is rotated internally or externally as it starts, and no matter in which plane it rises.

My personal preference would be to use the comparative anatomist's terms, for elevation seems to me extension and the anatomic position flexion. However, I shall avoid using "extension" and use "elevation" so far as I can in describing the relative position of the shoulder bones which is suggested alike by one's thought of either the Statue of Liberty or of Annette Kellerman.

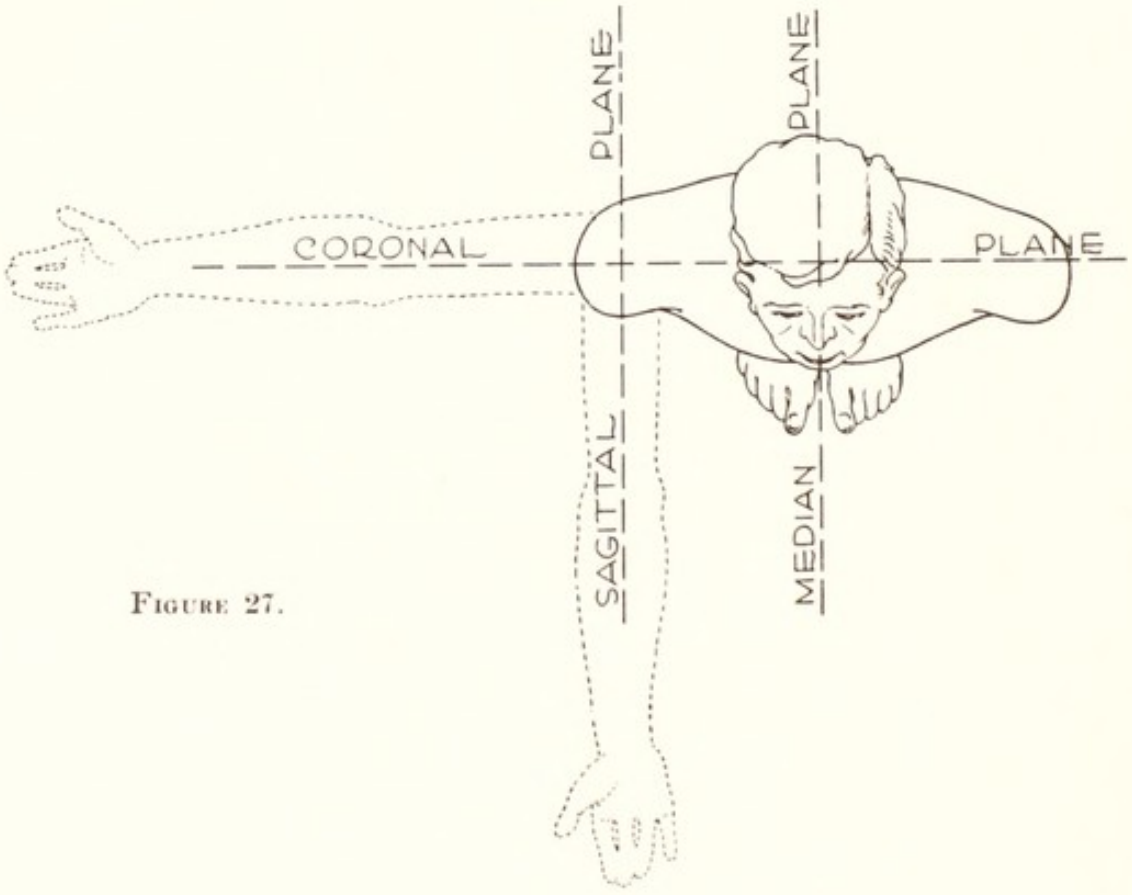


FIGURE 27.

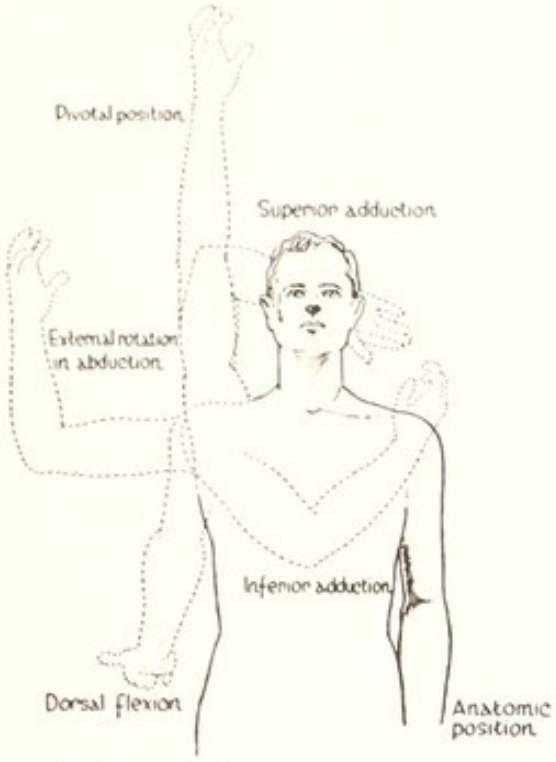


Figure 28. The Terms To Be Used in This Book

Should this man bring the right arm in its lower phase, which is "dorsal flexion," directly forward in the sagittal plane, he could not arrive in the pivotal position without rotating his humerus inward for about 90°. I prefer to call the motion in which he would endeavor, without rotating the humerus, to raise his arm forward in the sagittal plane "elevation in the sagittal plane," but it may be called *forward flexion*. In the extreme of this position, the axis drawn through the condyles of the lower end of the humerus would be the same as in the anatomic position, *i.e.*, transverse or coronal; in the pivotal position this axis changes 90° (*i.e.*, becomes sagittal), and the inner condyle points forward. When I say that a patient "elevates his arm" I do not especially indicate whether he rotates it externally and carries it up in the coronal plane, or whether he rotates it internally and carries it up in the sagittal plane. Usually he elevates it in some intermediate plane, and the rotation of the clavicle and of the humerus on their long axes occurs without his knowledge or observation. The only justification for using "forward flexion" is that it is the opposite of dorsal flexion, which is true extreme flexion as in the jumping horse. Forward flexion is really only extension.

Adduction would be used to describe a mid-position between "superior adduction" and "inferior adduction" of the scapulo-humeral joint. These terms may be further qualified by stating whether they are posterior or anterior. They are characteristic motions in youth, and it is remarkable to what a degree they may persist in some adult women. One rarely finds a laboring man who can perform superior adduction; many cannot even attain a vertical position.

We have by no means settled the terminology which should be used in describing the motions of the shoulder, but we have, I hope, arrived at a fair understanding of the terms to be used in this book.

Now that we have the terms to use for descriptive purposes, we may study the motions of the shoulder under the following headings:

(1) Rotation on the long axis of the humerus.

(2) The relative parts played by the three different joints in the motion of elevation.

(3) Lateral elevation in the coronal plane.

(4) Antero-posterior motion in the sagittal plane.

(5) Circumduction.

(6) The motion at the acromio-clavicular joint.

(7) The scapulo-humeral rhythm in which all the structures coöperate.

Rotation on the long axis of the humerus. When the patient is standing in the anatomic position, with his arm at the side and the palm forward, the humerus is in almost a mid-position between internal and external rotation. By turning the palm as far as one can outward, the humerus has moved to extreme external rotation. By turning the palm inward until it has faced backward and again outward, the plane of the palm has traversed nearly a complete circle (360 degrees), owing to rotation of the humerus and scapula (180 degrees), plus rotation of the radius (nearly 180 degrees). When one reaches up as high as possible, the humerus arrives at a fixed position where it can no longer be rotated, although the palm will turn nearly 180 degrees. Now in all of these motions, the turning of the palm is deceptive, because the palm turns almost a half circle by the motion of the radius over the ulna—the rotation of the humerus on its long axis being only a part of the motion. In order to get rid of this deception in testing the scapulo-humeral motion, one should flex the forearm to a right angle, and retain it in this position throughout our tests. Since the elbow joint does not move laterally, one can discount the motion of the palm and judge the rotation of the humerus (and scapula) by the angle through which the flexed forearm (*i.e.*, the ulna) moves inward and outward. No matter what position the palm assumes, the motion of the humerus is not affected so long as the forearm is flexed at a right angle, but when the elbow is extended the humerus may rotate synchronously with the radius without our perception.

Keeping the forearm flexed at a right angle and keeping the elbow at the side, one can rotate the humerus from the anatomic position outward about 90 degrees. Keeping the elbow at the side and placing the flexed forearm behind the back, one can rotate the humerus inward about 90 degrees. The total arc of rotation, there-

fore, is at least 180 degrees in a normal person of medium age, provided that the arm is at the side in the anatomic position, for the degree of rotation diminishes as the arm is elevated (p. 44). This estimate is not perfectly exact because the whole scapula rotates a little also. It is slightly in excess of 180 degrees in young people, particularly in girls, and is slightly beneath this in elderly persons —although occasionally, elderly persons, particularly women, retain a large range of motion.

I wish to warn the reader not to get the idea that rotation of the humerus is a motion which occurs in the true shoulder joint only. The subacromial bursa, particularly its subcoracoid portion, is as essential in rotation as in upward motions of the arm. (See Fig. 33.)

And now we come to a curious paradox which I have only recently observed, although I have studied the motions of the shoulder for years. *You can prove that the completely elevated arm is in either extreme external rotation or in extreme internal rotation.*

(1) Proof that it is in external rotation.

a. Raise your hand as high as you possibly can toward the ceiling.

b. Without moving the humerus, flex your elbow to a right angle, and your forearm will lie across the top of your head.

c. Holding your elbow still flexed at a right angle, let your humerus descend slowly, without rotation on its long axis in the coronal plane to the side of your body.

d. The forearm will be pointing directly outward from your body in extreme external rotation of the humerus.

e. Since you let it descend, without rotating it at all, it must have been in external rotation all the time. Q. E. D.

(2) Proof that it is in internal rotation.

a. Raise your arm to complete elevation.

b. Flex your elbow to a right angle and the forearm is again over your head.

c. Let the humerus (without rotating on its axis) descend in the sagittal plane.

d. The forearm will be across the front of your body pointing to the opposite side; *i.e.*, in internal rotation of the humerus.

e. Since you let it descend without rotating it at all, it must have been in internal rotation all the time. Q. E. D.

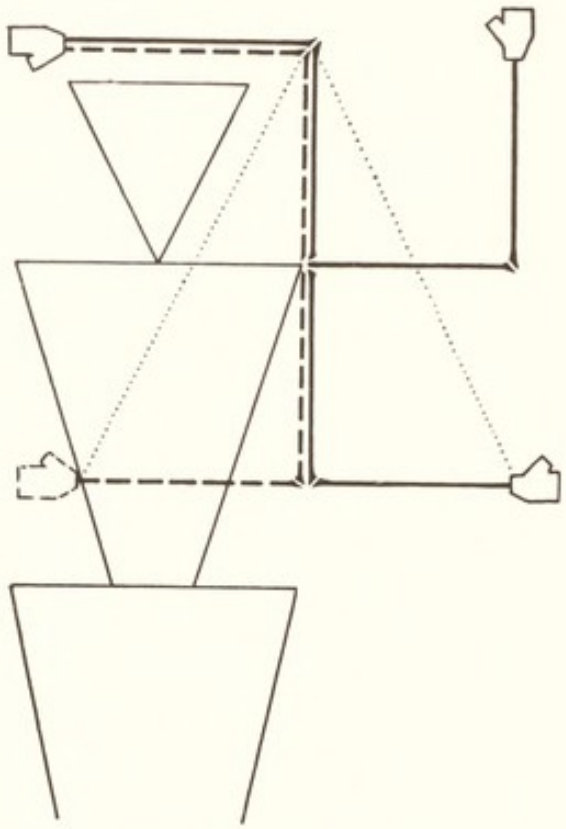


FIGURE 29. THE PIVOTAL PARADOX

A diagram to illustrate the pivotal position and the diminution of the possibility of rotation of the humerus as the arm ascends from the anatomic position. If external rotation is performed in the anatomic position the arm may be raised in the coronal plane, as illustrated by the solid line. If it be rotated internally while in the anatomic position it can be raised in the sagittal plane, as illustrated by the dash line. The triangle completed by the dotted lines indicates the degrees of the ability of the humerus to rotate, as the arm proceeds upward. Notice that in whatever plane the arm is elevated the flexed forearm arrives at a position over the head, where scapulo-humeral rotation practically ceases.

Still another point has been formulated only recently. *The range of rotation of the humerus diminishes as it is elevated.*

In the anatomic position at the side the extent of rotation is roughly 180 degrees; when raised to the level of the shoulder in abduction (*i.e.*, the scarecrow position) or in mid-forward flexion (*i.e.*, the normal quadruped anatomic position), it is about 90 degrees, and in complete elevation it is practically nil, for the humerus cannot be further rotated externally or internally. The loss of range is gradual between these points.

I find it difficult to explain the paradox of the ability of the elevated arm to descend without rotating and come at will to the side in either external rotation or internal rotation. It makes the puzzle more clear to hold a humerus in your hand and observe that the lines of bearing made in these two motions on the articular surface would meet each other. Also you may see that the glenoid surface comes to a

point at its upper extremity. However, this does not answer the question of whether the completely elevated arm is to be considered to be in external or internal rotation. I favor internal rotation because the internal condyle points forward, but will call it *a pivotal position* to make sure. From the pivot it can descend in either way.

Analysis of relative parts played by the three joints during elevation of the arm. Elevation of the arm may occur in many different planes and the behavior of the scapula and humerus varies somewhat in each plane.

The rotation of the humerus on its axis, which we have just been considering, occurs in either plane if we start from the anatomic position. As has just been pointed out, in complete elevation the humerus arrives in a pivotal position in which little, if any, rotation is possible. When one reaches upward in any plane, three joints move. (1) The scapula rises on the chest wall owing to about 45 degrees of motion at the sterno-clavicular joint. (2) The acromion tilts upward under the tip of the clavicle owing to about 5 degrees of motion at the acromio-clavicular joint. (3) The humerus moves on the glenoid about 140 degrees.

Notice that the sum of these somewhat doubtful and variable figures is greater by 10 degrees than the one fixed and readily observed amount of 180 degrees, which the vertical axis of the humerus has passed through to become inverted. In other words, the full extent of motion in each joint has not been used.

In childhood and in youth the axis of the humerus may pass the vertical and proceed toward the median line at either end of its course for about 20 degrees.

Possibly by some feat of mathematics these motions may be accurately measured in the future, but the sum of the ranges of vertical motion possible in each joint will always be greater than the total of range accomplished when they all move together.

The explanation of this paradox lies chiefly in the facts that (1) each of the bones rotates in its relation to the others; (2) the clavicle also passes backward; (3) there is lateral movement as well as elevation of the two movable centers of motion; *i.e.*, the outer end of the clavicle may pass backward while the glenoid moves forward. (4) Each joint is capable of a greater degree of motion than it uses in the combined motions required to elevate the arm.

If you will hunch your shoulder and then elevate your arm you may feel the outer end of the clavicle drop, pass backward and rotate slightly in a left turn. In other words, the clavicle can be raised

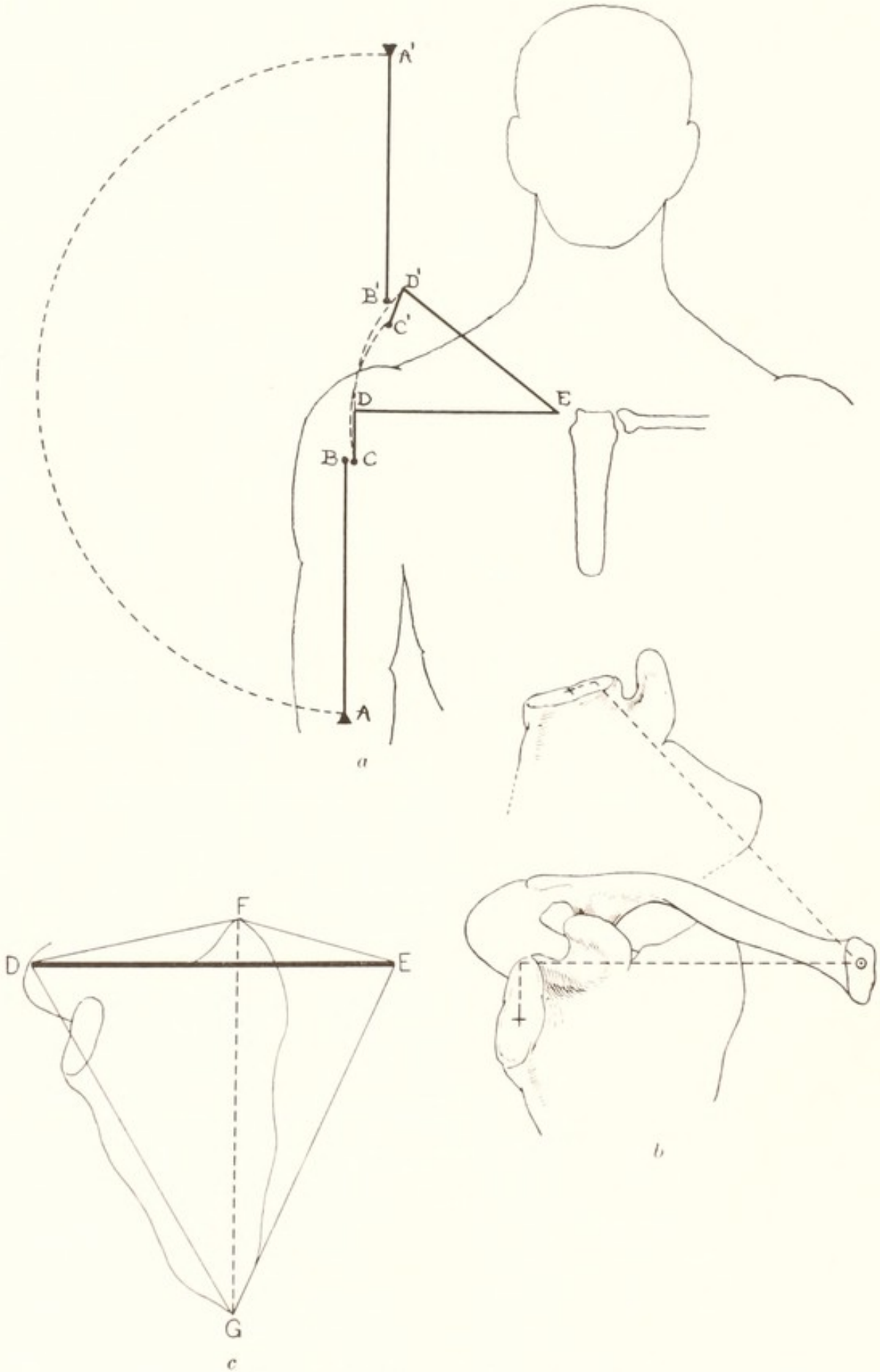


FIGURE 30. ELEVATION OF SCAPULA

FIGURE 30

Strictly speaking, any motion of the arm should be described in two ways; *e.g.*, stating the degree of abduction of the clavicle and also the degree of abduction of the humerus on the scapula. Usually when there is abduction of the scapulo-humeral joint there is, at the same time, abduction at the sterno-clavicular joint, but this is by no means always true. We may, for example, adduct the clavicle, and at the same time abduct the humerus. However, for clinical purposes it is best to consider the arm and shoulder as a whole. The diagrams on p. 46 suggest some of the phases of the coöperative movements of all the shoulder bones.

A—lower end of humerus. B—upper end of humerus. C—glenoid. D—outer end of clavicle. E—inner end of clavicle.

To elevate the arm straight toward the ceiling so that the axis of the humerus becomes inverted; *i.e.*, travels 180°, three joints are used. The clavicle E-D does not move directly up to E-D′, for, as it rises, it passes backward and at the same time twists anticlockwise on its own long axis about 45°. There is a negligible amount of motion at D in the acromio-clavicular joint, but the rotation of the clavicle carries the offset glenoid forward and upward; *i.e.*, the motion of D′ is backward away from the plane of the paper, but the motion of C′ is at the same time coming forward toward the plane of the paper. Meanwhile the humerus A-B is constantly moving on the movable center C. When we subtract the upward motion of the clavicle, *i.e.*, 45°, from the obvious 180°, we have about 135° of scapulo-humeral motion.

But how about the offset glenoid, which has also moved about 45° by *rotation* of the clavicle? If B and C were firmly united, A would be raised halfway to the level of C′ merely by this rotation. This would subtract 45° more from the 135°, leaving only 90° of pure scapulo-humeral motion. Thus authorities may disagree on the true amount of scapulo-humeral mobility according to whether or not the glenoid is considered to be fixed or in motion. It seems to the writer that the movement is coöperative and cannot be fairly subdivided among the three joints.

(*b*) In complete elevation, the scapula rises in nearly "one piece" with the clavicle. It is not quite "one piece," because its union with the clavicle through the coraco- and acromio-clavicular ligaments permits a certain amount of motion, the extent of which is so slight that we may ignore it in the interpretation of this diagram, which is particularly planned to suggest the idea that the motions of the clavicle are transmitted to the scapula, not only in elevation, but in rotation. However, the slight mobility of the joints between the scapula and the clavicle permits exaggeration of any one position when force, active or passive, is applied to the limb in that position.

The axes of the clavicle, the glenoid, the blade of the scapula and the acromion meet one another at varying angles, although in the main at about 45°. It is well nigh impossible to construct a diagram which would be actually correct in its attempt to show the complicated normal movements which may take place among these irregularly shaped bones meeting at such angles.

(*c*) If the reader will experiment with a block of wood shaped like a three-sided pyramid, the apex of which, D, represents the tip of the acromion, and the base, E-F-G, the lines drawn from the upper and lower angles of the blade of the scapula to the center of the sterno-clavicular joint, he may obtain a very good idea of the effect of any movement of the edge which represents the axis of the clavicle, whether that edge be raised or rotated. Such an experiment will make him thoughtful in regard to the X-ray interpretation of lesions of the shoulder.

The plane of the glenoid meets nearly all the faces of the pyramid at about 45° to 60°, and in the anatomic position it also assumes about this relation to the sagittal and coronal planes of the body. Place your pencil point at the center of the glenoid in this figure and hold the axis of the pencil at 45° to the plane of the paper and perpendicular to the plane of the glenoid. Then imagine rotation and elevation of D-E, and conceive of the effect on the axis of your pencil still held in the same relation with the glenoid.

The heavy line D-E is the border which represents the clavicle. Rotation of D-E for 45° on the point E shortens the X-ray projection of F-G and of all the other lines. It raises the plane of the glenoid 45° and therefore the axis of a humerus, which we may assume is moving on the glenoid. Meanwhile elevation of the clavicle for 45° raises the projection of the humerus 45° more.

When we discuss the pure scapulo-humeral motion shall we consider the plane of the glenoid to be fixed or in motion? Even without using the clavicle, tilting that plane by the use of your spine or by bending a knee makes a great difference in the relation of the long axis of the humerus to the floor.

higher by hunching than by elevating the arm. Rotation of the clavicle, slight as it is, tends to throw the glenoid forward and face it more directly upward. Consider that the clavicle is a rod fastened near its end to the coracoid and acromion which join together firmly at the glenoid. Forget the rest of the scapula. Twist the clavicle, which is about 7½ inches long, a few degrees on its long axis, and the glenoid, which is offset two inches, will move a good deal even assuming no acromio-clavicular motion.

As the arm is elevated it is very easy to feel on your own shoulder that the scapula not only rises but rotates on the chest wall. The lower angle which is easily palpated may travel an arc of over 45 degrees on the circumference of the chest. Part of this motion is made possible at the acromio-clavicular joint, but most of it is due to rotation of the clavicle at the sterno-clavicular joint.

To sum up, we may say that during the motion of elevation the humerus is rotating on its own vertical axis, pivoting on a fulcrum which is rotating and passing forward in relation to a center which is rising, moving backward and slowly rotating on a left turn.

Taking all these facts into consideration, it is quite possible that by astronomic calculation from motion-picture X-ray films of a living skeleton, the "relativity" of these compound motions might be made out. But when this has been done, calculations for the motions of the spinal column must be added, for in life it is scarcely possible to move the shoulder without compensatory curving or twisting of our spines. This is particularly true in patients with sore shoulders.

Lateral elevation of the humerus in the coronal plane cannot be accomplished as far as a right angle with the axis of the body, if the arm is first put in internal rotation (*i.e.*, arms akimbo). However, if it starts in the anatomic position it may be raised to the horizontal or perhaps a little higher without any external rotation. This includes motion of the scapula on the chest wall through motion at the acromio-clavicular joint and at the sterno-clavicular joint. Only a part of this 90 degrees is performed by the shoulder joint, perhaps less than a half. Full lateral motion or elevation in the coronal plane can only be accomplished in external rotation. In internal rotation, the tuberosity meets the acromion, but as the arm is externally rotated, the shape of the head and neck under the acromion changes, and the head rolls in under the acromion by a very narrow margin at just about the region of the bicipital groove. At the same time the scapula tilts so that the glenoid turns upward. However, elevation in the sagittal plane *can* be accomplished in internal rotation.

So far as *antero-posterior motion of the humerus* in a sagittal plane is concerned, *i.e.*, when the patient brings the elbow as far backward (dorsal flexion) and then as far up and forward as possible (forward flexion or elevation in the sagittal plane), the range is also nearly 180 degrees, being greater forward than backward. This is an important motion to remember, for one can palpate much more of the head of the humerus forward of the acromion when the elbow is carried as far backward (Laocoön's left arm) as possible. In this position the range of palpability of the head of the humerus is greater in some subjects than in others, depending on the shape of the acromion process and the position in which they habitually carry their shoulders. Sloping shoulders are more difficult to palpate in dorsal flexion than are square shoulders.

When the arm is carried as far forward and upward as possible *in the sagittal plane*, the entire humeral head, tuberosity and all, disappears beneath the acromion; the arm will be elevated to about 45 degrees above the horizontal and cannot be rotated further outward. Then if it is rotated about 45 degrees inward it will rise the remaining 45 degrees to complete elevation. But if the arm is elevated in the coronal plane it will have to be rotated outward to attain complete elevation. The bronze Mercury of Bologna shows the right arm in forward flexion, while Laocoön's right arm is abducted in mid-rotation. The former would have to rotate inward to attain complete elevation, while the latter must rotate outward.

In other words, complete elevation of the arm from the anatomic position in a sagittal plane is not possible; in order to arrive at complete elevation the humerus must rotate on its long axis externally at the start of this motion or internally at the latter part. As a matter of fact, in raising one's arm, one is unconscious of this rotation of the humerus on its long axis. Although the humerus can only rise to within 45 degrees of complete elevation in the anterior phase of this motion in the sagittal plane, it can proceed 45 degrees farther backward in the posterior phase than it can when it is rotated externally. Therefore the long axis of the humerus in the whole antero-posterior motion can also pass through an arc of 180 degrees, but the semi-circle described by the lower end of the humerus ends at a lower level in front, and passes further behind the body, than if elevation occurs in either external or internal rotation. As in motion in the other planes this movement, too, is partly accomplished by rotation of the scapula on the chest wall; that is, by motion at the sterno-clavicular joint.

Circumduction is a deceptive motion in the shoulder. One can swing the arm around and persuade oneself that the humerus is moving like a spoke in a wheel. Watch a man circumducting his arm and you will swear that the arm is rotating round and round with its hub at the shoulder joint. You do not observe that he is rotating his body, that his clavicle is circumducting through a small circle, that the scapulo-humeral joint is a moving pivot at the point of a cone, and that the humerus is rotating on its own long axis as it passes around the periphery of a compound cone, centering at the sterno-clavicular joint.

It will be a good mental exercise for the reader to trace on imaginary coronal, sagittal, transverse, median and equatorial planes the points he can touch with the tip of his elbow. Or he may employ a mechanical draughtsman to plot the points on the inside of a sphere which his elbow may reach without bending his spine. He will be surprised to find how like a conventional shield this figure will be.

We may roughly say that the tip of the elbow can be made to touch any point within most of a small hemisphere, and that the elbow cannot touch any part of what would be the opposing hemisphere. If the elbow and wrist are allowed to move, a still larger portion of a sphere can be swept by the hand. Then if you will allow your spine to rotate and bend, you can very nearly sweep the inside of three-quarters of a globe.

The acromio-clavicular joint. The exact manner of, and the exact amount of motion in this joint, still elude my curiosity. When one hunches the shoulder, the scapula rises several inches on the posterior ribs, and the spine of the scapula rises to a higher level than does the clavicle; *i.e.*, the scapula rolls forward. At the same time the angle between the spine of the scapula and the clavicle (as seen from above) appears to diminish to some extent. Furthermore, the vertebral border of the scapula remains only about 15 degrees off its normal parallel with the axis of the vertebral column, and yet the angle of the vertebral border with the clavicle as seen by X-ray diminishes. These facts all indicate that in hunching the shoulder there must be 5 or 10 degrees of motion at the acromio-clavicular joint. Incidentally you will find that the clavicle rises considerably higher when you hunch your shoulder than when you reach upward.

However, X-rays of a completely elevated arm show very little change in the right angle which the vertebral border forms with the clavicle, as compared to the angle in the anatomic position, although in elevation the vertebral border has reached an angle of 45 degrees with

the spinal column. If the reader will make a little model of sticks, using one for the clavicle, one for the vertebral border and one for the spine of the scapula, he may understand that a very slight rotation at the sterno-clavicular joint may greatly change the relations of the other bones as seen in any X-ray projection, without any movement taking place at the acromio-clavicular end. After reading various works on anatomy and making some observations by dissection and checking with the X-ray, I have come to the conclusion that the acromio-clavicular joint moves very little indeed, but this motion may occur in many different planes. Its surfaces slide a little, rotate a little, tip apart a little and act like hinges to some degree.

It appears that nature abhorred a perfectly fixed fusion at this point in her architectural plan, for it would oblige a very small point to bear the stress for a whole extremity. So she compromised by fixing the scapula near the outer end of the clavicle, by binding the latter firmly to the coracoid process and using the acromio-clavicular joint to steady the protruding tip of the clavicle on the acromion process. She bound the tip of the clavicle which extended an inch or two beyond the coracoid to the side of the acromion, not to its very tip. The ligamentous binding of coracoid to clavicle, though very strong, would not be truly rigid; the acromio-clavicular joint must take up the play or be rigid, and if rigid, be very much subject to injury. Therefore nature made it slightly movable in order that it might: (1) Swing a little (as seen from above downward—p. 10, Fig. 6) so that the angle between the axes of the scapula and clavicle could be slightly diminished or increased. (2) Rock a little (as seen from the side) so that the lower angle of the scapula could follow the curve of the ribs as it moves about them when the scapula proceeds forward and backward. (3) Twist a little so that the face of the glenoid can point a little more upward when the arm is elevated. (This occurs with No. 2.) (4) Slide a little inward when pressure is made on the outside of the shoulder. (5) Act like a hinge when the shoulder is shrugged.

All these motions are very slight in degree and checked by the ligaments which bind the coracoid to the under side of the clavicle, more than by the relatively weak capsular ligament which surrounds the acromio-clavicular joint itself. In other words, the range of the acromio-clavicular joint is only the range of the pliability of the coraco-clavicular ligaments themselves, which are very cunningly arranged to bear the stresses which occur in any of the directions in which the joint can move.

Owing to the obliquity of the joint itself, force coming from below the acromion (for instance, if the lower angle of the scapula were pulled forcibly outward or upward as by passively raising the arm in internal rotation in the coronal plane) would throw all the stress on the coracoid ligaments. In the same way, force from behind the acromion makes supporting contact in the joint because the clavicular surface faces backward as well as downward. These considerations are important in a study of the mechanism of dislocation.

The greatest weakness of this little joint seems to be to downward force applied to the acromion without touching the end of the clavicle which, however, protects it by projection above it. Fortunately, none of the ordinary uses of the joint subject it to a strong downward force, but a blow or a fall on the tip of the acromion *without at the same time striking the clavicle*, commonly ruptures the acromio-clavicular ligaments without tearing the firmer conoid and trapezoid strands.

On the whole, although the acromio-clavicular joint may move a little in any direction, and is necessary for perfect shoulder motion, it has a very slight range in any direction, and may be ankylosed without serious alteration of the use of the shoulder. It contains an intermediate fibro-cartilage which is often incomplete and sometimes absent.

The scapulo-humeral rhythm. There has been a great deal written in anatomic text books and journals, and also in articles concerning fractures and dislocations in this region, about the successive stages in the motion of abduction or elevation. I have never yet found what seemed to me an accurate description.* Authors divide the motions into primary and secondary. They say, for instance, that the humerus first moves on the scapula and then the scapula on the chest wall, and later the humerus on the scapula again. My own opinion is that in the normal motion of elevation no one can tell just

*Footnote. Since writing this chapter, I have found an admirable article by an English anatomist (R. L. Lockhart), from which the following paragraph is quoted:

"In raising the arm from the side to the vertically upright position, from the very beginning to the very end of the action, there is continuous movement of the scapulo-thoracic, acromio-clavicular and sterno-clavicular joints, and continuous activity of the associated muscles, whereas traditional teaching, by authors of repute, past and present, holds that abduction of the arm to the horizontal is secured by the full action of the deltoid causing complete movement of the shoulder joint, subsequent vertical elevation of the arm being made by trapezius and serratus rotating the scapula up on the chest, while the humero-scapular angle remains constant." (Movements of the Normal Shoulder Joint. R. L. Lockhart, M.D., *Jour. Anat.* (English), 1929-30, Vol. 64, p. 298.)

when each of these motions occurs, for they all occur simultaneously. This is the way to prove this statement.

Watch the back of a thin, naked man and concentrate your attention on the motion of the vertebral border of the scapula from start to finish of elevation, performed slowly. You will find with your finger on the vertebral border that you must follow the scapula continuously during the whole period of motion and that this motion does not occur in a jerky, but on the contrary, in a perfectly steady manner. Incidentally, therefore, the scapula cannot be performing its function in an independent way in relation to the shoulder joint. Next, concentrate your attention on the shoulder joint, with your fingers so placed that they can feel the motion of the head of the humerus on the scapula, and you will find that the motion of this joint, too, is symmetrical and steady throughout the whole of elevation. Still further, you will find that the motion is steady, whether accomplished from the start in internal rotation, or external rotation.

Now concentrate your attention on the sterno-clavicular joint. It, too, moves steadily through its special arc. The clavicle not only rises, but it passes backward and rotates, so that although its motion is oblique, it is steady throughout the whole period of elevation.

Then concentrate your attention on the level of the top of the shoulder relative to the other one that does not move. It, too, rises smoothly and continuously.

Therefore, Q. E. D., if each movement is steady and continuous, each joint is moving *pari passu*, although the arc of one, the scapulo-humeral joint, is much greater than that of the others and attracts your attention fully three times as much. After seeing many shoulder cases, one must conclude that this rhythm of motion as the arm is elevated is the normal action. When you see a scapula that halts and does not move symmetrically the probability is that that shoulder is abnormal. Almost all lesions of the shoulder joint and bursa exhibit this sign which I speak of as "loss of scapulo-humeral rhythm" in many places in the text. (See p. 147.)

At this point it seems wise to pause for a review. Most of the motions to which I have called attention are perhaps novel to the reader. You must try these motions for yourself and verify them or disprove them. Be fair to me. It is not only necessary to try them, but to try them accurately, and accuracy is very difficult, for in spite of good intentions you may make unconscious auxiliary motions. For instance, it is almost impossible to hold one's spine

rigid as one moves the arm into extreme positions. One must also be very careful to maintain the specified positions very precisely. In studying rotation, for example, one may allow the forearm to relax from its position at a right angle with the humerus, and without this guide, a very slight degree of rotation may occur which would permit greater freedom in elevation. Furthermore, it is next to impossible to disassociate movements of the scapula itself from the scapulo-humeral motions. In spite of one's will, the scapula will move a little on the chest wall and will give a deceptive freedom to the humerus. You think, for instance, that you can, with the humerus in internal rotation, perform abduction of the scapulo-humeral joint as far as the horizontal, but as a matter of fact, the degree of scapulo-humeral abduction will be very meager. If your scapula were nailed to your chest in the anatomic position, I doubt if your arm could move 45 degrees from your side.

To recapitulate, I believe it is important to understand that: (1) the humerus is in external rotation when the arm is at the side and the palm turned as far as possible forward and outward; (2) a considerable scapulo-humeral adduction (quadruped flexion) is possible on the part of the patient, so that the angle of the spine of the scapula and the axis of the humerus reduces some 20 degrees; (3) reaching as high as possible (elevation) does not mean the extreme degree of "extension" of the shoulder, for some degrees more may be gained by lowering the arm back of the head toward the other shoulder; (4) the young patient has so much greater motion in all directions than does the man or woman above middle age; (5) the sterno-clavicular joint functions more in shrugging than in elevation; (6) in elevation the clavicle moves upward and backward and rotates at the sterno-clavicular joint; (7) there is only a slight degree of motion at the acromio-clavicular joint, which is not very important (many times this joint becomes ankylosed in laboring men, without special pain or disability, and motion is not seriously interfered with); (8) a spiral motion is made by the humerus as it ascends, rotating and rising at the same time; (9) the motion of the hand at the wrist does not involve the use of the shoulder joint, and yet when the arm is straight out by the side, the humerus usually rotates without our perception; (10) in the normal subject the scapula, the humerus, and the sterno-clavicular joint move in a rhythm so beautifully coördinated, that they all perform a steady elevation of the arm at the same time; (11) with the forearm flexed to a right angle on the humerus and raised in the coronal plane in extreme internal

rotation, the humerus cannot rise quite to a right angle with the body, and that as soon as the forearm is thrown into external rotation, the humerus is in a position in which it can be elevated 90 degrees more; (12) trying to watch the three bones at once, one necessarily shifts his gaze, but in the meantime each point is still moving, and the scapulo-humeral motion, being three times as great in its arc as either of the others, attracts most of our attention, for the speed is also three times as great.

I think it is not generally appreciated that the shoulder joint is unique in so many ways. Compare its simplicity of construction with its most complicated muscular mechanism. It may be likened to the universal joint in an automobile so far as its function is concerned, but its anatomy is more simple; *i.e.*, it is a rounded knob, loosely fitting on a small, shallow, slippery fulcrum. It is dependent for its accuracy of motion in every direction on a group of muscles which must be absolutely coördinated and *always* work together to some extent, each in turn acting as stabilizers, and each becoming at times the dominant muscle which either the intellect or blind habit may order. Moreover, each muscle must be prepared to substitute for a neighbor in case of slight injuries or all together to go on strike until the injured one is healed. Each and all must be able to play their parts with the greatest speed under all conditions, and at all angles as the moving fulcrums change positions.

Have you ever tried to back an automobile with a trailer attached? The usual connection with a trailer is about as simple a mechanism as is the human shoulder joint. When you back your trailer your brains take the place of the neuro-muscular apparatus of the shoulder. You back perfectly straight and the trailer goes back straight for a moment, but instantly may swing to the right or left. You correct this by swinging the rear of your car in the opposite direction and find your trailer at once going too far in the other way. Now the mechanisms of the muscles of the shoulder are more alert than we are; they could back a trailer behind another trailer. Here is an illustration of the mechanical problem when you reach up and put your finger on the ceiling. Pressing your finger up is like backing an auto with eight trailers.

Through all these motions the subacromial bursa is in constant use. It is in fact a joint without articular cartilages but quite as indispensable. When its surfaces are thoroughly adherent, half the extent of elevation is lost and nearly 140 degrees in rotation and the same amount in dorsal and frontal flexion. Rupture of the supra-

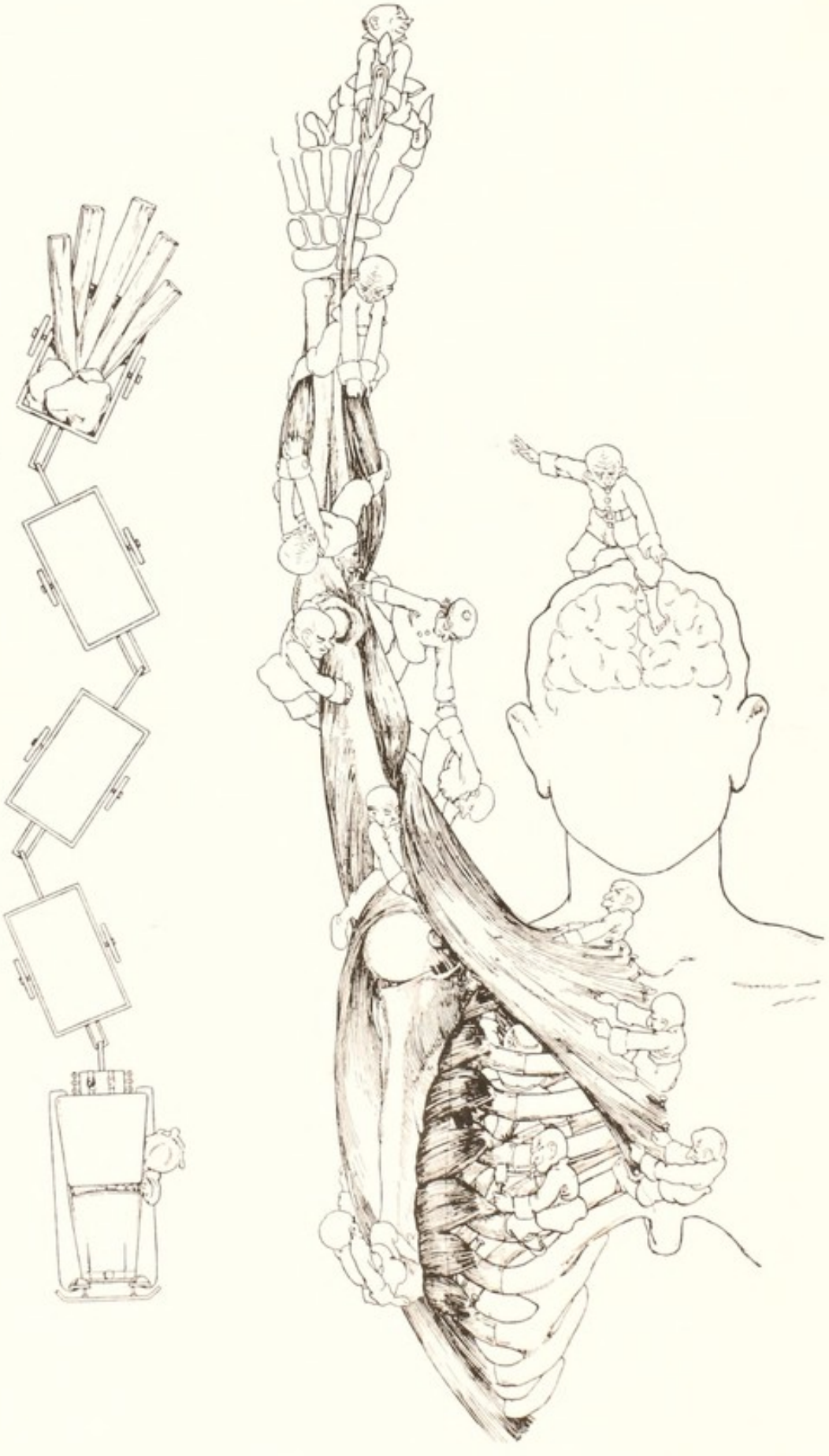


PLATE I. COÖPERATION OF ALL MUSCLES

spinatus tendon destroys the even convexity of its hemispheric base and wrecks the normal scapulo-humeral rhythm. Soreness at the insertion of this tendon or in the bursa produces spasmodic locking of the joint.

From what has already been said in this chapter, it is apparent that the shoulder motions can be divided pretty sharply into the motions of the scapula on the chest wall and the motions of the arm on the scapula. In the former set of motions, the subacromial bursa and the supraspinatus tendon are not brought into play, for they could be made with an ankylosed shoulder joint, but in the motions of the scapulo-humeral joint, constant use is made of the bursa.

There is frequent clinical opportunity to see how much motion the arm can perform when the shoulder joint is ankylosed by bony ankylosis or by fibrous adhesions. In such a joint the elbow cannot move above a horizontal line drawn through the shoulders, so that the hand can barely be applied to the forehead or nose. The hand cannot be applied closely to the abdomen, nor can it be carried outward much farther, if as far, as directly in front (in the sagittal plane) of the shoulder joint. It cannot be placed behind the back farther than the hip pocket of the same side. In other words, the motions which are restricted by the lack of use of the scapulo-humeral joint are chiefly those in which rotation of the humerus occurs. Elevation of the whole arm is limited by fully 90 degrees, but even though one cannot reach anywhere nearly as high when the shoulder joint is ankylosed, this particular motion is not missed very much if the other arm is well. It is the inability to reach objects to the right or left or behind the body that chiefly annoys the patient. In other words, if the true shoulder joint is ankylosed, man loses his purely human shoulder motions (adduction, abduction and circumduction)

PLATE I

A man can undoubtedly raise his arm until he has reached a point as high as seems to him possible, and then with an effort he can push directly upward against that point. We may compare the mechanics of this process with the automobile and trailers shown in this diagram. Even if a driver succeeded in backing these trailers as accurately as suggested, we cannot conceive of his getting them in a line sufficiently straightened to enable him to push backward on some other object. Yet man can, without effort or strain, perform this miracle by the alignment of the bones of his arm in such a manner that a further contraction of his muscles will straighten the line of his bones and transmit the force directly away from its base.

The right-hand figure suggests a comparison with the work of a rigger who is erecting a complicated arrangement of spars, which at the final moment he can raise to a vertical position by the unanimous coöperative efforts of the crew in charge of each portion of the structure. When we consider that in each muscle, the gnomes at work are thousands, each operating a special muscle fiber, it fills the mind with admiration of the accomplishments of evolution, but with humility with regard to man's ability to undertake to know in detail such a delicate mechanism. Have we even a right to attempt gross adjustments?

and is even worse off than if he had the extent of motion given to the quadruped, for he lacks the forward and backward motion which in animals would be extension and flexion. Fortunately he retains his human clavicular motions. The limitations due to complete adhesion or destruction of the bursa are practically the same as those due to destruction of the joint. (See Figs. 15 and 17, Chap. I.)

A surgeon who deals with shoulder cases cannot understand these points too well, because in the most serious forms of injuries about the shoulder, he must always have the choice between recommending fixation of the joint and an excision of the joint. Excision permits rotation below the level of the shoulder so that the patient can put his hand in the opposite pocket, in front and behind his body. In fact, a patient with excision of the shoulder can turn a door-knob behind his back better with the injured arm than he can with the well one. As compensation for this mobility in rotation, he lacks the power to raise weights in abduction which is possessed by the man whose shoulder is ankylosed, who can lift with great strength within the arc of motion which he still retains; *i.e.*, to a little higher than the level of his shoulder.

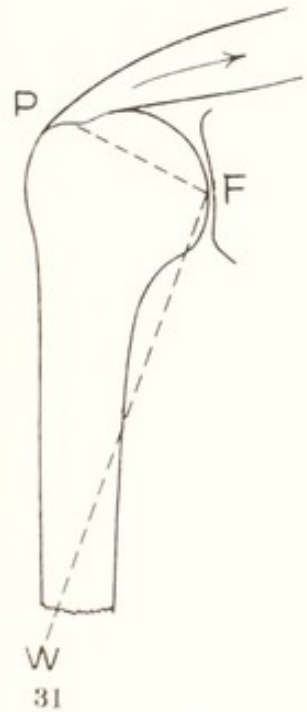
Unfortunately the clinical fact is that between these two extremes there is no halfway station. Patients with shoulder joints which are partly damaged are usually worse off than either patients with flexible excised joints or those with stiff ankylosed joints. Patients who have had excision or ankylosis soon come to a painless condition with the definite limitations spoken of above. On the contrary, if the scapulo-humeral joint is imperfect and works irregularly, it is apt to be more useless and troublesome than either the ankylosed or excised joint, because it is not only mechanically imperfect but painful, and pain creates spasm which destroys the normal scapulo-humeral rhythm and inhibits effort. Thus a joint with a few degrees of motion is usually worse than one without any. When a shoulder is injured in a way to destroy its wonderful mechanical perfection, it is apt to go from bad to worse, and while in the beginning it may have a fair degree of motion short of normal, it sooner or later tends toward ankylosis.

Prognosis in shoulder conditions is therefore very important. If one is able to recognize at once those cases which will eventually become ankylosed, and separate them from those which are sure to get well, it will be of great advantage to patients who might suffer through many months and years only to end with a stiff joint. When the soft structures of tendons or ligaments only (as in bursitis or

rupture of the tendons) are injured, permanent ankylosis will not occur and repair may be hoped for; but if the articular surface of the humerus itself is injured or destroyed by disease so that it becomes even partially adherent to the soft parts, practical ankylosis is almost sure to follow. It is for this reason that operative treatment of complicated fractures of the shoulder joint is so unsatisfactory, and has been a discouraging field for surgical endeavors. Such operations to be successful must be done immediately after the accident. Every surgeon should know well the respective advantages and disadvantages to be expected from excision or from ankylosis of this joint. In general, patients who must do heavy work with the arm are better off with ankylosis, while excision is preferable in those of the white collar class.

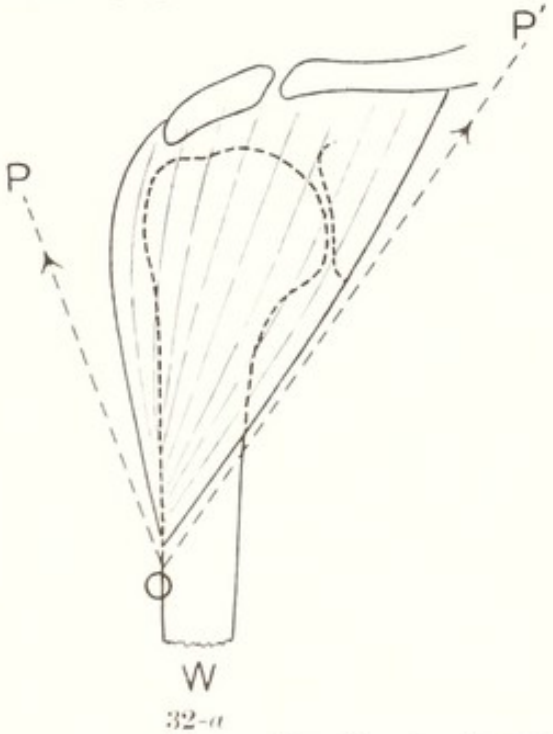
Fortunately ankylosis does not occur from rupture of the supraspinatus tendon, for the excess of fluid which develops in these cases prevents adhesions, and although the cartilage may be exposed to friction and be eroded, it does not become adherent.

We have hitherto been considering the motions of the skeletal part of the shoulder without attempting to determine the parts played by the individual muscles in more detail than given in the quotation from Gray on page 4. There is, however, a certain obligation for me to attempt to describe the special part played by the supraspinatus. For this purpose I present two diagrams with the accompanying text from one of my former papers.

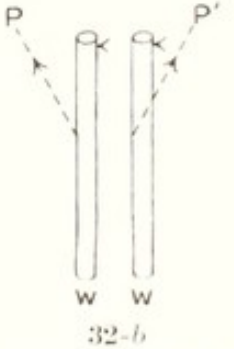


31

Figure 31—Showing how power is applied by supraspinatus.



32-*a*



32-*b*

Figure 32—Showing how power is applied by deltoid.

"In explaining this subject to students I find that many do not realize that during abduction (elevation) of the arm the greater tuberosity actually passes beneath the acromion and coraco-acromial ligament. They appear to think that the motion is limited by the impingement of the tuberosity on the acromion. I think this false conception is due to the usual method of articulating skeletons used for anatomic study, which does not allow the tuberosity to pass under the acromion. The diagrams (Figs. 31 and 32) illustrate the importance of the supraspinatus as an abductor. It is generally supposed that the deltoid is an abductor of the arm, but I believe it can be shown that the deltoid is not an abductor unless accompanied in its action by the supraspinatus. The deltoid alone tends to press the head of the humerus up under the acromion process.

A consideration of Figures 31 and 32 will demonstrate the importance of the function of the supraspinatus in abduction of the arm. In Figure 31 is shown how the supraspinatus applies its power on the short arm of the lever P. F. A mechanically perfect sliding fulcrum is obtained on the glenoid.

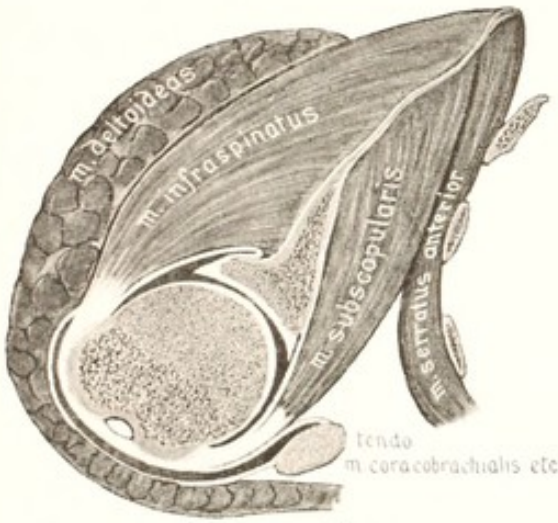
In Figure 32 is analyzed the application of the power of the deltoid to the point O. If the power were applied by the outer fibers O-P, a fulcrum could not be obtained on the glenoid but would be obtained on the acromion, which, since it is not provided with articular cartilage, is obviously unfitted for this function. On the other hand, if the power were applied by the inner fibers O-P′, the glenoid could only act as a fulcrum in adduction. The resultant of the action of all the fibers tends to drive the head of the bone directly upward under the acromion.

When, however, both muscles act simultaneously, the power applied by the supraspinatus furnishes a fulcrum for the power of the deltoid. (Fig. 17-*a*.) As the lines of force of the two muscles approach one another (Fig. 17-*b*) the deltoid is able to obtain a fulcrum on the glenoid. In normal action the glenoid bears all the weight, and the power of the supraspinatus keeps the tuberosity from seeking a fulcrum on the acromion and coraco-acromial ligament. The function of the bursa is to avoid friction at this point.

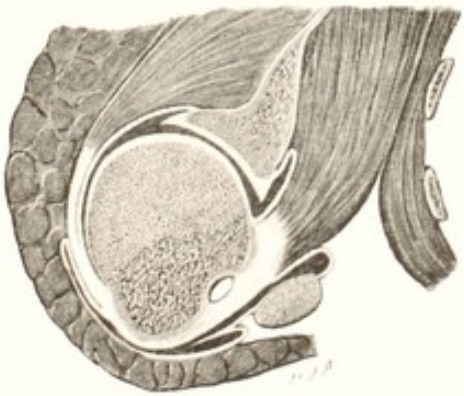
Soreness in the bursa, or of the tendinous insertion of the supraspinatus, throws the latter out of action and the deltoid alone cannot perform abduction. Duchenne has reported a case of circumflex paralysis where the supraspinatus alone was strong enough to perform abduction. A similar case is reported on p. 393.

The combined action of the two muscles is so habitual that one cannot voluntarily disassociate them in abduction, although one frequently uses the power of the deltoid alone to raise or square the shoulder, and *vice versa*, in lowering the shoulder to the position of round or sloping shoulder, one abducts the scapula on the humerus by using the supraspinatus. Even in the latter motion one cannot disassociate contraction of the deltoid."

As the years have passed I have seen no reason to withdraw any of the above statements, and I have seen many individual cases which confirm and illustrate the importance of these observations. I have seen a number of cases which confirm the observation of Duchenne, that the supraspinatus alone can perform abduction. This is referred to in Chapter XII, p. 393. On the other hand, every case of rupture of the supraspinatus confirms the remarks in regard to



a. EXTERNAL ROTATION



b. INTERNAL ROTATION

FIGURE 33. ROTATION IN THE SHOULDER JOINT

A semi-diagrammatic drawing by Mr. Aitkin from a frozen section taken transversely through a right shoulder joint. One is surprised to see how bulky the muscular mass of the shoulder seems from this point of view. One may also understand, from observation of the attachment of the serratus, how easy it is for the scapula to rock forward or backward; in other words, to rotate on its vertical axis. This rotation occurs so readily that it is difficult to determine the actual share which the scapulo-humeral motion performs when the humerus is rotated on its vertical axis in the anatomic position. In these diagrams the scapulo-humeral motion is indicated as about 90° between external rotation (*a*) and internal rotation (*b*), but these are probably not the extreme positions which the joint is capable of assuming. The range is probably nearly 180° in young subjects.

The diagrams illustrate the mobility of the peripheral parts of the bursæ and joint. The bursa subscapularis becomes part of the joint in external rotation and the bursa infraspinati unfolds in internal rotation. In a similar way the subcoracoid portion of the subacromial bursa rolls out in external rotation, while in internal rotation its loose periphery forms a nictitating fold as indicated here by a sharp angulation just external to the coracoid and which corresponds to the front view shown in Figure 49. These nictitating folds occur in other portions of the periphery as well, and are quite variable in position. When the bursa is blown up or injected they largely disappear.

the function of the supraspinatus in furnishing a fulcrum for the deltoid. All cases of rupture of the supraspinatus illustrate the tendency of the deltoid to lift the arm on its vertical axis.

While it is very interesting to speculate on the exact action of any one muscle or group of muscles related to the shoulder, one must constantly bear in mind that the actions of these muscles vary greatly according to the tension in the surrounding muscles. To study the action of the supraspinatus alone, without considering the combined actions of all the short rotators, would be short-sighted. Nor can we consider the action of all the short rotators together without taking into account the positions of the whole shoulder blade on the chest as maintained by the muscles which stabilize it on the body. J. H. Stevens (*Amer. Jour. Med. Sc.*, Dec., 1909. "The Action of the Short Rotators on the Normal Abduction of the Arm.") has discussed in a very interesting way the relation of the action of the supraspinatus to the other short rotators, so that it would be quite unnecessary for me to repeat here what he has to say, for I agree with him almost to the letter. He maintains that while the supraspinatus has the functions which I allude to above, the other short rotators have an important action also in holding the head of the humerus from below. In this I agree.

Stevens in the above article quotes me as holding that the supraspinatus initiates the motion of abduction. If I have expressed myself thus, I have changed my mind now, and as stated on page 52, I at present believe that the scapulo-humeral rhythm in elevation of the arm and shoulder together is uniform from first to last, all the muscles coöperating to some degree. It is probable that the reason that the motions of the shoulder have been misinterpreted so long, *i.e.*, that it is almost universally accepted that the humerus moves before the scapula, is a simple little point. When the model stands erect preliminary to the order to raise his arm so that the motions of the bones can be observed, his muscles are relatively relaxed. As soon as his mind obeys the order, all the muscles related to the scapula have to work in some degree in coöperation. The very first observable motion in the scapula is a slight movement of the inferior angle toward the vertebral column. Shortly after this, the inferior angle proceeds slowly and steadily in a curved line toward the axilla. I interpret the little preliminary motion toward the median line as an effort of the secondary shoulder muscles to fix the scapula so that the short rotators and muscles of the arm can begin to work. The phenomenon may be just as well interpreted in the reverse way; that

is, that the sudden assumption of scapulo-humeral action throws weight on the scapula and therefore tips the inferior angle toward the back bone. It does not seem important to decide which of these sets of muscles first begins to operate, but it is important to know that there is this little hesitation at the beginning of the motion, for I think that it is this brief period of preliminary stabilization of the whole shoulder which has led to the deception that the humerus begins to move on the scapula before the scapula begins to move on the body. At any rate, I feel quite certain that the supraspinatus must exert its power before the deltoid can act as an abductor, and that in a normal person abduction of the scapulo-humeral joint is due to a combined effort of the two muscles. As reiterated in other places in this book, I am also sure that elevation can be freely and haltingly performed, even when the supraspinatus has been evulsed, for the other short rotators can to a certain extent hold the head of the humerus on its fulcrum, the glenoid.

A few principles should be briefly emphasized before leaving the subject of the action of the muscles about the shoulder. In the first place many of the muscles in this region are fan-shaped, and therefore their fibers pull in greatly different directions. The trapezius, for instance, is really a compound muscle. Each bundle of fibers is almost a separate muscle and in fact the extreme ones are almost antagonistic. The lowest fibers pull the shoulder down and the uppermost pull the shoulder up. The deltoid itself is fan-shaped, with the edge of the fan curved. The subscapularis, infraspinatus, latissimus dorsi and pectoralis major and minor are also fan-shaped. All the handles of the fans point toward the head of the humerus. How extremely sensitive must be the coöperation between these varied muscles, all the parts of which must act together in almost any motion which we perform with our hands, for not only are the various nerves which we know as branches of the brachial plexus concerned, but the subdivisions of these nerves after they have proceeded to the individual bundles of muscular fibers. In raising the arm, for instance, a ripple of impulses must run through the bundles composing the trapezius, and similar ripples must take place through the fan-shaped origins of the latissimus dorsi, serratus, pectorals, etc. Relaxation of some fibers at appropriate moments is quite as important as stimulation of others. In fact, as one makes the simplest motion to raise the arm to touch a point on the wall with the finger, there must be a perfect symphony of coöperating relaxing nerve impulses harmonizing with contractions from various other groups of muscle

fibers, which, if analyzed in detail, would be more complex than the music written for a whole burst of harmony from an orchestra.

In studying the motions of the shoulder one must always bear in mind the serratus, the action of which is concealed. It is of great importance and is necessary to complete elevation of the arm. Remember that the majority of the animal world walk, run and leap on their serrati, for they have no clavicles to make skeletal contact.

I do not intend to attempt to review in detail the action of the other individual muscles about the shoulder. At best I could not equal the wonderful descriptions of their actions written by Duchenne in his "Physiologie des Movements," published in Paris in 1867 and remaining unsurpassed today. He was greatly interested in the study of the paralyses of these various muscles, and his analysis is much influenced by this point of view. More recently Mollier has published his lectures addressed to art students (S. Mollier, Plastische Anatomie, Verlag von J. F. Bergmann, München, 1924). I have been able to find no contribution of importance in the literature during the 60 years that intervened.

Curiously enough, neither of these writers shows any interest in the subacromial bursa, which I regard as an indispensable part of the human joint. They do not mention it. I have frankly given all the space I can spare on anatomy to my own point of view to bring out what I feel is the relative importance of the bursa, compared to the joint itself. I have tried to show that the destruction of either joint or bursa takes away not only the motions which quadrupeds possess, but most of those remarkable additional features of the mechanics of the joint which have come to us and to the monkeys to fulfill the need for abduction and adduction.

I have described in these two chapters some aspects of shoulder motion which I have not seen mentioned elsewhere, but Duchenne and Mollier have between them covered about everything else now known about this joint, including a great deal that I have not alluded to above.

We simply must close this chapter, for there is no end to the beautiful complexities of motion which this joint performs, but if the reader has not understood and agreed (or rationally disagreed) with what I have said here, he had better not finish the book.

REFERENCES

Acknowledgment has been made in the text to the works of Duchenne, Lockhart, Mollier, and Stevens.

Chapter III

THE PATHOLOGY OF THE SUBACROMIAL BURSA AND OF THE SUPRASPINATUS TENDON

I do not propose to review in detail the pathology of such general conditions as tuberculosis and syphilis, which, of course, may occur in this region, but merely to attempt to describe such pathologic changes as I have myself seen at operation in about two hundred explorations of the bursa on living patients. I have kept no record of the number of times I have studied the bursa in the cadaver, but I am sure I have opened over five hundred subacromial bursæ, perhaps a thousand. It is significant that most of the pathological changes which I have noted had not been previously described, at least, so far as this particular bursa is concerned; and, *per contra*, the conventional pathologic conditions, such as suppurative inflammation, "rheumatic changes," syphilis, tuberculosis, new growths, etc., have seldom been observed by me. In the shoulder these lesions are relatively rare. On the other hand, lesions peculiar to this region are so often present that I have observed some of them in probably one-third of all the bursæ I have examined in the dead, and, of course, in nearly all of the living cases. Fortunately, within the last few years, I have had the privilege of studying a much more systematic series of observations made by Dr. I. B. Akerson of the pathologic staff of the Harvard Medical School and pathologist to the Long Island Hospital of the Institutions Department of the City of Boston. We have published together in the *Annals of Surgery* for January, 1931, an article on "The Pathology Associated with Rupture of the Supraspinatus Tendon." The following quotation is from this paper:

"The present study has been made from autopsy material obtained at a large municipal hospital for chronic conditions. The patients sent to this hospital are people who are aged and down-and-out, and owing to the chronic character of their diseases, cannot be cared for at the other Boston hospitals. Dr. Akerson has made a study from one hundred specimens taken from fifty-two consecutive autopsies, and the percentage of cases showing evidence that the supraspinatus tendon had been ruptured at some time during the lives of these patients is high—39%. It may, therefore, be taken as a maximum, and it may be expected that pathologists performing routine autopsies

in general hospitals for acute diseases, where the ages average considerably younger, will find a decidedly smaller percentage. We have made no attempt to correlate the past histories of these patients as to trauma or occupation with the autopsy findings. It would have been hardly possible under the circumstances.

"As a routine method of examination of these cases, we recommend the pathologist to employ the form of incision which we use in operations on the living. A cut is made on the anterior aspect of the shoulder joint from the acromio-clavicular articulation downward for about two inches. The fibers of the deltoid are separated and retracted and the roof of the bursa is incised between two pairs of forceps as one opens the peritoneum. When the lips of the wound are retracted, the surface of the floor of the bursa can be made to pass in review by rotating the humerus. The base of the normal bursa is smooth and spherical and almost as colorless as the cartilaginous surface of the head of the bone, though it lacks the bluish luster of cartilage. Most rents in the tendons of the short rotators usually appear in this base as communications directly into the joint and are readily visible. Occasionally the tendon is ruptured beneath the base of the bursa, which is left intact. In the ordinary autopsy it would take but a few minutes' extra time to examine both bursæ. If lesions are found, the head of the bone with the insertions of the short rotators can be removed and studied. This was the method used by Dr. Akerson and his findings are good evidence that some sort of pathological process has been at work in the subacromial bursæ of these patients during their lifetime. One must understand that these findings are those accumulated by each individual subject in many years. They are end-results, not acute lesions."

Dr. Akerson has continued his observations and at the present date (January, 1933) has recorded his findings on 200 shoulder joints in 102 subjects. I am greatly indebted to him for the opportunity of studying this material and the many microscopic sections he has made from it.

Doctor Akerson's Findings in 200 Shoulder Joints

	First Series	Second Series of 100
Both shoulders examined	48	50
Single right shoulders	2	
Single left shoulders	2	
	100	100
Total autopsies	52	50
Males, 37; females, 15; total	52	
Ages: 46-59 . . 7 cases 60-79 . . 35 cases 80 and over . . 10 cases — Total	52	
Males, 37; females, 13; total		50
Ages: 51-60 . . 9 cases 61-70 . . 28 cases 71-80 . . 12 cases 80 and over . . 1 case — Total		50
Right shoulders, 50; left shoulders, 50; total	100	100
Rupture of Supraspinatus Found		
Right, 23 (46 per cent); Left, 16 (32 per cent); Average	39%	
Right, 17 (34 per cent); Left, 8 (16 per cent); Average		25%
Degree of Ruptures		
Large enough to show the articular surface on opening bursa	21	12
Smaller than this but still quite apparent	18	13
Total	39	25

The supraspinatus was ruptured either alone or in combination with adjoining tendons as follows:

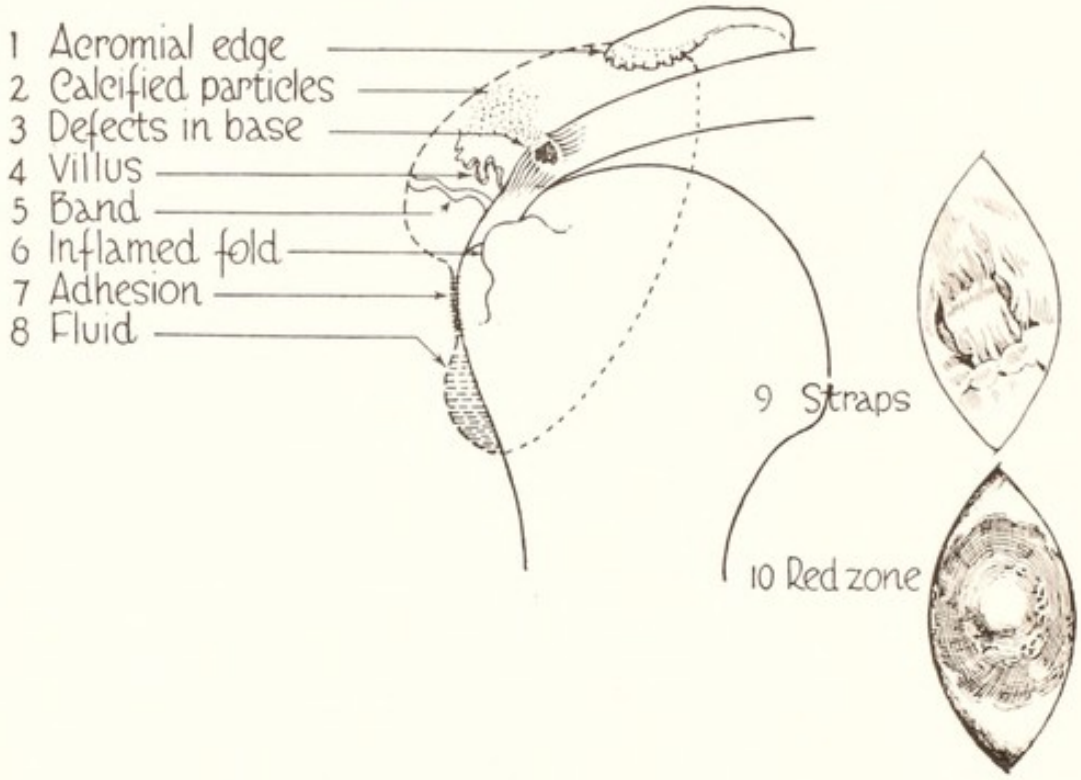
With portion of subscapularis	6	1
With portion of infraspinatus	10	4
With infraspinatus and teres minor	2	1
With all other short rotators (in same patient) (3 cases)	2	4
Condition of Biceps Tendon in 50 Cases		
Exposed to view in gap	1	1
Could be pulled into gap	2	—
Evulsed from glenoid	6	5*
Flattened	4	3
Frayed	5	1
Adherent to groove		3
Adherent to conjoined tendon		2
Total	18	15

It seems best to list under five headings the pathologic conditions which may be found at autopsy or at operations on the living:

Changes occurring: (A) In the bursa itself; (B) In the musculo-tendinous cuff itself; (C) In the tuberosities; (D) Within the joint; (E) When the musculo-tendinous cuff has been ruptured so that a free communication has been established between the joint and the subacromial bursa.

* In four of these cases the tendon was not found (*i.e.*, had retracted).

A. CHANGES WITHIN BURSA



Normally the incised roof of the bursa is as thin or thinner than the peritoneum, but when acute or chronic inflammation is present it may be as thick as blotting paper. It is apt to be especially firm in old, chronic cases of complete rupture of the cuff, when fluid is present. Normally it is transparent and film-like. In acute cases it may be opaque and reddish in color. In chronic cases it is whitish and firm.

(1) The roof of the bursa seldom shows any pathology, but in cases of long-standing bursitis from complete rupture of the supraspinatus, one frequently sees by X-ray, or one can palpate from within the bursa, *hypertrophic changes at the acromial edge*. Such changes may also be found in muscular laborers without symptoms.

(2) *Calcified material in the bursa.* Calcified deposits do not form in the bursa, but in the tendons. They may remain in the tendon substance for years, but the usual sequence of events is that they gradually work through to, or enlarge enough to reach, the base of the bursa, where they produce a mound-like swelling resembling a little boil, with a white center and a turgid zone about it. Up to this time there may have been no symptoms or very slight ones. Then some slight accident or unusual effort or even some customary action, as in turning over in bed, causes tension in the tendon resulting in rupture of a few superficial fibers, so that the little crater discharges into the bursa, where the calcified particles at once become diffused and may be readily delineated by the X-ray. An

acute bursitis is produced; fluid is secreted with which the calcified particles mingle; fibrin appears and the specks of calcium become entangled in it. I have several times operated at this stage within a few days of the perforation and removed from the bursa a delicate film of fibrin shaped like a watch glass. Grossly the film is whitish in color and the microscope shows calcified particles scattered throughout its substance like stars in the sky.

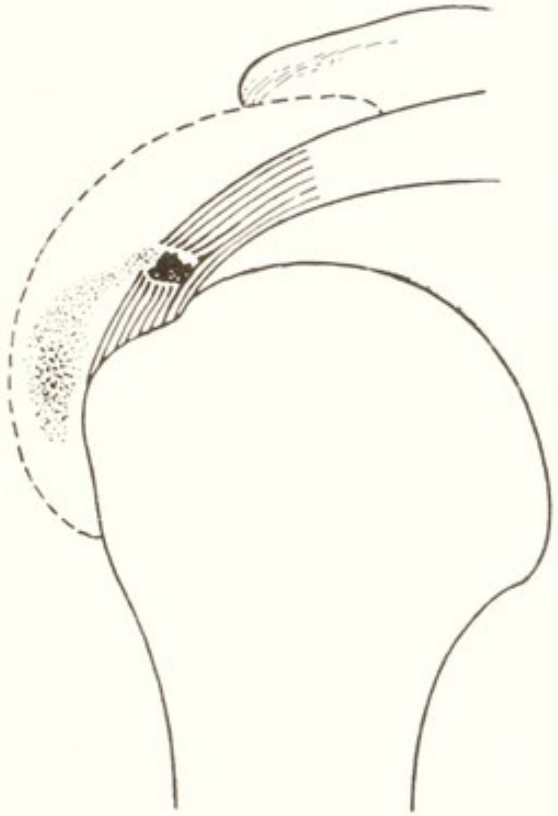


FIGURE 34. PERFORATION INTO BURSA

Diagrammatic illustration of a calcified deposit bursting into the subacromial bursa. This should be compared with Fig. 4 in Plate II, which was taken from the case alluded to below.

Soon after the material is thus forced into the bursa and becomes enmeshed in fibrin, the particles are rapidly eliminated, probably by the prompt action of leucocytes. This is not the case while they still remain in the tendon. When the operation on these cases was within a few days of the perforation, I have been able to readily remove the film of fibrin almost intact, but in one case, on the sixth day after perforation, I found the film so firmly adherent that no attempt was made to remove it, because I should have had to use a curette and feared that I might destroy the membrane and get obstinate adhesions in its place. Through the kindness of Dr. H. F. R. Watts, who followed the patient with weekly X-ray pictures, I found that absorption of all the calcified material took place within three weeks of the perforation. The patient was then free from symptoms. I was encouraged in making this decision in this case by the fact that I had previously had a number of cases in which rapid

disappearance of the particles had taken place without any operation. I now feel that perforation is nature's method of getting rid of this condition and that the average case, where perforation into the bursa has occurred, if left entirely to nature without any treatment, would follow this course. I think this fact accounts for the encouraging results sometimes obtained by all sorts of treatment, whether diathermy, foreign proteids, alpine lamp or surgery. This favorable course is not always the outcome, however. The inflammatory reaction may be too intense, and adhesions and consequent "frozen shoulder" result. It may be months before the adhesions loosen and full motion returns. Sometimes the whole deposit is not discharged into the bursa and the residue may give trouble later.

Under this heading we have discussed only the bursitis caused by the perforation of the calcareous material into the bursa. The study of the deposits themselves will be taken up again under Section B.

(3) *Defects in the base of the bursa.* These defects are nearly always at the same critical point, almost in the center of the base, where the red zones, calcified deposits and "straps" occur. The description of these defects properly belongs under the next division of our subject, for they consist of lesions of the tendon itself, whether partial or complete. Should the reader undertake to verify my statements by observing the conditions found by incising the bursa in a series of autopsies, he will be surprised at first by the variety of appearances which tears in the tendons may cause. After he has seen the inside of fifty or a hundred bursæ, the secondary appearances due to the synovitis set up by the lesion will cease to distract him and his surprise will then consist in seeing how much alike the fundamental lesions are.

In other words, the superficial appearances of these defects vary with the extent of the rupture, the amount of joint cartilage exposed through the rent, the position of the lesions, the proliferation of the adjacent membrane, the extent of exposure of the biceps tendon, the degree of involvement of the adjacent tendons, the duration since the accident, the extent of formation of a falciform edge, the amount of recession or hypertrophy of the tuberosity, etc. Yet after sufficient experience, rupture of the tendon of the supraspinatus will be seen to be the basis of almost all the lesions. His chief doubt will be as to whether degeneration of the tendon always precedes rupture, although he will be convinced that rupture has certainly occurred whether the tendon was previously sound or senile in texture.

(4) *Synovial villi.* These are the most common evidence of pathology one encounters in the bursa itself. It is difficult to say that a given degree of roughening of the serosa is abnormal. Is callus on the palm pathologic? It may be that in the same way some of these villi represent overuse rather than inflammation. On opening the bursa one sees strands of filmy tissue crossing the space after the air has been let in. Sometimes instead of being attached at their ends to both floor and ceiling, one end will be free so that the capillary villous will drift about in the bursa. In such cases the whole floor of the bursa is apt to be velvety with lesser villi and thickened nictitating folds. The center of this process is usually over the attachment of the supraspinatus to the tuberosity. In fact, this point seems to be the *locus minoris resistentiæ* of this bursa. We seldom find lesions of any kind in which this point is not involved. It seems to be the place where there is the most mechanical stress. It is here that the supraspinatus is at the greatest disadvantage in overcoming inertia in starting the upward motion of the arm. The sudden contraction of the deltoid may jam this point on the acromion or coraco-acromial ligament if the supraspinatus is not on the *qui vive*. Here also would come the greatest friction in occupations requiring repeated elevation of the arm. At any rate, lesions of the bursa as a rule are found on the base at this point. I have recorded none on the roof except a general synovial thickening.

There is a vast difference in the appearance of these synovial villi in life and after death. In life they are often pink or red, but in the cadaver they are limp and colorless and do not readily attract notice. In my opinion their presence is evidence that there has been inflammation and adhesion of the bursal surfaces, followed by resolution of most of the adhesions. They are probably homologues of pleural bands and are remnants of organized exudate. They are very frequently found and are to be expected in the bursæ of the type of human being usually found in the dissecting room.

(5) *Bands.* In a few cases at operation I have found thick, cord-like bands of fibrous consistency similar to the bands one frequently finds in the olecranon or prepatellar bursa. One very typical case was in a man who had for years climbed electric light poles, usually carrying heavy weights as he did so. A very painful, chronic bursitis with loud friction sounds developed. Excision of the bands restored normal use of the arm. Such cord-like bands are relatively uncommon in this bursa although frequently found in the prepatellar bursa. They must not be confused with the film-like

synovial villi spoken of above, nor with the "straps" alluded to under No. 9.

(6) *The normal nictitating folds* formed by double layers of the movable periphery and described in Chapter I, page 27, may become inflamed and swollen. I have occasionally seen them bright red, resembling inflamed conjunctiva. They are sometimes the sole cause of a mild, chronic, non-adherent bursitis, giving intermittent, elusive symptoms such as twinges in certain motions.

(7) *Adhesions.* As stated previously, I have come to believe that the bursa, like the peritoneum, possesses the function of rapidly forming protective adhesions to confine inflammation, and also the ability to absorb them and thus restore, in whole or in part, the mobility of the adjacent tissues. For instance, the diaphanous folds which often partially separate the subacromial and subcoracoid portions of the bursa may, when inflammation arises in one portion, wall off the affected part after the manner of water-tight bulkheads in a modern steamer. It is not uncommon to find blotches of calcareous deposit, which have escaped into the bursa, thus localized.

In my experience adhesions never occur in the strictly subacromial portion of the bursa, and are most common in the subdeltoid portion and in the fold partially separating this from the subcoracoid portion. They are seldom very dense in any situation. They are always absorbed or rendered pliable in time by nature's own processes. Frozen shoulders from this cause invariably recover. I have probably relieved the minds of more patients by this assurance than by my best operative efforts on their bodies.

(8) *A slight amount of fluid in the bursa* may occur in any acute or chronic bursitis, but if on opening the bursa and abducting the arm, a large amount, *i.e.*, several drams spurt out, one may be quite confident that the supraspinatus is ruptured and that there is a free communication between joint and bursa. As in other joints where there is synovitis, we may assume that nature supplies this fluid to reduce friction.

(9) *Straps.* These are damaged tendon fibers. One sees just over the supraspinatus insertion a circular area about an inch in diameter, which seems to be worn as if from friction. It is ragged and frayed. On close inspection there is a thin strap of fibers running in the same direction as the supraspinatus fibers and really composed of tendon tissue. The strap is perhaps a half inch square, attached above and below to the supraspinatus and lying on it or in it, but separated from it by a space. It is usually separable into

a half dozen parallel strands. It can be lifted up except where it is attached at its two ends and perhaps by filmy material on its sides. It is very thin but quite firm. I have not been able to make out positively whether these lesions are caused by friction and consequent inflammation, or are remnants of the effect of the discharge of a calcified deposit in years gone by, or are the results of partial ruptures. If you abduct the arm this little strap will buckle up off the tendon on the top of the greater tuberosity like an inch worm. Section of such a case showed that the deeper parts of the tendon were a series of such "straps" on a smaller scale. The tendon seemed to be split in layers.

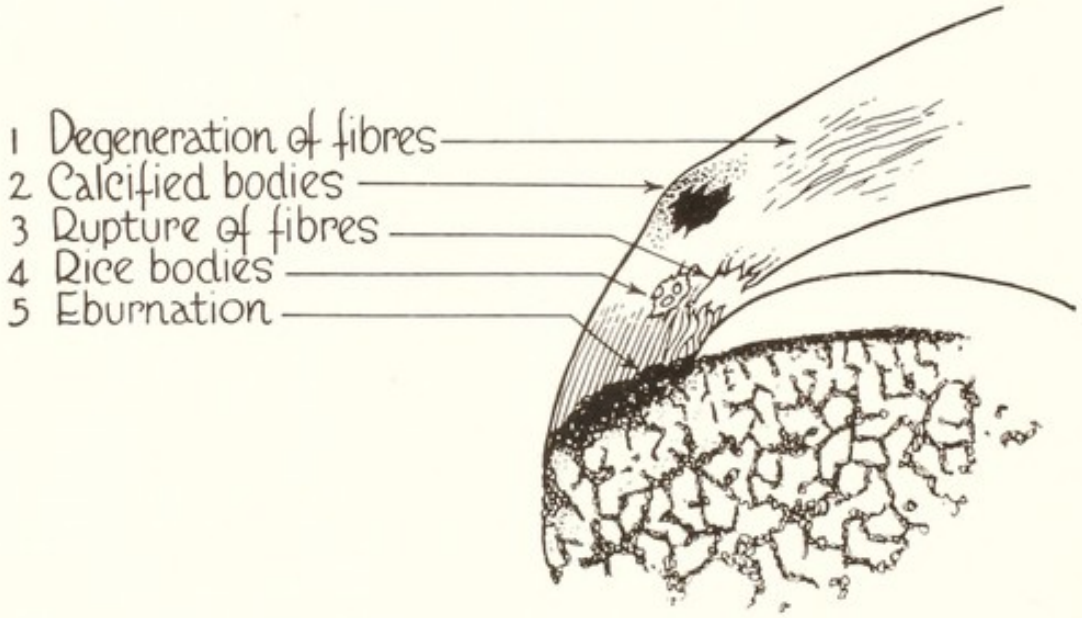
Dr. Akerson is studying a series of normal supraspinatus tendons at different ages and has kindly allowed me to have some of his sections. At all ages there appears to be a tendency for the tendon fibers to lie in loosely attached planes or laminations parallel to the joint surface as in Fig. 9. In the pathologic tendon these rifts or separations become complete and "straps" are formed. One receives the impression that normally these bands of tendon may each in turn take the maximum strain as the arm is elevated. Also that any one of them singly may tear or evulse from the denser portion of the attachment.

(10) *A deep red, more or less circular zone* of turgid membrane lying on the base of the bursa in the region of the supraspinatus facet is a not uncommon finding in the class of cases described on page 216 as tendinitis. Often this zone has a whitish central area which corresponds to the portion of the tendon just proximal to its insertion. The calcified deposits are usually surrounded by similar red, circular zones. Those referred to under this heading differ, because no white deposit is obtained when the whitish area is incised with the point of a knife, nor does any show in the X-ray. In one such case after puncture with the knife a few little rice-like grains of material popped out through the opening. Under the microscope the rice bodies appeared to be bits of necrotic tendon. In another case a little fluid came out under tension. In several other cases I have excised bits of tendon and found invariably the peculiar degenerated condition illustrated in Plate VI.

It will be more appropriate to consider the cause of this condition in the tendon under heading B. The point which I wish to make under this heading is that the red, circular zones are visible from the bursa in both these cases and in the calcareous cases. The deep red, circular zone seen on the base of the bursa is due to con-

gestion of the subsynovial vessels. It is very superficial and does not extend into the tendon, which itself is very anæmic. In fact, the gross appearance of the whitish center in both cases suggests there has been an infarction. The reader should refer in this connection to page 232.

B. CHANGES OCCURRING IN THE MUSCULO-TENDINOUS CUFF ITSELF



(1) Degeneration in the collagen of the tendinous fibers is so common that it is difficult to find in elderly subjects an example of normal tendinous substance. Even in the case which I have used as an illustration of the normal insertion of the supraspinatus (Fig. 9), a few of the fibers showed early changes. Fig. 5, in Plate VI, is taken from this case. It shows small areas of collagen which take a deep red stain with hematoxylin and eosin, and at first sight resemble muscle fibers.

In practically all the bits of tissue which I have taken from the tendons at operation or from the specimens Dr. Akerson has examined, these same changes are found in some degree, whether the specimens came from tissue adjacent to calcareous bodies, partial or complete ruptures, or from the non-calcareous cases which I am disposed to call "tendinitis."

Little pathologic work has been done on lesions of tendons in any part of the body, so there is not much in the literature to help us to interpret our findings. Indeed, there is little to say beyond the fact that hyaline degeneration occurs and is not accompanied by invasion of lymphocytes or leucocytes, or other usual inflamma-

tory signs. The degenerated areas vary greatly in their staining reactions, which may be as blue as cartilage or as red as muscle with hematoxylin and eosin.

I am convinced that some of the central fibers of a tendon may tear without those which are superficial to it on the bursal side, or beneath it on the joint side, being ruptured. In one case in the living, I opened up a little cavity in the tendon which contained fragments of degenerated or necrosed fibers. There was no apparent opening from the cavity which contained these little bodies, either toward the joint side or the bursal side. They were not calcified. It seems to me highly probable that the calcareous deposits represent nature's failure to heal such internal lesions in the tendon. However, one might take the opposite view that central degeneration first took place; then fragmentation of the degenerated areas; still later calcification of the fragments; finally, discharge into the bursa and, soon after, absorption as described on page 69.

We must at present confess that we do not know what the first step in the retrograde process is. It may be traumatic infarction, or perhaps due to a general toxic condition.

It stands to reason that there must be a stage in the course of the calcified lesions just prior to the time when X-ray evidence can be obtained, when necrotic tendon fibers would be found without any calcium in them. Perhaps some day we shall be able to diagnose such cases, and by simply puncturing the tendon remove the tension and obtain prompt relief. This is certainly possible now as soon as the calcium is dense enough to show in the X-ray. In such cases the little cavity contains only a milk-white fluid.

(2) *Calcified deposits.* These peculiar and interesting areas are probably the next most common lesions to villi, although in the dissecting room, partial ruptures of the supraspinatus will be found much more often. This is because the deposits are temporary and undergo natural absorption, leaving no evidence behind, unless perhaps defects in the tendons. Many partial and all complete ruptures, on the other hand, leave a permanent defect. In living patients the deposits are probably more common or at least more often diagnosed, because, when pronounced, they show in the X-ray. I am convinced, however, that many are small and escape notice, for I have seen some in very clear films which are not larger than a pinhead and others which cast but a faint shadow, easily missed if they had overlapped the contour of a bone. Many escape notice entirely and perhaps never give symptoms. Some are microscopic. (Plate VI.)

FIGURE 1 (Case 140)

FIGURE 2 (Case 140)

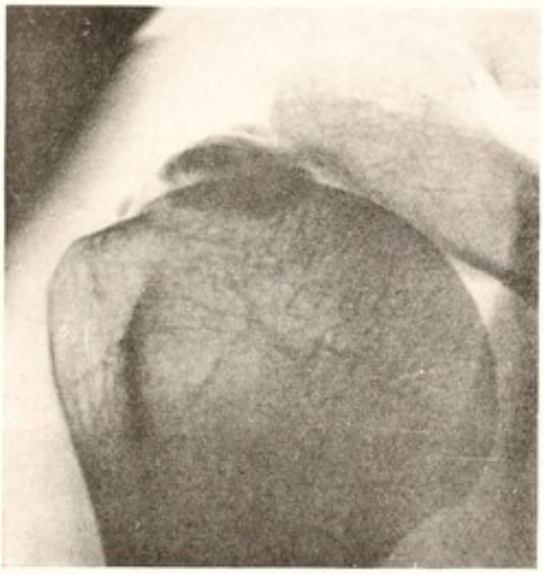


FIGURE 3 (Case 68)

FIGURE 4 (Case 139)

FIGURE 5

FIGURE 6 (Case 84)

PLATE II

PLATE II. CALCIFIED DEPOSITS

Figure 1. The deposit appears to be double. The larger mass lies in the supraspinatus tendon, while the smaller mass is in the portion of the infraspinatus tendon which lies, in part, anterior to it. (See Chapter I, Fig. 6.) The long diameter of the latter is probably perpendicular to the plane of the plate. The motions of this arm were free, and without symptoms, although the patient had acute symptoms in the other shoulder, shown in Fig. 2.

Figure 2. The shadow of the deposit consists of a diffuse portion and a small dense portion. The diffuse portion was due to calcified material in the supraspinatus tendon, which had not yet perforated into the bursa, although it was on the point of doing so at the time of operation. It had infiltrated the substance of the tendon and muscle far beneath the acromion. In both shoulders the shadow of the deposit corresponds very well with the normal position of the tendon. The dense portion was probably in the infraspinatus.

Figure 3. Shows a large, dense deposit in a patient with subacute symptoms, who still possessed a normal extent of motion in his shoulder joint, although he had acute pain during elevation when the mass impinged between the tuberosities and the acromion process. The calcified material had not yet escaped into the bursa, although the X-ray shows outlying specks of calcium in the superficial parts of the tendon. As is the case in most instances, the deposit was made up of small, dense masses. The tuberosity shows characteristic atrophy in the trabeculæ. At operation in both this case and in that shown in Fig. 2, the floor of the bursa showed a red zone with a white center. Contrast these three figures with Figures 4 and 5, which show the bursa distended with the material.

Figure 4. Should be compared with Fig. 34, for it shows a similar outline of the extruded particles. In some cases, *e.g.*, Fig. 44, the lower portion of the bursa appears to be bilocular, the particles having accumulated by gravity in the fluid at the bottom of these pockets. One should understand that in these figures we detect merely the outline of the bursa, as if in section, but that in reality the bursa lies also in the antero-posterior plane and forms a concavo-convex cap over the tuberosities.

Figure 5. As in the last case, one may be sure that the deposit has burst into the bursa if the bulk of the material shows external to the tuberosity in *the unrotated antero-posterior view.*

Figure 6. Illustrates a case alluded to in the text (p. 78) in which a second operation was done. There is a remnant of the deposit still left in the infraspinatus after the first operation, which was on the supraspinatus, but the long, oval deposit is in the subscapularis and was removed at the second operation. Compare this figure with another case where the deposit was in the subscapularis and is shown in the outline drawing, Fig. 43.

The deposits do not arise in the bursa itself, but in the tendons beneath it. Most instances occur in the supraspinatus tendon close to its insertion, but a considerable number are found in the tendon of the subscapularis and in the tendons of the infraspinatus and of the teres minor. I formerly did not identify them in these tendons. As will be seen later, one does not see these tendons in operating, and at first I contented myself with removing the deposit and making no particular effort to determine its exact situation. It took me some years to realize how easy it is to determine the situations of the tendons when operating by placing the arm in exactly the mid-position between external and internal rotation and palpating the bicipital groove. Since, after the bursa is opened, one usually has no difficulty in locating the deposit by the circle of blood-red congestion about its whitish or yellow center, it has seemed superfluous

to ascertain which tendon contained it. I later learned that there might be two or more deposits each in a different tendon. In one patient failure to remove both at the first operation resulted in a second attack of acute symptoms and a second operation. On page 230 the importance of accurate X-ray localization is emphasized.

The size of the deposit varies greatly, ranging from that of a pinhead to a shadow, two or three inches long, of the supraspinatus tendon and muscle as in Plate II. Such large shadows are very rare, and the deposit is seldom bigger than a lima bean. The consistency varies also and may be that of a milk-white fluid or of a hard, yellowish crystalline substance. Usually it closely resembles in color and consistency ordinary zinc ointment. Sometimes it is cheesy and yellowish, like the contents of a wen. In acute cases it is not difficult to remove, but in some chronic cases of long standing it is very gritty and seems incorporated in the fibers of the tendon so that one cannot curette it all out without removing much semi-normal tendon with it. One sees whitish, stringy fibers in the little pit from which it is being removed. Sometimes it shells out very easily with a definite contour. In my own operations I have avoided removing any tendon, preferring to leave some of the deposit in the fibers, than to excise tendon which might return to normal and become of use in future function. Brickner has removed enough for sections, and Moschcowitz has studied them. Harbin has not hesitated to remove some tendon and reports good results in spite of this. On a few occasions I have found the deposit surrounded by a small amount of granulation-like tissue, largely composed of foreign-body giant cells, but in these cases the deposit had probably perforated into the bursa.

Several times I have had careful chemical analyses made.

Dr. James L. Stoddard made for me an analysis of the material from one of these cases.

"The following report on the bursal deposit shows it to be a mixture of calcium phosphate, calcium oxalate, and organic matter, mostly fibrin.

Gross: A pasty, white mass, in part blood tinged, springy and a little elastic on stretching, moist, with an extremely fine, even texture, and with no evident structural elements which hinder division at any point.

Microscopic: Small ovoid colorless bodies, varying enormously in size. Some are at the limit of vision with an oil immersion lens, and show marked Brownian motion; all gradations exist up to bodies

several times the diameter of a red cell. They are all the same in qualitative appearance. They are slightly refractive, less so than fat, have usually a somewhat irregular oval shape, but the edge is always smooth and even. Nothing but the bodies is visible in the specimen.

Chemical—Qualitative: No color with Sudan 3; hence not fat.

Insoluble in acetic acid, alcohol or ether.

Soluble for the most part in fairly strong HCl.

On heating to yellow heat, chars, smokes, and burns briefly with a yellow flame, and gives a burnt feather odor. Leaves a gray ash.

Ash dissolves easily in weak acetic acid with evolution of CO_2, showing original substance to have contained oxalate.

Gives no murexide test for uric acid. No test for cholesterol (Lieberman-Burchard test).

Solution in strong KOH acidified with acetic acid gives off slight trace of H_2S, detected on lead acetate paper. This is probably due to presence of fibrin, which also explains the consistency of the deposit.

Solution of ash gives heavy phosphate test with ammonium molybdate.

Chemical—Quantitative: A small sample of the deposit was ashed, weighed (weight 86 mg.), dissolved in HCl, filtered from char, ammonium oxalate added, the solution neutralized, and the precipitate analyzed quantitatively for calcium as in the blood calcium method (Clark's modification of the Kramer-Tisdall method).

The filtrate was made acid with acetic acid, and a calcium acetate solution added to precipitate the excess oxalate which would interfere with the phosphate determination. The filtrate from this precipitation was analyzed for phosphate by the blood phosphate method (Brigg's modification of the Bell-Doisy method).

"Result: Calculated back to $Ca_3(PO_4)_2$ and Ca_2O_4, H_2O gives respectively: calcium phosphate 55.8%, and calcium oxalate 44.2% of the inorganic matter of the dried, unashed specimen. The amount of organic matter was not determined. Of the ash there was 14% not to be accounted for by figuring all the phosphate as calcium phosphate, and the remaining calcium as carbonate. Part or all of this 14% was due to char, and part may have been due to other inorganic matter (perhaps some magnesium salts, etc.). (The ashing was done at the lowest possible heat, but of course some of the calcium carbonate may have decomposed into oxide, which would raise

a little the undetermined fraction.) One of the most interesting features is the peculiar microscopic appearance of the specimen, with this non-crystalline precipitate of fairly definite shape."

Maxwell Harbin (*Arch. of Surg.*, Vol. 18, No. 4, P. 1491, Apr., 1929) reports a case which from the X-ray film is very clearly a case of perforation of one of these deposits into the bursa. He made a rather extensive excision and gives the following report of the chemical analysis of the specimen, together with a low power photomicrograph.

"The bursa, as well as the necrosed tendon (Fig. 8), was excised. It is of interest to note the considerable variety of earthy substances which exist in the necrosed tendon. Inorganic phosphates were present to a considerable degree. Calcium was easily discernible with a trace of calcium, iron, carbonates and chlorides. The specimen was composed of tough, white fibrous substance covered in certain areas by a cream-colored cheesy substance. A few grit-like particles were found in the cheesy material. Some hardened blood was present. Analysis of tissue removed from the supraspinatus tendon showed the following:

Fats	considerable
Cholesterol	positive test (small amount)
Cheesy Substance	
Xanthroproteic	+
(after extraction with ether, a white chalky substance remained).	
Biuret	+
Millons	+
Hopkins Cole	slight
Extracted with Warm Diluted Nitric Acid	
Inorganic phosphates	considerable
Chlorides	trace
Extracted with Warm Diluted Hydrochloric Acid	
Calcium	present
Calcified Deposit	
Magnesium	trace
Iron	trace
Carbonates	trace
Sulphates	none

"The white fibrous substance was apparently dense fibrous connective tissue. The yellow cheesy substance was composed of fat, protein and a fair amount of calcium phosphate." Comment by E. A. C.: Confessedly Dr. Harbin had excised some tendon and fat, and the analysis was not made from the pure deposit as in my case.

One of my assistants, Dr. T. W. Stevenson, made the following observations:

Study of a Calcareous Deposit

A little of the material was suspended in normal saline and observed under the microscope. Many small bodies of variable size, mostly round or oval in shape, were seen. The smallest were the size of bacteria or smaller and the larger ones were about the size of white blood cells. Scattered over the slide were many large, round or oval bodies 10-15 times the size of a white cell. They presented a peculiar appearance in that they were concentrically striated. The appearance resembled that of the gall stones pictured in MacCallum's Pathology. The center of these bodies is slightly clearer than the periphery. This central nucleus(?) resembles a white cell and is about that size. The concentric rings are very distinct, but two or three in each body are more pronounced than the others.

When a little of the material was suspended in concentrated sodium hydroxide solution, the bodies were turned slightly more yellowish. The striations were more marked. No dissolution or other change occurred up to one hour.

Concentrated nitric acid caused no change.

Glacial acetic acid caused no change.

Thin smears were made and fixed by heat. They were stained and the following results noted:

Gram's Stain: No organisms were seen. The calcareous bodies decolorized and were stained an even red with no striations visible.

Methylene blue stained the bodies an even blue and concealed the striations.

Ziehl-Neilson stain showed no acid fast organisms or bodies.

Stern of Cleveland has reported that these deposits are modified fat, but I can find no evidence to support his contentions.

Dr. Robert Chambers was kind enough to investigate some of the small bodies for me with his dissecting microscope. He found, when he attempted to pull them apart, that they were quite elastic and sprang back to their original shape.

I have repeatedly made microscopic examinations of fresh specimens of the material removed and they always consist chiefly of the little, round and ovoid bodies above described. I have never succeeded in dissolving them in ether. Their real character remains a mystery to me, but I am satisfied they are not fat.

Moschcowitz in 1915 said: "The calcification is present either in the form of discrete, sharply circumscribed, sand-like masses, embedded as it were in a cavity within the tendon; or as diffuse, finely granular masses, shading imperceptibly into the surrounding tissue structure; or as finely granular amorphous material, lying within the tendon or the granulation tissue." Concerning another case he said: "In no specimen was the slightest tissue reaction noted, in the form of round cell infiltration, granulation tissue or the formation of new blood vessels."

It seems to me that the latter statement is more characteristic of the early uninflamed stages in the tendon when the trouble is forming and the bursa is not yet involved. The first description is that of a more acute case when the deposit has caused a reaction in the base of the bursa. It seems to me probable that new blood vessels, etc., proceed into the tendon from the synovia. In such cases, unless the bursa is involved, it is striking that blood vessels, lymphocytes and the usual microscopic phenomena of inflammation are lacking, and yet there is clear evidence of degenerative change. I hazard the suggestion that it may be found that tendon repair differs from the repair of other tissues in this respect; *i.e.*, that necrosis must first proceed to a point where it involves an adjacent vascular tissue before vascularization will take place.

Moschcowitz did not definitely state his opinion on the question of precedence of lesion or trauma, for he was evidently influenced by Brickner's belief that this calcification process was a prompt reaction to a trauma. My own belief is that the deposit has probably been present long before the trauma with which it is associated by the patient.

Moschcowitz continues: "The necrotic tissue can have only two sources: (1) Degeneration of the blood and serum consequent upon the primary trauma. (2) The breaking down by coagulation necrosis of the tendinous structures." He excludes the first because there is no blood pigment; the tissue is too homogeneous and plastic in appearance; the line of continuity of the necrosed portions with the solid portions is a gradual one. He also states that it used to be generally accepted that calcification never occurs in any but dead

or inert tissue, but that in consequence of recent experiments, which he quotes, it seems that calcification may be a matter of only a few days in areas rendered anæmic by tying arteries. Yet he says it does not follow experimental suture of tendons.

It seems to me that we are indebted to Moschcowitz for the best statement of the pathologic findings, but that he has been too strongly influenced by Brickner's clinical belief that in these cases calcification is an immediate result of trauma. He also quotes me as believing this, but I do not recall that I ever did believe it, and I certainly did not state this in my writings. I did believe, and do still, that some slight injury, long before, may have pulled the fibers apart and started the process. Since tendons contain few blood vessels, evidence of hemorrhage is not required to support the likelihood of this theory.

There are two other interesting points in Moschcowitz' paper. One is that he was unable to find in the literature of pathology that any effort had been made to study the minute pathology of these lesions. He was only able to find Wright's report (on one of my cases, and which I had quoted) and another by Wrede, which was inconclusive. The second is that he prophesies that since in other tissues where one finds calcification one eventually finds some ossification, we should presently find ossification in an instance of this lesion. I have not yet been able to find such an instance proved microscopically. One must not be deceived by bits of the tuberosity in a retracted supraspinatus tendon.

Similar formations are sometimes found in other tendons, as in the quadriceps and the gluteus medius, and I know of one case in the flexor carpi ulnaris. Dr. J. H. Wright told me that a similar pathologic process was found in the heart valves.

The question of whether infection has anything to do with the condition has ceased to interest me, as I have always closed the wounds and have never seen even mild sepsis develop. Many times I took cultures which were always negative. Nor have I been able to definitely associate the condition with distant foci as evidence of toxæmia. Of course, many patients have bad teeth, but in no instance has there seemed to be any association, for as soon as the deposits are removed the patients promptly recover, whether the teeth have been extracted or not. In one instance extraction of a tooth was at once followed by acute symptoms in the shoulder, which soon subsided and disappeared altogether. However, I strongly recommend every patient with bad teeth to have them attended to,

although I do not consider the causative connection scientifically established.

There is no constitutional condition which I have associated with the lesions, for they occur in the lean or the fat, in the active or sedentary. My chief reason for believing that they are primarily due to trauma or overuse is that their pathology may best be accounted for on this hypothesis. Furthermore, I do associate them to some extent with occupation, for they occur in people who habitually use their arms in semi-abduction, as stenographers, accountants, machine operators and surgeons. I think that automobile driving may have a tendency to encourage this formation. In fact, any occupation or avocation which requires undue use of the abducted or elevated arm.

(3) *Defects due to ruptured supraspinatus tendons.* In spite of my known interest in the subject and the goodwill of many friends in active surgical practice, I have never seen, either on the operating or autopsy table, a fresh rupture of the supraspinatus tendon. It merely happens that no one recognizes these cases in time. I have seldom operated within three weeks after the injury, although I strongly believe in immediate operation. The earliest case was three days after the injury, and in this case, unfortunately, there had also been a prior one. Decided changes occur in the first few weeks, so that one finds the ends of the torn surfaces covered with fresh tissue which disguises the exact anatomic relations of the parts. However, experience in operating at varying intervals after the trauma, combined with dissections on the cadaver, has given me a fair idea of the method of healing which nature attempts. We must examine again the normal anatomy.

The continuity of the semi-circular conjoined insertion of the four muscles (see p. 17) is interrupted by the groove for the biceps tendon which separates the two tuberosities and theoretically the insertions of the subscapularis and supraspinatus. The facets of insertion of these two muscles are widely separated in anatomic diagrams, but as a matter of fact, their common expansion bridges the groove and makes them continuous, although thin just over the groove. Ruptures almost always involve this portion over the groove and it is very often the internal limit of the rupture, which as a rule extends from this point outward, involving the portions of the cuff made up of the supraspinatus and part of the infraspinatus expansions. Occasionally the tear crosses the bicipital groove and involves the subscapularis portion of the conjoined tendon. In at least one

dissecting room specimen the tear extended inward from the groove and involved the subscapularis portion alone.

When one opens the bursa in these cases the biceps tendon may or may not be exposed, according to whether or not the tear extends across above the bicipital groove. It is usually to be found just under the edge of the inner side of the rent. If it is exposed it is seen as a bright pink band crossing the bluish surface of the shining articular cartilage. It is not only pink and inflamed looking, but may be twice its natural size. One finds that below and above the point where the biceps tendon is exposed by the retraction of the ruptured supraspinatus, it is still of normal size and white and shiny. When not exposed, but lying just under the edge of the portion not ruptured, it is not abnormal looking. Frequently it cannot be found, for it has been torn off its attachment on the glenoid and has been pulled by its own muscle belly down into the bicipital groove. The proximal end sometimes sticks in the upper portion of the groove, and at other times retracts way down it and curls up in a coil just above the muscle belly. Occasionally it is found to be much frayed, which probably indicates repeated jamming between the head of the humerus and the acromion. Sometimes the frayed edges are adherent in the groove, and have little rice-like bodies attached to them.

From what I have said about the cuff of conjoined tendon in which the tendons of each muscle and the capsule are welded intimately, and since ruptures always occur in this portion, it will be more readily understood that when I speak of any one tendon as rupturing I mean the portion of the cuff represented by that tendon. Since the gap of the rupture is held on each of its edges by the conjoined tendon, the retraction of the muscle makes the rent triangular. The interweaving expansions of the tendon on each side still hold it to a certain extent, and the pull of the muscle is distributed on the conjoined tendon on each side as if it had a Y attachment. The middle of the inverted Y retracts farthest and the sides slope toward the base on the tuberosity. The shape of the whole rent is therefore roughly an equilateral triangle, with its base on the tuberosity and its apex disappearing under the acromion. The width of the tear is the width of the base; restoration would be made if the apex were sutured to the middle of the base. The base itself is inflexible, being either the bony seat on the tuberosity from which the tendon was evulsed or an inelastic stub of tendon still attached to the tuberosity. The size of this triangular rent varies from a half inch to an inch and a half base. Sometimes only a few fibers are torn, and at other times most of the conjoined tendon. In

one case I found that the whole cuff with all the tendons had been evulsed, and that the head of the humerus was free under the deltoid. (See case report on pp. 389, 390, 391.)

A skin wound of triangular shape healing by second intention, *i.e.*, granulation and epithelialization from the edges, becomes rounded before it heals; so do these triangular defects in the supraspinatus, so far as the edge of the rent is concerned. They differ from the skin defect because there is no bottom or bed of granulations, so must heal entirely from the periphery, and complete closure of the hole is therefore unlikely. In the skin ulcer this peripheral healing is epithelial and spreads over the surface of the granulations on the base as ice, beginning at the edges, skims over a pond. In the tendon defect the peripheral tissue is fibroblastic not epithelial, and fibroblastic reconstruction is dependent on a new formation of blood vessels instead of mere multiplication of cells to skim over another tissue which will feed them. Now it happens that tendon is a tissue in which there are exceedingly few blood vessels to form the basis of new granulations, so that the conditions for healing are still more unfavorable. I have already stated that it is my own belief that tendon repair does not usually take place from within, but from the synovial or areolar tissues adjacent to it. It is likely, therefore, that the synovial covering acts in these cases something like epithelium to cover the edges, and also as an agent to expedite the new supply of blood vessels. At any rate, I have had the opportunity to see on the operating table the appearance of these rents at various stages from a few days to two years after the accident. Even three weeks after the rupture, the triangular appearance is sometimes a little rounded by bridging of the corners and extension from the sides of an almost transparent new tissue, presenting a falciform edge toward the center. In later cases this edge is harder and more extensive, but always presents the falciform margin, which lies on the cartilaginous joint surface in the manner of the semilunar cartilages of the knee joint—clearly to avoid friction.

Obviously, as this circle gets smaller, either by the contraction of the tissue of which it is composed (cf. contraction of surface scars), or by more tissue forming toward the center, the smaller and more circular the defect becomes, until possibly the hole between the bursa and the joint might be entirely closed. Assuming that there is still a stub of tendon attached to the tuberosity, it seems possible that healing in this manner would eventually occur in young, well-vitalized tissues, but in the aged, with *barely vitalized* tendons, little change would take place after the first few weeks. This sup-

position is strengthened by reference to the age table, which shows that I have never operated on a young man and demonstrated a rupture of the tendon.

In those cases in which the tendon is actually evulsed from the facet, a complete circle with a falciform edge is not formed as healing progresses, for the bone forms a defect in the circle. The base of the former triangle is the bone of the tuberosity. It may be bare and as a rule has no granulations; sometimes there is a little velvety tissue on it. On microscopic section of the bare facet, one finds a little skim of fibrous tissue over the spongiosa, and that is all. As a rule there is a little plaque of dense bone beneath this, and at the periphery of the defect some raised "productive" bone. These bony changes are shown in the X-ray. Healing in these cases would appear to be at still more of a disadvantage than when a stub of tendon is left on the tuberosity. It is not unusual to find both, some stub and some bony defect. From the operator's point of view a stub of tendon is a great advantage, for where evulsion has occurred he must drill the tuberosity in order to suture the tendon. A knowledge that a falciform edge is present is also important for the operator, for in most cases he must remove it and pare down to the real tendon tissue before he sutures. The new tissue in this falciform edge is seldom strong enough to hold a suture, and would be in the way of the sound tissue. (Fig. 12.)

Referring to Fig. 6 will help the reader to understand the points at which rupture or evulsion is likely to take place. The figure shows that the insertion of the tendon into the bone is very superficial, and the shell of the surface of the bone is very thin. Under high magnification very many minute prolongations of the tendon are inserted into this thin shell of bone. (The structure suggests to my mind a room full of people hanging to the ceiling by having all their fingers deeply thrust into the plaster as into finger holes in a bowling ball.) As a practical matter this network of attachment to the tuberosity does hold against excessive pull except in elderly people when the attachment is pulled out. In most of the cases I have seen at operation it was apparently evulsed at the line of junction, but soon after the injury a stub of tendon would probably have been found. Such stubs probably slowly disappear.

Referring again to Fig. 9, it will be seen that stress on the supraspinatus tendon in the direction of its normal pull may result in four different forms of break in continuity:

a. The tuberosity, facet and all may be pulled away. This may occur without making a communication between the base of the

bursa and the joint. Since the fixed portion of the base of the bursa is on both tuberosity and tendon, it is likely that the whole bursa follows the displaced tuberosity, and the bony gap is filled in by callus from the peripheral torn periosteum. (See p. 317.) I am quite sure that this is the rule, although my opinion is formed on that very untrustworthy guide, "clinical experience." Cases of fracture of the tuberosity get well much more promptly and with less pain than do ruptures of the tendon, and differ in their minor symptoms from the latter. Moreover, in the two cases cited I have opened the bursa and felt beneath its unruptured base the fragment

FIGURE 35. RETRACTED FACET AND DROPPING SHOULDER

I have found this Röntgenogram, unlabeled among my papers, so that I cannot give, as I usually have in my other illustrations, a statement that the condition was proved by operation. However, the picture is so characteristic of a retracted supraspinatus facet that I do not hesitate to use it. Such a condition is far more serious than most of the major fractures (Fig. 59). Even without knowing the history in such a case I should be confident in saying, from the X-ray alone, that the patient has a post-dislocation condition, where there is "dropping shoulder" due to deltoid paralysis and where the supraspinatus facet of insertion has been pulled by the muscle beneath the acromion. It also shows some bone formation from shreds of periosteum retracted to a lesser degree by the adjacent tendons. As in cases 71 and 115, the dislocation has probably not been really reduced. These appearances indicate operation.

of tuberosity. In most fracture cases the bursa is not involved because it is higher than the usual line of fracture. (See p. 273.) It seems to me probable that in all cases where the tuberosity is involved, whether it is broken off alone or with several fragments, that the line of cleavage in the bone is just external to the firmly attached base of the bursa, which in fact strengthens the bone locally like a patch. In other words, I think that if one opened the bursa by my routine incision in a series of fresh cases of fracture of the tuberosity, he would *not* find the bursa full of blood and communicating directly with the two raw fractured surfaces and thus into the joint. Sometimes this would be true when the fragment as shown by the X-ray is drawn inward as far as in Fig. 35, but not in the usual case as in Plate IX.

b. Evulsion may occur at the point of insertion and the superficial part of the facet be carried inward by the retracted tendon. Since this little shell of bone would show very faintly in the X-ray, and also since it would have been absorbed in the long-standing cases which form the bulk of my experience, I cannot speak very positively about it. In a few cases I have seen remnants of it attached to the retracted end of the tendon, and I have often noted its absence on the tuberosity. In some cases it seems that the minute, finger-like processes are evulsed from the bone without taking the bone with them. Occasionally part of the tendon is evulsed, leaving bare bone, while the remaining portion leaves a small stub on the tuberosity.

c. True rupture of the tendon leaving a stub on the tuberosity usually occurs at the narrowest place shown in Fig. 6, leaving the broad, semi-cartilaginous fibro-cartilage still attached to the bone as a stub. This amount is barely sufficient to permit a good hold with a stitch. I have occasionally seen a larger stub, perhaps three-quarters of an inch in length. In these few cases this larger stub was very vascular and of deep purplish red color like a cock's comb. On one occasion such a stub popped out of the wound as soon as the roof of the bursa was incised. The vascularity of such a stub suggests that there must be a considerable blood supply in the bony facet. The desiccated bone shows many vascular channels in the sulcus, and near the upper end of the bicipital groove.

Both those cases in which there is evulsion and those in which a stub is left necessarily have a direct communication established between the joint and the bursa. In cases where bursa and joint communicate there is always more or less straw-colored fluid in the joint which washes back and forth into the bursa, rising in the bursa when an effort is made to raise the arm, and falling back into the joint as the arm is relaxed and lowered. This process dilates the bursa and in extreme cases leads to hygroma of the shoulder, as described on p. 478.

d. A fourth condition of great clinical importance consists of those cases in which a portion of the tendon is torn to a degree insufficient to tear the base of the bursa itself, so that a film of tissue is left between the joint and the bursa. I allude to these cases as "partial" ruptures or "rim rents." Repair takes place to a certain degree from the thickening of the film of bursal base. I have found few such cases in the living, probably because they do not cause a degree of disability to make me advise operation, but I have frequently seen this condition at autopsy. If one removes the head of the humerus with the short

rotators attached and examines the sulcus which surrounds the articular edge, one finds the attachment of the supraspinatus extremely thin, so that when held up to a bright light the attachment is found to be Y-shaped and the central portion between the limbs of the Y is diaphanous. The sulcus at this point is correspondingly broad instead of being a cleft, as it is in the normal joint (Fig. 10). It appears that the portion of the tendon on the joint side, together with its joint synovial lining, has pulled off, leaving the bone bare.

I believe this fourth division accounts for many of the industrial shoulder injuries which recover after a few weeks or months, and which I have in the past classified as relatively trivial cases of traumatic subacromial bursitis (see Plates III and IV, pp. 101 and 102).

It seems legitimate to assume that in such injuries as almost or quite cause a dislocation of the shoulder, the synovial reflexion and the portion of the tendon on which it is reflected, might be torn away from the articular edge, leaving the bone trabeculæ bare at this point. There would be slight bleeding, which might gradually fill the joint and distend it. This hemorrhage would in turn be replaced by an excess of synovial effusion, which might be kept up by the individual continuing to use the arm. Motion might prevent the formation of fibrin to act as a basis for new granulations and fibrosis in the bed of bone. Even if pain were sufficient to start scapulohumeral spasm, the effusion might hold the parts apart and prevent proper healing. If the patient were old, and the tissues already weak, healing would be even more unlikely. At any rate, it is very common to find at autopsy that the sulcus is bare.

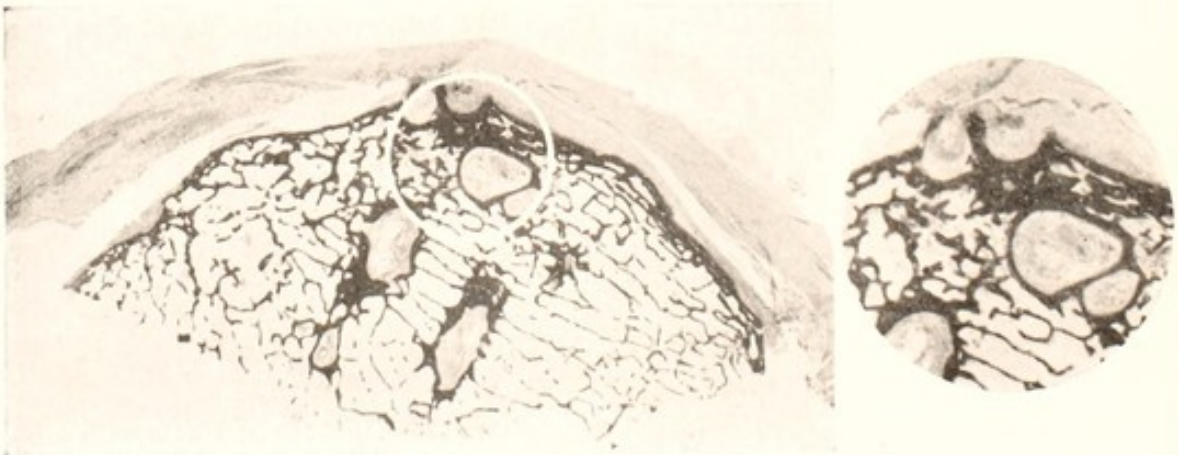
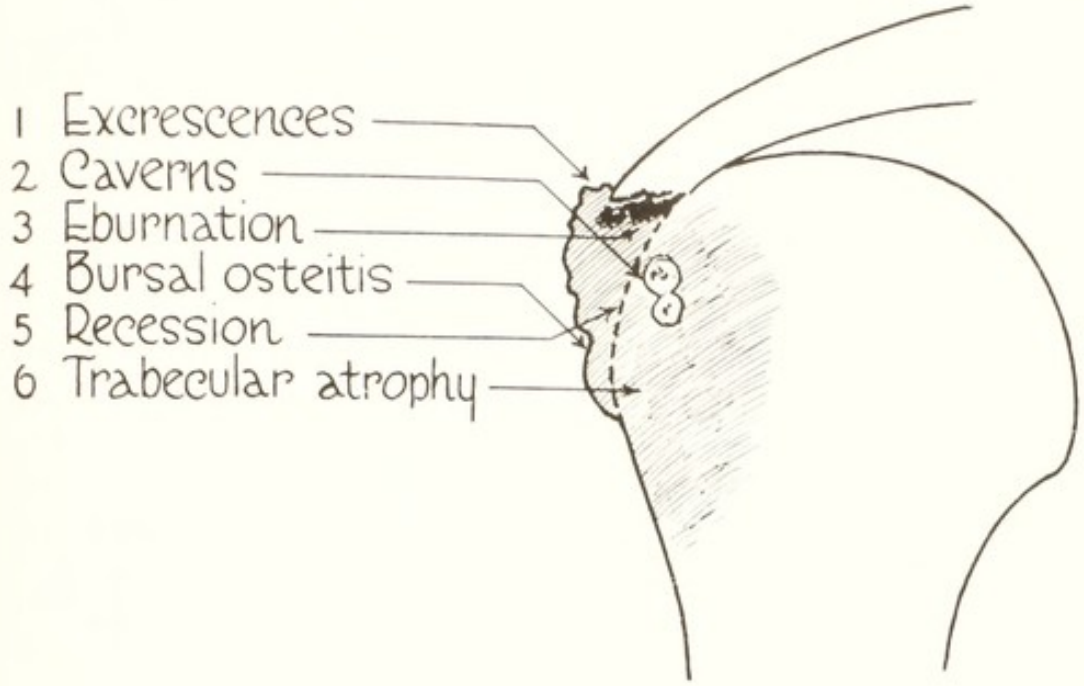


FIGURE 36. VOLCANOES AND CAVERNS

Deeper section through the same tuberosity as that shown in Plate V, Figure 1. The ends of the canals of invasion of vascular tissue appear as caverns. As these caverns and volcanoes are frequently found in X-ray films in cases of tendinitis, as well as in cases primarily due to rupture of the tendons, intensive study should be given to them.

C. CHANGES IN THE GREATER TUBEROSITY



(1) *Excrescences.* Pathologic changes in the tuberosity may readily be detected by the X-ray. In many cases of old, complete ruptures of the supraspinatus and in some cases of partial rupture, the relative thickness of the tendon and of the tuberosity is changed, so that there is a notch in the base of the bursa. Thus the tuberosity forms a distinct eminence and may cause a jog and friction as it passes upward under the acromion. The result is a stimulation of the bone cells in the tuberosity and irregular excrescences form at the region where undue friction occurs. This new formed bone is very spongy and probably later atrophies, because in the very old cases one finds that complete recession of the whole tuberosity has taken place. Atrophy of the tendon without actual rupture might cause enough irregularity in the base of the bursa to begin a little irritation. A vicious circle of atrophy of tendon, inflammation of tuberosity, osteitis, excrescences, greater jog and greater friction, greater atrophy, etc., might be established. At the same time hypertrophic changes on the acromial edge are stimulated.

(2) *Caverns.* Very frequently where this superficial osteitis occurs on the tuberosity, there is also absorption of the bone beneath its surface. This absorption of the bone results in the formation of little caverns which contain vascular tissue. I am inclined to think that they represent the early stages of what I call recession.

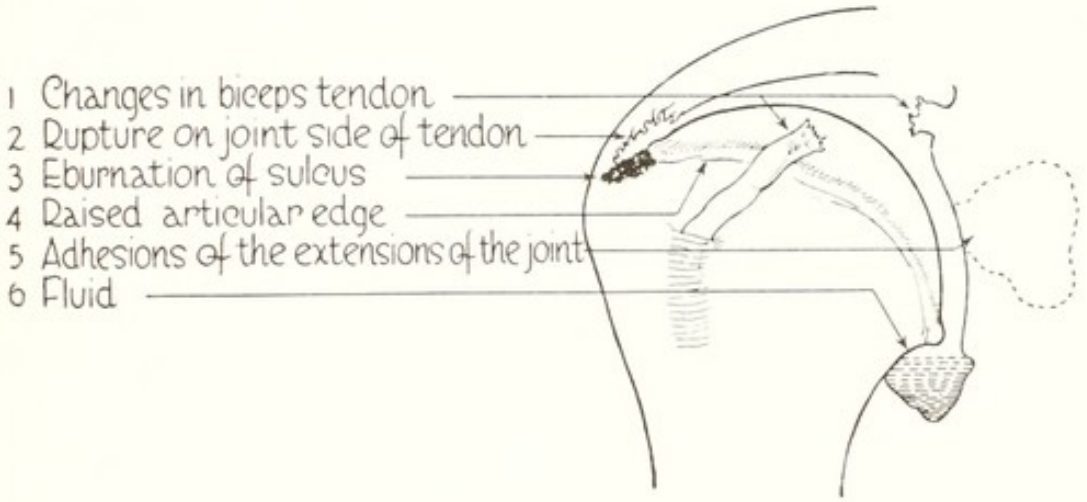
(3) *Eburnation.* By eburnation I mean an increased deposit of lime salts which result in the hardening of a definite area of the bone. Such areas of eburnation are frequently found at the insertions of these tendons in working men. In the X-ray a heavy plaque of bone is shown where the tendon is inserted in the sulcus. In some cases where the fibers of insertion have been damaged, there appears to be an increase of this bony deposit, so that when this is found in a case of suspected rupture, it is a confirmatory sign. It is not, however, of much importance practically. In my opinion, this appearance is evidence of the existence of low-grade, chronic inflammation, and I think that it is possible that the increase of this bony substance may interfere with the nutrition of the finger-like processes which hold the tendon in the bone at this point and tend to weaken the hold which these processes have there.

(4) *Bursal osteitis.* Osteitis caused by this irritation on the tuberosity may sometimes involve the outer side of the tuberosity as well as its tip. The base of the bursa may be the seat of chronic inflammation, so that irregularities will be shown by X-ray on the appropriate portion of the tuberosity, external and below the insertion of the tendon, on the outer aspect of the bone.

(5) *Recession.* I have referred above to the atrophic changes which take place in the sulcus and tuberosity long after ruptures of these tendons have occurred. It is highly probable that most ruptures occur at the line of junction of the tendon tissue with the palisade-like fibrocartilage adjacent to the bone shown in Fig. 9. When I have operated within a few weeks after the accident, this stub of tendon was usually present. In the very late cases, however, the tuberosity has usually been found to be quite bare, and not only bare, but atrophic. Instead of showing a normal sulcus and tuberosity, these structures are more or less leveled off, or, as I prefer to say, there has been a gradual recession of the tuberosity after the stub of tendon has worn away or been absorbed. The sulcus and tuberosity were no longer of use, but were actually an impediment to elevation of the arm. Without the pull of the tendon to fulfill its normal functional demand, the tuberosity had gradually disappeared. In the very old cases, such as many of those Dr. Akerson found at autopsy, or the case mentioned on page 108, the recession of the tuberosity was so marked that one could hardly tell, by superficial examination, where the old cartilaginous edge ended, or where the area normally occupied by the sulcus and tuberosity had been. This region now was smooth, covered with a thin

fibrous layer, and continued the rounded contour of the articular head. This recession of the tuberosity may be detected also by the X-ray. Note the dotted line in Diagram C and also Plate IV.

(6) *Trabecular atrophy.* In all long-standing lesions of the bursa, whether or not they are due to rupture of the tendons, the X-ray shows that the trabeculæ beneath the base of the bursa are more or less atrophied. This produces a dark area in the X-ray film which corresponds with the portion of the tuberosity on which the base of the bursa rests. In the normal bone the trabeculæ are, to be sure, less dense in this region than in other parts, but where the atrophy is pathologic, the outline of this more translucent area is indefinite and not as plainly marked as in the healthy bone. I presume that the trabeculæ are absorbed because there is chronic congestion of the small vessels of the marrow spaces in the region. This kind of atrophy is sometimes very marked in acute cases of bursitis due to calcified deposits. In such cases it slowly disappears after the lesions have healed.



D. CHANGES WITHIN THE JOINT ITSELF

(1) The common lesions of the long tendon of the biceps have been alluded to on p. 85.

(2) *Rupture on the joint side of the tendons (rim rents).* We have already considered under B, lesions of the tendons at this point, but since they do occur here, they necessarily involve the joint. Normally the true joint cartilage forms a very sharp angle with the insertions of the tendons, as shown in Fig. 10. Fig. 9 shows some of the fibers beginning to tear at this synovial reflection. Diagram D shows an extensive tear so that the rent has come through to the most super-

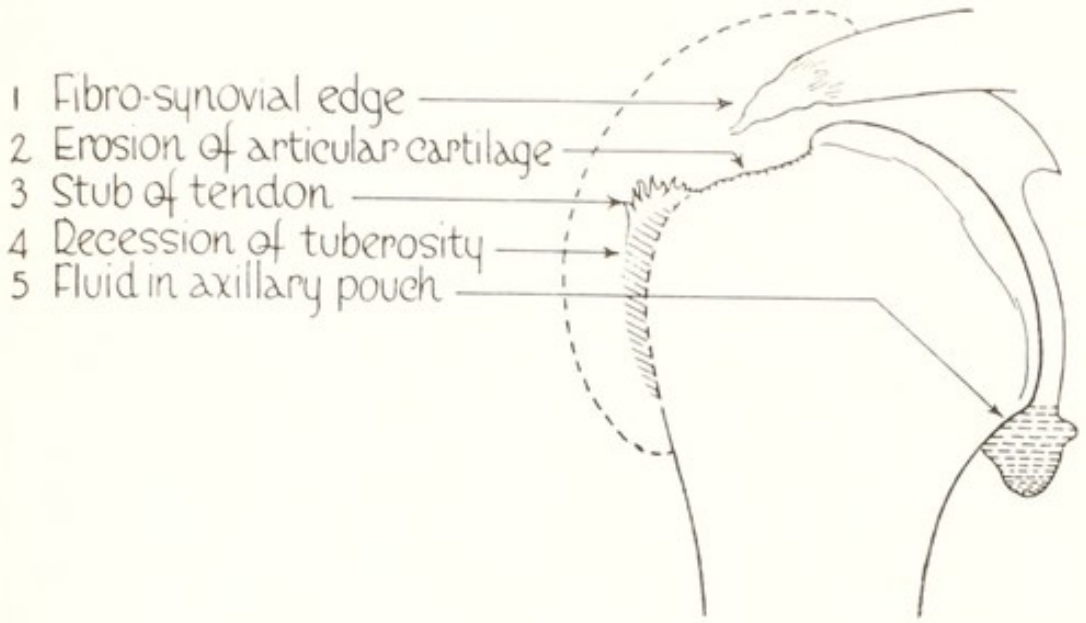
ficial fibers of the tendon. The reader should visualize this vertical section so as to understand that the rent also extends along the curve of the edge of the joint cartilage to a considerable extent, leaving the sulcus bare, perhaps for an inch or more. This condition I like to call a "rim rent," and I am confident that these rim rents account for the great majority of sore shoulders. It is my unproved opinion that many of these lesions never heal, although the symptoms caused by them usually disappear after a few months. Otherwise, how could we account for their frequent presence at autopsy?

(3) *Eburnation* of the sulcus has already been spoken of under the last heading, but it is unavoidably listed here again because it lies at the periphery of the joint.

(4) *Raised articular edge.* Not infrequently we find in elderly subjects, a very slight, rounded, corona-like elevation of the edge of bone under the articular cartilage. It is difficult to say whether it is really pathologic in character. It might be regarded as evidence of a chronic arthritis. However, it is frequently present as shown by X-ray, in joints of which the patient makes no complaint. So far I have been unable to attribute any clinical significance to it.

(5) *Adhesions of the extensions of the joint.* The reader will recall the so-called bursal extensions of the joint which lie beneath the infraspinatus and subscapularis. It is highly probable that the margins of these, especially the margins of the one beneath the subscapularis, may be stretched or torn so that habitual dislocation may readily occur. On the other hand, adhesions between their surfaces would necessarily limit the motions of the joint. I mention these extensions because some future investigator may be able to make some observations of clinical importance concerning them, but my personal experience with them is nil, for I have not even studied the question systematically on the cadaver. Albert (1893) mentions a case of tumor in the axilla caused by distention of the subscapularis bursa, but I have never observed such a case.

(6) *Fluid* in the shoulder joint is considered on page 115. The chief point which I wish to make in regard to it at present is, that in elevation of the arm the lower portion of the capsule is stretched tight and fluid is forced upward into the rim rents or, if there is a communication through the whole tendon, into the bursa itself. Theoretically, if there is fluid in the joint and the tendon is not ruptured, elevation of the arm should also tend to dilate the bursæ beneath the subscapularis and infraspinatus.



E. WHEN THE MUSCULO-TENDINOUS CUFF HAS BEEN RUPTURED SO THAT A FREE COMMUNICATION HAS BEEN ESTABLISHED BETWEEN THE JOINT AND THE SUBACROMIAL BURSA

The considerations under this heading are merely presented as a review of the others, with the particular idea of accentuating what I believe is nature's method of compensating for these lesions, *i.e.*, dilatation of the bursa by joint fluid, gradual absorption of the tuberosity and atrophy of the stub of tendon in order to form a new, smooth surface.

(1) Indicates the fibro-synovial edge which is formed very soon after the injury and which must be pared away before the tendon is sutured.

(2) *Erosion of articular cartilage.* It is obvious from what has been said that after rupture of the musculo-tendinous cuff, more or less articular cartilage will be exposed on the base of the bursa, and that the cartilage at this point would be subject to contact with the acromion when the arm is elevated. In old cases at operation or in specimens of extensive tears found at autopsy, one frequently sees a superficial erosion of the joint cartilage with clearly marked outlines separating it from the undamaged surface. These areas of erosion are consistently limited by the size of the rent. Perhaps it is better to say that these areas of erosion are always somewhat larger than the rent, because in different degrees of rotation of the humerus different parts of the articular surface can be exposed through the same rent to pressure on the acromion as the arm is

elevated. In practically all the cases where an extensive rupture exists, there is some osteitis on the edge of the acromion, which would increase the wear and tear made by the articular surface touching the acromion through the gap. This erosion of the joint surface is always superficial. It is possible that in some cases the process is reversed. It may be that the rough acromial edges wear through the tendon.

(3) Is the stub of tendon which remains on the tuberosity for some weeks or months after the injury, but since it is functionless, eventually disappears.

(4) Represents the dotted line which limits the recession of the tuberosity.

(5) Is the fluid in the dependent axillary portion of the capsule.

In the above description of the pathologic changes which I have observed in this region, I have endeavored to confine my remarks to the essential features which I am sure that any one who is earnestly studying these joints will be able to verify. Such a student may also find many other atypical little irregularities in the tissues close to the lesion in the tendon, which I have not thought it worth while to describe. However, when one has examined a considerable number of joints, these changes will seem relatively unimportant and entirely secondary to the lesions described above. One must not allow one's self to be deceived by these secondary changes, although sometimes the congested, inflamed synovial folds are the most striking lesions visible.

Before closing this chapter it is well to add a word about the question previously brought up as to whether some form of necrosis in the tendons themselves, such as age induces in the walls of arteries, is largely responsible for the various lesions or at least for their failure to repair. The facts that most of my cases are aged, many are bilateral, and that I have observed no cases in young people are strongly suggestive of this idea. Moreover, the traumatic cases as well as the calcified cases show a similar degeneration of the tendons under the microscope, although many of the latter are in somewhat younger people.

It seems possible that these lesions may occur merely from gradual deterioration of the musculo-tendinous cuff. The frequency with which they are found at autopsy would suggest this, as well as the reasons spoken of in the last paragraph. When a tendon has disintegrated to a great degree of weakness, even a slight effort might break away

the rest of it. However, this is not usually the case, for all but two of my operated cases of complete rupture of the supraspinatus gave a very clear history of serious trauma. Dr. Wilson's cases also had histories of trauma, with one exception. The findings at operation in the three exceptions were similar to those in definitely traumatic cases. Moreover, the results obtained by operation were excellent; in fact, both my cases were demonstrated at a medical meeting as perfect results. How can we account for this combination of a non-traumatic history with typical operative findings and excellent results? If the lesions were caused by a progressive disease we should not expect good results.

It happens that in both of my cases I strongly suspect that the injury had occurred when the patients were drunk. Since I believe that the usual cause of this injury is a sudden attempt at elevation of the arm to maintain balance during a fall, I think it is quite likely that such an injury may occur to a reeling drunkard. Therefore, I feel that before we say that these lesions are ever non-traumatic, careful inquiry should be made into the possible chance that the injury occurred while the patient was intoxicated. The early symptoms are not necessarily very marked, and would readily be masked in a case of a steady drinker.

Goldthwait has suggested that faulty posture underlies the weakness of the tendons. I am convinced that overuse of the arms in abduction without giving the tendons time to let their circulation do its duty, because they are perpetually stretched, has much to do with these lesions. I have no doubt that trauma *may* rupture a healthy tendon, but I think rupture from trauma occurs in the great majority of cases in aged tendons made weak by overuse, age, or toxic conditions. Dr. Akerson suggests that long-continued *disuse* might weaken aged tendons to such an extent that they would break under very slight strains and perhaps without much pain.

Whether or not injury precedes or follows degeneration in the tendons, it is a fact that complete rupture of the supraspinatus tendon may be diagnosed and even in late cases may sometimes be successfully repaired by surgery.

PATHOLOGIC HISTOLOGY

In studying the lesions that have been discussed, we should first have some idea of the normal microscopic landmarks which may become altered by injury or disease. The diagram (Fig. 37) should be compared with Fig. 9 in Chapter I, which shows an enlargement of

the normal insertion of the supraspinatus tendon in the region where pathologic changes usually take place.

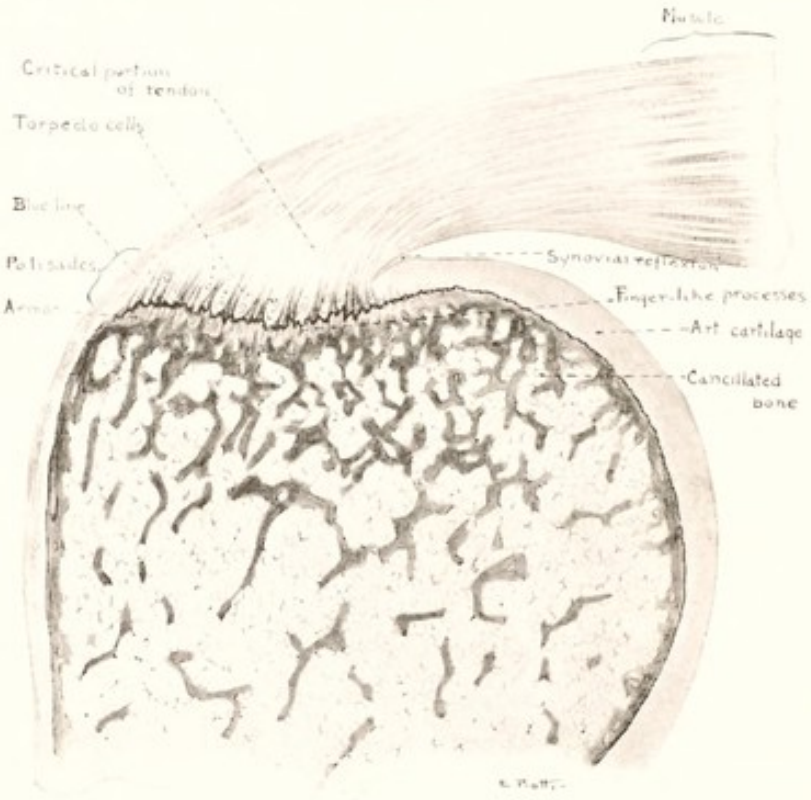


FIGURE 37. DIAGRAM OF MICROSCOPIC REGIONAL LANDMARKS

The relative sizes of the structures and cells have been altered in order to simplify the figure, which should be compared with a photograph of an actual section of this region (Fig. 9).

1. "The Critical Portion" of the tendon is the half inch proximal to the palisades. It is in this region that calcified deposits gather and where compiete rupture frequently occurs, although partial ruptures usually begin at the edge of the cartilage. As has been said, even under the microscope one cannot distinguish, in this part of the tendon, between joint membrane, capsule, tendon substance, and bursal floor, because they are here welded into one single structure. Nor is there a clear line of demarcation on the proximal side of the short tendon, where the muscle begins.

2. "The Torpedo Cells" are so called from their shape. They are probably merely modified cartilage cells which lie in the palisades with their long axes parallel to the columns. They closely resemble the cells of the adjoining articular cartilage, but their intercellular substance is more fibrous. They seem to function in making the columns, or palisades, and also in forming the blue line.

3. "The Palisades," *i.e.*, the portion of the tendon inserted in the tuberosity, is thus named because of its columnar fibers. In cases

of complete rupture of the tendon, the stub, which often remains on the tuberosity, represents this structure. It is fibro-cartilaginous in consistency and contains many "torpedo cells."

4. "The Blue Line." With the hematoxylin and eosin stain there is always, in adults, a blue line (of course, really a surface) where the palisades are inserted into the bone. The blue line is not present in infancy and even in a specimen from a girl of eighteen (Plate VI), it is not well developed; *i.e.*, the cells stain blue, but there is little deposit outside the cells as compared to the amount of blue non-cellular material one finds in older patients. The blue line is very close to the surface of the bone, but appears to be fenestrated for the insertion of the tendon.

5. "The Armor of the Sulcus" is the superficial layer of bone on the facets which we find in the dried specimen, and is really the cortical bone beneath the blue line. Like the trabeculæ it takes a pink stain. The armor in a normal tuberosity should be almost smooth on its surface; *i.e.*, in section it should give a definite regular outline. It is a surprisingly thin layer considering the stress it must bear; beneath it are marrow spaces and cancellated bone.

6. "The Synovial Reflection" is the angle between the edge of the articular cartilage and the palisades. It is described on pages 15-16, Chapter I, and is illustrated in Figs. 9 and 10. In dissecting room subjects this angle is seldom normal; there is almost always evidence of rupture of some of the columnar fibers of the palisades, as in Fig. 9.

7. "The Finger-like Processes" pass through the blue line, and form a singularly strong attachment to the bone beneath. These processes usually contain cells similar to the torpedo cells but apparently charged with calcium, for when close to the bone they take a deep blue stain.

8. "The Articular Cartilage" definitely begins at the very edge of the sulcus where the tendon is inserted. There is a sharp line between it and the "palisades"; *i.e.*, the synovial reflection.

9. "The Cancellated Bone," or Spongiosa, forms the bulk of the head of the humerus. The spaces usually contain fat and scattered marrow cells, and are not normally very vascular. Both the blue line and the armor are continuous with similar but less pronounced structures, beneath the articular cartilage, where there are no finger-like processes and where there appears to be a cleft between the blue line and the bone under the cartilage.

PLATES III AND IV

The cuts in Plates III and IV are made from direct photographs of some of Dr. Akerson's slides of old lesions found at autopsy. Those on the left are enlarged two diameters, and those on the right about four diameters in each case.

PLATE III

Figure 1 shows a beginning tear of the fibers at the synovial reflection, a little more extensive in degree than that shown in Fig. 9 in Chapter I.

Figure 2 shows a still more extensive rupture of the deep fibers, *i.e.*, a "rim rent." More than half of the tendon has been torn away, leaving portions of the columnar fibers (palisades) still attached to the tuberosity. The little hook which is in the center of the small circle is all that remains of the former synovial reflection. One small cavern is present in the tuberosity, which also shows evidence of slight excrescences.

Figure 3. A section from the same specimen as that shown in Plate V, Fig. 6, but taken near the edge of the gap, so that there is continuity of some of the superficial portion of the tendon with the external part of the palisades. It would be hard to explain this section or No. 4 by erosion from contact with the acromion process.

Figure 4. Another section from the same specimen which shows a partial rupture in the central portion of the tendon, and yet the fibers are continuous with the palisades, superficial to, and also beneath, the torn fibers. Most specimens show, when sectioned, this same irregularity in the extent of rupture in the different layers of the tendons. Compare the frontispiece, and imagine the degree of tearing in each layer which would be shown by a section at the left of the rupture.

PLATE IV

Figure 1. A very old complete rupture in which recession has taken place; no evidence of either sulcus or tuberosity left; the whole surface of the tuberosity has become rounded off to avoid friction. It is not enough to say by friction. The process of recession is a response to friction rather than a result of it. The fibrous layer which occupies the place of the tendon is not true tendon substance, but a portion of the roof of the bursa, the entire floor of which has disappeared.

Figure 2. A recessed tuberosity where no sign of sulcus or eminence remains. In the falciform edge of the torn tendon is a small oval speck of bone which formerly was the facet of insertion of the tendon. This is pretty definitely proven, not only by the gross appearance of the specimen, but by the fact that under high magnification one can still see evidence of the blue line on the periphery of the fragment. In this way nature manages to reduce friction even where a fragment of tuberosity is evulsed.

Figure 3. Section through an excrescence which has deformed the armor and the blue line in this region; yet under high power there are none of the usual evidences of inflammation, such as lymphocytes or increased fibrosis of the marrow spaces. The synovial reflection is relatively normal and so are the palisades, internal and external to the excrescence.

Figure 4. A rim rent where all the tendon is torn away from the sulcus except the superficial portion which extends into the periosteum. Partial recession has occurred. Some of the columnar fibers are still present in the deformed sulcus.

Figure 1

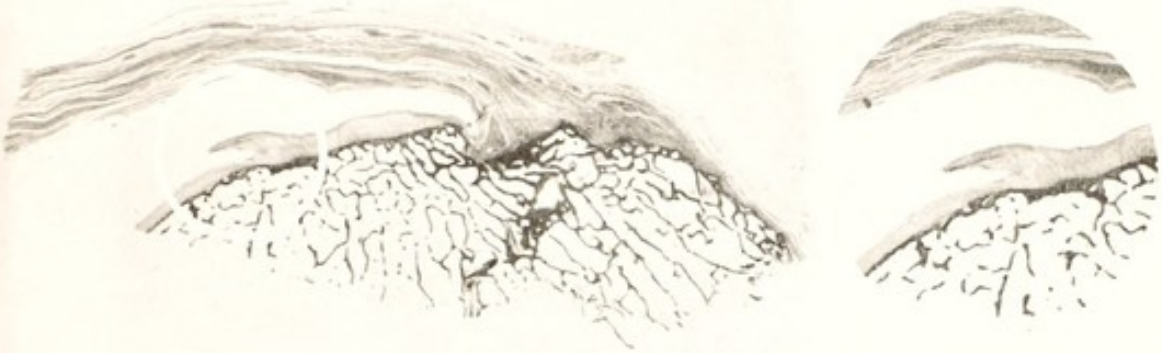


Figure 2

Figure 3

Figure 4

Plate III. Photomicrographs of Common Pathologic Findings

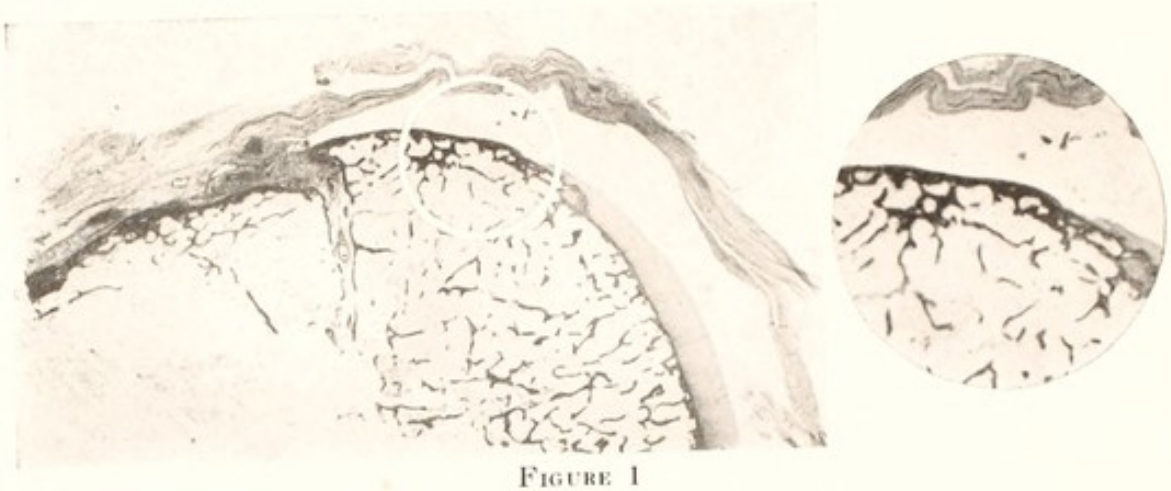


FIGURE 1

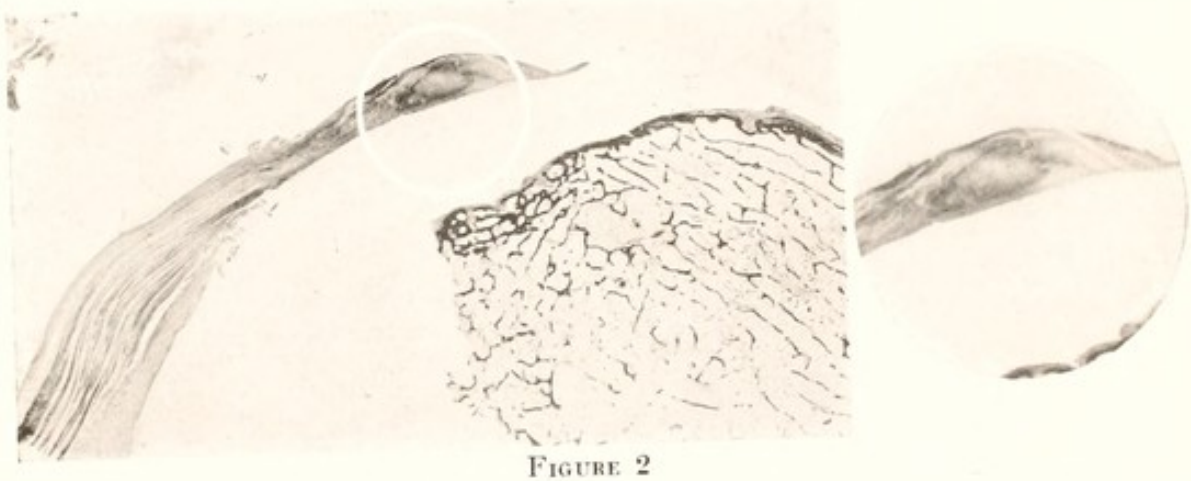


FIGURE 2

FIGURE 3

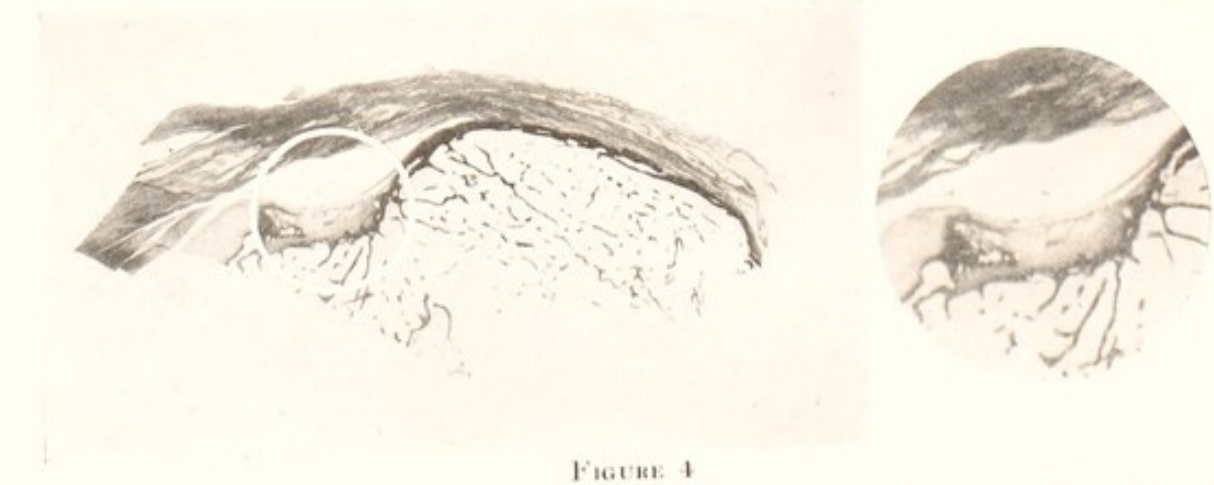


FIGURE 4

PLATE IV. PHOTOMICROGRAPHS ILLUSTRATING COMMON PATHOLOGIC FINDINGS

FIGURE 1

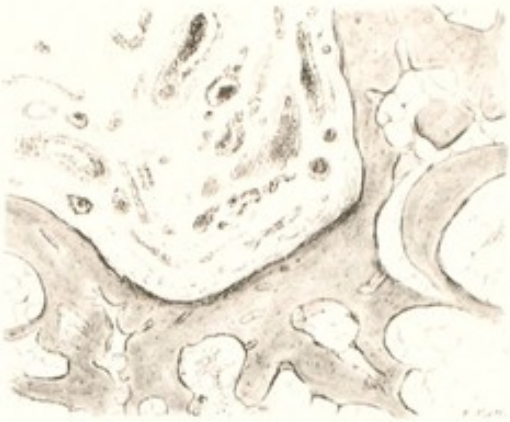


FIGURE 2

FIGURE 3

FIGURE 4

FIGURE 5

FIGURE 6

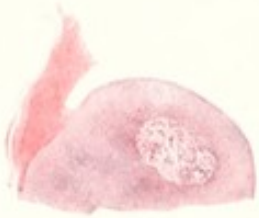
PLATE V. DRAWINGS OF COMMON PATHOLOGIC FINDINGS

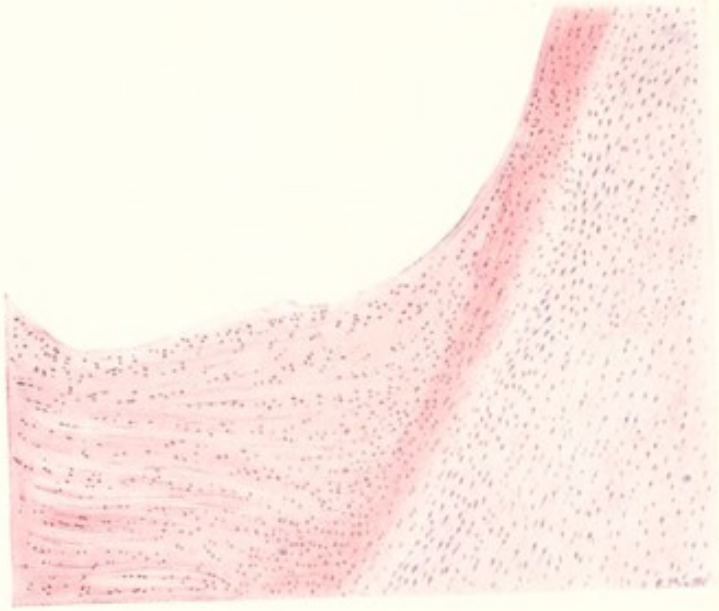
Figure 1. Section of the greater tuberosity in an old case of rupture of the supraspinatus. It shows the presence of "caverns" containing vascular tissue. The section is taken through an excrescence or "volcano." A section of the same specimen taken in a more transverse plane shows the caverns as rounded, cystlike spaces.

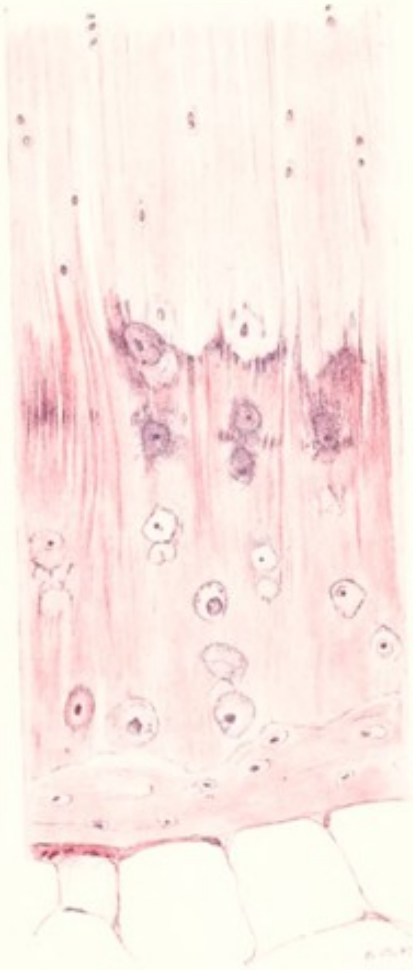
Figure 2. High power picture of the lower end of one of these caverns, indicating the vascularity of the tissue within.

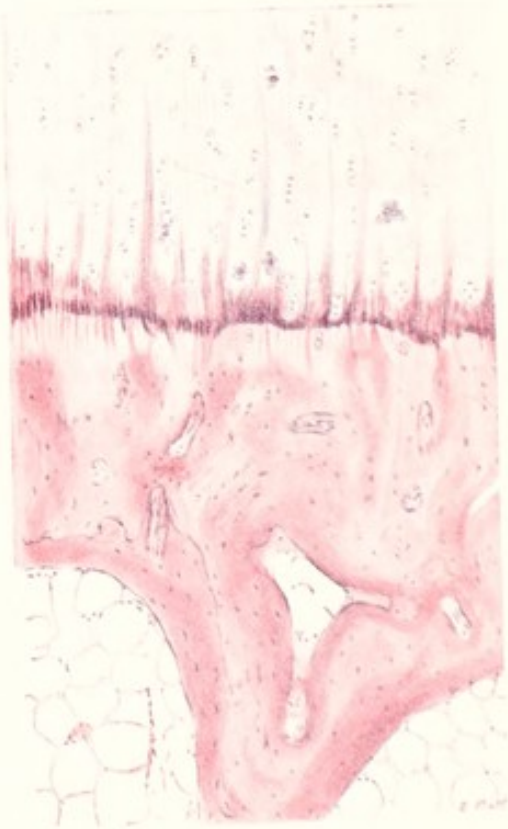
Figures 3, 4 and 5. Illustrations of a few of the varied appearances presented by villi found in chronic inflammation of the bursa. Fig. 3 shows fibrous, vascular and fatty changes, and Fig. 4 shows richly cellular areas. The section, Fig. 5, showing infiltration with polynuclears was taken from the case reported on page 247. There was no gross evidence of suppuration in this wound, but old silk stitches were causing irritation of the tissues.

Figure 6. Section through an old rupture of the supraspinatus tendon. The tuberosity is in process of recession and has become atrophied except for an excrescence at the outer margin. The blue line has almost disappeared. The armor is lacking because for years there has been no functional stimulus. Eventually such a tuberosity would become almost completely rounded off.









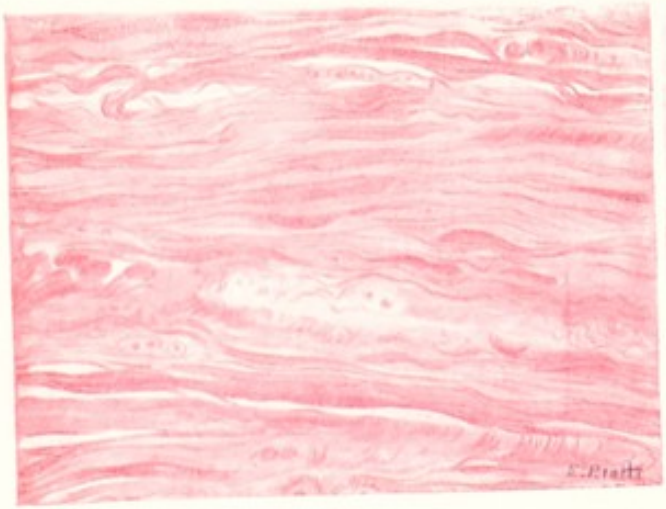


PLATE VI. HISTOLOGY OF TENDINOUS INSERTIONS

PLATE VI

1. Section through the attachment of the supraspinatus tendon of an infant. The center of ossification of the articular head is present, but those for the tuberosities have not yet appeared. The tendon takes a bright pink stain, while the cartilage, which is to form both the articular surface and the tuberosity, is purple.

2. Higher magnification of the "synovial reflection" in the same specimen. It shows the line which separates the tendon from the cartilage which is later to form the tuberosity. The coloration of the intercellular substance (*i.e.*, the acidity or alkalinity of the tissue), rather than the morphology of the cells, is the distinguishing feature. Sections of this region from children up to at least the age of twelve show no very definite blue line of demarcation between these two structures, but in sections from a girl of eighteen (Fig. 3) there is the suggestion that the blue line is in process of formation. It therefore probably begins to form somewhere between twelve and eighteen.

3. Section through the cortical (armor) bone of the tuberosities in a girl of eighteen. In this section the blue material appears to be chiefly in and about the cells (bone corpuscles?) in the armor. It suggests that these cells are instrumental in causing the blue deposit. They appear to leave it behind as a kind of shellac for the finished adult surface of the armor of the tuberosity. Whether they die when they have finished this work or whether they leave the deposit behind and proceed upward to become fibrocartilage "torpedo cells" is a matter for histologic research. The present theory is that they do not proceed up into the tendon. In this young girl, the bone below the blue line is rich in cells, but in adult life (Fig. 4) there are relatively few living torpedo cells in the armor. Dr. Wolbach tells me that the formation of this blue line resembles to some extent histologically the formation of enamel in a tooth.

4. This section shows what is usually found in an adult specimen. The blue line appears to be entirely extracellular and the fibrocartilage cells, which I have called "torpedo cells," in the palisades are very conspicuous. There often occur little masses of purple, crystalline collections, as indicated in this picture, just above the blue line. They are sometimes found even further up in the palisades. Their appearance suggests that they may be somewhat abnormal deposits, due to the failure of the cells to leave their blue staining material in the region of the surface of the bone. It is possible that a knowledge of their formation would throw light on the formation of the peculiar calcified deposits which occur in the tendon at a higher level. In both Figures 3 and 4, notice the finger-like processes which pass down (or it may be pass up?) through foramina in the blue line. Evulsion of these fibers may occur or a portion of the blue line may be retracted with the tendon.

5. A section taken from the supposedly normal adult tendon shown in Figure 9. On study with high power it shows deeply staining areas in the tendon substance which give evidence of abnormality of the collagen. It also shows abnormal changes in the torpedo cells.

6. Early changes in the character of the collagen of the fibrocartilage which composes the palisades. When the collagen is in this condition, the degenerative portions may be indicated by either a bluish or a pinkish stain with H and E.

We may now briefly take up the changes which are often found in these structures.

1. The critical portion of the tendon may show many shades of stain according to the degree of necrosis. It may contain large calcified areas or minute specks or merely necrosed fibers without any deposit. It may show rupture of all the fibers or of only those of the central or peripheral parts.

2. The torpedo cells undergo very interesting alterations. They may become calcified or enlarged into peculiar giant cells, or they may fade.

3. The palisades often show irregular staining qualities indicating necrotic changes; broken fibers, rice bodies, etc.

4. The blue line is a curious structure which is present at most points in the body where cartilage or tendon is attached to bone. It may become irregular on its surface or embossed by the formation of bony excrescences beneath it. It may be torn out in patches by the tendons.

5. The armor may become greatly thickened (eburnated) in laboring men from excessive use of the tendon. It may also become thickened from chronic inflammation in the tendon. Where the blue line is destroyed, the armor is thin; *e.g.*, after recession of the tuberosity it is usually quite thin, and the blue line has disappeared.

6. The synovial reflection is almost always changed to some degree as rim rents form. In most elderly patients we find at the angle of reflection that the columns of the palisades are more or less torn.

7. The finger-like processes may also show evidences of necrotic changes if staining reactions may be considered evidence. I strongly suspect that increase of the calcium deposits in the blue line may choke off these fibers so that the tendons may be more easily pulled out.

8. The articular cartilage may show thinning and superficial loss of substance but usually appears unchanged even to the very edge.

9. Vascular caverns may be formed in the spongiosa near the facets.

There are many other interesting and suggestive points about the pathologic histology of these lesions, but the above-mentioned changes may be readily confirmed by any one who has access to autopsy material. This is an uncultivated pathologic field except for Dr. Akerson's work. Correct interpretation of the common findings is greatly needed so that our clinical results may be improved. To my mind one of the most promising leads for the pathologist is that concerned with the extreme sensitiveness which the tendons show to hematoxylin

and eosin stains. Sometimes the necrotic portions are pink or red, and sometimes blue or purple. Notice the staining of the section from the infant's humerus. Why should the contrast be so pronounced? In the adult why is the bone red and the blue line blue? What phases of necrosis attract the blue or the red stain? Could these tissues be acidified by either local or general use of drugs? Could the pathologic process in tendinitis be checked by increasing or diminishing the acidity of the blood or in any way by altering its constituents?

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Brief references to the pathology of this region are also made in some of the papers listed at the end of Chapter VI.

Chapter IV

ARTHRITIS, PERIARTHRITIS, AND BURSITIS OF THE SHOULDER JOINT

In papers which will appear in future on the class of cases which I have made the chief subject of this book, the main criticism of my point of view may be that I have belittled "rheumatism" in the shoulder joint, and that the cases which I have called ruptures of the tendon, are merely the results of rheumatic joint disease, called "arthritis" nowadays. The findings which Dr. Akerson has published, and which can of course be duplicated wherever a similar class of material is found, may be used as demonstrating arthritis. There will be endless discussion as to whether disease in the tendon is the primary condition, or whether trauma, either chronic or acute, is the cause of the defects. I may say that years ago, when I first began opening these bursæ, I ignored many ruptures of the supraspinatus because I thought they were merely "rheumatic joints." As I grew more familiar with the appearance of the ruptures seen at operation at varying periods of time from weeks to months after the accident, I began to realize that such old lesions as depicted in Plate VII, which are found at autopsy, were merely the results of arthritis in consequence of a rupture, or a sequence of trauma-arthritis-trauma-arthritis, and so on. Thus my present belief is that trauma and arthritis play alternating parts, but that trauma is the chief cause of the damage.

The change in my views as time has gone on, may be illustrated by a description of an examination which I recently made at autopsy through the kindness of Dr. Wolbach, Pathologist at the Peter Bent Brigham Hospital. The subject was a man of 83, who entered the hospital moribund and could give no history. He was afflicted with an extreme grade of general arthritis, so that there was distortion of most of the joints in his body. I was permitted to examine the shoulder joints. The findings on both shoulders were essentially the same. I will describe the condition of one. On making the usual incision into the bursa, a little fluid escaped and the bursa itself was found to be extremely large, extending beyond the normal limits in every direction, so that the whole head of the bone, tuberosities and all, well down on the neck externally, formed the base of this large sac. There were no synovial folds between the three different portions, and the finger could be easily passed beneath the acromion as far

as the glenoid edge, and far under the coracoid process as well. The posterior limits extended well beyond the insertions of the infraspinatus and teres minor. The attachments of the supraspinatus and part of the infraspinatus had parted from the tuberosity, and an eroded joint surface was exposed in the gap thus made. Much of the greater tuberosity was rounded off. The amount of eroded cartilage corresponded to the portions which could be exposed through the gap. The tendons of the subscapularis and the teres minor were normal in appearance, but rather thin and feeble. The edges of the gap were falciform. The sulcus and eminence had disappeared, that is, were rounded off and covered by a thin, cartilage-like layer of fibrous tissue. The left shoulder joint showed a similar appearance, except that the degree of evulsion of the tendons was not quite as great. The appearance in both shoulders was very much like that in Fig. 8, Plate VII.

Years ago I would have simply said this is a case of arthritis, and therefore of no interest so far as the pathology of the bursa is concerned, but I feel quite the other way at present. My interpretation of the condition would now be that the patient suffered from some toxic disturbance which rendered his joint tissues and the adjacent tendons sub-normal, and even partially necrotic, so that slight traumata which would not affect a normal tendon, easily tore their fibers. Perhaps at first the change was merely degeneration of the structure of the tendon. Presently some slight extra effort, as in putting on a coat, may have torn a few of the fibers, creating some local soreness and disability, and starting up a mild inflammatory process in the adjacent membrane. The patient would have refrained from using the arm for a time, and still further atrophy and degeneration of the tissues would occur. Then another minor accident may have pulled out a few more fibers and more lameness have been experienced. Presently enough fibers may have been torn to allow the fluid of the joint to pass through into the bursa. Continued use of the arm would have produced a mild synovitis with an increase of fluid, and the fluid itself would have lubricated the tissues sufficiently so that friction under the acromion would have been allayed. However, each time the patient raised his arm the fluid would have been forced more into the bursa, so that eventually the bursa would dilate in all directions. As this process went on, more fibers of the tendon would be torn out and the weaker it became, the more of a vicious circle would be established, until eventually it arrived at the condition in which I found it at my examination. Meantime, the portion

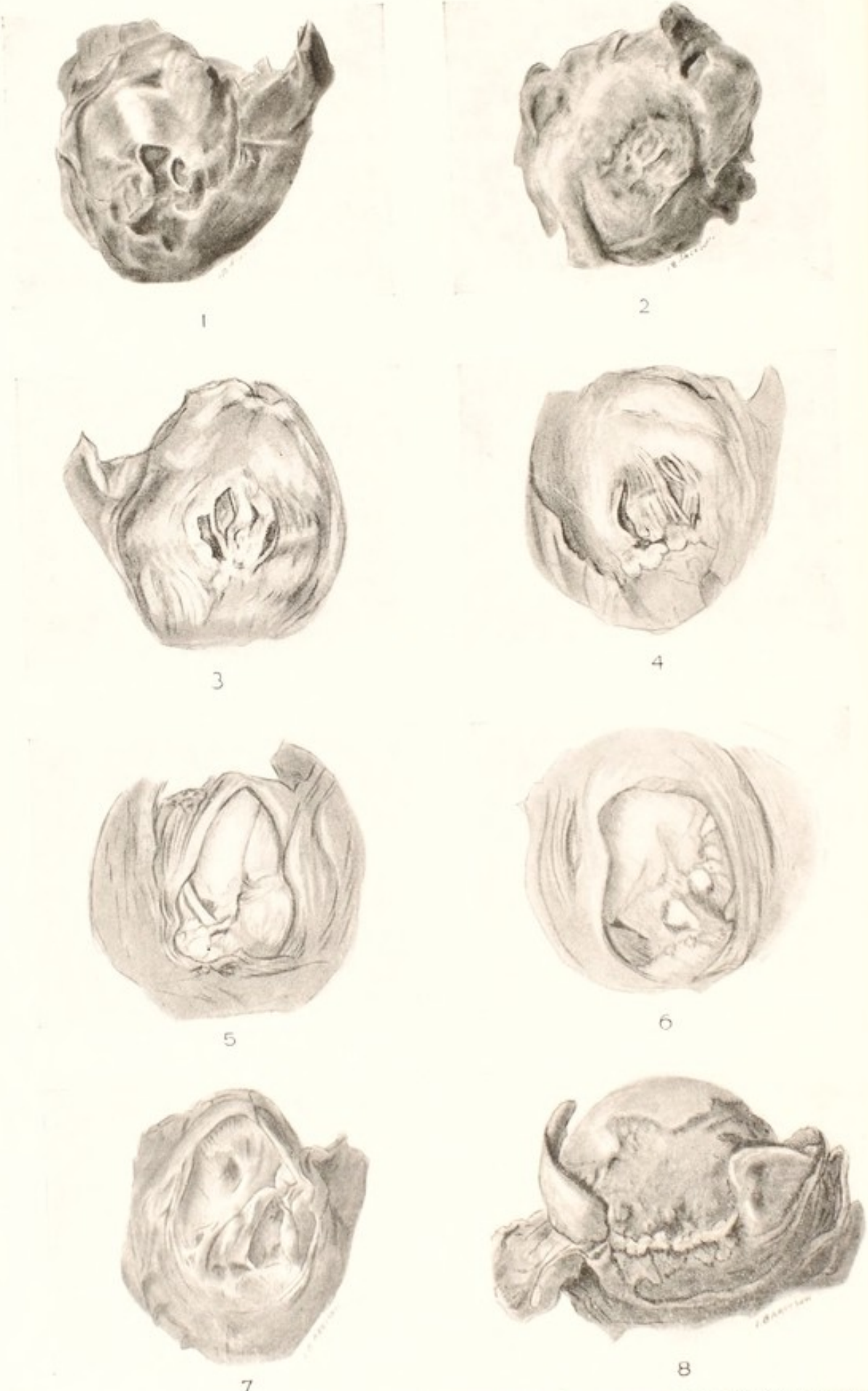


PLATE VII. ASPECTS OF BASE OF BURSA IN OLD LESIONS OF THE SUPRASPINATUS

PLATE VII

These drawings (reduced nearly one-half) were made by Dr. Akerson from specimens which he had removed at autopsy. They represent old, chronic lesions, the results of accidents or disease suffered many years before death. The superior aspect of each specimen is shown; *i.e.*, one is looking down on the base of the bursa and sees the defects in it caused by destruction of the tendon of the supraspinatus. The lower margin is anterior in all cases.

The plate, as a whole, gives a very fair idea of the general aspect of specimens obtained in this way. At first sight one would not recognize that they are all instances of the same lesion. Unless one examined them in detail one would say they were instances of chronic arthritis. Without the knowledge obtained from operation on living cases, the writer himself would not have been able to interpret these specimens as the results of ruptures, but with increasing experience he feels quite confident that each lesion should be so interpreted. Owing to the large number of Dr. Akerson's specimens from which we could choose, we have been able to use this plate to illustrate two quite different and antagonistic points. By arranging them in sequence, the first six figures show a steadily increasing degree in the extent of the defect, although each pair came from a single cadaver. Second, by choosing pairs from the same patient the series also illustrates the frequency of the finding of a bilateral condition, which is a strong argument for considering that the original process may be one of disease or perhaps of overuse. The casual pathologist on finding such conditions would hardly suspect injury as the basis of the lesion, yet this conclusion must be reached after earnest study, even if the rupture may have occurred in degenerated tissue.

No. 1 illustrates the condition of the base of the bursa in a case where the rupture is partial and has made only a small opening between the joint and the bursal cavity, although a chronic bursitis has been set up.

In No. 2 the destruction of the tendon beneath the base of the bursa was more extensive, so that the degree of bursitis is greater.

In No. 3 we see the condition I have called "Straps," where many fibers of the tendon have torn away irregularly. The joint surface is seen in the openings between the straps.

In No. 4 this condition is more advanced so that one sees the eminences on the bare tuberosity from which some of the fibers have been evulsed.

No. 5. The cartilaginous surface is exposed through the more or less triangular rent, of which the bare tuberosity and joint cartilage form the base.

No. 6. The condition is even more pronounced. The upper half of the gap shows cartilage, the border of which is irregular. In the lower half of the gap is seen the bare tuberosities with their eminences of new formed bone.

No. 7 and No. 8 show the extremes of the series. The base of the bursa, with chronically inflamed bands, is present on the left, while the right shows the effect of a severe injury which tore away the supra- and infraspinatus and later, erosion of the cartilage over the area of the articular surface, which was exposed in the gap, had occurred.

Nos. 5 and 6 are typical "complete" ruptures, while the first four and also No. 7 were "incomplete," although small communications existed between joint and bursa in each case. No. 8 represents such a case as that described on p. 109.

of the tuberosity to which the tendons were formerly attached would have become absorbed and rounded off as explained on page 92.

It is quite possible also, that instead of this process occurring by gradual changes, there may have been at any time a fall downstairs or other injury which would have torn the fibers of the tendon to a considerable extent, and from that time on, he may never have had proper use of his arm. Perhaps he had two separate injuries—each to one arm—or perhaps, on the other hand, the process may have been so slow and insidious and accompanied by so much greater disturbance in other joints, that no history could have been obtained of a shoulder injury, even if the patient had been able to give one.

Since this patient presented an extreme arthritic condition in most of his other joints, one might say that the condition in the shoulders was simply a part of the whole process. Undoubtedly, this is to a certain extent true, but my contention is that without traumata, even though trivial, the partition formed by the musculo-tendinous cuff, between the bursa and the joint, would have remained intact. This contention is amply justified by the fact that in many of my operated cases such lesions were unilateral. In a few early cases, I have found a good, thick, normal appearing tendon with a transverse rupture and so little separation, that I could readily and successfully suture the tear.

This was an extreme case of chronic generalized arthritis, and for the sake of argument I think it is fair to postulate that most of the operated cases had some degree of "arthritic" degeneration in their musculo-tendinous cuffs before their accidents. Yet the fact remains that I sutured their ruptured tendons and obtained good function in some cases. Even in this old man, there may have been a time, years ago, when prompt suture of his tendons, combined with attention to his foci of absorption, might have saved him the use of his arms.

Although cases of arthritis which involve one shoulder joint and do not involve other joints, must be very rare, they do occasionally occur. With the exception of one case of a probable gonorrhæal infection, and several cases of tuberculosis, the following case is the only one which I have confirmed by an exploratory operation.

Case No. 7. An unmarried woman of 27, who was born in New Brunswick, but who had done housework in the United States for some years, entered the Massachusetts General Hospital in December, 1909. She stated that she had always been well except for scarlet fever as a child, and pneumonia five years before entrance. Ever since the pneumonia she had frequently had sharp pain in her shoulder at night, but not during the day.

In January, 1909, the pain was especially severe one night and the next morning she found she was unable to raise her right arm above her head, and a "jumping pain" started in the arm and shoulder. At the suggestion of her doctor she carried the arm in a sling for three months, but without relief. In September, the arm was immobilized in plaster, but this did not relieve her pain.

The patient was thin and nervous. There was atrophy of the muscles about the whole shoulder, especially of the deltoid. There appeared to be no motion at all in the scapulo-humeral joint. X-ray showed that the true joint was involved and the cartilaginous area greatly damaged. A diagnosis of tuberculosis was made.

At operation the bursa was found to be adherent throughout, except in the portion beneath the acromion. The tendons of the short rotators were not thickened but were atrophied. There was no œdema or tuberculous granulation tissue seen at any time during the operation. The tendon of the subscapularis was divided and the true joint opened. It was found that only a central island of normal articular surface was left on the head of the humerus. There was no pus or fluid in the joint. The inferior surfaces of the tendons of the short rotators were adherent to the bone beneath them over a circular area about one-half inch wide, which took the place of the normal periphery of the articular surface. With a finger in the joint the amount of remaining articular surface was demonstrated to be nearly exactly that shown in the X-ray. After these manipulations, external rotation and abduction were found to be about two-thirds normal. Abduction was limited by contraction of the teres muscles and it was thought best not to risk rupture. A piece of the synovial membrane just at its reflection to the subscapularis tendon was excised for section. Gross observation showed no evidence of tuberculosis nor did the microscopic report by Dr. Whitney. The tenotomy of the subscapularis tendon was not repaired. Muscle loosely closed with catgut and skin with s. w. g. The wound healed by first intention.

A year later the condition of the shoulder was found to be the same as before operation. There had been no improvement at all. I was unable to trace the patient afterward.

My opinion at the end of the operation was that the patient had caries sicca, but the pathologic report did not confirm this and merely showed chronic inflammation. The fact that the symptoms began soon after a pneumonia suggests that there may have been a pneumococcus infection. Since the wound healed by first intention it is very unlikely that the infection was due to any of the ordinary pyogenic organisms. The striking differences of the symptoms in this patient from those of the ordinary case of "frozen shoulder" were absolute immobility of the joint, and a more obtuse angle formed by the axis of the shaft of the humerus and that of the scapula.

What is arthritis in any joint? Inflammation from any cause, whether primarily infection from pathogenic bacteria or from me-

chanical derangements due to changes which are congenital or traumatic.

Most of the phenomena of idiopathic arthritis are best illustrated in the hinge joints, the cartilaginous surfaces of which remain in contact at some point and in which the lateral movement is prevented by tense ligaments. Static weight-bearing joints also are readily deformed by the results of inflammation, so that permanent damage with distortion results.

Pathologically, acute arthritis is characterized by swelling of the membrane, increase of joint fluid, œdema and congestion of the ligaments and periarticular structures, and spasm of the neighboring muscles. Chronic arthritis results in areas of absorption of the joint cartilages, lipping of the edges of the bone adjacent to the edge of the joint cartilage and shrinking or thickening of the ligaments, and often redundant fibrous changes in the synovial membrane, which may encroach on the cartilage.

You will find it difficult to demonstrate these changes in an untraumatized shoulder joint, and the reason is that the shoulder differs from all the other joints in its mechanics, especially in two respects. Its surfaces are not held in contact by ligaments and its ligaments are never tense, except in the extremes of motion. It is not a static weight-bearing joint, and only at intervals is it even a power-bearing joint. Therefore, it is to be expected that the phenomena of arthritis will not be pronounced, even when the joint has been infected or traumatized. Since it does not have to bear weight, it can be relieved of duty on the slightest onset of soreness. The hip, the knee, the ankle, when inflamed, must bear a burden even during the momentary pause as the patient limps. Pressure is brought to bear on the affected cartilage, and the sway of the body tenses the tight ligaments and induces fibrous reparative changes.

When the shoulder joint is involved *and other joints are not*, it may be taken for granted that the trouble is not due to arthritis of constitutional origin.

The shoulder joint does become involved in some of the general infections, as in cases of acute and gonorrheal rheumatism and in some of the exaggerated cases of general chronic arthritis of septic or toxic origin, but it is noticeable that the shoulder usually clears up before the weight-bearing or hinge joints do. It is seldom left permanently damaged. Isolated arthritis in the shoulder joint is most unusual. How common it is in the knee or the great toe joint, or even in the hip, compared to its incidence in the shoulder! Dealing

with shoulders as much as I have for thirty years, I can recall but one case of a permanently damaged shoulder from a local non-tuberculous arthritis.

Consider by itself in relation to this joint each of the individual changes characteristic of arthritis.

Fluid. Did you ever recognize an excess of fluid in the shoulder joint? You have many times in the knee or in the ankle. I have rarely seen it, although I have been on the lookout for it. When it occurs you may recognize it in two ways. First, by the position of the humerus in relation to the scapula. The arm and scapula are fixed at an obtuse angle, readily demonstrated by comparing the axis of the spine of the scapula with that of the humerus. The tense capsule forces the bones into this position. This sign appears only in very severe cases when the fluid is under tension. Careful palpation beneath the posterior edge of the acromion or in the axilla is the other way, for a rounded swelling can be felt and the sensation of fluctuation obtained. A third way should be by the X-ray, for the joint surfaces would be forced apart, if the amount of fluid is great. One may also, with a sudden push upward of the humerus, knock the joint surfaces together. When joint and bursa communicate through a rupture in the capsule, you may find what I call the fluid sign. (See p. 155.) Fluid in the shoulder joint never causes anterior swelling unless the bursa is also involved.

Why is distention of the capsule of this joint so rare? In the first place, because there is room for so much fluid, that long before a large amount forms, the irritation commands the joint to rest. We do not limp around on a lame shoulder as we do on a knee. In the second place, there are no normal protruding tags of membrane in the shoulder joint, as about the alar ligaments in the knee, to get caught, bruised or twisted and thus to start synovitis.

If *congestion of the tissues* should start in the shoulder, it has plenty of room, and the spasm initiated in the short rotators would protect it until the inflammation subsides. Thus the shoulder joint, in cases of general arthritis, recovers more readily than do the tense joints held by firm ligaments.

Villous conditions are likewise unlikely, because normally the whole synovial lining is smooth, and no clefts have to be filled as in the knee during normal flexion and extension. Hence we rarely find fibrosis of inflamed villi and "lipoma arborescens" to impinge in the shallow surface of the glenoid.

Serious *œdema and thickening of the ligaments* are scarcely possible, since there are no thick ligaments and but a thin capsule.

Absorption of the cartilage does take place in some old cases of severe rupture of the supraspinatus, but this is usually a local area where pressure is exerted on the joint surface by the under side of the acromion, as the head of the bone gains a fulcrum when the arm is abducted. This cannot occur unless the supraspinatus tendon has given way, for normally it protects the cartilage from this contact. It is very significant that Akerson found joint erosion localized by and proportionate to the extent of the ruptures in the tendon. I doubt whether cartilage erosion occurs in the shoulder joint unless the supraspinatus has been damaged or when suppurative inflammation, which is rare in this joint, has occurred.

I have not found cases where a "pannus," as described by Nichols in the other joints, has invaded the cartilage from the joint edge and gradually reduced its extent. The loss of cartilage in the gap caused by retraction of the tendon is symmetrical with the exposed areas and is quite superficial.

Deformity of the articular surface does occur in the shoulder, but only after very destructive traumatic lesions or suppurative infection, tuberculosis, fracture, or operation. Most deformities of joints are due to combinations of disease (softened bone and tissue), with weight bearing or power bearing in sufficient degree to cause one side of the joint to yield. The shoulder joint is relatively free from this combination. With the slightest symptom of inflammation, all weight or power bearing is instinctively stopped. As a whole, arthritic deformities in the shoulder are rare, and most of the old museum specimens which show deformities are probably the late results of fracture and dislocation. I have never seen a monarticular arthritis of this joint sufficiently severe to cause deformity. Even in the generalized case detailed above, the heads of the bones were mere rounded knobs, not much misshapen and with no hypertrophic excrescences.

Lipping of the articular margins is the most characteristic appearance in cases of hypertrophic arthritis in all joints. It occurs exactly at the border of the articular cartilage. In most hinge joints the lateral ligaments are inserted just at this point, and hypertrophic spurs or rims may go up into these ligaments, or even into the attachments of tendons. In the shoulder joint, since there are no lateral ligaments, there is very little tendency for lipping, so far as the articular surface of the humerus is concerned. In the glenoid,

there may be some lipping due to bone formation in the fibrous rim of the shallow joint surface. The finding by X-ray of such instances of lipping of the glenoid is usually accidental, and generally accompanied by no symptoms. I doubt if it is significant at all, so far as arthritis is concerned. Examination of many desiccated shoulder blades shows that the glenoid surface is quite variable, and such instances as there are of unusually misshapen conditions of the edge, are probably due to relaxation of the joint from conditions such as paralysis of the adjoining muscles, recurrent dislocation, or looseness of the capsule due to rupture of some of the short rotators. These conditions would permit the head of the bone to ride over unduly on the edge of the glenoid and cause bone proliferation there. On the humeral head in occasional desiccated bones, one sees a very slightly raised rim on the edge of the articular cartilage. This appears to be the only evidence of arthritis that one could determine by X-ray. Such appearances are frequently found in elderly people who have never had a symptom. I wish to call especial attention to Diagram C, which shows why hypertrophic appearances on the tip of the tuberosity, or external to it, are to be attributed to lesions of the bursa and not to arthritis of the joint. They are not in the joint.

Synostoses. Bony ankylosis of the shoulder joint rarely occurs except in children, following tuberculosis and osteomyelitis. Both of these conditions are very rare in adults. In many of the other joints the severe grades of arthritis end in fixation, but the shoulder joint is of such a structure that bony ankylosis can only be produced with difficulty even when it is the intention of the surgeon to procure it. The amount of surface for bony contact is so small in proportion to the degree of leverage exerted by the arm, that it is a real achievement to get a synostosis. The tendency of the joint, even in severe infections, is to slowly form fibrous adhesions which permit only a small arc of motion.

We have now reviewed the individual changes characteristic of arthritis in other joints, and made it clear that in the shoulder these pathologic conditions either do not exist or have a tendency to remain at the minimum. We cannot say that there is no such thing as non-traumatic arthritis in the shoulder joint, because inflammation of the joint can and does occur, but we can see that owing to the structure of the joint it does not readily occur and, if it does, the lesions are at a minimum compared to those which occur in the other joints. In fact, we might say with much truth that there is no such

condition as a chronic arthritis *per se* of the shoulder, for, owing to the structure of the joint, it would always be a periarthritis; that is, inflammation can only show itself in the tissue which is the important one in the joint; namely, the fused musculo-tendinous cuff which forms the major part of this articulation. I contend that the defects in this cuff so frequently found at autopsy were originally largely traumatic, although unhealthy tendon may have suffered the trauma.

Periarthritis. The structures besides the musculo-tendinous cuff which might be involved in this condition are the prolongation of the synovial lining of the joint down into the bicipital groove, the bursæ under the infraspinatus and subscapularis, the subacromial bursa, and the glenoid half of the capsule. It would be a very severe periarthritis which would affect to any considerable extent the structures external to these. There is occasionally some secondary atrophy of the muscles further from the joint (*e.g.*, the deltoid, the pectorals, and the teres major), due to prolonged spasm or inaction. The reader may refer to Chapter I, p. 14, which discusses the outer and inner set of muscles in the shoulder. It is the inner set which is involved regularly, but the outer set which operates the scapula is as a rule not atrophied.

In periarthritis, what is the exact condition which prevents the patient from performing active motion, or the surgeon from performing passive motion? Such a case might have all the following changes:

1. Adhesions between the roof and floor of the subacromial bursa involving (*a*) the subacromial portion; (*b*) the subdeltoid portion; (*c*) the subcoracoid portion.

2. Adhesions between the bicipital groove and the tendon of the long head of the biceps.

3. Necrotic changes and inflammatory stiffening in the musculo-tendinous cuff.

4. Chronic inflammation in the synovial membrane of the joint and of its capsule.

5. Adhesions in the extensions (bursa subscapularis and bursa infraspinati) of the joint underlying the infraspinatus and subscapularis.

Different cases may have varying proportions of pathology in these structures, extreme grades in any one of which would suffice to limit the motion of the joint; *e.g.*, firm adhesions at any portion of the subacromial bursa might be the major factor, or joint motion might not be complete owing to adhesions between the intact biceps tendon and the sheath in the groove, even if no other parts of

the periarticular structures were involved. Likewise, the bursa might be free; the sheath of the biceps might be free, but restriction of motion might be maintained by the inflammation in the musculotendinous cuff.

I am inclined to believe that all these factors are present to some degree in the worst cases, but I am very sure that in many cases the adhesions in the bursa are most significant and important from the point of view of treatment. However, I believe that the adhesions in the bursa are very often primarily due to the peculiar form of inflammation characteristic of the tendons. This subject of the tendinous involvement is more fully discussed in Chapter VII.

When I first began my work on "Stiff and Painful Shoulders," the usual diagnosis assigned to such cases was "periarthritis." Following my articles drawing attention to the anatomic characteristics of the bursa and the importance of its recognition, the term "subdeltoid bursitis" replaced that of "periarthritis," and since my second paper, the adjective "subacromial" has largely replaced that of "subdeltoid." The present status is, that the bursitis has been accentuated rather than the tendinitis. I now feel that in most cases the bursa, like the peritoneum, is only secondarily involved, and that the commonest causes are: (1) in traumatic cases, a rupture of the fibers of the supraspinatus tendon; and (2) in spontaneous cases a necrosis in this tendon, and even in these cases possibly an initial severe trauma to the tendon. My present view might be expressed as follows: The starting point of most lesions of the shoulder centers in the tendon of the supraspinatus. Thence it involves the bursa and the adjoining tendons of the other short rotators, but the inflammation of the bursa gives the most pronounced and often the only painful symptoms.

Thus, from arthritis, we have passed through periarthritis to bursitis.

Bursitis. Formerly I described bursitis as consisting of three types, the acute, the chronic adherent and the chronic non-adherent. These are still useful clinical divisions. I now feel that an attempt to describe bursitis as an entity would be equivalent to attempting to describe peritonitis as an entity. I believe that the bursa, like the peritoneum, has a very great capacity for the formation of protective adhesions which later may disappear. It is not a structure where disease starts, so much as a structure which limits disease in the adjacent structures by temporary adhesions, causing fixation of the parts. Later its physiology is such that adhesions disappear

and two shining, adjacent, frictionless surfaces are again formed to allow motion between the parts. After inflammation the single sac may be replaced by a number of smaller spaces. Lesions in the supraspinatus tendon are the common cause of bursitis, just as lesions in the appendix are the common cause of peritonitis.

Since the success of treatment depends in most conditions on our knowledge of the exact pathology, a surgeon treating shoulder lesions should try in each individual case to picture in his mind the relative proportions of the elements of the joint which may be involved. Personally, I believe that the sheath of the biceps tendon is less apt to be involved than are the other structures. I have never proved its involvement in a single case. I think that the substance of the tendon of the supraspinatus is the most often involved. In many cases there is a secondary bursitis, but in some there is none. I am quite sure that patients may have small ruptures, small calcified deposits, and small areas of necrosis in the tendon, without any bursitis being caused.

I am willing to admit that I cannot readily define just what signs give me the impression in individual cases that any particular structure is especially involved. It is a matter of touch which can only be learned with experience, and deceptive even after much experience. The difficulty of estimating the proportions in which each structure is pathologic is made still greater by the degree of spasm present. In acute cases of bursitis, such as those caused by perforation of a calcified deposit from the tendon into the bursa, spasm alone may hold the whole joint rigid, although on the previous day the full arc of motion could be performed. A month later the joint may be wholly free again, or perhaps even six months later the spasm may have become at a minimum, and yet the joint will be just as rigid from adhesions and stiffening, which are wholly mechanical and prevent relaxation even under ether. All degrees between these extremes exist, because stiffening from spasm fades into an adherent condition by imperceptible degrees.

I may summarize by saying that owing to the peculiar mechanics of the shoulder, the avascular and inert supraspinatus tendon is the most vulnerable part of the joint, and that inflammation in it is apt to be painless until the adjacent subacromial bursa is involved, which, being abundantly supplied with vessels and nerves, produces the symptoms of which the individual complains.

The explanation of the relative weakness of the supraspinatus is in my opinion to be given on evolutionary grounds by the change from the horizontal to the vertical animal. This led to obliging the

supraspinatus to lift the arm instead of swinging the foreleg. As suggested on page 7 the bursa is a relatively newly developed structure. However, we must not lose sight of the fact that the tendons of the other short rotators are often involved in all these conditions.

Furthermore, although the capsule of the joint is only tense in extreme positions, the anatomic position in which we usually carry our arms as we walk or sit, does put some strain on the upper portion represented by the insertion of the supraspinatus. Lockhart has pointed out that in the cadaver this portion of the capsule will hold the head of the humerus up, even if all the remaining capsule is removed. He uses this fact as an argument against the accepted theory that air pressure helps to maintain the joint in position. While I do not agree that this is true, I do believe that in the upright position there is always *some* tension on the tendon of the supraspinatus, for this is almost an extreme position.

Definitions and Classification of the Various Forms of Subacromial Bursitis

An *acute subacromial bursitis* is one in which there is sharply localized tenderness and protective spasm of sudden onset. Acute bursitis rarely arises from any cause except one of the following:

(1) A direct bruise on top of the anterior part of the shoulder when the arm is in dorsal flexion. This type is not proved by actual observation on the operating table. It is merely supported by the clinical fact that such bruises are followed by acute local tenderness over the bursa, accompanied by scapulo-humeral spasm. Such cases promptly recover and do not have a chronic phase. They are rare.

(2) Prolonged (an hour or two) hyperabduction. The best instance is bursitis following hyperabduction of the arm in operations, as for cancer of the breast. Instances of this form are not common, and there is no absolute proof that the strain of this position does not primarily affect some other part of the joint. Such cases may become subacute but not chronic (lasting over six months).

(3) Inflammation extending from about a calcified deposit which has either burst into the bursa or has approached the bursal surface closely enough to inflame its lining. This is by far the most common cause, and is abundantly proved by the findings at operations. These cases may be acute only, or last for months, but they rarely give symptoms for over two years, even in the worst cases. Many recover within a few weeks after perforation.

(4) A rupture of the supraspinatus tendon by trauma of sufficient degree to make a direct opening between the joint and the

bursa through the gap in the tendon. This must lead to bleeding into the bursa and more or less distention with joint fluid. I have never yet opened a bursa immediately after such a trauma, but more or less acute bursitis must necessarily occur in such a case, for the floor of the bursa is involved. The acute phase is brief. Complete ruptures always produce a permanent chronic bursitis because the bursa is continually irritated by friction on the irregular base.

(5) Minor ruptures may occasionally produce an acute bursitis, but they may be almost symptomless and usually are subacute in character, and sometimes become truly chronic. In my opinion the great majority of sore shoulders come in this class as subacute cases.

Any other conditions which suddenly produce an acute bursitis must be very rare. I have seen only one suppurating acute case from pathogenic bacteria. Even this case was doubtful and was probably to be explained by contamination of the culture.

Subacute or chronic subacromial bursitis may be divided into two classes:

(1) Those in which there is no restriction of motion, but in which there is a painful point causing a wince or jog in the motion of elevation.

(2) Those in which there is limitation of motion from adhesions, or contractures, although spasm may still be present.

The two classes are not perfectly distinct, but serve in a general way in the study of the subject.

The cases of the *non-adherent type* are caused by (*a*) inflamed villi, folds or bands. These cases do not often have an acute phase. (*b*) Irregularities of the base due to calcified deposits in the tendons, about which there is only a little chronic inflammation. These cases may have already passed through an acute phase or may at any time exhibit one. (*c*) Defects in the base of the bursa due to a partial or complete rupture of the supraspinatus tendon. The acute phase is brief and occurs only immediately after an accident.

The *adherent type* of subacute and chronic cases seldom follows types one and two of the acute cases. It is a common sequence of the third type; is very infrequent in the fourth type; not uncommon in the fifth type, especially in those cases where the trauma is trivial and which will be discussed later under "Tendinitis."

REFERENCES

I know of no references in the literature of arthritis which would be of any particular help in studies of the shoulder. If the reader wishes to inform himself as to the modern conception of what little is known about "arthritis" and "rheumatism" in general, he is referred to the number of the *New England Journal of Medicine*, for May 18, 1933, Vol. ccviii, no. 20, which prints several papers read at a recent symposium on the subject; with these he will find numerous references.

Chapter V

RUPTURE OF THE SUPRASPINATUS TENDON

Now that we have considered the shoulder from the anatomic and pathologic points of view, we come to the clinical study of the lesions which may be identified by special groups of symptoms as definite entities. It seems to me that the practice of medicine might be greatly simplified if an official list of clinical entities was constantly maintained by some great medical association. Our literature and our methods of medical education are greatly hampered by synonyms. McCarthy has recently pointed out in *Surg. Gyn. and Obst.*, February, 1932, that there is great need for such a list of malignant conditions. Pathologic entities and clinical entities are not the same. Clinical entities are the practical working diagnoses on which rational treatment may be based. I feel that the Registry of Bone Sarcoma has served such a purpose so far as the nomenclature of bone tumors is concerned, and that this fact alone has done much to crystallize our working knowledge of the diagnoses and treatment of bone lesions.

I shall try in this book to make a similar list of the lesions of the shoulder which have such distinctive characters that they may be recognized clinically and given appropriate treatment. For instance, I recognize as significant clinical entities, complete rupture of the supraspinatus tendon, partial rupture of the supraspinatus tendon, calcified deposits in the tendons of the short rotators, and tendinitis of the short rotators; and I do not recognize muscular rheumatism, neuritis, or idiopathic monarticular arthritis of the shoulder, as entities of sufficient clinical frequency or importance to make them demand special forms of treatment, although these terms are much more frequently used as diagnoses on which physicians base their therapeutics. If an official list existed I would ask to have my new entities added and the old ones at least put in small type. The reader is referred to the Index.

CHART CONTRASTING FOUR COMMON CAUSES OF PAINFUL SHOULDERS

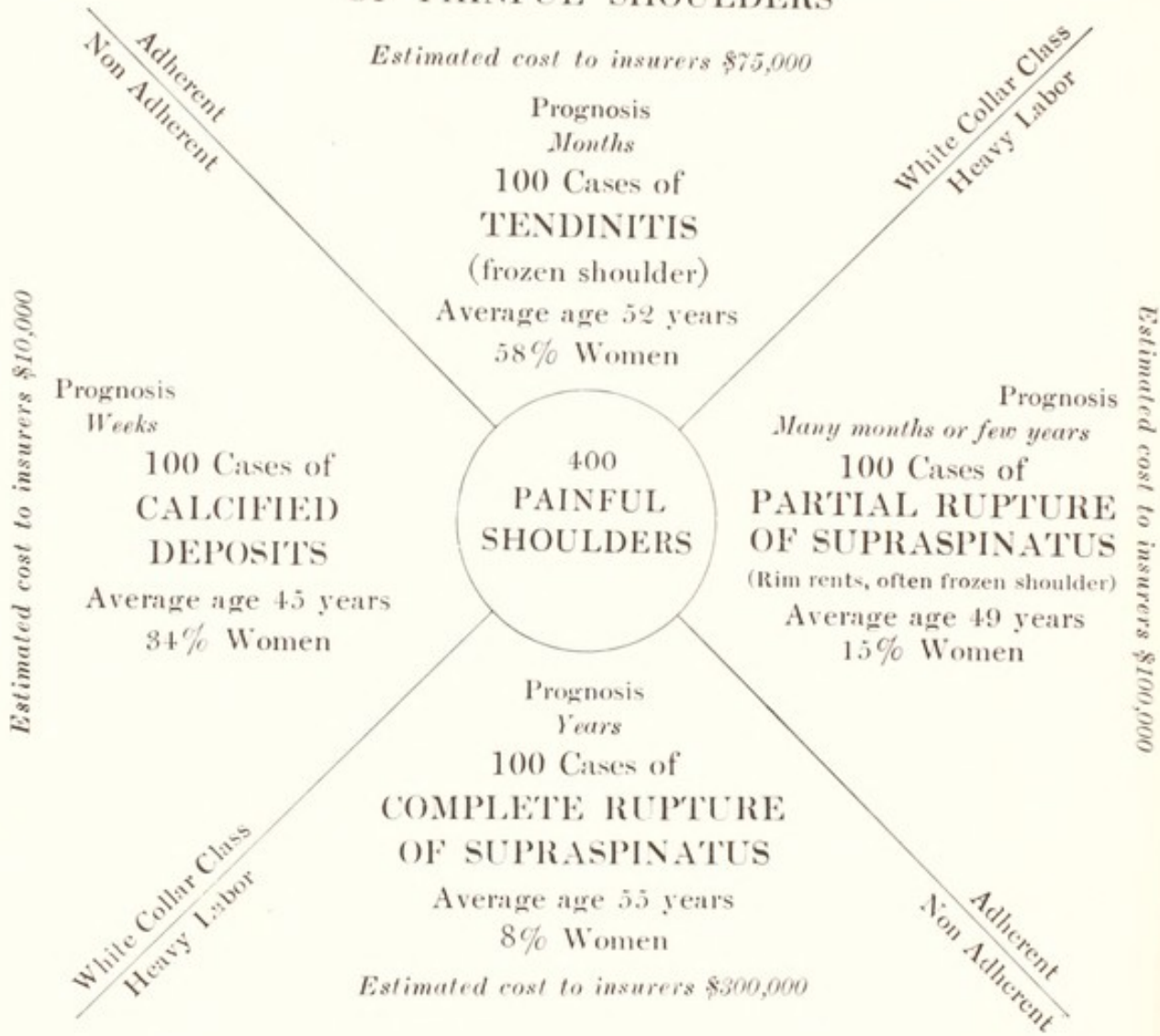


CHART I

Excluding obvious diagnoses such as tumors, fractures and dislocations, most painful shoulders may be classed under the four diagnoses indicated in large type, although each entity merges into the two which adjoin it. For example, calcified deposits are probably a product of tendinitis, but if large in amount, they alter the clinical picture, both in prognosis and in the character of treatment required. If the deposits are very small, they may be negligible, and the clinical picture be that of a "frozen shoulder" due to tendinitis. Tendinitis may also be confused with its other neighbor, because a "rim rent" may precipitate the inflammation which results in the frozen shoulder. In a similar way the line between partial and complete ruptures is difficult to draw; *e.g.*, some cases of partial ruptures do not show much restriction of motion. Even complete ruptures may be confused with calcified deposits, as in Case 76, where the tendon was torn through a partially calcified area. Pathologically, too, there is some reason to believe that calcified deposits may be caused originally by small ruptures, and that the defects left after the deposits have disappeared may later lead to rupture. Nevertheless, although these entities are difficult to separate in borderline cases, typical instances are very clearly distinguished for purposes of treatment as well as in prognosis. There are also suggestive variations in sex, age, and occupation among the patients subject to these conditions.

This chapter discusses the most serious of these entities. As far as I know, I was the first writer to call attention to this lesion, and it seems to me that I can best introduce the rest of the book by reprinting here my first article, so that we may thus start at the beginning, so far as the history of this particular entity is concerned.

Reprinted from the Boston Medical and Surgical Journal, Vol. clxiv, No. 20, pp. 708-710, May 18, 1911

Complete Rupture of the Supraspinatus Tendon. Operative Treatment with Report of Two Successful Cases

In a paper on "Stiff and Painful Shoulders," published in the *Boston Medical and Surgical Journal* for May 31, 1906, in speaking of rupture of the supraspinatus tendon in connection with subacromial bursitis, I presupposed, on anatomic grounds, the probable symptoms of such rupture as follows:

"One theoretical symptom (since the supraspinatus is put out of action) should be the persistence of passive and loss of active abduction. I am not ready to say this as yet, however, because in most cases the pain is so great that spasm prevents even passive motion, and later adhesion takes the place of spasm. When rupture of the tendon does take place, it probably is only partial and a Y-shaped attachment still remains to perform part of the function. In a similar way, the quadriceps may extend the femur when the patella is broken if the lateral expansions of its tendon are not torn. Nevertheless, I believe that the active function of the supraspinatus is important in elevation of the arm."

Since this paragraph was written I have had two cases of complete rupture of the supraspinatus tendon on which I have operated, and in both of which I was able not only to demonstrate the existence of the anatomical lesion in conjunction with the above symptoms, but succeeded by suturing the tendon to the tuberosity in bringing about complete restoration of the function of abduction. I have also, in a number of cases, verified the clinical diagnosis of a partial rupture such as depicted in the accompanying figure, which was produced in the other articles which I have written on this subject. (The reader is referred to the articles appearing in the *Boston Medical and Surgical Journal* for Oct. 22 and 29, Nov. 5, 12, 19 and 26, and Dec. 3, 1908. The figure alluded to is not repeated here.)

This partial rupture is the common lesion, and, as I have explained, the remaining portion of the tendon is competent to take up the function when the sensitiveness due to the reparative process

about the point of rupture has disappeared. The curious deposits of lime salts sometimes shown in the X-ray, and spoken of by Painter and Baer, are, I believe, faulty attempts at repair of these tiny ruptures of the tendon.

The following two cases are quite definite because they present complete rupture of the tendon of the supraspinatus. Therefore, the function of active abduction was almost entirely lost.

CASE 1.—Mrs. J. A. Aged fifty-two. Born in Scotland. Referred by Dr. Kent, of Dorchester, March 7, 1909.

Patient had always been a healthy, hard-working woman and had practically never been sick. On Oct. 3 of the previous year, *i.e.*, five months before I saw her, while she was hanging recently washed clothes on the line in her yard, she endeavored to toss over a heavy blanket and felt something snap in her shoulder as she threw her arm up. She felt an intense pain and her arm fell and "hung by her side for a while." She was examined later by Dr. Kent, who found no thickening or ecchymosis. An X-ray was negative. Since then she had not been able to abduct the arm and had suffered much pain at night and somewhat during the day. The symptoms were in general those which I have described as the symptoms of adherent subacromial bursitis. The following points were, however, noticeably different.

(1) There was little atrophy of the deltoid, and it even appeared hypertrophied on account of the apparent swelling of the joint beneath. (2) Under the fibers of the deltoid, beneath the acromion and over the joint, there was a distinct deep fluctuation as if the whole bursa were full of fluid. (3) The ability to start abduction was absent, but when the arm was passively abducted to about 140°, the patient, by a strong contraction of the deltoid, could prevent the arm from falling for an instant, but the slightest downward pressure made it drop to the side. External rotation was about one-half the normal.

Operation.—March 11, 1909. Usual incision between fibers of deltoid. Roof of bursa abnormally thick with granulation-like bodies on its under surface. Escape of straw-colored fluid, about one-half ounce. The floor of the bursa was found communicating with the joint, because practically the whole supraspinatus was torn from its insertion and retracted inwards. The biceps tendon was exposed over the articular surface, but was apparently uninjured. For about one-half inch on the lower visible portion it was bright pink; the rest of it was normal in appearance. By holding the bursa wide open,

pulling down on the arm and raising the elbow from the table, the retracted end of the supraspinatus could be seen. This was caught with a tenaculum and pulled down enough to suture with four heavy silk threads to the remaining portion still attached to the tuberosity. This could not be done exactly, but was done nearly enough so that it seemed possible for repair to take place along the silk sutures. A little gap was also left on each side, which was not covered with tendon substances. It was in a sense a *suture à-distance*. When the operation was completed it seemed as if there was a fair possibility of the supraspinatus obtaining enough attachment to enable it to perform its function, although the base of the bursa would necessarily remain a rough instead of a smooth surface. Dr. Kent gave ether, Dr. Vincent assisting. Dr. J. J. Putnam and Dr. M. P. Smithwick present.

June 9, 1909. Looks very strong and well. Sleeps well. Arm aches at times but not much. Pain in stormy weather (after use). Gets good use out of arm and does her own work. Can button back of dress and do her own hair. *Real* free abduction to 135°. Fair rotation. Muscles developing well and deltoid is strong. Very much pleased with result.

This patient was demonstrated to the Interurban Orthopedic Club, March 25, 1911. The arm is perfectly well and the function is perfect. The only abnormal sign is that the deltoid is unusually prominent due to the presence of joint fluid in the bursa.

Case 2.—Mr. D. R. Aged forty. Hostler. Referred by Dr. John Homans, Dec. 21, 1910.

Patient is a strong, wiry Irishman. He has always been well. About three and one-half months previously, he had been saddling a horse in the stable and while tightening the girth he felt something in his shoulder give way and he fell to the floor. There was immediate loss of power in the arm, but he managed to finish saddling the horse without raising that arm. That night he consulted Dr. Luce, of Canton, who found no ecchymosis, but thought there was slight crepitus. An X-ray a little later was negative. The hip was also hurt by his fall, so that for some time he was more bothered by that than he was by his shoulder and he was obliged to use crutches for several weeks. At the time he presented himself to me for examination the symptoms were at first sight those of the adherent type of subacromial bursitis, but on more careful examination the same signs that were present in the previous case were demonstrable, that is, (1) Relatively slight atrophy of the deltoid and an appearance of

hypertrophy. (2) Fluctuation over the region of the bursa beneath the deltoid producing a "*verwölbung*" of the latter. (3) The persistence of nearly normal passive abduction with no active abduction. When the arm was passively abducted, the patient by a strenuous exertion of the deltoid could hold the head of the bone on the glenoid and thus prevent the arm from falling immediately. (4) In this case a distinct depression could be felt just above the tuberosity at the point where the tendon was torn away from the latter. A definite diagnosis was made and the members of the Boston Orthopedic Club invited to examine the case and witness the operation.

Operation.—Jan. 10, 1911, at the Massachusetts General Hospital.

As in the previous case, when the fibers of the deltoid and the thickened roof of the bursa were incised, there was an escape of straw-colored fluid and the bursa was found to be in communication with the true joint. The supraspinatus had retracted so far that at first it could not be seen, and one looked directly at the articular surface of the bone with the uninjured biceps tendon lying across it. With some difficulty the supraspinatus tendon was caught with a tenaculum, freed and pulled forward. It was then sutured "*à-distance*" to the tuberosity with heavy silk prepared with paraffine after the manner of Lange. As in the previous case, the retracted tendon could not be entirely united, but enough strands of silk were put in to make it possible for the function of the tendon to be replaced.

Convalescence was normal and the patient was not allowed to use the arm in abduction for three weeks, but since then has been using it with more or less freedom. He was shown to the Interurban Orthopedic Club on March 25, and the following condition noted at that time:

Patient is working every day—can chop wood and do other "chores" without pain. He can easily place his hand on top of his head or behind his back. Full abduction of the humerus on the scapula is, however, weak, and although he can elevate his arm, he cannot hold it in an abducted position against a downward pull of even moderate force. The strength of the arm in other respects is excellent and the patient is well satisfied. The function of the supraspinatus is fully as good as it was in Case 1, at the same length of time after the operation.

I have seen only one other case in which I have made a diagnosis of complete rupture of the supraspinatus, and as I have not been able to persuade this patient to allow me to operate, his present condition is very instructive as compared to the two cases mentioned above.

In spite of the fact that the patient is a powerful man with an extremely well-developed deltoid, he is now, four years after the injury, still unable to start abduction. As in the other cases, however, when the arm is passively abducted so that the patient's deltoid acts in the same line of force as the axis of the humerus and the remaining short rotators (*i.e.*, subscapularis, teres minor, infraspinatus), the head of the humerus obtains a fairly firm contact with the glenoid so that the weight of the arm can be held by a great effort of will on the part of the patient. The slightest pull downward on the arm, however, will overcome what little power he has, and as soon as the fulcrum on the glenoid is lost, the arm drops to the side.

It will be necessary for those readers who are interested in this subject to refer to the articles mentioned above to thoroughly understand this one, but, best of all, they should look for themselves at dissecting-room subjects, because injuries to this tendon are so common that I have never had any difficulty in finding examples of it in a single set (20) of dissecting-room subjects.

The injury, as I have explained, is usually confined to a partial rupture of not more than one-quarter to one-half inch in breadth. Such complete cases as these three which I have reported are exceptional. The smaller ruptures, which are not of sufficient mechanical importance to interfere greatly with the function of the arm, are best considered with the subacromial bursitis which they cause. It must be understood that these ruptures are beneath the serous base of the bursa, which may or may not be torn through. If it is torn through, a communication is established between the bursa and the true joint.

In operating for subacromial bursitis, if on entering the bursa one finds straw-colored joint fluid, a careful search will usually demonstrate a small opening into the true joint at the point of rupture. In only one case has it seemed worth while to me to make an attempt to suture one of these small ruptures. Usually these heal satisfactorily if the inflamed portion of the bursa over them is clipped away with scissors. I am convinced, nevertheless, that suture is necessary in long-standing complete cases such as those cited above. The one which was not sutured has a decidedly impaired function and for two years was unable to work.

I have never seen the tendons of the other short rotators ruptured except in conjunction with that of the supraspinatus. Twice I have seen a longitudinal split between the tendon of the subscapularis and that of the supraspinatus. (End of 1911 paper.)

FIGURE 38. RUPTURE OF THE SUBSCAPULARIS

Sketch by Mr. Aitkin of a specimen found and prepared in the dissecting room by my former assistant, Dr. T. W. Stevenson. It illustrates a rupture of the subscapularis without rupture of the supraspinatus, and is instructive from several points of view. This is the only instance of an exception to the statement in the last paragraph which has come to my knowledge in the twenty-two intervening years. It gives a very good idea of how the insertion of the supraspinatus, which in this specimen was intact, normally fills the sulcus at the anatomic neck, and covers the tuberosity, thus leaving a perfectly smooth exterior contour beneath the base of the bursa. In this case the bursa has been thoroughly dissected away in order to show the superficial fibers of the tendons passing over the tuberosity and becoming continuous with the periosteum below. In Chapter X it will be shown how these fibers cover up and hold together the fragments in comminuted fractures.

The figure also gives a good idea of the manner in which the supraspinatus emerges from under the coraco-acromial ligament and acromion. The lower or inner edge of the muscle has been rather sharply dissected, but in the undissected specimen this edge blended with the upper portion of the subscapularis. Below this, one sees that most of the subscapularis has been torn away from the conjoined tendinous cuff, so that the biceps tendon, running through its groove between the two tuberosities, is exposed at the left edge of the gap. Internal to the biceps tendon we see the lesser tuberosity, from most of which the subscapularis fibers have been evulsed. The knobby character of the surface of the exposed tuberosity is shown; an appearance usually found in old cases where the tuberosity is exposed by evulsion of the fibers of any of the other tendons. (See frontispiece and Fig. 40.) In other words, this knobby look is the superficial appearance of the "excrescences" or "volcanoes" spoken of on page 91, and also shown in Plate V, Fig. 1.

In the upper half of the gap above the excrescences is the exposed cartilage of the joint. If this gap extended outward from the biceps tendon, instead of inward, it would represent the condition which we usually see; *i.e.*, rupture of the supraspinatus rather than of the subscapularis. It is not unusual to find in the dissecting room extensive tears involving both tendons, but it is very unusual to find the subscapularis involved alone, as in this case. One can readily picture how easy it would be to produce such a condition as this by forcibly performing external rotation in a case of "frozen shoulder."

This diagram also gives an excellent idea of the coracoid process, coraco-clavicular and coraco-acromial ligaments, as well as of the conjoined origin of the internal or short head of the biceps, and of the coraco-brachialis muscles. It also shows the insertion of the pectoralis minor, the tendon of which protrudes as a stub at the inner side of the coracoid process in this diagram.

Although it is over twenty years since the above paper was written, I have very little of importance to add or subtract from it. I followed the two cases for many years and the results continued to be satisfactory. Although the second case never had perfect function in his shoulder, he could do all sorts of farm and stable work without complaint. As he worked for a neighbor, I had frequent opportunity to observe him for over ten years.

There is a point in the quoted paragraph which might cause confusion. At that time, 1906, I did not realize that the stooping posture was such a great help in testing mobility in the scapulo-humeral joint. One may get the impression, when examining a patient in the upright position, that scapulo-humeral adhesions exist, and yet in the stooping posture, positive proof will be given that the joint is movable. It is important for the reader to understand at once that scapulo-humeral passive mobility is a *sine qua non* for the diagnosis of a complete rupture of the supraspinatus, and that in the stooping posture this mobility is much less inhibited by pain and spasm. I did not fully appreciate this point in 1906, and even in 1911 I had hardly grasped it, and did not accent it enough in the above paper. It will be discussed later in this chapter.

The only other point which I desire to correct is in the next to the last paragraph. I do not think I was justified in making such a general statement as "usually these heal satisfactorily if the inflamed portion of the bursa over them is clipped away with scissors," for I am still in doubt as to how to treat the incomplete ruptures.

I have really little more than I had in 1911 to give to the profession in this book, except that repeated experience with the same signs, symptoms, operative findings and follow-up have increased my confidence in the accuracy of my former observations and opinions. During these years I have only operated upon about forty belated cases, although I have made the diagnosis over a hundred times. My results have been good but by no means perfect, because I never see these cases in their early stages, when I am sure the operations would be easy and the results entirely satisfactory. This book aims to try to teach the practicing physicians, who see the cases soon after the injury, how to recognize this lesion immediately, and to rush the patient to a competent surgeon as promptly as if the patient had a broken arm—a much less disabling accident. As in acute appendicitis, early recognition and prompt operation are of the utmost importance. The remainder of this chapter will therefore be devoted to a more detailed discussion of the symptoms.

FIGURE 39. RUPTURE OF THE SUPRASPINATUS

A schematic posterior view of a case of ruptured supraspinatus, to show the posterior short rotators and the sulcus and eminence formed where rupture of the supraspinatus has occurred. The acromion has been sawed off at its base. The reader should study the frontispiece and the next figure in connection with this one.

The size of the rent in the tendon is an important factor since the degree of the severity of each symptom may vary with the extent of the rupture. It seems best to discuss first the symptoms of those cases where the rent is large, as in the two cases which I first reported and which I have called "complete." This means that at least that portion of the conjoined tendinous insertions supplied by the supraspinatus has been torn away, with or without portions of the adjacent tendons. These are the cases which should certainly have the benefit of immediate operation. I do not at present advocate operating upon incomplete cases, for it is likely that after a few months they may heal in whole or in part. On the other hand, there is good reason to believe that the complete ruptures which make an open communication between the joint and the bursa never do heal entirely unless sutured. In other words, the symptoms have had to be pretty pronounced in order to make me willing to operate. It is significant that almost invariably the rent in the tendon has been found to be larger than anticipated. I have perhaps been over-conservative in deciding to operate, but the reader must remember that I have taken the responsibility of doing an operation which is not generally practiced, and naturally I have been somewhat cautious. It is my sincere belief, however, that a small exploratory incision is harmless and that the practice of promptly making such an incision in acute, doubtful cases is to be encouraged, provided the operator has carefully studied the anatomy of the region.

Almost all surgical operations which are now standard procedures had similar histories. Many human sacrifices were required to teach us not to delay when the symptoms strongly suggested appendicitis, perforated duodenal ulcers or intestinal obstruction. The fact that death occurs when we procrastinate in these serious cases has made us, in the public eye, more to blame for delay than

for making negative explorations. The surgeon who does explorations on these injured shoulders might be criticized today and yet a few years hence be blamed for the failure to do them. Moreover, the laboring man with a shoulder injury has not yet been educated to dread this particular lesion as he has been to fear appendicitis.

FIGURE 40. X-RAY OF SPECIMEN SHOWN IN FRONTISPIECE

Owing to the fact that it had been dissected, air has entered both the joint and the bursa, somewhat after the manner indicated in the cover design. It suggests appearances which we might see if we used air or opaque fluid injections in the bursa and joint. It shows a little irregularity on the surface of the tuberosity, which in the painting gives the appearance that I have called a "volcano"; *i.e.*, a small eminence which has a craterlike place in its center. These little eminences are found in many old cases of ruptured supraspinatus. They may represent a productive osteitis due to irritation from contact with the acromion during elevation. The figure also shows two small caverns such as those illustrated in Plate V, Fig. 1 and Fig. 2. I am not sure just what these caverns indicate.

The account of symptoms given in my 1931 paper before the American College of Surgeons was presented in a twenty-minute talk, and while I still think it accurate, I am not satisfied with its arrangement nor with the amount of detail its time limit permitted. The immediate symptoms were not separated as they should have been from those that supervene later in the course of the disability. The early signs should have been emphasized, because success in treatment must depend largely on prompt diagnosis. It is easy enough to recognize one of these cases when atrophy has developed and the lapse of time has shown the persistent character of the lesion, but to make the diagnosis on the day of, or on the day after, an injury is quite another matter.

Probably insurance records would show that 80 or 90 per cent of employees complaining of shoulder "strains," return to work within three months. Certainly we could not recommend exploratory incision of the bursa in all of these cases in order to detect perhaps 10 or even 20 per cent where the rupture would be complete. When we have learned just what to do when we find minor ruptures or tendinitis, it may become wise to make such incisions as a routine, but at present the bill for negative explorations would be far too large. I contend that it is possible to detect the severe cases.

CERTAIN CONDITIONS, SYMPTOMS AND SIGNS WHICH INDICATE COMPLETE RUPTURE OF THE SUPRASPINATUS TENDON AND WHICH SHOULD BE PRESENT WITHIN TWENTY-FOUR HOURS AFTER THE ACCIDENT.

(1) Occupation—labor.
(2) Age—over 40.
(3) No symptoms in shoulder prior to accident.
(4) Adequate injury—usually a fall.
(5) Immediate sharp, brief pain.
(6) Severe pain on following night.
(7) Loss of power in elevation of the arm.
(8) Negative X-ray.
(9) Little, if any, restriction when stooping.
(10) Faulty scapulo-humeral rhythm.
(11) A tender point,
(12) a sulcus, and
(13) an eminence
(14) at the insertion of the supraspinatus,
(15) which cause a jog,
(16) a wince and
(17) soft crepitus as the tuberosity
(18) disappears under the acromion when the arm is elevated, and usually also, as it reappears during descent of the arm.

Here are eighteen conditions to be fulfilled—an especially exacting syndrome. If such a syndrome is present I do feel that not only is exploration indicated but that it should be strongly urged, *for immediate suture should be a simple and successful operation. Delay means retraction of the tendon and a much more serious problem.*

I feel confident that this syndrome must exist, although I admit that I have never seen one of these cases within twenty-four hours of an injury. My best way of knowing the immediate symptoms is from the accounts of the patients or of their physicians given weeks or months after the injuries. Moreover, since these same symptoms are found at varying periods from three weeks to many years after the accidents, and do not vary much with the lapse of time, either in quality or in degree, it is likely that they were present at first. In a case in which they were all typical I should be positive of the diagnosis, and should urge operation. If several of the conditions were not fulfilled, it would influence me against operation, but if there were doubt, a negative exploration, if correctly performed, is a trivial matter, although the patient must be hospitalized in case a rupture is found.

If suture is done he should remain in the hospital for about ten days; if the exploration is negative he might well be discharged in twenty-four hours. These eighteen points will be discussed in more detail in numerical order.

(1) *Occupation.* The great majority of cases must belong to the laboring classes, for I have seen only one case in a person whose occupation did not or had not required heavy work. This suggests that overuse as well as increased liability to accident may be a contributory cause. The occupations are given serially in the following paragraph because if they were tabulated the list would not give the same impression of sequence which is presented by patients as they come for examination. On looking over these occupations the reader should contrast them with those in the following three paragraphs which are the occupations of patients who have had calcified deposits, tendinitis and partial rupture.

COMPLETE RUPTURE OF THE SUPRASPINATUS
(100 Cases) Women 8%

Housewife, hostler, plasterer, street cleaner, housewife, coal-heaver, waiter, paper cutter, laborer, housewife, marble worker, currier, cooper, housewife, stationary engineer, two laborers, longshoreman, wrecker, teamster, two laborers, steamfitter, three laborers, truck driver, stock fitter, cook, stableman, painter, two laborers, truck driver, laborer, lineman, lather, farmer, three laborers, harness maker, wood molder, planer, electrician, plumber, mechanic's helper, laborer, roofer, laborer, longshoreman, riveter, two laborers, porter, cooper, two laborers, steamfitter, laborer, lather, steamfitter, laborer, stationary engineer, laborer, store clerk, carpenter, laborer, night watchman, longshoreman, laborer, taxi driver, lineman, laborer, painter, coal-heaver, laborer, foreman, truck driver, laborer, construction, painter, laborer, rubber worker, laborer, painter, laborer, carpenter, meat cutter, floor layer, stitcher, two laborers, housewife, foreman, laborer, store clerk, burnisher, teamster, laborer.

CALCIFIED DEPOSIT (100 Cases) Women 34%

Housewife, two no occupation, two physicians, chemist, physician, superintendent, two physicians, business, no occupation, business, manufacturer, architect, business, three physicians, milk delivery, supervisor, ironworker, housewife, filing clerk, physician, machinist, histologist, housewife, physician, business, no occupation, physician, no occupation, pipefitter, stenographer, no occupation, physician, garage, postman, business, waitress, musician, shipper, laborer, bookkeeper, machine tender, porter, teacher, laborer, housewife, broker, housewife, business, baker's helper, two salesmen, organist, weaver, housewife, shoemaker, forewoman, boxmaker, two laborers, housewife, shoe machinist, farmer, housewife, advertising, paper mill, housewife, surgeon, real estate, advertising, housewife, machinist, boxmaker, store manager, wool handler, laborer, physician,

truck driver, shoe worker, teacher, knitting mills, housewife, plasterer, laborer, shoe stitcher, clerical, physician, treasurer, investments, laborer, housewife, laborer, beauty parlor, manufacturer, salesman, manufacturer.

TENDINITIS (100 Cases) Women 58%

Two housewives, two no occupation, army officer, two housewives, carpenter, tailor, nurse, no occupation, tailor, nurse, business, P. O. clerk, secretary, three housewives, no occupation, physician, maid, coppersmith, merchant, housewife, jeweler, minister, harnessmaker, housewife, hostler, tailoress, storekeeper, priest, no occupation, photographer, housewife, professor, housewife, carpenter, starter, housewife, shoe laster, housewife, two no occupation, housewife, roofer, civil engineer, two housewives, cigar maker, judge, shoe manufacturer, no occupation, nurse, three housewives, physician, housewife, laborer, two housewives, physician, two housewives, store clerk, housewife, social worker, housewife, insurance broker, factory worker, two housewives, desk work, social worker, salesman, food checker, housewife, consulting engineer, banker, physician, invalid, two housewives, nurse, laundry, writer, surgeon, two housewives, two physicians, foreman, lawyer, grocer, executive secretary, nurse, public accountant, no occupation.

PARTIAL RUPTURE (100 Cases) Women 11%

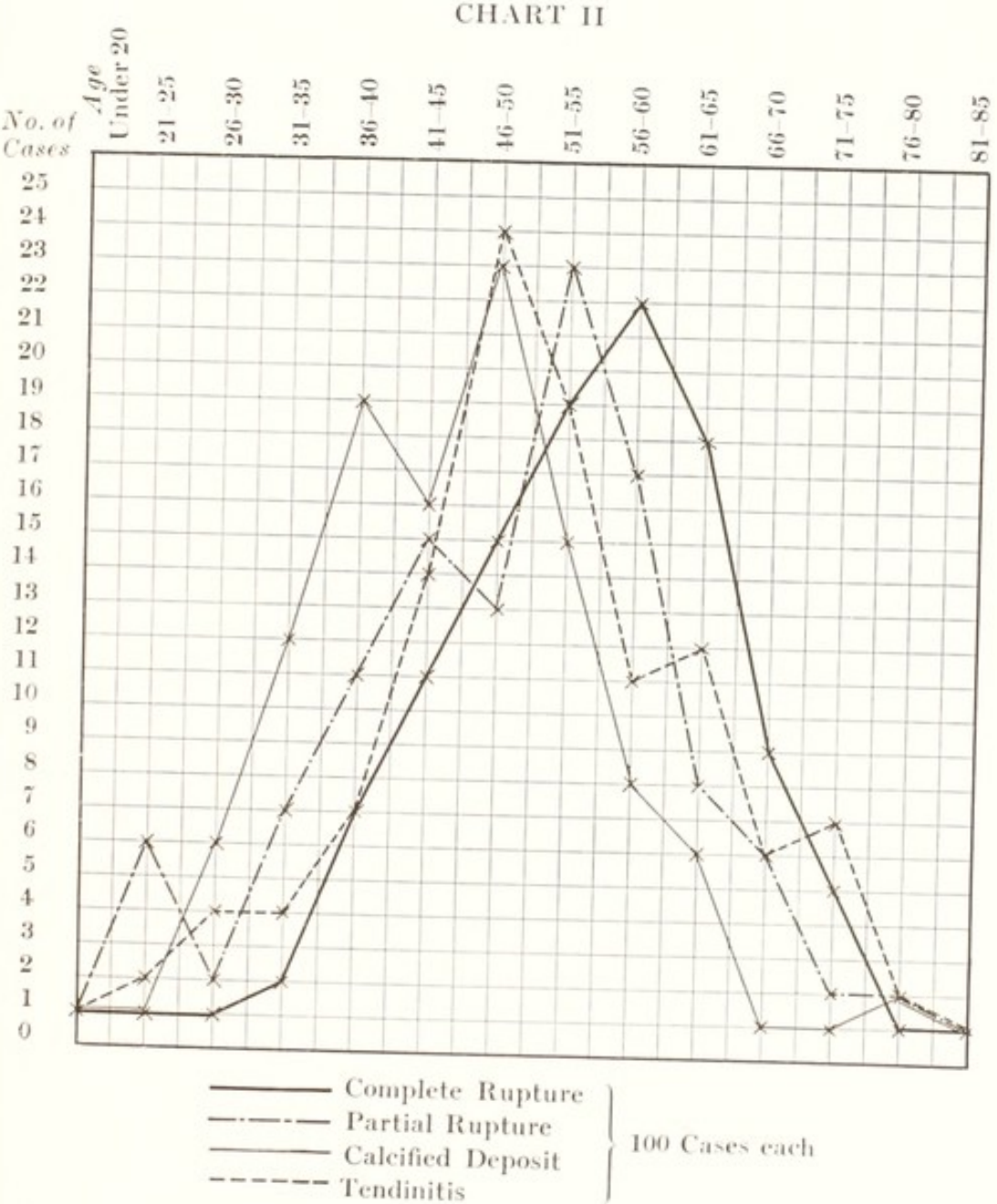
Three laborers, farmer, carpenter, eight laborers, baker, plumber, laborer, housewife, two laborers, carpenter, housewife, painter, laborer, mechanic, garage, two laborers, writer, three laborers, physician, laborer, carpenter, teamster, laborer, carpenter, laborer, machinist, laborer, housewife, teamster, cook, stone mason, bricklayer, carpenter, three laborers, store, housewife, laborer, mechanic, housewife, store clerk, cook, machine oiler, laborer, two housewives, insurance, store, business, janitor, hoisting engineer, painter, meat cutter, three laborers, florist, two laborers, ironworker, nurse, gardener, shoe factory, clerk, plasterer, lawyer, laborer, no occupation, student, bartender, manager, housewife, laborer, foreman, bricklayer, horseman, two laborers, nurse, farmer, machinist, real estate, gardener, laborer, housewife, tailor.

Both occupation and sex are of importance in the diagnosis of shoulder conditions. Men who have done heavy labor are typical subjects for complete ruptures of the supraspinatus, and women of the so-called "leisure class," for tendinitis (frozen shoulder). Calcified deposits are more characteristic of the class who have gainful but not laborious occupations (the white collar class); *i.e.*, they are not usually found in inactive people. Partial ruptures are also in the main characteristic of men of the laboring group, but they may occur in the more active and athletic members of the leisured class, both in men and in women.

These observations were already made from general impressions in the course of my practice, but they are in part confirmed by the above analysis of the occupations of 400 cases. The detailed accounts which each patient has given of his or her occupation and other activi-

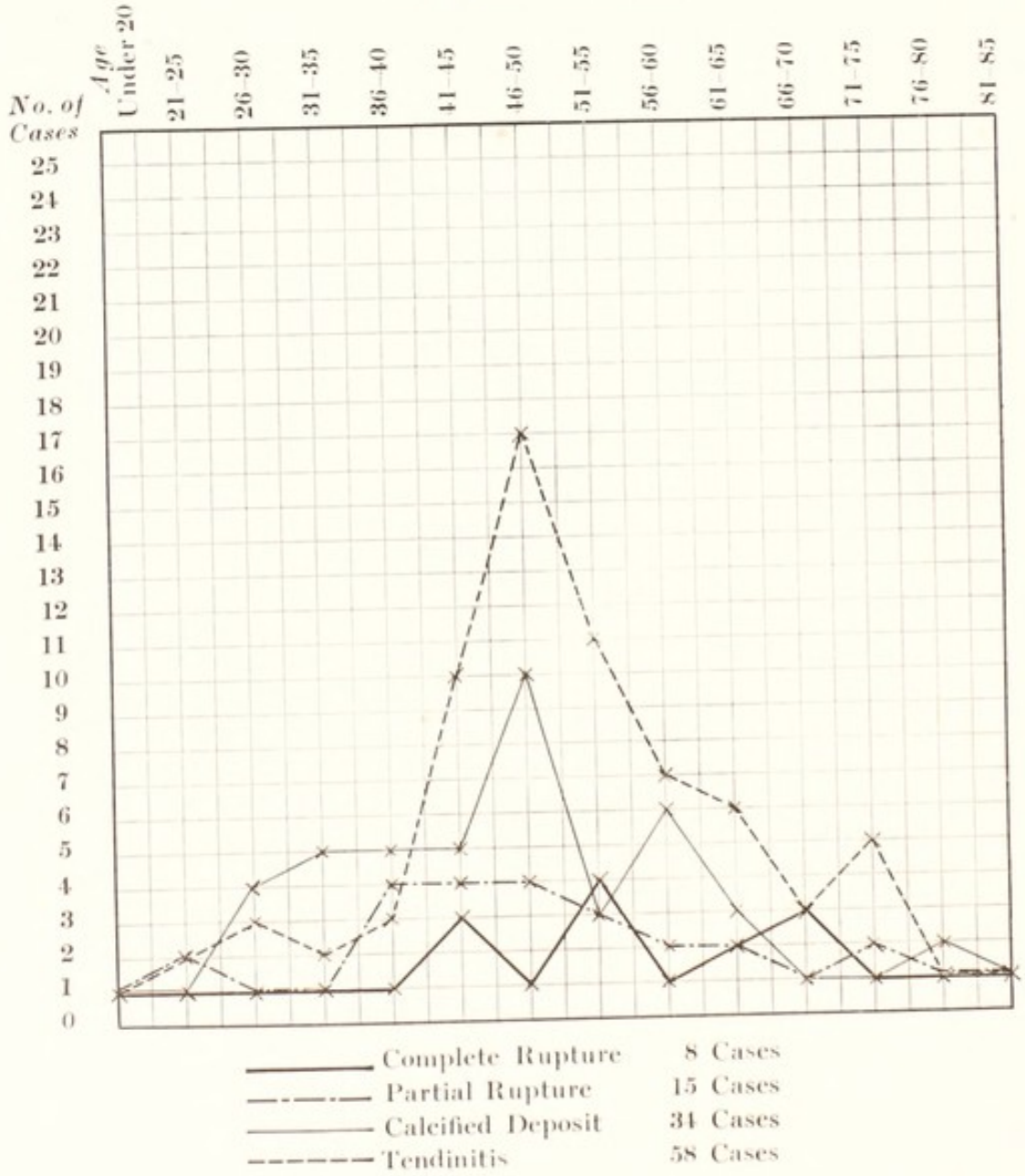
ties, are of even greater weight in confirming my own impressions. For instance, the term housewife may apply to a woman who does all the work and washing for a large family, or to a lady who scarcely uses her arms, or to an active wife who plays golf, sends out her washing and only occasionally uses her kitchenette. The percentage of women varies greatly in the different classes, eight per cent, eleven per cent, fifty-eight per cent and thirty-four per cent. The eight cases of complete rupture and eleven of the fifteen cases of partial rupture were women whose work was really laborious. The fifty-eight tendinitis cases were chiefly women of the leisure class and the thirty-four calcified cases were active single women or wives.

CHART II



(2) *Age.* The four clinical entities also affect different age groups. Young persons below twenty-five seldom have any of these conditions. Apparently in young people the tendon is stronger than the bone in which it is inserted, and stresses, which in later life would break the tendon, cause fracture of the bone or separation of the tuberosity. Chart I shows that the four entities affect, in the main, persons in the latter half of life, and that the occurrence of tendinitis both of the calcified and uncalcified forms precedes the peaks of incidence of the ruptures, partial and complete, by about five to ten years. The curves of the two forms of tendinitis, calcified and uncalcified, have their peaks at the same period, but the rise of the curve is distinctly earlier in the calcified form. These facts suggest that injuries

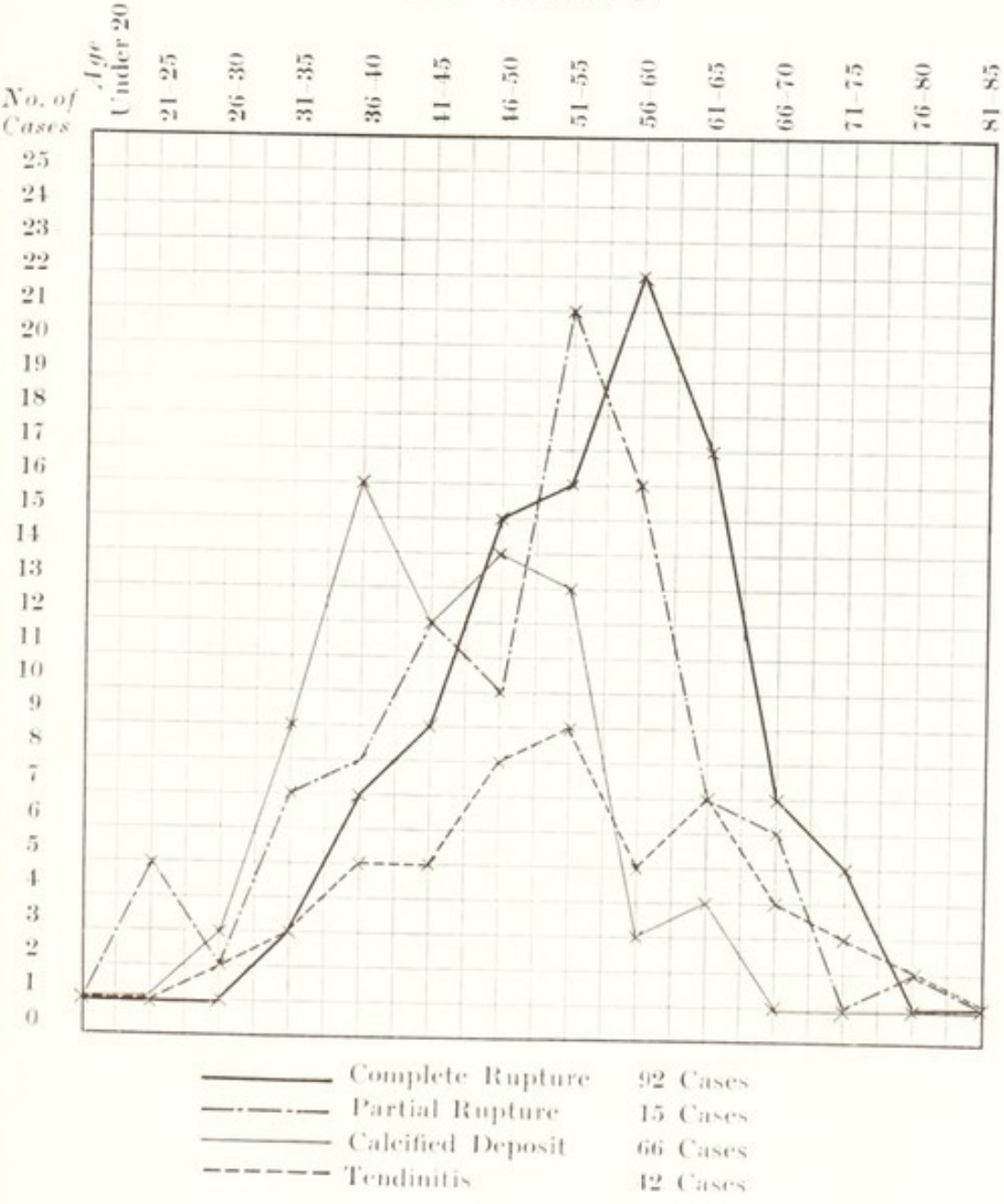
WOMEN—CHART III



of the tendon prior to the thirtieth year either are rare or that the tendons are capable of normal repair. Then follows a period when repair is uncertain and is apt to be complicated by the deposit of calcium. Later even this incomplete repair fails.

If we chart separately the male and female cases there is a marked contrast. The curve of the female cases suggests that tendinitis, both calcified and uncalcified, may be related to the menopause or to the age when the teeth begin to loosen. By comparison of the two charts we find that calcified deposits apparently occur somewhat earlier in males than in females, and their occurrence tends to diminish at the period when they are highest in the female. The contrast of Chart II and Chart III makes it very clear that the more serious forms of rup-

MEN—CHART IV



ture of the supraspinatus are characteristic of the male toward the end of his laboring days. The period of life shown in all the curves is that in which the teeth are usually in decay.

If these charts are made without separating the cases in four entities the contrast between the ages of males and females is even greater, so that the coincidence with the menopause is more striking.

(3) *The history of a previously painless and useful arm* is unreliable for two main reasons. First,—previous troubles may have been forgotten. Men of the laboring classes put up with a great deal of soreness and pain and forget it easily. Such a matter as an attack of bursitis years before, may readily be forgotten. I would rather have the history of previous troubles from a man's wife than from the man himself. I am inclined to think that in many cases where complete rupture has occurred, there may have been previous minor troubles, which might have indicated either partial rupture of a few fibers, or a "calcified deposit." However, my records do not show this, for in only nine of a hundred cases could a history of previous trouble be obtained.

The second reason is that men may lie as well as forget. The statements of employer and fellow workmen are sometimes more accurate than the patient's own. Patients may conceal previous trouble to avoid losing compensation.

The other shoulder should always be examined in these cases, for occasionally one finds signs of a partially ruptured tendon or of chronic bursitis there, although no complaint is made of symptoms. This would, of course, be very suggestive of preëxisting disease in the injured shoulder. However, we must not forget that a rupture may occur as a *bona fide* injury in degenerated or diseased tendon.

(4) *Adequate injury* seems pretty well illustrated by the following brief accounts of the accidents in twenty-one successive cases which were proved by operation, and in seventy-nine others in which the diagnosis was made but not proved.

ACCIDENTS (Operated Supraspinatus Cases)

57. "Fell taking down an old gallery."

75. "On this date (Aug. 25, 1922) he was engaged with others in hoisting lumber, when a plank slipped off the hoist and struck him in the side knocking him down."

83. "On Jan. 17th, 1923, he fell downstairs and injured his left shoulder."

88. "On Feb. 1st, 1926, while at work he was pulling a heavy case along the floor with a hook. The hook slipped and as he caught his balance he felt something snap in his shoulder accompanied and followed by intense pain."

89. "When getting off truck caught his hand in fly wheel. Cuts of hand."

96. "On April 4th, 1927, he slipped on a concrete floor and injured his left shoulder."

97. "On Nov. 8th, 1926, he was pushing a freight car with another man, using bars. His bar slipped and he fell down wrenching his left shoulder."

98. "Six weeks ago he had slipped on the ice while at work and had injured his right shoulder. He fell on his back striking his elbow, but had no bruise on the elbow. (It is probable he threw up his arm to get his balance.)"

102. "On Nov. 2nd, 1927, he was piling some 100-lb. sacks of beans with another man who stood above him and received the sacks as he threw them up. On one throw the other man failed to connect with the sack and the entire weight came on the patient's right arm. He felt something in his shoulder snap with a sharp pain."

106. "On January 20th, 1928, he was cranking his truck, which he uses to deliver cans of ice cream. The engine backfired and he felt a sharp pain in his right shoulder."

107. "On Jan. 18th, 1928, she fell on the floor of the kitchen where she works and dislocated her left shoulder."

108. "At some time in July (date uncertain) while at work he was turning a board and felt something snap in his right upper arm about the middle, in the region of the long head of the biceps. There was a sharp pain which went away in a few minutes."

112. "On March 10, 1928, he was wheeling a barrow up into a barn when he slipped and fell forward on the barrow, but did not let go of the handles."

115. "On August 21st, 1928, about 1.30 P.M., slipped and fell from piazza roof, striking the edge of the piazza floor with his right arm in abduction. Had immediate pain in shoulder and arm."

119. "Day before Thanksgiving, Nov., 1928, fell off staging three feet high. Walked backwards off."

123. "On Jan. 14th, 1929, he slipped on an icy platform and hit on his left elbow."

127. "On Oct. 17, 1929, was handling 2 x 4 lumber and stepped in a hole. Fell against left shoulder on pile of lumber, hitting on elbow."

128. "He slipped on the ice near a building and fell, striking the side of his right shoulder against a low step. This is what he says, but such a fall as that would probably be impossible. It is more likely that he threw his arm up as he fell."

129. "On October 24th, while piling some pipe, he was standing on a pile of pipe which rolled under his feet and he fell between two piles of pipes."

135. "He slipped on the ice and fell. After he got up, he found that he could not raise the left arm."

137. "On May 13, 1930, he had a fracture near the left elbow which healed satisfactorily and he went back to work about September 1st. On October 7th, in Andover, he fell and dislocated his left shoulder."

ACCIDENTS (Unoperated Supraspinatus Cases)

". . . he was doing some overhead work standing on a support twelve or fifteen inches high. The support slipped and he fell on his right hand and elbow, and then forward on his shoulder." ". . . he was pulling on a rope which suddenly gave way. He fell to the ground with his right arm below and behind him. He felt a sharp pain in the shoulder as if a bone had broken." ". . . he fell about ten feet while at work and injured his shoulder." ". . . he had been hit by an automobile, knocked down and taken to the Boston City Hospital. His shoulder had been injured." ". . . he was cranking his truck, and had a kick back. He thinks the handle struck him on the lower part of his upper right arm, but his shoulder was wrenched." ". . . when walking in a dark passageway, he stepped over some steps and in trying to save himself caught with his left arm on the wall, but kept his feet. He had a violent pain in his left shoulder but pulled himself together and went to his next job." ". . . he was struck on the left shoulder by a falling bale of hemp while at work. He was knocked down and much shaken up, but pulled himself together and continued to work the rest of the afternoon with his right hand." "Fell off wagon when unloading barrels. Hurt badly at time and went to Camb. Relief Hosp." ". . . he was lifting a barrel and something snapped in his shoulder, since which he has been unable to work." ". . . he tripped on a rolling log and fell injuring his left shoulder." ". . . he fell down some stairs and injured his right shoulder." ". . . the employee slipped on a loose plank and injured his left shoulder and right foot." ". . . he was pulling on a chain fall and something slipped in the right shoulder." ". . . he was jammed between a moving truck and the side of a building. Fortunately, he was near the corner of the building and the moving truck carried him around the corner, squeezing him from side to side as it did so. His left arm immediately became powerless." ". . . he fell from a staging and injured his right shoulder and has not yet recovered the use of it for anything requiring the function of abduction." ". . . he was filling a wagon with coal when the horses started and he fell in between the wagon and the side of the coal-pocket." ". . . he fell forward at the top of a flight of stairs and injured his shoulder as he supposed by hitting some beams." "A frame which he was moving dropped and to prevent it striking his feet stepped aside, losing his balance and falling to the floor." ". . . she felt something snap in her shoulder (left) when raising some wet clothes on a pole to put them in a laundry machine." ". . . while raking grass on a steep bank, he slipped and injured his left shoulder." ". . . as he was stepping out of a tip cart on the hub of the wheel, he slipped and fell heavily to the ground injuring his left shoulder and also his right shoulder to some extent." ". . . the employee was carrying a plank with another man, when one of the planks on which he was walking gave way, letting part of his body through the pier so that he sustained injuries to his left shoulder, arm and leg." ". . . she fell over a bag of soles and injured her right shoulder." ". . . he slipped off a plank and fell headlong to the floor, about four feet." ". . . large heavy car six or eight feet on the side. On this occasion the car skipped the track in spite of his efforts to prevent it, striking him on the left arm." ". . . he says that he was using his truck

to load heavy rubber bales weighing about 350 lbs. each. He had put the edge of his truck under one such bale and reached forward with his right hand to pull the bale toward him on to the truck. As he pulled, he felt something give in his right shoulder." ". . . he was helping with other men, to pull a heavy truck, when he felt something give way in his right shoulder." "He was carrying a roll of leather and fell on the floor in the room where he usually worked." ". . . he fell from his truck and injured his left shoulder." ". . . he fell thirty feet from a staging and was badly bruised all over." ". . . he was on a lumber pile helping to load a truck. He was using a pick to drag the lumber. The pick slipped and he felt a sudden pain and something snapped in his arm at the right shoulder. His arm fell limp at his side." ". . . he slipped on some stairs and injured his left shoulder. . . ." ". . . fell among some barrels in the hold of a vessel and struck his right shoulder again." Had had previous similar accident six years ago, and never full use of arm since. ". . . while working in a meat market in Nantucket, he slipped on going out of the ice chest and injured his right shoulder." ". . . while carrying rubbish on an incline in the factory where he works, he fell and injured his right shoulder. As he says, 'it went dead immediately.'" "He was piling wool at the time, lost his balance and fell from one bale to some other bales not far below. As he fell he felt something snap in his shoulder which did not hurt him very badly at the time. . . ." ". . . slipped on ice in street." ". . . while working on a flat-car he fell and struck his right shoulder on the side of the car." "While at work in the factory in Lawrence . . . he was in an elevator holding a heavy plank in both hands to steady it. One end of the plank was on the floor of the elevator and it stood vertically. Some one below started the elevator downward and then upward. As it went up it thrust the top of the plank violently against the top of the well. This shattered the heavy plank just above his hands with extreme violence and he was thrown into the corner of the car in a heap." ". . . he fell off the back of a load of straw and injured his shoulder." ". . . he again fell downstairs—only a few steps—and again injured his right shoulder." "Slipped and fell, and a box or case fell over on him." ". . . while directing some work where a floor was being replaced and the boards were up, he tripped on a beam and fell on his right side against another beam, probably breaking some ribs." ". . . he received an injury to his left shoulder when trying to move a large rock with a pitchfork. He felt something snap in his shoulder joint and suffered a sharp, severe pain at the same time." "He injured his right shoulder by falling from a truck." ". . . he was pulling some lumber off a truck and when it slid off quicker than he expected he fell backward and injured his left shoulder." ". . . he fell four feet, striking on the pavement, and injured his left shoulder." ". . . he slipped on an oily floor, and hurt his right shoulder and left shin." ". . . he fell from a staging about six feet to the floor below, and injured his right shoulder." ". . . he fell from a ladder and sustained injuries to the left shoulder and ribs." ". . . he was on a roof sawing a plank, and was standing on an extension ladder, which fell with him thirty-one feet to the ground. His right shoulder was injured, and his nose was cut." "Strap from machine fell off wheel and hit patient on right shoulder." ". . . he was working with others on a bridge in Rumford, Maine, helping

to receive some cement in a frame from a bucket. In some manner, the bucket swung in the wrong direction and the patient fell from the bridge a distance of fifty feet." ". . . he was lifting a concrete block and felt something give in his left shoulder." ". . . he fell injuring his left shoulder." ". . . he had started to crank a hoisting truck when the starter began to work and the crank flew round and struck him on the right shoulder." "Slipped on ice." "Fell yesterday and sustained a contusion over outer end of right clavicle." ". . . while helping to unload a truck, a derrick knocked down a platform above him, and something, probably a heavy plank, fell on him and others working with him. He was knocked out by the blow, and cannot describe exactly the manner in which the plank struck him, but he knows it injured his shoulder and elbow and there was blood streaming down his arm." "He was pulling hard on a gunny sack, which gave way, and he fell over and thinks he struck his right shoulder." ". . . she tripped and fell on the floor at her work. She dislocated her right shoulder, bruised her knees severely and cut her face." "He was standing on stony ground swinging a sledge hammer, when he had to step back quickly. As he did so, he lost his footing and the sledge hammer, which he was swinging, carried on around his body so that his left shoulder was in an awkward position. He felt something snap in the left shoulder."

One may interpret the mechanism which produces this injury in several ways, but a *sudden* character is common to all of the accidents, which are generally falls. It is my belief that the rupture usually takes place from sudden elevation of the arm in attempting to regain balance, particularly if the hand is at the same time grasping a heavy object. Under these conditions a tremendous strain must be suddenly thrown on this little tendon as it attempts to quickly overcome the inertia of the arm, and perhaps, in addition, that of some heavy object held in the hand. In my first case, the woman attempted to throw a heavy, wet blanket over a clothes-line. It seems to me that this case, like a "slowed down movie," typifies the kind of strain which occurred in most of the other accidents. I believe that the even more sudden effort to regain balance during a fall caused the damage, probably before the patients struck the ground. For anatomic reasons one cannot, in falling, strike on the supraspinatus, because it is protected by the acromion.

Undoubtedly, however, in some cases, the tendon may have been torn in conjunction with dislocations, because of the leverage of the humerus on the fulcrum of the acromion. This mechanism will be explained in Chapter IX.

(5) *A sharp pain* in the shoulder at the time of the accident is almost always spoken of, although occasionally complaint of it is not volunteered. Sometimes patients say that they have felt some-

thing actually snap and think that they have broken a bone. Sometimes they feel that something has struck them on the shoulder. It has been explained on page 9 in the chapter on anatomy that histories of striking on the head of the humerus are unreliable because the acromion intervenes, and on page 144 that in falling, the arm is usually raised before the top of the shoulder can strike the ground. Consequently it seems to me that these tendons must usually be ruptured by indirect violence or sudden efforts of the muscles to overcome the inertia of the dependent arm, especially if there is a weight as a pick or shovel in the hand at the time, or the hand grasps something to save the man a fall. Often the fall is so sudden and the man so confused that the only thing he can understand is that he has hurt his shoulder and attributes the pain to having hit something as he fell.

(6) So far as I can judge from histories, there is then usually an interim of a few hours after the acute, immediate pain has somewhat subsided before the more severe pain comes on. Often the employee does not even consult a doctor at once, but tries to work the day out, favoring his arm. Perhaps he does not report the accident to his foreman. *In the evening the pain becomes worse*, and later in the night intolerable. He calls the doctor, or sits up in a chair, or "walks the floor." Next day he is pretty sure to report that he cannot work, but may persuade an accommodating foreman to let him "hang around" for a day or two until he gets better. These patients usually think the injury of no great consequence and expect "to have it wear off." This hopefulness is generally confirmed by the doctor's opinion, who perhaps may never have heard that such lesions occur. This attitude of mind of both patient and doctor is the main cause of delay in diagnosis and appropriate treatment.

It seems to me that the following theory is the probable explanation of the interim between the sharp pain when rupture occurs and the intense pain which appears some hours later. These tendons are not very vascular, and when they tear, there is probably very little bleeding; what there is, would come from the tissue between the bursa and the tendon. The interim spoken of would come during the period it would take this slight hemorrhage to distend the joint and bursa somewhat, *i.e.*, enough to start a tension pain. This would create some spasm, and the tension caused by this would stop the slight bleeding. It would take several nights or perhaps a week for tension and spasm to subside and the hemorrhage to absorb. During this period the acute pain would continue.

(7) *Inability to raise the arm* is a constant symptom, but one must be on guard not to mistake unwillingness for inability. After almost any shoulder injury there may be pain when attempt is made to raise the arm, owing to the fact that the head of the humerus has to be forced upward to gain its fulcrum on the glenoid. The mere fact that the muscles have to exert tension to do this, causes pain in whatever structure about the shoulder may be injured. Therefore, the examiner must be sure that an honest effort is made to ignore the pain and elevate the humerus. It takes experience to tell whether such an effort is made, and one judges it by the degree of tension palpable in the deltoid. Even in the case of trivial injuries, such as ruptures of a few fibers of the supra- or infraspinatus, the symptom of inability to raise the arm may be pronounced, simply from the fact that the power to exert the appropriate muscles is inhibited by sensitiveness to pain. As explained on page 59, the deltoid needs the assistance of the supraspinatus and of the short rotators to hold the head of the bone on its fulcrum in order to have proper direction for its power. If the supraspinatus is torn, contraction of the deltoid brings the arm upward on the vertical axis of the humerus, and the amount the shoulder is raised will depend on the amount that the scapula, moving via the sterno-clavicular joint and at the acromio-clavicular joint, can rise and rotate on the chest wall.

Formerly I thought that it was necessary to have this symptom of inability to raise the arm absolutely demonstrable in order to make the diagnosis of rupture of the supraspinatus, but experience has shown that, even when the supraspinatus is torn across its full width, the other short rotators can sometimes hold the head of the humerus on its fulcrum sufficiently to permit the patient to weakly perform elevation. However, as will be explained under No. 10, this elevation is never accomplished with a normal scapulo-humeral rhythm.

(8) *A negative X-ray* is almost always reported after these injuries. I believe that in the near future we shall be able to make the X-ray of more use in this diagnosis, either by using injections into the joint of non-radiable fluid, or by developing a soft tissue technique which will show the rupture. However, at present, negative X-rays are the rule, for ruptures which do not involve the bony facet of insertion are not shown in the film. A negative X-ray is of some positive importance, however, for it rules out the two conditions which are likely to make confusion in the diagnosis, that is, fracture of the

greater tuberosity and the presence of calcified material in the tendon. In long-standing cases changes in the structure of the trabeculæ of the tuberosity which may be shown by the X-ray do take place. These are described on page 92.

(9) In the symptom complex of this condition, *lack of restriction* of motion takes a very important part, and this lack of restriction can be best determined when the patient is stooping from the hips with the knees extended. The patient should stoop (Fig. 47) to the horizontal position, letting the arms hang loosely toward the floor. In this position the deltoid is relaxed and there is no fulcrum needed in order to have the arm passively raised, *i.e.*, brought forward into complete elevation (quadruped extension); in fact, even if this is not passively done, the patient has to exert but little muscular power to swing the arm forward into this position. This he is usually able to do without much pain. The examiner may then hold one hand on the scapula and with the other raise the lower end of the humerus, so that he takes the full weight of the arm and permits the patient to stand upright with the arm still in complete elevation. Such a procedure effectually rules out restriction from adhesions. Even if the supraspinatus is torn, the patient can retain the arm in this upright position. If he stoops again he can lower it without much pain, and then if he rises to the upright position with the arm relaxed, the humerus will, by gravity alone, come into its normal position at the side of the body.

When I say that lack of restriction is an essential symptom, I must not be taken too literally, for there is often, in fact usually, in these cases, a very little restriction in extreme elevation and in rotation, probably due to the presence of fluid.

(10) *Faulty scapulo-humeral rhythm* is a *sine qua non* for this diagnosis. When one sees a patient who in raising the arm lets it ascend to the horizontal while maintaining (quadruped) flexion of the scapulo-humeral joint, and then slowly and painfully (perhaps with a little help) proceeds to complete elevation by motion in the scapulo-humeral joint, and finds that when the patient allows the arm to descend, he keeps the scapula and humerus fixed in (quadruped) extension until he reaches the horizontal, and then quickly flexes it, a presumptive diagnosis of rupture of the supraspinatus can be made. Ascent in flexion, descent in extension, might be a slogan for students to learn in this connection. To express this lack of scapulo-humeral rhythm in other words, we may say that the normal ratios of the movements of the joints in elevating the arm, explained

on page 59, disappear. Instead, in the first part of the movement only the motions of the scapula on the chest wall are concerned; then the relations of the humerus and scapula change, wholly above the horizontal. In the descent of the arm the reverse is the case—no scapulo-humeral motion takes place above the horizontal, but all below it. While the symptom is a *sine qua non* to the diagnosis of complete rupture of the supraspinatus, it is also present in most cases of minor ruptures and in many cases of calcified deposits. It therefore is an indispensable but not a pathognomonic sign.

(11) *The tender or sensitive point* is not complained of by the patient as a rule, and in fact he is unconscious of its exact location until the examiner finds it, when he usually says: "You have your finger right on it." Without your aid in locating it he will perhaps know that there is a tender point, but locate it deep under the acromion or even in the spasmodic deltoid muscle down near its point of insertion. In fact, the lower portion of the deltoid is usually also tender. Examination of this part of this muscle in all these patients nearly always shows that there is some thickening and sensitiveness, as compared to the normal side. In the old, chronic cases the sensitiveness at the point of rupture may not be very noticeable, and even when the exact point is pressed the patient will hardly admit that it is tender. Presumably in fresh cases it would be especially sensitive.

In speaking of the 11th, one is necessarily obliged to consider the remaining symptoms, since the tender point is at the gap between the

FIGURE 41. POSITION OF HANDS FOR EXAMINATION OF SHOULDER

The left thumb lies along the depression below the spine of the scapula and the tip of the forefinger is just anterior to the acromion. The other three fingers cross and hold the clavicle. Thus the shoulder girdle is firmly held and any motion of the scapulo-humeral joint is at once detected.

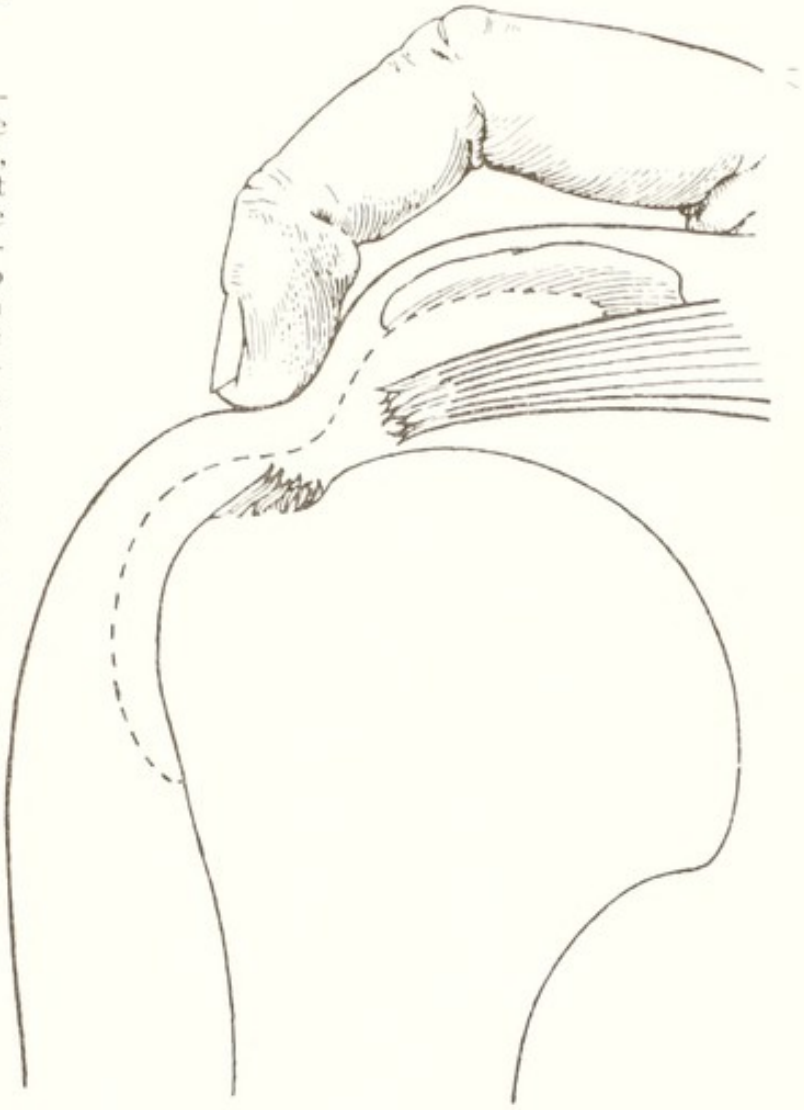


FIGURE 42. TIP OF FINGER PRESSING ON EMINENCE AND ON SULCUS

The plane of this diagram is halfway between the coronal and sagittal. It is, perhaps, the most important diagram in the book for the reader entirely to understand, for it is the ability to put the finger in this position which enables one to make the clinical diagnosis of rupture of the supraspinatus tendon. The dotted line represents the contour of the bursa. Compare this with Figure 44, which shows the contour of the bursa when filled with the calcified material, and also with Plate II, Fig. 3, which shows a large calcified deposit in exactly the situation in which the rupture lies in this diagram. In this one the sulcus is immediately under the tip of the finger and the eminence just external to it, but in Figure 3 the eminence would be just under the finger. Therefore, as explained on page 148, the tender point in a case of rupture is represented by a depression, but in cases of calcified deposit, by an eminence at the corresponding position.

ends of the torn tendon, and this gap is the reason for the sulcus and eminence, which may be felt just anterior to the edge of the acromion, when the arm is in dorsal flexion. If the examiner remembers his anatomy, the tender point, sulcus and eminence will be found to be at or near the insertion of the tendon of the supraspinatus. It is the passing of this irregular sulcus and eminence under the acromion and acromio-clavicular ligament which causes a jog, a wince, and a soft crepitus, as the sensitive, irregular base of the bursa disappears under the acromion when the arm is brought forward by the examiner. The two figures (41 and 42) present the condition, it seems to me, more vividly than could any description. However, I will give a few brief additional points under each one of the remaining headings.

(12) *The sulcus* is just about big enough to be filled by the tip of the finger, as indicated in the diagram. It is nearly always found to

be larger at operation than one would guess from palpation before incision.

(13) *The eminence* is an eminence only by contrast with the sulcus. It consists of normal tuberosity with perhaps a remnant of the tendon attached to it. In elderly men without injury to the shoulder one can often feel the tuberosity because the tendon is more or less atrophied, so that at times it is hard to be sure whether the tendon is torn or merely atrophic. However, in most cases of ruptured supraspinatus the eminence is conspicuously large and one is quite sure of its existence. It is well to say here that the eminence which is found in cases of calcified deposit is not on the tuberosity itself, but proximal to the tuberosity in the tendon at just the point where ruptures so often occur. Furthermore, the tenderness is usually greater on the eminence in cases of calcified deposit than it is on the eminence in the cases we are speaking of.

(14) As may be seen under Pathology, the supraspinatus is nearly always torn if any of the other short rotators are, but it is very common to have portions of the adjacent tendons torn, so that the tenderness, eminence and sulcus may be a little internal or external to the mid-point of *the insertion of the supraspinatus* itself. This latter may be determined pretty accurately by placing the forearm in flexion and drawing a line from the mid-point of the flexure of the elbow to the mid-point of the head of the humerus. The bicipital groove lies about its own width external to this line, and the supraspinatus is on the top of this line and to the outer side of it for about three-quarters of an inch. The insertion of the infraspinatus is just external and also partly in front of that of the supraspinatus, for, as explained in Fig. 6, the two insertions nearly cross each other. The insertion of the teres will be found nearly exactly on the mid-point of the head of the humerus on its outer aspect. Be careful, in determining this, that the forearm is flexed at the elbow and held straight forward. The insertion of the subscapularis can be determined by putting the arm in the anatomic position and placing the examiner's forefinger just external to the tip of the coracoid process, which is always palpable, as shown in Fig. 6.

(15) *The jog* is noticeable to the patient himself, and sometimes is visible as well as palpable to the examiner.

(16) The patient nearly always *winces* as the jog occurs, but in long-standing cases he may not do so.

(17) *The soft crepitus* is not like the crepitus in fractures. It is of a more velvety, gristly character. When one has become familiar with it, it is easily distinguished from the kind of crepitus often found in the shoulders of old working men, which resembles the crepitus one frequently feels over the prepatella and olecranon bursæ and about the other joints.

(18) When the sulcus, eminence and tender point have once *passed beneath the acromion* as the arm is elevated, there is a sense of relief on the part of the patient which is usually apparent in his countenance. When the arm is almost fully elevated (there is often so much fluid in the joint and bursa that absolutely complete elevation is not attained), the patient is relatively comfortable. His pain will appear again when flexion occurs at the level of the shoulder, after the arm has descended with humerus and scapula locked, to a horizontal position. At this time the jog and crepitus are usually again palpable. As this occurs the patient leans toward the affected side and lowers the whole arm quite suddenly.

These eighteen symptoms must be present soon after the accident, but the difficulty is to estimate the degree of the rupture at this time. Partial ruptures must give much the same symptoms as complete ones at this stage, and the degree of spasm must vary, as well as the courage of the patient as he makes a voluntary effort to raise the arm. Immediate diagnosis cannot be easy at this stage. However, the progress of the case makes the diagnosis easier and easier, although valuable time elapses. If exploration is not done these symptoms remain the same and the following points in the course of the case will tend to confirm the diagnosis.

Character of Pain. Practically every patient whom I have seen has given the history that during the first few nights after the accident the pain was severe or intense. Gradually this severe pain changes to a nagging, annoying one, sufficient to greatly interfere with the night's rest, but bearable without drugs. It is usually located near the deltoid insertion far below the actual lesion. Pain of this character continues week after week and with but little change for many months. It is aggravated by the attempt to work, and the patient's resistance to it is gradually lowered as he becomes more and more worn out by restless, painful nights. I am convinced that this pain is very severe as well as prolonged, for I have heard many strong laboring men state that they have never suffered such pain in their lives. It is more the persistence of it than the pain at any one time which wears them out. They often say: "If I could only get a good

night's rest I could work during the day." Practically they find that working during the day gives them bad nights, and therefore nearly all of them, in spite of their courage, give up work after a time. Of the series of a hundred cases, only eighteen stated that they had worked for even a brief period. They say that they may go to sleep for a while, but wake with pain in the shoulder or in the region of the insertion of the deltoid, and that they have great difficulty in getting the arm into a comfortable position again. When they do, they go to sleep only to wake up in a few hours for another change of position. Sometimes they get up and walk about or apply hot water bags or other household remedies. It is very characteristic of these cases to have complaint of pain out of proportion to the physical signs, and therefore they receive little sympathy.

Atrophy of the spinati, as shown by prominence of the spine of the scapula, always occurs after these injuries, but does not appear for about three weeks. After it has once appeared it persists, and is apparent for a long time even in the operated cases.

In very few of the cases that I have seen years after the injury was it absent. The atrophy may be more conspicuous in the infraspinatus, which is the larger muscle. Whether the fact that the infraspinatus is always also atrophied, is due to the crossing of its fibers of insertion with those of the supraspinatus so that they are always also torn to some degree, or is due to the fact that the two muscles are supplied by the same nerve, *i.e.*, the suprascapular, I do not know, but it is a fact that atrophy of both is a constant sign. Of course atrophy of these muscles occurs in any chronic condition of real severity affecting a shoulder joint, so that the presence of atrophy does not necessarily indicate this diagnosis, but its absence would be strong evidence against it. In a few long-standing cases I have seen only a small amount of atrophy. It is usually very pronounced. As a rule the deltoid is not much atrophied and may even be hypertrophied.

The general condition of the patient is a factor in diagnosis, for he gets into a vicious circle. He is out of work so that all the muscles of his body become enfeebled. He often cannot afford good food, and he may, therefore, be ill-nourished. Add to this the constant depletion of his energy from restless, painful nights, and we may readily account for the fact that while previously he was a strong, healthy man, he now appears haggard and unhappy.

The mental condition also is poor, for worry on account of inability to work, and that he may never be able to work again, is

enhanced by the fact that the physician he consults is unable to tell him the cause of his trouble, and all attempts to relieve him by ordinary remedies absolutely fail. Seeing these cases months after their accident, I am frequently told, "Nothing they have done has done it any good."

The actual physical deterioration from worry is still further aggravated by the doubt that is thrown on their veracity by the physicians employed by the insurer. Usually by the time they are sent to see me some months later, their attitude of mind is defensive, and they at once begin to express their disgust with being told that they ought to go to work and think less about the pain.

This attitude of mind becomes still worse when they are actually accused of hysteria or malingering. They say they want to work. "Do you think I would lie around like this if I could earn $24.00 a week?" They become embittered at their treatment by society in general in spite of the fact that they may still be receiving their compensation.

At length they may lose their self-respect, and brooding over their hard luck take to drink. My second patient was such a case. He had had a good job which he enjoyed; after his injury became discouraged, and evidently decided to let things go and to use up what money he had saved, in drinking as much as he had a mind to. The person for whom he formerly worked, instead of losing sight of him, looked him up, and, realizing that he must have some real trouble with the shoulder, sent him to a doctor who referred him to me. The result of the repair of his tendon was not only that he was able to work, but that he refrained from drinking and worked for ten or more years for the same people who formerly employed him.

Unfortunately the attitude of the relatives of such an old man with a disabled shoulder is apt to become somewhat like that of the doctors who have been unable to diagnose and relieve him. His own family after a while get to think of him as a burden, and since they can see nothing the matter, such as a limp or a deformity, are inclined also to think that he has "laid down" before his time. In recent years, however, we see more signs of sympathy, for the compensation such patients may receive will perhaps be the chief support of the family.

Undoubtedly many such cases eventually turn up as recipients of charity and eventually die in state institutions. It is not surprising to me that the material reported in this book, which

was accumulated by Dr. Akerson at a hospital for the indigent, shows such a high percentage of instances of these lesions.

I would venture to predict that if one should see the patients who are chronic nuisances to industrial insurance boards, and the physicians connected with the administration of compensation for industrial injuries, most of those complaining of shoulder disability would have this particular lesion.

Some patients may continue to work. There are rare individuals who, in spite of the disability, have the courage and otherwise sound health to continue to work in spite of the soreness, awkwardness, loss of power, and painful and restless nights. About one-fifth of my series attempted to work for a time before they gave in and sought compensation. I have no doubt that there are others who have never given in.

This is a lesion which tries a man's character, and, since it usually occurs in later life, is often the cause of permanent incapacity, for even if the use of the arm returns in good measure at the end of a year, the patient's habit of work has been destroyed, his muscles have become soft. If he has the courage to go to work again, he will find it difficult to get a job. Those courageous men who do work in spite of the lesion, become more or less free of serious symptoms in from two to five years. As has been explained under Pathology, compensatory changes take place so that the eminence absorbs, the sulcus partially fills, and an excess of fluid allays friction. After several years even the night discomfort disappears, and weakness in abduction, atrophy of the spinati, friction rubs, the fluid sign and occasional pain in certain positions may be the only aftermaths of the injury.

Hypertrophy of the Deltoid. Perhaps it would be better to make this heading "well-developed deltoid as contrasted to the spinati," for the hypertrophy is not striking except when compared to the condition of the spinati. It is a fact that, in the long-standing cases, the deltoid itself is as well developed, or even more so, than that of the other side. I explain this because it has to do most of the abducting work of the arm unaided by the supraspinatus. It not only misses its help, but acts at a disadvantage as explained in Fig. 3. Hence it retains its development or even hypertrophies. My third case had a deltoid like a ham, but at the end of five years he could only feebly perform abduction and could not raise even a slight weight in that hand above his head. He had refused operation.

The Fluid Sign. Among the auxiliary signs and symptoms I find some help from what I call "the fluid sign." I had studied shoulders for many years before I realized how fluid in the true shoulder joint behaves. When the arm is by the side, the fluid sags in the relaxed axillary portion of the capsule. When the arm is elevated the axillary portion of the capsule is stretched tightly below the rounded head of the bone, and the fluid is driven upward where the capsule is now relaxed. In case there is a rupture of the supraspinatus tendon, the fluid is forced through the gap and distends the bursa in the subdeltoid portion beneath the upper fibers of the deltoid.

Stand behind one of these patients, who is holding both arms as straight as he can toward the ceiling, and you will see that the contours of the two shoulders are quite different. When there is a considerable amount of synovial secretion, absolute complete elevation of the arm is prevented by the mass of fluid. Another interesting point is that when there is fluid the friction is largely prevented as the arm is elevated. When it subsides pain reappears. This phantom improvement by the formation of fluid is not uncommon.

Patients who continue to work in spite of their pain develop fluid, as do people with various affections of the knee. The fluid continually pumping in and out of the bursa dilates it and a true hydrops may result (p. 478). One finds in long-standing cases that the bursa is very large. (See case Fig. 44.) Some fluid, from a dram to an ounce, is a constant operative finding in these cases. It may not be noticed when the bursa is first opened, but if one elevates the arm and puts the axillary portion of the capsule on the stretch, the thick, straw-colored fluid runs out of the wound. Sometimes there is enough to distend the bursa even in the anatomic position, and when the first incision into the bursa is made, it obscures the field and has to be sponged out.

This behavior of the fluid was forced on my attention when I used to put the arm in abduction after suture. This caused the weeping wounds described on p. 248, and this serious and annoying complication led me to reason out the facts.

Incomplete Rupture

Although there is no sharp anatomic distinction between complete and incomplete rupture, there is the practical one that cases of the former will not recover completely unless the tendon is sutured, while cases of the latter may heal in a natural manner. One which is typi-

cally complete involves the whole supraspinatus tendon, with perhaps parts of the adjacent tendons. One which is typical of the incomplete form need neither involve the whole width nor run through the whole thickness of the tendon. In its minimum phase it may be only an evulsion of a few fibers from the tuberosity; in its maximum phase it would be a borderline case of complete rupture. In its acute phase it may be definitely due to trauma; in its chronic phase the tendinitis overshadows the traumatic history.

The reader who has the patience to finish this book will inevitably remain confused about the lines of distinction which I have attempted to draw among the six most common clinical entities which affect the shoulder, *i.e.*, complete and incomplete tendon ruptures, rim rents, calcified deposits, tendinitis and arthritis. In fact, I must leave the reader puzzled, for I am still puzzled myself. I can only confront him with the puzzle—show him that there is a puzzle. Personally, I believe that these rim rents and incomplete ruptures are the cause of the great majority of sore shoulders, and yet I believe they usually occur in degenerated tendons. For example, a man might have degenerative changes in both shoulders without symptoms and then, after trauma to one tendon, have severe local symptoms without any signs of trouble in the other shoulder.

One cannot even divide complete and incomplete lesions by the criterion of whether or not there is a direct communication from the bursa to the joint through the rupture, for the size of the opening is important also. Yet splitting hairs on a definition is not worth while. Practically, it is convenient to say that when one opens the roof of the bursa and finds the cartilage of the joint exposed to view through a rent in the floor of the bursa, we are dealing with a complete rupture. If there is no communication at all or only a small hole in the base of the bursa, we would class the case as incomplete. Of course the persistence of a direct communication between the joint and the bursa, even if small, is a most important point, because it alters the mechanics, as indicated on the cover of this book. Yet the size of the hole is also important if it is large enough to permit erosion of the joint cartilage (Plate VII, Fig. 8) by friction on the acromion.

It seems to me highly probable that incomplete rupture is much more common than complete. Since I have only operated upon cases where very pronounced symptoms existed, I have naturally found many more instances of the complete form, but in my observations on the cadaver, or on living cases in the clinic, I have much more often found incomplete lesions. I have not kept a numerical account and therefore cannot give the exact number or even the proportion.

Dr. Akerson's statistics give the best measure we have, but are subject to the criticisms on page 65. Similar observations may be made by any one having access to autopsy material. Moreover, when the economic importance of this lesion is appreciated, and the principles of efficiency become applied to the practice of medicine, such observations must be made.

I am inclined to think the table on page 469 also gives a low proportion of the incomplete ruptures, because so many cases in the other classes probably had this lesion besides the lesion which caused their classification. For instance, rupture of the supraspinatus is a not uncommon complication of dislocation and of circumflex paralysis, and many of the minor unclassified cases may have also been instances of this lesion. On the other hand, I have usually found at operation that the rupture was more extensive than I had previously thought, so that a good proportion of the supposed incomplete type may have been complete.

Referring to the chapter on Pathology, it will be seen that incomplete ruptures of four kinds are described:

(*a*) A few of the lower fibers on the joint side, together with the synovial reflection, may be torn out; these I call "rim rents."

(*b*) Some of the central fibers may be parted without tearing either the joint side or the bursal side of the tendon.

(*c*) The rupture may extend vertically through the whole tendon, making a communication between the joint and the bursa, without involving the whole breadth of the tendon.

(*d*) The fibers on the bursal side may be eroded without complete communication with the joint.

I believe that type (*a*) is synonymous with the cases so frequently found at autopsy where the sulcus is bare and eburnated, *i.e.*, the condition I speak of as "rim rents." I think that type (*b*) may be the precursor of calcified deposits. Types (*c*) and (*d*) may be among the cases which give "tendinitis" symptoms out of proportion to the traumatic history.

Since I do not intentionally operate on incomplete ruptures, what I have to say in regard to these minor lesions is more theoretic than what has been stated concerning the complete ruptures.

All four varieties should be distinguished from the complete form by the persistence of considerable power in elevation, because the mechanics of the pull of the supraspinatus are not greatly altered. I have not operated unless there was decided loss of power.

The other cardinal symptoms, jog, crepitus, atrophy, sulcus, eminence and local tenderness, will be present, although any one of

them or all of them may be found in less pronounced degree than in the complete cases. Type (*c*) might be recognizable, as the fluid sign should be present because joint and bursa communicate.

The diagnosis of the incomplete type is therefore made chiefly by the persistence of a considerable amount of power in the elevated or abducted arm and *some doubt* about the presence of some of the other symptoms and signs. Complete cases are usually so absolutely typical that no doubt remains. Complete cases do not form adhesions, while many incomplete cases do.

If the surgeon is sufficiently informed about the anatomy, pathology and surgery of this particular region, I believe that when in doubt he should explore the bursa through a half-inch incision. If he finds a communication through into the joint, let him close it, but if no opening exists, let him back out, for we do not yet know what to do to these incomplete ruptures.

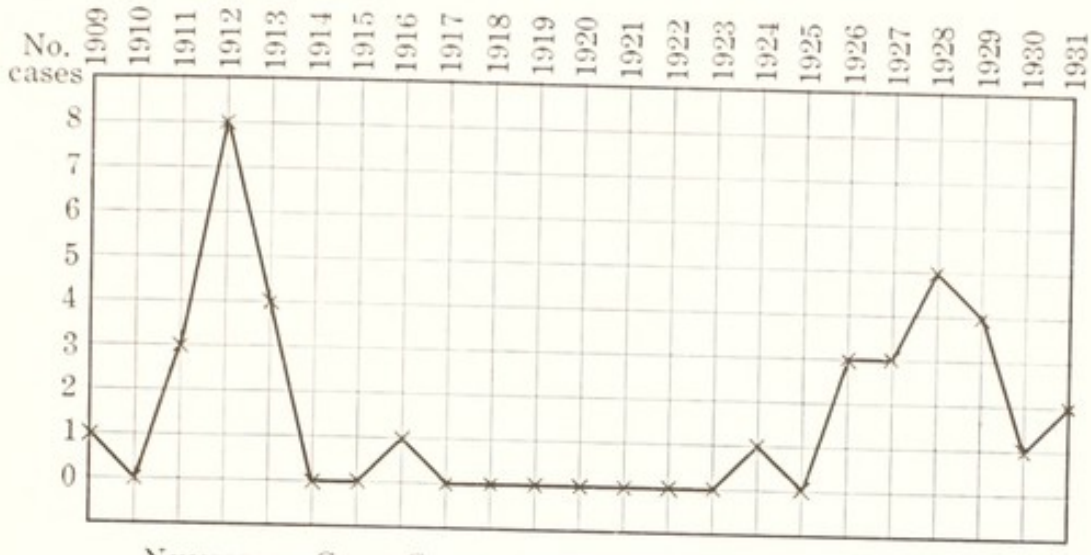
Before closing this chapter, I may say that I believe these lesions to be the most common form of shoulder injury. My slogan has been that *complete* rupture of the supraspinatus is the most common cause of *prolonged* disability from industrial accidents to the shoulder. I feel that I have proved this at least to my own satisfaction. I may now add that the *incomplete* form accounts for the majority of minor shoulder disabilities. This I have not proved, for I do not operate on these cases, but the frequent presence of broadened sulci found in any series of autopsies in elderly people is convincing to me. The clinical examination of employees with sore shoulders often shows symptoms suggestive of this lesion. Possibly opaque fluid injected in the joints might confirm the diagnosis. I have not felt justified in doing this, for exploration seems to me quite as free from danger. I certainly should try this experiment on patients now if I had in mind some definite plan for relieving them, if I did find broadened sulci indicative of incomplete ruptures, but I do not yet conceive of a plan by which to promptly cure them.

The appearance that I think opaque fluid in the joint would show in cases of complete rupture is indicated in the cover design.

FREQUENCY OF COMPLETE RUPTURE OF THE SUPRASPINATUS TENDON

There are certain reasons for believing that this is a far more common lesion than is generally appreciated.

1. The writer's personal experience.
2. The statistics of Dr. Akerson.
3. The lack of attention paid to lesions of the shoulder in general; *i.e.*, of end result studies of large series of shoulder injuries.



NUMBER OF CASES OPERATED UPON BY WRITER (BY YEARS)

1. *Personal Experience.* The above chart shows that the writer's experience in operating upon these lesions was confined to two periods of about three years each, when attentive study was given to relatively large groups of shoulder cases. In the first period (1911 to 1913 inclusive) an intensive study was made of the shoulder cases at the Massachusetts General Hospital. The author resigned from that hospital in 1914 and was not reappointed until 1929. Other activities and a period in the army during the war intervened, without opportunity to operate on such cases, although I continued to make occasional impartial examinations for the Industrial Accident Board. In 1926, owing to the interest of Dr. Henry C. Marble and Dr. W. A. Bishop, who are directors of Insurance Clinics, I again gave intensive study to a group of shoulder cases, and the chart again ran to a peak. This led to increased interest in other clinics, so that other surgeons (*e.g.*, Dr. Wilson) began doing these operations, which accounts for the decline at the end of my chart, although the rate of frequency, if we included other surgeons' cases, would not show this decline.

In other words, the chart shows that whenever I gave intensive study to a group of shoulder injuries, I was able to diagnose some of these cases and to demonstrate them by operation. My work received little attention after the first period but since the second, largely owing to the changes brought about by the Workmen's Compensation Laws, the importance of the lesion is becoming recognized by industrial surgeons in this state. I do not know just how many cases were seen during these two periods, and therefore cannot give the exact percentage of the cases examined which were instances of this lesion, although the limits may be stated pretty positively. For instance, the first fifteen cases were found among less than 200 patients, and the

second fifteen cases were from an uncertain number, but probably less than 100. This means that the frequency of occurrence was about 7½% to 15% of all shoulder lesions that came under my observation.

By referring to my records for the twenty years since I recognized the first case, I find that I have seen in all about one thousand shoulder cases. Of these more than one hundred have been positively diagnosed as instances of complete rupture and many more as incomplete. This would justify an estimate of between 10% and 20%. It would require an amount of labor disproportionate to the present object to go over these records to apportion the exact percentage, since it is sufficiently clear that I have made this diagnosis in not less than 10% and perhaps more than 20% of all shoulder cases. If cases of partial rupture were included the percentage would be at least doubled.

The figures in the annual Reports of the Industrial Accident Board help us only in a negative way, for this diagnosis is not classified. Their tabulations of the durations of disability are not carried over from year to year, so that we cannot obtain a list of the cases in which disability is protracted. This is unfortunate, for this list would probably be largely composed of cases of rupture of the supraspinatus.

The report of the Industrial Accident Board for the year 1928, Table 7, gives the following figures in regard to shoulder injuries. It is supposed to cover every accident occurring in industry in this state.

	Bruises, Contusions, Abrasions	Burns and Scalds	Cuts, Punctures and Lacerations	Dislocations	Fractures	Sprains and Strains	All Other	Total
Scapula					25			25
Clavicle				2	64			66
Shoulder Joint				123	24			147
Shoulder	279	15	29			528	192	1,043
Both Shoulders	4	1	2		1	8	6	22
Humerus				1	34			35
								1,338

The last two columns, "Sprains and Strains," and "All Other," are the ones which would probably contain most of these ruptures, although a large number must be included under dislocations and fractures. If we double the figures where both shoulders were injured, the total of the two columns would be 748, five per cent of which would be thirty-seven. During the same period, July 1, 1927, to June 30, 1928, I personally made the diagnosis in seventeen cases which were referred to me by Insurance Clinics or by the Board for "Impartial Examination." Since I probably see but a small fraction of these shoulder injuries of doubtful diagnoses which occur annually in this state, the estimate of thirty-seven is surely a minimum one. The maxi-

mum reasonable estimate would double this and also include twenty-five per cent of the 125 dislocations. It seems to me pretty well proved that not less than thirty-seven and very likely more than one hundred injuries of this type occur annually among Massachusetts employees.

At first sight thirty-seven, or even a hundred, neglected shoulder injuries in a state of 4,200,000 inhabitants does not seem a very serious matter, but as each insured case may cost the state $4,000 for compensation (aside from the pain endured and the economic loss of productive effort in labor), the amount of money involved is considerable ($37 \times \$4{,}000 = \$148{,}000$). This fact makes this obscure injury more interesting, and, if we multiply by 100, still more so. My opinion is that the larger figure is a more accurate estimate. If we included partial ruptures which heal after a few months, but which cause a large total of lost time, the annual expense to the state for injuries of this small tendon can be reasonably estimated to be over half a million. Furthermore, there must be many such injuries which are not industrial.

In addition to the above reasons for believing that this lesion is not rare, I may say that in a single set of twenty dissecting room subjects I have always been able to demonstrate one or more instances. When I have been asked to speak to small groups of doctors or students, I have usually been able to find a subject on whom to demonstrate the diagnostic signs of this lesion, by asking at adjacent clinics for cases complaining of inability to raise their arms. I feel quite confident that I can demonstrate (recognized or unrecognized) instances of this lesion by examination of the cases under treatment in the out-patient departments of any large general hospital in any city of the United States, and, in fact, probably in most parts in the world, where there are many persons over forty doing laboring work. This does not necessarily mean that there are large numbers, for each case is apt to be a patient for several years, meanwhile going from one hospital to another in search of relief.

2. Dr. Akerson's statistics, already given on page 67, give us a maximum estimate, because nearly all the observations were made on elderly subjects of the laboring class, in whom a previous injury could only be assumed, for there were no histories negative or positive of accidents. There is a splendid chance for a clinical research at the Long Island Hospital, in future, to correlate autopsy findings with retrospective histories of shoulder injury, taken from patients before death comes. It would be a time-consuming job, but it would help us to decide the important question of whether these lesions can occur

without injury, and it might lead to the saving of many thousands of dollars, not only in compensation, cost of medical care and economic loss, but in maintenance of the indigent. Dr. Akerson's work might in this way lead to a saving of more than the cost of his entire department. Similar opportunities exist in like institutions from one end of the country to the other.

3. *The Lack of Attention to Shoulder Injuries.* There is a voluminous literature on fractures and dislocations (especially on habitual dislocations) of the shoulder, but, with the exception of Dr. Meyer's work (*loc. cit.*) and an investigation now being undertaken by Dr. Fowler of Evanston, Ill., I know of no systematic observations from autopsy findings in this part of the body. Nor are there any serious studies of similar clinical cases. If the End Result system of organization existed in our hospitals and insurance clinics, these cases of prolonged disabilities would be automatically revealed, studied and eventually prevented by early operation.

Thus my confidence that these lesions are common is enhanced because no one has evidence to contradict me, and a few support me. It is clear that Dr. Meyer finds similar lesions, although he explains them as the results of attrition. Dr. Fowler will, I believe, as he has already told me, confirm some of my findings and give support to those of Dr. Akerson.

ARGUMENT

This book is not written to show that I have had good results in this class of cases. My argument is merely that *this lesion exists;* is not uncommon; *has characteristic symptoms which permit accurate diagnosis;* and, since I have been able by suture of the tendons to *greatly relieve a few cases*, even though the diagnosis was belated, it is highly probable that the *prolonged and trying symptoms* from which these patients suffer, when not operated upon, might be prevented by early diagnosis and the prompt use of a minor surgical operation, which should not be difficult, if promptly done.

If I can offer reasonable evidence for the clauses italicized, it seems to me that the conclusions in the last clause of the above paragraph would be sustained sufficiently to justify prompt exploratory operation when the symptoms suggest this diagnosis.

POSSIBLE ACCURACY OF DIAGNOSIS

I have already established the existence of this lesion, endeavored to estimate its frequency and have described the characteristic symptoms. Evidence is now offered to sustain the statement that accurate

diagnosis is possible. The following extracts, taken *verbatim* from my pre- and post-operative reports to insurance companies, show that a positive diagnosis of this lesion may be made. Every case operated upon during the last sixteen years, in which I have demonstrated a complete rupture of the supraspinatus, is given. Even in the cases operated upon prior to 1916, one may find that my definite pre-operative diagnoses in the records of the Massachusetts Hospital are correct, in all but five cases in which the diagnosis was simply "subacromial bursitis." With each of the abstracts given below the case number is stated. The reader will find further information about each of these cases by referring to these numbers in the Table on pages 255 to 260 in Chapter VIII.

Case No. 57.

Preop. Diag. "The injury which he sustained two years ago, I believe to have been a rupture of the supraspinatus tendon and the upper portion of the capsule of the shoulder joint."

Op. Findings. "As soon as the bursa was opened it was apparent that the entire supraspinatus tendon had been torn away, for the cartilage of the bone showed where the supraspinatus tendon should be. A small stub of the tendon still remained attached to the tuberosity. Further exploration showed not only that the supraspinatus tendon, but the upper portion of the infraspinatus tendon was also torn, but the subscapularis was intact. The long tendon of the biceps could be seen crossing the field just at the edge of the subscapularis. The proximal portion of the supraspinatus had retracted so far into the subacromial space that only a rather unsatisfactory portion of it could be pulled upward and attached to the stub on the tuberosity. It was evident that the atrophy which had taken place in the two years left practically no hope of the restoration of the complete function of the muscle or tendon."

Case No. 75.

Preop. Diag. "I have no hesitation in making a diagnosis of rupture of the supraspinatus tendon, nor in advising an operation to suture it and clean off the ragged vegetations at the edges of the old rupture."

Op. Findings. "Showed supraspinatus entirely torn off and retracted far under acromion. Biceps tendon lying at inner edge of rupture."

Case No. 83.

Preop. Diag. "I feel that the diagnosis of subacromial bursitis is correct, but that the underlying cause of the bursitis was a ruptured supraspinatus tendon. This has now partially healed—enough to renew the power of abduction but not enough to make the point of rupture smooth. The result is an irregularity on the base of the bursa which in abduction impinges on the acromion and acromio-clavicular ligament."

Op. Findings. "On opening the bursa the ruptured supraspinatus tendon was beautifully demonstrated, the tendon having been torn from the tuberosity, leaving none of the original attachment on the bone. It had retracted about a half inch and the torn end could be seized with a tenaculum."

CASE No. 88.

Preop. Diag. "He has the following signs indicating that his trouble is a ruptured supraspinatus tendon and his history is typical of this lesion.

"1. There is an acutely tender spot on the greater tuberosity at the normal point of insertion of this tendon.

"2. I can, I believe, feel at this point a sulcus where the tendon is torn.

"3. There is a painful jog in the motion as he raises his arm.

"4. There is velvety crepitus as he does this.

"5. After the arm has been elevated one can easily pull it down, although the deltoid muscle can be felt strongly contracted.

"6. There is a feeling of fullness under the upper part of the deltoid as if there was fluid in the bursa."

Op. Findings. "Free straw-colored fluid. Cartilage of head of humerus and the greatly swollen and injected biceps tendon visible on retracting the margins of wound in deltoid. The tear of the supraspinatus tendon was complete and the tendon of the infraspinatus was also almost completely torn. The proximal ends had retracted so as to be barely reached by Allis forceps to enable passing a suture."

CASE No. 89.

Preop. Diag. "Patient presents to me typical symptoms of supraspinatus. He can abduct his arm slowly and with a hitch which gives an obvious sudden change in the relative position of the humerus and the scapula. He cannot maintain his arm extended against any considerable pressure."

Op. Findings. "Routine bursal incision disclosed a very extensive tear of the supraspinatus tendon. The biceps tendon had disappeared and could not be located except possibly as a few fibers in the anterior part of the joint."

CASE No. 96.

Preop. Diag. "Examination shows pretty typical signs of a ruptured supraspinatus tendon, except that the friction rub is masked by the presence of joint fluid in the bursa. The outlines of the bursa are visible through a heavy deltoid as evidence of this fluid. It is well shown when the patient attempts to abduct the arm. The arc of abduction cannot be performed without a typical jog in the motion. The patient himself feels this and complains of pain during it. There is tenderness over the bursa."

Op. Findings. "Routine bursal incision showed a typical rupture of the supraspinatus tendon which did not involve either the subscapularis or the infraspinatus; making a gap pulled away from the tuberosity of about one-half inch width. Through this, on opening the bursa, the cartilage of the head of the humerus was visible. By retracting a little, the biceps tendon could be seen to the inner side, greatly congested."

CASE No. 97.

Preop. Diag. "Diagnosis. Rupture of supraspinatus tendon (left) complete and of severe grade. The same on right but incomplete and partially repaired. Probably rupture of the long heads of both biceps tendons."

Op. Findings. "The supraspinatus tendon was completely ruptured and the upper portion of the subscapularis also. The biceps tendon had disappeared, evidently having been torn off the glenoid and retracted into its groove."

CASE No. 98.

Preop. Diag. "Examination shows very typical tenderness, crepitus, abduction weakness and the jog in motion typical of a ruptured supraspinatus tendon in the right shoulder."

Op. Findings. "Routine bursal incision showed the supraspinatus was completely ruptured and also a part of the tendon of the subscapularis over the biceps tendon, and also a portion of the infraspinatus. The joint cartilage was exposed for about a half inch to the outer side of the bicipital groove and there was a tag of tendon still attached to the tuberosity just external to the groove."

CASE No. 102.

Preop. Diag. "Examination shows a decided atrophy of the muscles about the right shoulder, tenderness and irregularity of the tissue over the greater tuberosity, a painful jog in the motion of abduction, free external rotation, persistence of passive and loss of active complete abduction. These signs and the history are characteristic of rupture of the supraspinatus tendon. He also has a rupture of the long head of the biceps, if one may judge by the contour of the belly of the muscle."

Op. Findings. "Free joint fluid found in bursa, which was large. The supraspinatus tendon was about half torn away from the tuberosity and the torn tendon retracted only about one-half inch, exposing a small extent (about) one-quarter inch of the edge of the joint cartilage. The opening was not large enough to view the tendon of the biceps, so I made an incision about one-half inch long into the bicipital groove and identified the biceps tendon. It was large and normal in appearance, so that it is likely that the apparent atrophy of the outer belly of the muscle in this case was due to simple disuse from reflex pain."

CASE No. 106.

Preop. Diag. "Examination of the right shoulder shows typical signs of a rupture of the supraspinatus of considerable degree. One can feel the sulcus on the facet of insertion from which the tendon has been evulsed. There is fluid in the bursa."

Op. Findings. "The roof of the bursa was thickened. When incised, free straw-colored fluid escaped, about one dram. The supraspinatus was found torn from its attachment and retracted far under the acromion. About one-half of the insertion had evulsed from the facet. The other half was torn about one-half inch from the facet, leaving a thickened, irregular, whitish mass about the size of a large lima bean attached to the tuberosity."

CASE No. 107. Reported in text.

CASE No. 108.

Preop. Diag. "The diagnosis is fairly certain of a partial rupture of the supraspinatus, and also a rupture of the long head of the biceps."

Op. Findings. "The complete supraspinatus tendon had been torn close to the tuberosity and was retracted about an inch and a half. The infraspinatus and subscapularis were intact, but the biceps tendon had been torn completely off the glenoid and had retracted part way, instead of

wholly into the sheath, leaving a frayed end protruding at the edge of the articular surface. In repairing I used this by sewing the supraspinatus to it and the subscapularis, anchoring the biceps tendon in its groove as I did so."

CASE No. 112.

Preop. Diag. "Examination shows the following: A slight atrophy of the intrinsic muscles of the shoulder, a soft crepitus which occurs with a snap just as the tuberosity passes under the acromion in abduction, a tender point on the greater tuberosity at the insertion of the supraspinatus tendon, a tendency of the scapula to follow the motions of the humerus, a flaccid condition of the outer head of the biceps muscle. These signs indicate that he has torn the supraspinatus tendon from its attachment on the tuberosity, and probably has also torn the biceps tendon from its adjustment on the glenoid."

Op. Findings. "This displayed a perfectly typical complete rupture of the supraspinatus tendon without rupture of either the subscapularis or infraspinatus. The biceps tendon was slightly frayed at its inner edge where there were two small papilloma-like, whitish excrescences, the size of one-quarter of a pea. The stub of the ruptured tendon was still attached to the tuberosity, but had worn down to a falciform edge; similar falciform edges showed on the lateral aspect of the tear; *i.e.*, the unruptured edges of the subscapularis and infraspinatus. By lifting the edge towards the subscapularis, a pinkish, inflamed biceps tendon was demonstrated. A few fibers of the outer portion of this had been ruptured as spoken of before. There was only a moderate amount of joint fluid seen at any time. The edges of the torn surfaces were refreshed, and the parts sutured in place with three heavy braided silk sutures."

CASE No. 115. See history on page 389

CASE No. 119.

Preop. Diag. "Typical ruptured supraspinatus tendon. Atrophy, crepitus, jog, fluid, loss of power very great. Biceps O. K. Rotation free, tender sulcus."

Op. Findings. "The tendon was much thicker than usual and less retracted, so that the triangular gap was longer transversely than vertically. . . . There was quite a stub on the tuberosity, enough to suture. The biceps tendon was so badly damaged that there were but a few strands of it left and these were adherent to the roof of the joint. Most of the tendon had retracted into the groove and become adherent there. None was left crossing the joint surface and none was seen in the joint, although it might have been imbedded there behind the head."

CASE No. 123.

Preop. Diag. "This patient has the typical symptoms of a rupture of the supraspinatus tendon of considerable extent, but not enough to prevent his having a fair amount of power in abduction—although with pain. He has atrophy of the spinati, scapulo-humeral spasm, a jog in the motion of abduction, the fluid sign with limitation in complete abduction, a tender, palpable defect on the greater tuberosity where the tendon should be attached."

Op. Findings. "An exploratory incision a half inch long was first made and it was at once apparent that the supraspinatus and the infraspinatus tendons were evulsed from their facets, leaving no stubs of tendon. The biceps tendon, which was double at this point, was greatly swollen and inflamed and presented as soon as the roof of the bursa was opened. To its inner side the subscapularis and the inner portion of the tendinous expansion of all the tendons lay intact, and to its outer side was bare joint cartilage and the top and posterior portion of the greater tuberosity from which the tendons had been torn. The facets were smooth and the superficial parts of them had not been carried away by the tendons. The incision was enlarged to an inch and a half and a good view of the field obtained. It was found that the teres minor had also been evulsed. The whole posterior part of the capsule and conjoined tendon of the above muscles was retracted downward and outward. It was impossible to draw this back and to suture it in place."

Case No. 127.

Preop. Diag. "At my first examination I did not make the diagnosis in this case, although it had been suggested by Dr. Marble, but on my second examination, seventeen days later, I wrote, 'H—— is better, but I have changed my opinion about the diagnosis. I am now quite confident that he has a ruptured tendon and should be operated upon. I think Dr. Marble was correct in his original diagnosis.'"

Op. Findings. "A small exploratory incision was first made over the point of tenderness. As soon as the roof of the bursa was incised, an irregular, whitish mass of tendon about as large as a thumb nail popped out of the incision. It proved to be the proximal portion of the torn tendon and was about one-quarter inch thick. It evidently turned on itself as the arm was abducted and caused the catch which the patient himself could accurately localize. There was about a tablespoonful of straw-colored fluid in the bursa. The incision was then enlarged to about two inches and a good view of the lesion obtained. This case differed from others on which I have operated in that the tear did not involve the whole thickness of the tendon, but left a very thin layer toward the joint surface so that there was not a demonstrable communication with the true joint. However, the layer was so thin that the cartilage could be seen through it. The ruptured portion formed a little flap about a square inch in extent, attached only to the proximal end and evulsed on the distal end from the facet of insertion. I have seen similar cases in the cadaver where the distal end had not evulsed. It is a fact that lamination of the tendon is found in many cases, but this is the first one on which I have operated where the laminated portion evulsed without the lower surface also tearing. The free portion seemed rather necrotic but was clearly rather recently ruptured. The torn surface was rather bulbous, as if partially healed. Near the lesion and to its inner side was a streaked, red area which was excessively tender when touched, but the torn end itself had no sensation and was not so made by the novocaine. It is to be assumed, therefore, that the pain in these cases is not due to the tissue pinched but to the uninjured tissue on the tuberosity which does the pinching."

CASE No. 128.

Preop. Diag. "My opinion is very strong that he has a ruptured supraspinatus, but I am a little uncertain as to whether it is not a renewed injury in an old lesion. X-ray is very strong evidence of this. Clinically, I should not suspect it. In either case I should advise immediate operation as soon as arrangements can be made."

Op. Findings. "On opening the bursa there was found to be evidence of old inflammation in the bursa, as well as recent light adhesions. The supraspinatus was torn away from the tuberosity to the extent of about one-half inch, and little of the bursal floor remained untorn, but there was a gap directly into the joint through which the cartilage could be seen. This gap was about one-quarter of an inch in extent. The tendon had retracted very little and I was able to close it with four silk sutures, two of heavy silk and two of light silk."

CASE No. 129.

Preop. Diag. "Signs of ruptured supraspinatus tendon were present at my previous examination, but they are much more prominent now than they were at that time. He has a very distinct crepitus, sulcus, eminence, scapulo-humeral spasm, tenderness at the border of the tuberosity; localized tenderness, atrophy of the supraspinatus, sign of fluid when both arms are elevated. I agree with Dr. Marble in the diagnosis and would advise operation in this man's case."

Op. Findings. "The bursa was first opened by a half-inch exploratory incision. A small amount of free fluid escaped and it was readily seen that the supraspinatus tendon was ruptured and greatly retracted. . . . The tendon was partly evulsed and partly ruptured at the 'palisade junction.' The complete tendon was involved with possibly some of the infraspinatus. As in another recent case (H——) there was still some tissue remaining on the joint side so that the cartilage was partly covered with a turgid, deep-red, softish tissue containing a few obvious whitish tendinous bands. One of these was especially noteworthy because it was very sensitive, and the patient winced when it was touched. I excised it because it did not seem to be of sufficient strength to make up for its sensitiveness. The biceps tendon lay to the inner side of the rupture buried in inflamed synovial folds, but not injured, unless the strand of tendon above alluded to had been stripped off it. The bulk of the proximal end of the supraspinatus tendon had retracted far under the acromion and could barely be drawn into the field."

CASE No. 135.

Preop. Diag. "I feel very confident that he has a rupture of the supraspinatus tendon. I am not sure that there was not a previous rupture and that this latter accident merely increased the trouble. However, there is no question in my mind but that his chances will be much better if his bursa is explored and suture is done, if the tendon is ruptured."

Op. Findings. "No blood or fluid on opening bursa. A complete rupture of the supraspinatus, with extension across the bicipital groove, about one-half inch into the subscapularis. Definite indications that the rupture was fairly recent; *i.e.*, very little separation between the distal and prox-

imal portions. Stub of tendon still attached to the tuberosity over most of its extent. No new-formed falciform edge. Biceps tendon flattened and superficially reddened in portions beneath rupture. Parts satisfactorily sutured together with pedicle silk; five stitches. In my opinion, the condition found indicated a recent rupture of a tendon which had already been partially ruptured."

CASE NO. 137.

Preop. Diag. "Examination shows that this patient has a symptomless, chronic bursitis, with crepitus in the *right* shoulder. The *left* shoulder shows the typical signs and symptoms of a rupture of the supraspinatus tendon. Since he has considerable power in abduction—although the abduction is painful, and accompanied with a jog and crepitus—I am inclined to think that the rupture is not extensive enough to involve the adjoining tendons. Even though this man is sixty-two, and it is four months since the injury, I would advise him to have the tendon sutured to the tuberosity."

Op. Findings. "A one-half inch exploratory incision was made and the bursa opened. Rupture of the supraspinatus throughout its whole extent was disclosed. The rupture extended also, to a slight extent, into the insertion of the infraspinatus. The sulcus and tuberosity were bare, except for a very slight amount of tag of tendon near the edge of the bicipital groove. The incision was enlarged to two and one-half inches to permit suture."

ERRORS IN DIAGNOSIS

But a skeptical reader may now ask for a statement of how many cases I have operated upon under this diagnosis and have *failed* to find a complete rupture of the tendon. I give below a similar set of *verbatim* extracts from the records of nine cases during the same sixteen years, the only ones in which I have made a positive diagnosis and have *not* been confirmed by the operative findings.

CASE NO. 76.

Preop. Diag. "I believe this patient has a ruptured supraspinatus tendon, at the point of the insertion of the tendon in the tuberosity. This diagnosis is consistent with the X-ray appearances seen in the films taken by Dr. George. These films show an irregular opacity in the area of the bursa, which may be due to extravasated blood or to crumbs of bone torn off the tuberosity."

Op. Findings. The operative notes in this case have been lost, but I clearly remember that the condition proved to be a case of calcified deposit which had recently ruptured into the bursa, leaving a defect in the tendon very similar to a true rupture, but containing blood and calcified material.

CASE NO. 80.

Preop. Diag. "This is unquestionably a case of ruptured supraspinatus tendon of considerable extent, but the main symptoms are due to a tuft of synovial granulations from the remnant of the supraspinatus tendon still attached to the tuberosity."

Op. Findings. "The walls were much thickened and there were synovial tags in the region over the tuberosity, showing more or less chronic inflammation. The tender point, which could be felt prior to the operation and was thought to be a tag of tendon remaining on the tuberosity, proved to be a rounded elevation, markedly circumscribed and about one-half inch in diameter, slightly longer in the axial direction than the transverse. This was acutely tender when pressed on. It was incised and found to be the insertion of the tendon of the supraspinatus attached to an osteophyte at the edge of the joint cartilage. There was no direct evidence of a rupture of the supraspinatus tendon. If there had been one, it must have healed. No communication with the joint was demonstrated. The essential pathology was probably the hypertrophic arthritis which was evident in other joints. (Patient's age was 77.)"

CASE No. 91.

Preop. Diag. "The history and symptoms are typical of a ruptured supraspinatus tendon in the right shoulder, with some adhesions of the bursa, or blocking of the motion by the remains of the torn tendon impinging on the acromion. I think the latter."

Op. Findings. "The base of the bursa was deeply congested and swollen, but there was no demonstrable tear of the tendon of the supraspinatus. There were light adhesions in the bursa which were broken up with the finger. The short rotators and capsule were greatly contracted and were slowly stretched until full external rotation and abduction could be attained, and the wound closed."

CASE No. 104.

Preop. Diag. "I have little doubt that this patient has a rupture of the supraspinatus tendon of considerable extent."

Op. Findings. "Incision through the roof of bursa with the escape of about one dram of clear, straw-colored fluid. Exploration revealed an intensely inflamed zone on the greater tuberosity, approximately the size of a half dollar with red periphery and white center, resembling the condition seen in calcified cases. An inflamed fold moved back and forth over this as the patient's arm was moved. This fold was excised. The point of the knife was used to puncture the tendon in several places in the inflamed area with the purpose of allowing a new blood supply by attaching the scar to the inflamed base. There was no indication that the supraspinatus tendon had been ruptured."

CASE No. 110.

Preop. Diag. "The whole tuberosity is tender and it is hard to find a definite spot from which one can say the tendon has been torn. With the exception of this, his symptoms are fairly characteristic of a ruptured supraspinatus. From the relaxed condition of the biceps muscle and particularly of its outer head, I am inclined to feel that the biceps tendon is torn from its attachment on the glenoid. Indeed this may be the main lesion.

"I should advise inspection of his bursa under local anæsthesia and suture of the tendons if they are found torn. If there is no tear in the base

of the bursa, I should advise opening the joint between the supraspinatus and the subscapularis and inspecting the tendon of the biceps.

"Remarks. This case is atypical in the age of the patient (17). Ruptured supraspinatus lesions are usually in elderly men. The character of the accident is unusual; sudden abduction efforts are more common as causes. There has been comparatively little pain, especially in the first week, and usually one has a history of swelling over the bursa in the first few days. Palpation over the tuberosity is a little atypical. For these reasons I cannot be as positive as I sometimes am, but I am sufficiently certain of a rupture to feel that in the boy's place I would want to have an exploration. Little harm would be done if this is negative, which I am confident it will not be."

Op. Findings. "The exploratory instrument was first introduced into the bursa, which was found to be full of adhesions and consisted of a series of pockets of walled synovia. Through the exploratory instrument it could be definitely determined that the bursa had been subacutely inflamed, but that there was no tear in the supraspinatus tendon. There was no fluid in the bursa. As both Dr. Baker and I thought that the biceps tendon was probably torn, the incision was enlarged to about one and a half inches, and the bicipital groove was incised. It did not contain the tendon, which evidently had retracted downward. The arm was put through the accustomed motions of rotation and abduction while a finger was introduced into the bursa to break up all the adhesions existing there. When the adhesions had been broken, which was easily done, the motion of the arm was normal. No steps were taken to repair the biceps tendon."

CASE NO. 111.

Preop. Diag. "The shoulder presents typical signs of a ruptured supraspinatus tendon of a considerable degree. He has my fluid sign—characteristic velvety crepitus, a jog in motion, a tender irregularity on the greater tuberosity and weakness in abduction. He is able, however, with a little coaxing, to abduct the arm, but has very irregular action of the humerus on the scapula as he does this. There is still some ecchymosis in the anterior portion of the arm over the bicipital region, as if after the injury blood may have escaped down the tendon sheath; the tendon, however, appears to be intact."

Op. Findings. "Exploration of the bursa showed no fluid. No communication was found between the bursa and the joint. The bursa was adherent over about half the extent of the lower portion. On separating the adhesions it was evident that the supraspinatus had been torn, but had spontaneously healed; the tissues were red and swollen with white patches here and there. I therefore closed the wound in the usual manner without doing anything in the way of an operation."

CASE NO. 120.

Preop. Diag. "The left shoulder presents very typical signs of an extensive rupture of the supraspinatus and possibly of the long head of the biceps. There is slight but decided atrophy—consistent with a history of two weeks. There is a very tender point on the tuberosity, scapulo-humeral

spasm, a jog in motion and the fluid sign. In the stooping position the patient can abduct and hold the arm abducted as he straightens up."

Op. Findings. "Routine bursal incision. Adhesions prevented easy access, for roof was stuck to base over an area as large as a quarter over the greater tuberosity. These were separated and showed that the tendon of the supraspinatus was bright red, swollen, and covered with tags of vascular adhesions. The line of demarcation of the affected area was very sharp on the outer side, so that the contrast between the yellowish white to the right and the turgid red at the left was very decided. It suggested an infarction. Although there was some free fluid in the bursa there was no communication through the inflamed area to the joint. The supraspinatus tendon, if ruptured at all, must have been torn beneath the base of the bursa without communicating with the bursa. Several small incisions were made with the point of the knife to relieve tension in the tendon. Through one of these a tiny bit of white nondescript tissue protruded. This was saved for pathologic examination; I am pretty sure it was necrotic tendinous substance. The wound was closed as usual without endeavoring to suture the roof of the bursa. The pathology found at this operation may be explained in two ways, or as a combination of both ways. There was certainly an acute bursitis with recent adhesions, but it was impossible for me to say positively that this localized acute inflammation over the site of the supraspinatus tendon was due to a rupture of the tendon beneath the base of the bursa. It might have been due, as suggested by the evidence of osteitis shown in the X-ray, to a chronic necrotic process in the tendon. The extrusion of the bit of necrosed tendon from the incision also supports this. So does the symptomless condition in the well shoulder. It seems to me that if we accept the man's history that the condition of the shoulder was O. K. before he helped lift the case, that the whole picture can be explained by the supposition that there was a necrotic process going on in the tendon which had weakened it so that it partially evulsed without tearing into the bursa. This would explain all which was found at the operation, and my opinion is that this theory truly does account for the facts. I have had several other similar cases and have frequently seen evidence of such lesions in the cadaver."

CASE No. 126.

Preop. Diag. August 12, 1929. "I have little doubt that this patient has a mild rupture of the supraspinatus, but as the power in abduction is good, operation need not be considered. It is probably not an extensive rent. I suspect both from the clinical signs and from the X-ray that he has had an old lesion here for some years. It seems to me that the best plan of treatment would be to let him rest a few weeks and to have him attend your clinic three times a week for massage. If he does not feel able to go to work again in a month, let me see him again." Sept. 22, 1929. "Examination is as on August 19th. My opinion that he has a small rupture of the supraspinatus is somewhat strengthened by the fact that the symptoms have not improved. As he is only forty-two, I am inclined to advise an exploratory incision under novocaine to confirm the diagnosis and, if necessary, to suture the tendon. The spot at which he complains of tenderness is so localized that it seems likely we shall find some lesion at this region."

Op. Findings. Oct. 23, 1929. "Under local anæsthesia no rupture of the tendon was demonstrated. The walls of the subacromial bursa were thickened and the bursa contained a little fluid. There was nothing found which indicated any recent injury other than the presence of this chronic bursitis. The wound was closed after taking out a small specimen of the synovial lining for pathologic examination." (This showed chronic inflammation.)

CASE NO. 131.

Preop. Diag. "In my opinion, he has a chronic subacromial bursitis in consequence of a rupture of the tendon of the supraspinatus."

Op. Findings. "A half-inch exploratory incision showed that the roof of the bursa was thickened and adherent to the floor beneath. The wound was enlarged to about one and one-half inches and the adhesions freed, partly by cutting and partly by tearing with the finger. Several small bursal sacs had replaced the usual large one. These sacs lay beneath the adhesions which permitted a certain amount of motion. The adhesions were red and inflamed looking. There were one or two small calcified deposits which were wiped out. The periphery of the bursa outward and backward under the acromion was more normal in appearance and not adherent. The total area involved by the adhesions was about the size and shape of a fifty-cent piece over the greater tuberosity and insertion of the supraspinatus tendon. The most acute tenderness was felt at this point, although there was a little sensitiveness over the bicipital groove. Two or three tags of inflamed serosa were removed for examination and a very small bit of tendon at the point where the adhesions centered was clipped out for examination."

In most of these nine cases, although there was not a complete rupture, there was some evidence of partial rupture beneath the base of the bursa. However, I did not confirm this supposition by cutting through the base, for it seemed to me that its tissue formed a bridge over the gap which would assist in healing the tendon. I am inclined now to think that I have been too conservative, for several of these cases had long convalescences, and it might have been wiser to explore beneath the base of the bursa.

At the danger of repetition, I wish to state that I do not intentionally operate, unless I think the ruptures are complete, because I feel confident that where the rupture is complete, healing will not take place unless suture is done. I am not sure enough of what the treatment of partial ruptures should be, to make me urge operation, even if I were sure of the diagnosis. Therefore, the above nine cases may be considered to be errors in diagnosis, although by no means as negative explorations. The difficulty is to tell beforehand whether or not there is actually a complete rupture which needs suture. I think the fact that during a period of sixteen years I made the diagnosis correctly twenty-one times, and incorrectly in only nine cases, shows

that the diagnosis is not difficult. When in doubt, such a trivial operation as exploration would be justified, even if the proportion of errors was three to one.

In several of the nine cases it is to be noted that the mistake could have been avoided by more reliance on the fact that if there are decided restricting adhesions the diagnosis is not established. In the first case the X-ray indicated the presence of a calcified deposit and should have warned me not to make the mistake, although I should have operated at any rate. In several of the other cases where a mistake was made, the notes indicate that I was in decided doubt about the diagnosis before the operation, and, therefore, the incision as a matter of fact was "exploratory."

END RESULTS IN OPERATED CASES

We next come to the clause "since I have been able to relieve a few cases even though the diagnosis was belated." By referring to pp. 255-260, the reader may see that, excluding cases where there was a coincident fracture of considerable extent, I have operated on thirty-seven cases. In four of these the tendon was retracted to such a degree that I could not even attempt a suture. I have been unable to trace two patients operated upon about twenty years ago. Deducting these six cases we have thirty-one in which an attempt was made to suture the tendon. The results have been as follows: poor, four; fair, seven; good, twenty. That is, two out of every three operations were successful in that the results enabled the patients to return to work, although in most cases only after the elapse of months.

A very few, but not many, of my results have been perfect in the sense that the arm was just as useful and just as painless as before the injury; but many of them have been good in the sense that the arms have been relatively painless and quite, if not completely, useful. When I have written "good" I mean that the operation was well worth doing. When I have written "fair" it should convey that, in my opinion, there was some improvement from the operation, but that it was hardly worth doing. "Poor" means that the sum total of relief of the suffering of the patient was not sufficient to compensate for the pain and trouble which were incident to the recovery. There have been no bad results in the sense that any joint was made worse by the operation, but in at least two of the "fair" cases (Cases 83 and 112, see p. 175) the patient's point of view would have been that the result was poor, because his symptoms were not improved in spite of his post-operative pain.

There are many factors to account for the lack of perfection in the results, such as the age, the length of time the operation has been delayed, coincident disease, and above all, the desire of the patient to get well. The non-industrial patient is more readily pleased by improvement which lacks perfection than is the employee, who prefers compensation and no work, to double the pay *plus* work which would undoubtedly cause some pain and discomfort.

This compensation factor always causes some delay in the convalescence. Hard times have the same effect.

However, my argument does not need to show that all results should be perfect. I could give many excuses for the failures, but will be contented to present the following table.

Result	Case	Age	Elapsed time inj. to op.	Comments
Poor	15	70	5 mos.	Unsatisfactory repair. Weeping wound.
	83	48	3 yrs.	Ossification of coraco-acromial ligament.
	88	50	6 mos.	Weeping wound. Worked 13 months afterward.
	112	62	3 mos.	Secondary operation, p. 247.
Fair	57	63	2 yrs. 4 mos.	Too extensive to make good repair.
	89	65	5 mos.	Sabre-cut displacement. Worked afterward as watchman.
	102	57	2 mos.	Complicated by ulcer of stomach.
	107	52	2 mos.	Many complications.
	108	69	8 mos.	Complicated by cancer of stomach. Death 14 mos. later.
	127	60	6 wks.	E. A. C. considers good results, although patients are not known to have returned to work.
	137	62	4 mos.	

For further notes on all these cases see Table on pp. 255-260.

In three of the four poor results, the tear was very extensive and the repair unsatisfactory; three had weeping wounds from excessive fluid drainage without frank sepsis (see p. 248), and all four had secondary operations. The seven cases of fair results were all improved to a considerable extent but have not actually gone to work again permanently.

There was one death which might possibly be attributed to the operation. This patient, Case No. 123, was drowned while in bathing at one of the beaches three weeks after I did an exploratory operation. The operation had shown that the tendons were too badly injured to admit of any attempt at repair. The wound healed by first intention, and as the patient enjoyed going in bathing I permitted him to do so about ten days after the operation. On the twenty-first day he was drowned. He was known to have had a heart lesion. The medical examiner reported "Death by drowning," and did no autopsy. I myself

can see no connection between the operation and the drowning, but two doctors offered testimony at the Industrial Accident Board that the patient had had pulmonary embolism from the operative wound. The Board, however, did not accept their testimony, because there was no autopsy or other reason to support their diagnosis.

I feel that the proportion of good results is a sufficient reason for advising this operation, even in late cases. With greater experience and improved technique it is likely that the percentage of good results will be increased, but, as in many other surgical conditions, prompt recognition and treatment will be of greater importance than the difference in surgical skill between experienced and inexperienced operators, *provided the pathologic condition is thoroughly understood.*

END RESULTS IN UNOPERATED CASES

As most of my cases have been "industrial" the question of compensation becomes an important factor in estimating the period of disability. Cases who have no prospect of compensation will unquestionably go to work sooner; for instance, Case 119, a painter who had his own shop, began doing some work within a month after his operation. On the other hand in some cases, the compensation becomes equivalent to an old age pension. I have spent much time and money in tracing the unoperated cases of both partial and complete rupture, and have obtained enough information to venture the estimates on page 124 as to the probable costs of each. My estimate of $300,000.00 for 100 cases of complete rupture is also sustained by figures on ten patients on whom I did operate and therefore established the diagnosis. In five of these (delayed) cases the operation was a practical failure, but the patients were no worse off in consequence, and therefore were in a similar status to those in which no operation was done. In the other five the operation was essentially successful.

(The law limits total compensation to $4,000* so that these figures do not cover the period the patient is still disabled after his compensation ceases. The figures also include all medical and surgical charges.)

COSTS OF TEN CASES OF COMPLETE RUPTURE OF THE SUPRASPINATUS

Five Cases Not Improved			*Five Cases Improved*		
Case No.	88	$ 4,002.14	Case No.	98	$ 302.31
" "	89	4,481.90	" "	106	982.94
" "	96	1,393.66	" "	127	1,224.31
" "	102	3,484.25	" "	128	533.66
" "	115	4,063.73	" "	129	774.47
		$17,425.68			$3,817.69
Average		$ 3,485.13	Average		$ 763.53

* Since 1927, $4,500.

Case 96 in the first group had a delayed convalescence but eventually was able to go to work again as a steamfitter. Case 127 in the second group was a similar one. The reader may shift either case to the other group if he is disposed to exaggerate either average figure. Although it would not be fair to use the second group at all in computing the cost of 100 unoperated cases, by using it we may obtain an average minimum figure — $2,143.00. Therefore, the figure for 100 cases must be somewhere between $200,000.00 and $400,000.00.

Pain and disability are difficult to estimate in figures; these estimates are presented to impress on the reader that this lesion is important.

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So far as I know there is at present no literature on the subject of this chapter which deserves serious study, except that comprised in the above references. I have no doubt that Doctor Meyer and I refer to the same lesions which were studied by Doctor Akerson and can be found by any one who wishes to look for them in similar material. Doctor Meyer's attention has been focused on the long head of the biceps; mine has been focused on the supraspinatus. Probably both of us are in a measure right, and in great measure wrong, but at least we have opened up the field for future students who wish to relieve persons incapacitated by lesions in this region of the body.

Chapter VI

CALCIFIED DEPOSITS IN THE SUPRASPINATUS TENDON

As far as I know, the first patient to be operated upon for this condition was Miss McM., age 24, Massachusetts General Hospital, No. 127430, June, 1902. I assisted Dr. F. B. Harrington to incise and drain what we took to be an abscess of the subacromial bursa. A culture of staphylococcus aureus was obtained, but the wound showed no sign of infection and promptly healed. I believe now that the culture was contaminated, for I have never been able to obtain a culture from one of these cases since. On reading the record now the description seems impossibly stupid, for in spite of the fact that I was even then interested in the anatomy of the bursa, and also had been an enthusiastic pioneer in using the X-ray for six years, I did not put two and two together and realize that the condition was what we now call a "calcified deposit." It was not until May 17, 1905, when Dr. C. F. Painter asked me to see with him another doctor who was in such acute pain that he begged to be operated upon, that we suddenly understood almost as much as we know now about the condition. Yet I think that even then we were the first surgeons to realize what these cases were and how easy it was to relieve them.

I mention these experiences to illustrate how blind the human mind may be to facts which are perfectly obvious, and when such facts are clearly demonstrated how difficult it is to diffuse them so that they may be generally applied. Röntgen's discovery had for several years made the lesson we learned from this case plain for any surgeon to read, yet no one had seen it in just the right light. Those who happened to know about the anatomy of the bursa may not have happened to be able to read the X-ray, or to have had an appropriate patient, who, like our friend Dr. D., urged us to "try anyway." We tried and relieved him at once, yet today right in this city there are probably patients with the same condition being treated week after week as "neuritis." Dr. Painter and I have both written about our experience; other surgeons have confirmed what we say; we have continued to get excellent results from this operation; yet twenty-seven years have elapsed and some cases are still treated without even the diagnosis being made or the X-ray even used. Dr. Painter has reported this case in his original article, March 21, 1907; I also reported it in my paper published June 9, 1908, as follows:

"The patient, Dr. D——, is a man of 37 years of age, of heavy (200 pounds) physique and plethoric constitution. His previous history is unimportant, except that ten years ago after playing baseball all one summer he used to have a great deal of pain and soreness in his right shoulder joint. Since that time he has had at intervals a great deal of pain and tenderness in the joint. The pain bothered him particularly at night and was at times severe enough to prevent his sleeping well. He stated that until within a few days he had been able to move his arm without any trouble in all directions; for instance, behind his neck, behind his back, and over his head, without especial pain. There have been times, however, when it was difficult for him to raise the hand over his head, but when it was once put in this position it was comfortable for a time. He used to occasionally put it in this position at night; after lying with the hand behind his head for some time, he would then put it down by his side again. In doing this there was always pain during the motion. He recalls at times having his wife wake him just as he was going to sleep with his hand behind his head and tell him to put it down by his side, so that he would not go to sleep in that position and find it stiff on waking. External and internal rotation of the arm could be gone through without giving any trouble whatever.

Eight days ago while in New York he suddenly noticed that the point of his shoulder was tender and painful and that the motions were much restricted. He could with difficulty get into his clothes, but could not reach his hip pocket as he had been accustomed to. During these days the soreness had persisted and there had been great pain, sufficient to prevent sleep at night and to require large doses of morphine; the only comfortable position was sitting with the elbow supported. The pain was so great as to make him desire immediate operation.

Examination showed that the external and internal rotation of the humerus on its long axis is restricted about one-half the normal arc. Abduction or elevation of the arm is impossible on account of the pain caused in the neighborhood of the greater tuberosity. Attempts at passive elevation of the arm showed that the scapula moves in conjunction with the humerus.

An X-ray from the posterior view shows a pyramidal shadow in the region of the subacromial bursa just above the greater tuberosity and external to the tip of the acromion, the base of the mass being downward and about an inch in length. Between the base and the greater tuberosity is a translucent band about a quarter of an inch wide, occupying the position normally occupied by the tendon

of the supraspinatus. The mass is apparently in such a position that the tuberosity could not ride under the acromion without compressing it.

Operation, by Dr. C. F. Painter, May 17, 1905.

Under the impression that the mass shown in the X-ray was caused by a calcification either of the bursa or its base, a vertical incision of about two inches in length was made through the skin and between the fibers of the deltoid on the point of the shoulder over the greater tuberosity. When the fascia beneath the deltoid was reached a small opening was made in the subdeltoid bursa, and this was enlarged with scissors to an extent great enough to admit the forefinger. The aspect of the bursa was not abnormal inside except for some firm adhesions anteriorly which made it difficult to separate the plane of the bursa from the neighborhood of the bicipital groove and the anterior part of the greater tuberosity. With the finger in the opening, it was found that during the rotation of the humerus, a mass corresponding to the shadow seen in the X-ray could be felt to pass beneath the finger. This mass evidently lay at the base of the bursa on the top of the greater tuberosity. An incision was made into it permitting the escape of about half a drachm of sebaceous material resembling the contents of a dermoid cyst. The cavity, which resembled a wen or dermoid, was thoroughly curetted and the walls excised. The base of it was evidently the tuberosity and tendon of the supraspinatus; the roof was the base of the bursa. When the sac had been entirely removed, it was found that the motion of the joint under ether was perfectly normal, rotation and abduction being easily performed. The incision in the bursa was not closed and the skin was drawn together with silkworm gut and the arm put in the ordinary sling position.

Remarks:

The point of particular interest in this case is the persistence of the function of rotation in spite of the limitation by abduction. It is obvious that the position of the cyst allowed rotation with comparative freedom, but that attempts at abduction would crowd the cyst between the greater tuberosity and the acromion process and caused pain and spasmodic rigidity of the joint. The acuteness of the symptoms within the last eight days is explained by an inflammatory condition in the cyst. The previous attacks were probably due also to the same cause. That this inflammation did not involve the bursa to any great extent is shown by the persistence of rotation. Since the bursa was not inflamed it would be readily seen that the symptoms which the patient complained of in regard to going to

sleep with the arm behind his head can be explained, for the cyst having once got under the acromion was relieved of pressure. In other words, it was only when the cyst was inflamed by over exercise that its presence was noticeable, unless elevation of the arm crowded it between the acromion and the tuberosity.

The relief obtained by this operation was immediate and permanent.

It will be seen by comparing these notes with the description of the operation by Dr. Painter that the calcareous deposit seemed to me to lie not in the bursa, but beneath its base, and in or adjacent to the tendon of the supraspinatus."

It was difficult to find a heading for this chapter, and the one chosen is not satisfactory. An explanation of why it is not satisfactory may help the reader to understand the subject the caption is supposed to define. "Calcified subdeltoid bursitis" is a term in common use for the same condition, but this could not be accepted as a title for three reasons. First, the calcified deposits are rarely in the bursa, but lie beneath its base in the substance of the tendon of the supraspinatus or in one of the tendons of the other short rotators. To be sure, occasionally after inflammation has occurred the softened tissue containing a deposit bursts (See Plate II, Fig. 4) and the calcified particles are diffused throughout the bursa, the outlines of which may then be delineated by the X-ray. Fig. 44 shows an instance of this, but Figs. 45 and 46 are instances of the usual condition where the deposit is in its place of origin in the substance of the tendon, entirely beneath the base of the bursa. Therefore, "calcified subdeltoid bursitis" is a misnomer. It is also a wrong term because, as is explained on page 18, subacromial is a better term than subdeltoid. A third reason is the use of the word bursitis, as the noun which "calcified" is used to qualify. The bursitis is secondary to the pathologic process in the tendon. One may find no inflammation of the bursa and yet a large deposit beneath it may be demonstrated by the X-ray. It is only when the deposit is large enough, or the inflammation about it is great enough, to cause interference with the function of the bursa, that the symptoms of "bursitis" appear.

Our chosen title is also unsatisfactory because the offending deposit may not be in the supraspinatus tendon but in one of the other tendons underlying the base of the bursa, for instance, in that of the subscapularis. The infraspinatus and teres minor tendons may occasionally be involved. (See Figs. 45 and 46.) Sometimes two, three

or even four tendons in the same patient are the seats of deposits. It seems to me highly probable that in most cases which show the deposit in one tendon there may be also a "tendinitis," or at any rate a degenerative change in the other tendons, which is not accompanied by much calcification, and therefore is not demonstrated by the X-ray. Furthermore, I am confident that I have seen instances in which the same tendinitis has produced bursitis by involving the base of the bursa before any calcification dense enough to be demonstrated has taken place. We might change the title to "Calcified and Uncalcified Changes in the Tendons of the Supraspinatus, of the Subscapularis, of the Infraspinatus and of the Teres Minor; the Secondary Changes they may cause in the Subacromial Bursa overlying them, in some cases producing the Clinical Condition known as 'Subdeltoid Bursitis,' which should be called 'Subacromial Bursitis.'"

Even this extensive heading can be criticized, because there are still many cases never diagnosed as bursitis, but which pass as "neuritis." I might head the chapter "The Usual Cause of Neuritis in the Arm and Shoulder."

In 1908 I wrote as follows regarding subacromial bursitis:

"The cases which are to be discussed in this paper are by far the most common lesions of the shoulder joint. It is the writer's experience that more patients seek hospital treatment for lesions involving the subacromial bursa than for all other lesions of the shoulder joint, including tuberculosis and fractures, added together." "Not only is this true, but it is also true that more cases of subacromial bursitis seek hospital treatment than all the cases of the supposedly more common forms of bursitis such as 'miner's elbow,' 'housemaid's knee,' and 'weaver's bottom.' In those clinics in which these statements are apparently not true it will be found that these cases are passing unrecognized under the diagnoses of brachial neuritis, periarthritis, muscular rheumatism, circumflex paralysis, contusion of the shoulder, fibrous ankylosis, gout, rheumatism and other vague terms."

These convictions have gained strength with the years, although I now wish that I had expressed them in better English. It may be almost taken for granted that cases diagnosed as neuritis, rheumatism, and arthritis of the shoulder, are really instances of subacromial bursitis; those with a distinct traumatic history being usually cases of bursitis due to rupture of the supraspinatus, and those of spontaneous origin being instances of the process I have tried to

a *b* *c*

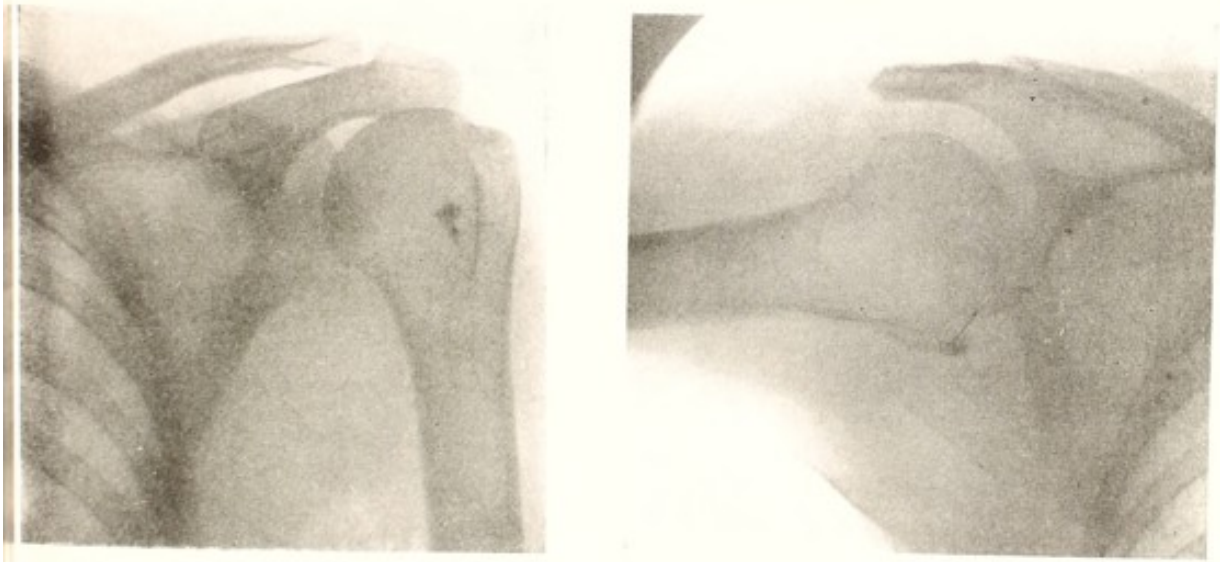
(Case 117)

FIGURE 43. VARIED VIEWS OF A CALCIFIED DEPOSIT IN SUBSCAPULARIS

Tracings from three different films of a case in which a calcified deposit had formed in the subscapularis and had caused acute bursal symptoms. The deposit lying in the substance of the subscapularis tendon of course did not move about in relation to the lesser tuberosity; the apparent change of position is entirely due to changes in the relation of the bones in consequence of rotation of the humerus. The first film was taken in the anatomic position, and the deposit is overshadowed not only by the contours of the humerus but by the acromion. The second film was taken in internal rotation and the deposit appears to have moved over to the top of the glenoid. The third figure shows the deposit when the arm is akimbo; *i.e.*, partly abducted and internally rotated. The deposit appears to have traveled to the lower edge of the glenoid.

This patient was operated upon by the writer, and the deposit removed. The incision was the same as in cases in which the deposit is in the supraspinatus; the affected area was just as readily brought under the incision by external rotation of the arm.

Deposits in the subscapularis are more frequently horizontal in their long axes than when they are in the other tendons. Compare Plate VI, Fig. 6. Sometimes they are not horizontal as in another case shown in *d* and *e*.



d *e*

In order to make the condition plain the shadow of the deposit has been slightly intensified in these cuts. In the anatomic position (*d*) the deposit shows as a small speck internal to the bicipital groove, which in most antero-posterior X-rays can be located by the dense line of the outer edge of the lesser tuberosity. This line is an important landmark in X-ray interpretation, for by its position one may determine to a certain extent the degree of rotation of the humerus, and therefore make a reasonable guess at which facet shows on the contour of the greater tuberosity.

Figure *e* is taken with the humerus held nearly horizontally and rotated inwardly as far as possible; *i.e.*, the arm is in a high akimbo position. The deposit now shows in profile on the lower margin of the head of the humerus. Compare this plate with Figure 26, which shows an arm in the hammock position, in which the profile of the lesser tuberosity appears superior to the head of the bone.

define above. Perhaps this seems a sweeping statement to the many physicians who still believe there is such a thing as brachial neuritis in otherwise healthy people; yet the statements are made in all sincerity by one who has studied the shoulder intensively for twenty-five years.

The relation of trauma to the calcified cases is not yet clear. Since I first studied the subject I have always felt that it was highly probable that an acute or chronic injury to the tendon must occur long before the deposit appears. The pathologic histology of the lesion itself might be accounted for by faulty repair of an injury in tissue which has little vascularity and would be slow to restore itself. This was J. H. Wright's view from some tissue which I gave him. On the other hand, the fortuitous finding of the specimen shown in Plate VI, Fig. 5, indicates that the first change may be degeneration in the interstitial part of the tendon. Moschcowitz feels that the primary process is a tendinitis. Meyer, investigating from anatomic material, feels that both the biceps tendon and that of the supraspinatus may be attenuated from friction in overuse. I take issue with Meyer on the ground that I believe that destruction by friction *per se* does not occur in living tissue directly. Friction would first produce inflammation with resulting attenuation and atrophy of the part. I would expect in such tissue, if the local area became sufficiently poor in synovial lubrication, that congestion, tenderness and protective reflex spasm of the adjoining muscles would occur long before friction *per se* could do any damage. Whether the attrition be directly from friction or indirectly from atrophy following inflammation due to friction, the fact is that we rarely find ruptures of the supraspinatus in any but the aged laboring classes. The calcified cases on the contrary occur in young or middle-aged people, and moreover, they are usually not in people who do heavy laboring work, but in the more highly educated classes who do sedentary work, using the arms in a slightly abducted position for hours (See p. 135, Chap. V).

Since the calcified cases are, as a rule, in the middle-aged, and the rupture cases in persons at least elderly, some relation may exist. Perhaps aged cases in which rupture occurs had the tendinitis and calcified deposits in their earlier years, and thus had their tendons weakened and made prone to rupture. Or perhaps in youth strains which would rupture the tendons in older people result in merely the rupture of a few fibers. The capacity for repair is then great and restoration results. The same force in middle life might cause a slightly greater damage with hemorrhage into the tendon fol-

lowed by faulty repair and calcification. Still later in life the same force might tear the aged tendon hopelessly. A partial parallel to this hypothesis is found at the knee, where the same kind of force in early youth avulses the tibial tubercle; in middle life the patella is the weakest point and gives way transversely, but in the aged the quadriceps extensor tendon yields in many cases. In other words, the relative tensile strength of the tissues has changed with age.

Although it is the rule that cases of calcified deposits recover with no known sequelæ, I am more and more inclined to think that they must result in some atrophy of the tendon, whether they are absorbed naturally or are removed. I cannot believe that complete and sound repair takes place. This belief arises chiefly from a study of the microscopic changes which consist largely of a necrosis of the fibers. I incline to the belief that these same tendons may be prone to rupture in later years.

The Workman's Compensation laws (through the pertinent questions they put into the mouths of the members of Accident Boards and of Insurance lawyers) are sharpening the wits of modern Industrial surgeons in many ways and are having their effect on shoulder surgery also. As these little calcified deposits now cause loss to the insurance companies, they are assuming more importance than when they only bothered the individual patient. We are confronted with cases in which a slight trauma is alleged to have occurred and in which, after a lapse of a few days or weeks, disability has appeared and the X-ray shows the shadow of a deposit. Did the lesion precede the trauma? If so, did the accident aggravate the symptoms enough to make the difference between inability and ability to work? May the occupation which called for unusual or repeated use of the affected tendon have caused the lesion prior to the alleged accident? Since in cases not associated with accident at all the symptoms frequently come on suddenly, might not the association with a trivial accident be merely fortuitous?

The answers to these questions can be founded on so little in the way of statistics or demonstrable facts that they come into the realm of expert testimony and are matters of opinion only. I will try to set down my own opinions and what few reasons I have to support them.

Precedence of trauma or lesion (*i.e.*, of a single definite trauma). Since in a general way we know that the process of calcification of dead or injured tissue requires a considerable time, it is pretty clear that the lapse of only a few days after the trauma before the X-ray

was taken would indicate that the lesion had existed prior to the trauma. The same would probably be true a week or two after the trauma, but there would be increasing doubt as the interval progressed from weeks into months. I incline in general to think that the lesion precedes the trauma even when the X-ray is taken months later because (1) I have seen no instances where an early X-ray showed no deposit and yet a later film demonstrated one. (2) Because I have seen many cases where the X-ray did show a deposit soon after the trauma. In other words, in my experience the calcification usually preceded the trauma and I have never been able to satisfy myself in any case that it followed the accident.

It not infrequently happens that the X-ray will show a quiescent deposit without any symptoms in the supposedly well shoulder, as well as in the traumatized one. In fact, bilateral deposits are so common as to make us doubt whether acute trauma is ever a cause. They suggest that the primary cause is a general one. For instance, a systemic one; an attitudinal one; a congenital morphologic one. Yet in persons who use to excess both arms in their daily work, a chronic traumatic cause might be postulated even in bilateral cases.

Aggravation by trauma. Any one who has seen the bursa opened at operation and beheld the angry, red tumefaction with its white or yellow summit would agree that a blow on the inflamed area or even a sudden excess of tension on the tendon might aggravate the condition. One can readily see that the softened area might burst and spread its contents into the bursa under such circumstances. And it is clear that a trauma need not be very severe to do this. Yet to have the deposit burst into the bursa is in my opinion nature's way of curing it (Fig. 34). The acute symptoms are often soon followed by complete and permanent relief!

Excessive use as a cause. I am inclined to believe that this is the most common contributory cause if not the primary one. There are many instances besides "writer's cramp" and "housemaid's knee" to support the statement that in our division of labor the abuse or overuse of particular organs or tissues results in pathologic changes. Millions of years of heredity have not prepared the modern stenographer or machine operator to keep their supraspinatus tendons stretched and under tension, on the *qui vive* hour after hour, day after day. While only a few individuals in these employments have a severe tendinitis or bursitis, I believe that could we see the supraspinatus tendons from a series of stenographers who have worked steadily five years or more, some pathologic change would

be found in them. Miners do not all have "miner's elbow," but I venture to say that all those who habitually work on their elbows would show hypertrophic and degenerative changes in their olecranon bursæ. It is therefore my opinion that when disability arises from these calcified deposits in industry, the presumption should be that they are "Industrial," unless it can be shown that the occupation is not of a nature to demand overuse in this region of the body, or that the patient at home pursues an avocation which causes such abuse. Nevertheless it would require many months or years of such abuse to produce a typical "calcified deposit," for the minute pathology of these lesions is of a chronic type, although the inflammation about them may be very acute.

Coincidence of onset with trauma. It is certainly true that many cases have a sudden onset of acute symptoms with no history of trauma. It is easy to suspect in any given case that trauma was only an afterthought to get compensation. I have suspected this was so in some cases, but in others there was a clear history of accident, leaving little room for doubt that the accident precipitated the symptoms. It seems to me that no rule can yet be formulated and each case must be decided on its own merits; the more vague the story, the less likelihood that the course of the trouble was influenced by the trauma. The character of the occupation, the condition of the other shoulder, the duration of the kind of work, and other circumstances should be taken into account. At present my tendency is to consider such cases as instances of coincidence, even when the symptoms followed soon after the accident and the X-ray was taken weeks later. It is so easy to hit any sore spot in the body and to attribute the soreness to the blow which drew attention to it! Add to this the incentive to obtain compensation and the psychologic fact that it is human nature to influence one's self into any belief in which one's fancied interest lies. Nevertheless I would not deny that a single trauma (if well proved and of sufficient force) could be the direct cause of the onset of disabling symptoms. Furthermore, in doubtful cases it is only just to remember that it is likely that the occupation itself may have been the underlying factor.

Symptoms. Symptoms may be absent entirely and yet the X-ray may show a large deposit. This fact is particularly striking in the bilateral cases, for one side may be symptomless and the other the cause of agony. *Radiologists not infrequently find these symptomless deposits accidentally.* Yet these facts do not prove that the disease may run its entire course without symptoms. On the con-

trary, since we often see a large deposit in a case which has had symptoms for only a day or two, it is clear that a symptomless case may at any time present symptoms. Therefore, we doubt whether any case ever runs its whole course without symptoms. Most of the bilateral cases I have seen had one symptomless shoulder at first, but perhaps a year or two later had typical symptoms come on in the other. I have yet to see a case in which a good-sized deposit disappeared without ever causing pain or restriction. Beyond question the X-ray demonstrates that spontaneous absorption takes place in most cases, although some such instances may be errors of X-ray interpretation. Some certainly discharge into the bursa and are absorbed from there. Whether there are actually cases which never give symptoms and run their whole course without complications, I do not know. Many never give severe symptoms.

Most cases run a subacute course, a few are exceedingly acute, and a few are truly chronic. An attempt to classify cases strictly in this way results in confusion, for we find many cases which are borderline and others which in their course pass through all three stages.

The usual history is not of acute pain at the beginning. The first symptom noticed is a slight, painful "hitch" during certain movements of the arm, such as in raising one's arm to put on a coat or in placing an object on a shelf. Sometimes reaching across a table for some object, as a pen or a salt cellar, causes this feeling. Women complain that they cannot do their hair. Little attention is paid to this until it becomes associated with every such motion. The slight pain becomes expected and eventually dreaded before such motions are performed, so that the patient corkscrews his arm in a peculiar way to avoid the "hitch." Instinctively he learns to rotate outwardly as he abducts and thus avoids pinching the inflamed point under the coraco-acromial ligament as the arm is raised. Presently the sensitiveness reaches such a point that involuntary scapulo-humeral spasm is produced, and the normal, smooth division of motion between the movement of the scapula on the chest wall and the elevation of the humerus on the scapula is destroyed.

Loss of Scapulo-humeral Rhythm.

Normally when one raises the arm to a position pointing straight toward the ceiling, much of the motion is performed by rotation and elevation of the scapula on the chest wall, while the remainder is performed by the true joint. (See Fig. 25.) The two motions go on *pari passu*, so that as one watches from behind it is impossible to

say that either motion proceeds faster than the other. In nearly all affections of the joint or bursa, this even distribution of motion is destroyed, because the sensitive point, unwilling to move, sends its reflex telegram to the short rotators to lock the joint in a fixed position and to hold it there by spastic tension. This phenomenon is one of the most important for the student of shoulder conditions to learn, and its behavior in these cases of calcified deposit is very characteristic. At first it is not complete, the spasm being semi-voluntary in the sense that the individual can control the inhibition of it to a certain extent. For instance, in mild cases the spasm holds the joint fixed as the arm makes its upward journey for about ninety degrees. Then the individual semi-consciously relaxes it and the joint surfaces themselves carry out the remaining ninety degrees to complete elevation. When the sensitiveness is still greater, even a strong effort of will cannot relax the spasm and the arm only ascends so far as it can through the scapular motion—almost straight out from the shoulder laterally. Later the spasm holds continuously night and day until firm adhesions form, producing a fibrous ankylosis which effectually splints the inflamed parts until they heal. When healing has taken place the adhesions gradually stretch and motion of the joint slowly returns. The course of this whole process in the most severe case is about two years. The fixation is never permanent. The great majority of cases do not have this severe course. Probably none would unless delayed by injudicious treatment.

The cardinal symptoms of the whole picture are pain, spasm, limitation and atrophy. These four symptoms may make a variety of complexes according to their relative severity. In one case pain may be the predominant one; in another, spasm; in a third, adhesion. I have seen cases which ran an almost painless course as if the spasm was started at once without the sensation of pain being experienced. The painful stage when the spasm is intermittent or semi-voluntary was absent; spasm from the early days was complete and adhesions took place before pain was felt in any considerable amount. The ankylosis stage persisted for months, the patient's chief complaint being from the limitation of movement. Some cases have relatively little spasm, no adhesions and constant pain. As a rule the amount of pain is inverse to the amount of restriction. The physician may relieve the pain by fixation of the arm and allow the adhesions to occur at once, but as I shall show later this is bad practice, unless the patient is one who can afford to avoid using the arm for many months. In most cases the "hitch" stage persists for many weeks

with gradually increasing pain until past the stage of semi-voluntary spasm when restriction dominates the picture.

Pseudo-neuritis. If there has been a long, painful stage all the adjoining nerves become sensitized and the phenomena we call "neuritis" supervene even when fixation has occurred. The pain or hyperesthesia, originally mainly felt in the circumflex distribution about the shoulder and near the insertion of the deltoid halfway down the arm, becomes more diffused. It shoots up into the neck, on to the back of the shoulder blade and down the distribution of the radial nerve to the thumb and forefinger. Even the ulnar and median nerves may become sensitized, the hand and fingers swell and the skin over them becomes glossy. The picture is that of "brachial neuritis," but removal of the calcified deposits relieves it!

When the patient has hugged this painful, tender, useless arm to his side for weeks it presents a most unnatural, misshapen appearance. The back of the hand and upper flexor region of the forearm are swollen, while the anterior portion of the wrist is not. The fingers are partially flexed and cannot be extended even passively. Meantime the sleepless nights, incessant pain, and the dread of incapacity and uselessness have had their dismal effect on the patient, who may reach a very pitiable condition. Drugs have only too often added to the unhappiness of the individual—the family has a querulous invalid to care for.

Fortunately this picture is not the usual one nor is it frequent at present in this community, for nowadays the X-ray is sought for most joint conditions and locates the deposit; the condition is treated more hopefully and intelligently than when the diagnosis of "neuritis" was more common. Mild cases are far more frequently seen today.

Pain at night and inability to get into a comfortable position is the most uniform and constant complaint in all stages of this disease whether in acute or chronic form. Even the adherent cases cannot sleep for long on the affected side. The average patient tells you that he could put up with the pain in the day if he could only get a good night's sleep. In the early stages before there is much spasm, relief may be obtained by sleeping with the hand behind the head. In this position the tender point lies beneath the acromion, where it has plenty of room and where the supraspinatus tendon is relaxed. It is probably the most favorable position for the blood supply to repair damages. Women seem to assume this position more readily than do men. If a patient told me that she had pain in doing her hair and

could not get her arm comfortable at night without putting her hand behind her head, I should be pretty certain that she had a calcified deposit in her shoulder. If she added that there was pain in the lower fibers of the deltoid near its insertion in the humerus, I should feel still more certain. If I found a raised, tender point about the size of the tip of the finger on the greater tuberosity which moved as the humerus was rotated, I should consider the clinical diagnosis established, although subject to confirmation by the X-ray.

The location of pain in the region of the lower fibers of the deltoid six inches or so below the real lesion, is a curious but very constant phenomenon in these cases, in all stages of their course. The complaint of pain in this region is almost diagnostic without other symptoms. I am uncertain whether it is to be explained as reflex pain or as directly due to spasm of the lower deltoid fibers. If one compares the two arms by taking the lower parts of the deltoids between fingers and thumbs of both hands simultaneously, one finds a distinct thickening, and there is often tenderness, too, on the affected side, such as would be produced by a tonic spasm of the fibers. The pain located here may be subordinated in the acute cases to the local pain in the bursa. One can in acute cases mark out the exact limits of the inflamed bursa by carefully pressing with a pencil tip and obtaining the patient's coöperation in locating the line of transition to acute tenderness. This is a very definite line and it may sometimes be vaguely seen when the bursa is distended with fluid and fibrin. Even when the bursa is the maximum tender point, the lower deltoid region may be the seat of maximum pain. There may be no local tenderness over the bursa and yet much pain and some tenderness in the lower deltoid.

Acute cases are characterized not only by the above-mentioned, distinct, local tenderness, but by excessive spasm and pain on the slightest motion of the joint. An acute attack may at any time appear in the course of a subacute or chronic case, but the most severe attacks usually appear without warning or known cause, though sometimes following a slight trauma. They represent the onset of bursitis from extension of the inflammation about the deposit in the tendon beneath the bursa. The symptoms are those of an acute bursitis and are homologous with similar acute inflammations in other serous cavities, *i.e.*, congestion, friction, pain, protective spasm, fibrinous exudate, adhesion, cicatrization, partial absorption. The acute stage is therefore brief, lasting a week or two. It is during this period when the bursa is full of fibrin that it may be outlined as

described above. The patient may wake at night with agonizing pain in the shoulder and be unable to move the shoulder joint more than a few degrees. Tenderness over the bursa is exquisite. Spasm has fixed the joint rigidly so that the arm is held in the sling position and the patient cannot be persuaded to move it in the least. One can take the patient lightly by the thumb and he will follow submissively where he is led, his whole body on the alert to avoid using the joint in the slightest degree. He cannot by any possibility sleep on the affected side and usually tells you that since the pain began he has "walked the floor" or had to sit up in a chair. For several nights no ordinary remedies give relief and considerable doses of morphine may be required. Exploration of the bursa at this stage usually shows that it contains fluid and fibrinous exudate overlying the boil-like elevation on its base with its crimson, turgid periphery and whitish center. Until one has seen this appearance it is hard to realize how much these patients suffer. It is in such cases that the relief by operation is particularly striking, but they often get well promptly without operation or any treatment at all.

FIGURE 11*a*. BURSA DISTENDED BY FLUID CONTAINING CALCIFIED PARTICLES (*Natural size*) Case 111

a. Shows the greatest distention of the bursa which I have ever seen from this cause. The film was taken by Dr. A. W. George, who referred the patient to me. I have, however, seen many bursæ as much distended as this, in cases of rupture of the supraspinatus, where the constant influx of fluid from the joint during efforts at abduction has dilated the bursa. Notice the gravitation of most of the calcified material to the bottom of the sac, which is bilocular, and yet enough particles have clung to the lining to cause its outline to show in the film. Case 141.

b. Schematic drawing to show that a distended bursa is a concavo-convex cap beneath the deltoid and placed in an antero-lateral position over the insertions of the short rotators.

FIGURE 44*b*. A DISTENDED BURSA IS CONCAVO-CONVEX
(*Size reduced one-half*)

Such acute symptoms subside in one to two weeks, some cases being followed by adhesions of the bursal surfaces which last many months. More often, especially if the physician has not tried to help the patient by binding up the arm, the spasm subsides, the soreness disappears, normal motion returns, the "hitch" reappears and the condition is the same or better than before the attack. There are various stages between these two extremes; in some cases the attack seems to clear up the whole trouble, but often more or less spasm and restriction remain for months. I am quite positive that in the cases which clear up, the soft material has perforated into the bursa and been absorbed there. In those cases which have been through an acute attack and on which I have operated long after, I have found the deposit drier and firmer and more crystalline in composition and more intimately combined with the fibers of the tendon than those operated on in the acute stage. The deposit is likely to be firmer in chronic cases which have never had an acute attack. Apparently in acute attacks invasion of leucocytes about the deposit produces a sterile pus which mixes with the finely divided calcareous matter and softens it to the consistency of cream or butter. This creates tension in the substance of the tendon and tends to make the softened matter point into the base of the bursa. For some unknown reason perforation does not occur into the joint. It is the inflammation of the synovial base of the bursa which produces the symp-

toms so suddenly and starts the sequence of friction, pain, spasm, exudate, etc. At operation one usually finds the softened material under tension, so that when the white or yellow head of the boil-like center is nicked with the knife the material is squeezed out, as if it had been pent up. The operation gives relief to the tension and is effective in the same manner as in opening a boil. It is an interesting fact that under local anæsthesia the patient may express relief as soon as the tension is lessened. It seems that in these acute cases there are two kinds of pain, a deep, severe ache due to tension in the tendon, and the friction pain of the inflamed bursa.

I hesitate to describe a chronic form of this condition, since I have seldom known of an instance of more than three years' duration. Nor do I know how long symptomless deposits may exist. What I mean when I speak of a chronic case is one which lasts many months without going through an acute or adherent or neuritic stage. A case in which throughout its course the symptoms are confined to the painful "hitch," pain in the lower deltoid and a distinct rotating sensitive point, I should call chronic. In such cases the amount of the deposit as shown in the X-ray is usually small, perhaps minute, or even not demonstrable. Probably many such patients never consult a doctor. I see them more often among my friends who tell me casually about their symptoms, having heard that I am "interested in shoulders." Patients who consult me and have X-ray proof of the diagnosis and are told that the deposits are no harm in themselves, and that no operation is necessary unless the pain is severe, seldom come back to report when they are well. So that I can only give my impression that mild chronic cases do exist and that their duration, like the subacute ones, is seldom more than three years. As this was being written a patient on whom I had operated for a severe acute attack in his right shoulder, nearly eleven years ago, called to see me about his left shoulder. The right had remained well. Reference to my old notes showed that X-rays had revealed a deposit in his left shoulder at that time. He had been conscious that the left shoulder was not well, but it gave him no real bother until about six weeks ago, when mild symptoms began. They are still mild and he is taking diathermy. X-rays show that there is no deposit at present and there is a slight roughening of the tuberosity at the facet of the supraspinatus tendon, but there is no such roughening on the right side, which was operated upon. Evidently in this case the left ran a truly chronic course without complications. This one case, so far as it goes, indicates that the ultimate damage to the tendon may be greater if the deposit is not removed.

Atrophy of the spinati is found in all cases which have had severe symptoms for more than a few weeks. I have observed it in acute cases as soon as three weeks after the onset, and it doubtless may occur even earlier than this. Looking at the patient from behind one sees the spine of the scapula on the affected side more distinctly. In adherent cases this appearance is very pronounced and the shrinkage of the muscles may be so great that complete paralysis is suggested. I am inclined to think that in the worst cases the muscular bellies are in a condition resembling that of the muscles of the forearm in Volkmann's paralysis, which in fact is an ischæmia. However, they regenerate better under appropriate treatment. In such cases I am satisfied that rough manipulation under ether may rupture the muscles themselves rather than the adhesions in the bursa and joint. Atrophy of the deltoid is also usual; I have known of cases being mistaken for circumflex paralysis. The biceps also is as a rule more or less weak, and in long-standing cases all the rest of the muscles of the arm show the result of lack of use. The teres group is usually found in spasm and it is difficult to tell the amount of atrophy, for they are contracted in contrast to the spinati which atrophy in a stretched position. When one stands back of the patient and with forefingers in the axillæ and thumbs behind the posterior axillary folds, and one palpates the muscles near the border of the scapula, one finds that those of the affected side are thicker and firmer than those of the normal side. Nevertheless, there may be atrophy of these muscles. The fact is that the spastic teres muscles are shortened by adduction of the arm and thus appear thick on palpation. Yet they may be shrunken as a whole although short and thick from constant spasm. These spastic muscles are always somewhat tender. This thickening and local tenderness of the teres group should not mislead us, for the cause of their sensitiveness and spasm is not local in these muscles.

In examining for shoulder lesions of any sort it is important to note the condition of the spinati. Atrophy of these muscles is an accompaniment of all lesions of the shoulder joint of a serious inflammatory nature. I do not find it in cases of malingering and of hysteria. Furthermore, its degree is a fair gauge of the gravity of the lesion, and also serves to gauge the prognosis.

It may be seen from what I have said about the symptoms caused by calcified deposits in the tendons, that a strict division of cases into acute, subacute and chronic cannot be made. I cannot take my own series and pile them in three groups of records after this

manner. Neither can I separate them into adherent and non-adherent cases satisfactorily. Restriction of motion may be slight or complete and of all degrees between. Yet these divisions are all clinically useful, though ill-defined, and mean something, at least for a time, in individual cases. A corollary can be drawn from this that prognosis is uncertain. One cannot tell in a given case exactly what its course is to be. This makes advice as to treatment and comparison of results very difficult.

Restriction of motion is usually in one or both of two different arcs of motion, *i.e.*, in external rotation and in elevation. When elevation is restricted and external rotation is free, one should suspect at once a condition in which the inflammation about the calcified deposit is great enough to make a mound in the base of the bursa, but has not yet created irritation of the synovial lining of the bursa. Rotation is painless because the inflamed, raised spot can rotate under the deltoid without striking any hard or any firm tissue. On the contrary, when abduction or elevation is performed, the inflamed mound is pinched between the tuberosity and the acromion or coraco-acromial ligament. The pain thus caused instantaneously produces scapulo-humeral spasm and the upward movement is restricted. Let this patient stoop over as far as possible with his shoulder muscles relaxed and the arm abducts on the body by gravity. The tender point rides easily under the acromion in this position because the supraspinatus is not acting to produce tension about the deposit, nor is the deltoid pulling the humerus vertically upward against the acromion. If the patient then fixes his shoulder voluntarily, he can stand upright with the arm elevated.

Such cases as I have just described can usually be separated from malingerers, because one can always find the rotating tender point where the deposit is, for it is easily felt as it passes beneath the deltoid fibers. Furthermore, having made the patient stand with the elevated arm, he will be seen to have a "hitch" or a period of scapulo-humeral spasm as he lowers it from the vertical position, and the tender point in its downward journey again passes the narrows under the acromion. This sign is also a *sine qua non* of complete rupture of the supraspinatus, but in the latter no calcified deposit is shown by the X-ray.

A symptom which is occasionally seen in these cases is what I call the "down-it-will-go" symptom. It is rather infrequently complained of, but often acknowledged if asked for. Patients with complete rupture also show it. A patient will say that the arm is para-

lyzed or that he is afraid it is going to be paralyzed. He reaches for some small object or is carrying one, and "down-it-will-go." It seems to be an involuntary sudden relaxation of the grasp. It frightens the patient, although it is usually spoken of diffidently or with an apologetic laugh. It is irregular in its occurrence. I cannot explain it, but my theory is that it is a phenomenon associated with position sense. I have already stated that in some cases spasm starts without pain. In a case of circumflex paralysis which I observed, and on which I operated under local anæsthesia to see whether the supraspinatus was torn, I found that the interior of the bursa and the joint were completely insensitive although the skin sensation was present. In a doctor on whom I was operating under local anæsthesia for a calcified deposit I could at will start spasm without pain by touching the base of the bursa. These observations indicate that the bursa and joint are supplied by nerves which do not transmit ordinary tactile pain but serve to excite the scapulo-humeral reflex. It may be that this "down-it-will-go" symptom is something caused by this set of nerves, when, in reaching for the object, the sensitive point comes against the acromion. Without consciousness the grasp relaxes in order to get rid of the weight which, by leverage, increases the pressure on the tender point. In this connection we also have the phenomenon that the pain in these cases is as a rule attributed to a point near the lower insertion of the deltoid.

FIGURE 45. CALCIFIED DEPOSIT IN THE TERES MINOR

In interpreting X-rays of calcified deposits one must be sure of the position in which the film is taken in order to say positively that the calcified material has escaped from a tendon into the bursa. This is particularly true in cases where the deposit is in the teres minor, as in this picture. The insertion of the teres is very low and if, as in this case, the view is taken with the head of the bone in inward rotation, the deposit appears to lie in the bursa, when in reality it is still confined within the tendon substance.

FIGURE 46. CALCIFIED DEPOSIT IN THE INFRASPINATUS

Figure 46 was taken with the plate on top of the shoulder and the rays nearly parallel with the long axis of the humerus, in order to bring out the bicipital groove. The prominent deposit in this case was in the infraspinatus, as is shown by the fact that it is about one-half inch exterior to the bicipital groove. There is also some deposit in the supraspinatus which shows poorly in this picture, for it is overshadowed by the tuberosity.

There should be some differences between the symptoms of cases of deposit in the subscapularis and in the supraspinatus. The location of the tender point, whether inside or outside of the bicipital groove as shown by the maximum spot of tenderness, is my usual way of differentiating. Careful study of the X-ray is confirmatory. Whether the symptoms themselves differ I am not sure. One would think that external rotation would be limited in subscapularis cases, but I have not found this true enough to rely on. A recent quite acute case had no such limitation. Another case of bilateral subscapularis deposit which occurred in my own household had symptoms quite like those in a supraspinatus case except in the location of the tender spot. My series of cases is not large enough to make comparison, but the point is important because it helps in performing the operation.

The X-ray should confirm a diagnosis of calcified deposit, but one should be very exacting in demanding excellent technique and films made from several angles before making a negative diagnosis when the clinical picture is typical. This is particularly important in ruling out a deposit in the subscapularis tendon. A recent instance was only positively diagnosed by a fourth film which showed the deposit in profile. The deposit was large, but in the first three positions the shadow overlaid the bone in such a manner as to elude experienced eyes, although after the fourth film it could be identified in each picture. A very slight rotation of the humerus may throw the shadow over the bony outline, especially if, as often happens, there is only a mere fleck of calcareous material. Even a large recent deposit of little density is easily overlooked. The Röntgenologist must learn to take pains to get each facet of insertion in profile.

Even before calcareous material is demonstrated by the X-ray, the diagnosis on clinical signs alone may be made with a considerable degree of certainty.

Treatment.

Since the prognosis of these cases is always eventually a good one, and since in any given case the probable course cannot be foretold with certainty, no treatment of a dangerous nature can be considered. Even a risk of any permanent minor damage would be unwise. All cases, whether treated or not, in which I have known the late result, have recovered without permanent complications of any kind. Recovery is only a question of time. The object of treatment is purely one of the relief of symptoms. Any one who wishes may endure the whole course of the trouble without other treatment than palliative remedies such as occasional doses of aspirin or the application of an ice bag or a hot water bottle. Many acute cases following perforation recover in a few weeks. Some persons prefer to endure daily pain month after month rather than to submit to a minor operation or go to the trouble and expense of prolonged treatment by physiotherapy. My personal opinion is that surgical removal of the deposit is practically free from danger, sure to relieve the severe symptoms at once, and, in fact, that it generally will relieve all really troublesome symptoms within a few weeks. It is not a painful operation; it can be done with local anæsthesia; it requires only a few days of hospitalization, and there is very little postoperative pain as a rule. It seems to me that the patients whom I have followed through an acute attack suffer more in forty-eight hours than does a patient who has been operated upon in his whole convalescence. I have performed the operation on a number of doctors, who have previously had acute attacks, and they have been of the same opinion. Furthermore I have seen, in subacute and chronic cases, the constant neuritic type of pain with consequent loss of sleep, reduce patients to a very serious physical condition, the relief of which would justify a much more dangerous operation. It therefore seems to me that it is better to have the deposit, if large, removed as a routine, than to run the risk of either an acute attack or of severe chronic pain. It is quite reasonable for a patient to say, "But I may never have any severe pain, and then the operation will have been unnecessary." This is quite true and the answer is that there is little objection to waiting until the actual pain makes the prospect of operation more welcome. It would be hard to say for another individual how much annoyance and loss of sleep is equiva-

lent to the operative pain and discomfort. Certainly pain sufficient to prevent work is enough to demand an operation of the trivial character of this one. Statistics as yet cannot show in a given case what the chances are that serious pain will ensue.

I think it is fair to say that the severe pain is relieved at once by the operation and that the patient may expect the return of full use of his shoulder in from three to six weeks, although he may feel twinges of pain in extreme positions of the arm for several months.

In general my attitude is: "If I had your trouble I would prefer operation as the best way to avoid pain and disability. You will get well anyway, but your total pain and discomfort is pretty sure to be worse and likely to be much worse, if you are not operated upon. I advise you, but I do not urge you, to have this deposit removed. There is no danger of your having a crippled arm or any permanent trouble, whether treated or untreated."

Many patients, on receiving this advice and reassurance that they will not lose the use of their arm, prefer to endure the pain, annoyance and discomfort. Others ask what other treatment there is apart from surgery. The answer is that aspirin and similar drugs will help them bear the pain, particularly at night. Morphine will relieve the pain, but its use for more than a few nights is of real danger, for the pain may last many months and a pitiable habit result. No drugs are known which will cause absorption of the deposit when taken internally. Since the deposit is deep in the tissues and covered by skin, fat, fascia, muscles and blood vessels, in which the blood is circulating, no local applications as ointments, linaments, iodine, etc., can reach the lesion and do good. The most that can be expected would be slight, temporary relief from counter irritation, such as is gained by hot or cold applications. Massage and manipulation and active or passive exercises might be harmful unless most judiciously performed. After the acute symptoms are gone these agents may be helpful in restoring motion if it has become restricted. Very acute cases are apt to be very brief cases, and their spontaneous recovery may be coincident with surgical or other therapy.

I know of no form of electrical treatment which might be expected to remove the lesion except diathermy. It is rational to hope that the use of this form of physiotherapy might help nature to absorb the deposit and resolve the exudate about it. It is held that diathermy heats living tissues through and through, and the effect of this heat on such deposits has been claimed to cause absorption. Since these deposits are *naturally* absorbed in varying periods, it

is difficult to be sure that their disappearance under treatment by diathermy is not a coincidence. No attempt so far as I know has been made to check up a series of cases so treated with an untreated series. The same criticism applies to the results of surgical removal, but we know then, that the deposits, and symptoms as well, are removed. In some cases I have operated after the patient had been treated with a course of diathermy, and yet found a large deposit. In others so treated the X-ray has continued to show the deposit for some months after the symptoms were relieved. However, it makes little difference whether or not the deposit is removed, if the pain disappears. I have known of a few cases in which this has happened after a few weeks or months of treatment by diathermy. They were relatively mild cases. In others I have operated when diathermy failed to remove either deposit or pain. Furthermore, after perforation in the bursa has occurred the deposit absorbs spontaneously within a few weeks. Yet if a patient wishes some treatment other than operative, I advise diathermy. I do not use it in my own work for two reasons. *First*, being inexperienced with its use, I should fear the danger of burning the patient more than I would the danger of surgery. *Second*, I know that operation permanently cures; I have some faith that diathermy will assist the natural process of repair.

Dr. E. B. Mumford of Indianapolis, who is a prominent orthopedic surgeon, believes that diathermy is the treatment of choice (*Jour. A. M. A.*, Vol. 97, No. 10, 1931, pp. 690-694). He gives details of his technique. "All of our cases became well by this conservative form of treatment and in all cases with a deposit shadow, the deposit either entirely disappeared or became much smaller in size and less dense in the X-ray. In none was surgery necessary. The average number of treatments was twenty-one, given daily for the first two weeks and then twice a week. Relief from pain was obtained in from three to ten days. The end result was complete restoration of function without pain. Ten cases have been checked at the end of one or more years and no recurrence noted."

I am far from denying that diathermy is effective. It is too bad that comparative tests of different forms of treatment cannot be made. One thing is greatly in favor of diathermy, and that is that it is necessary for a surgeon to know the anatomy and pathology of this particular region if he is to obtain uniformly excellent results by operating.

There is one form of treatment not uncommonly given, which I believe is to be heartily condemned, *i.e.*, fixation by strapping or

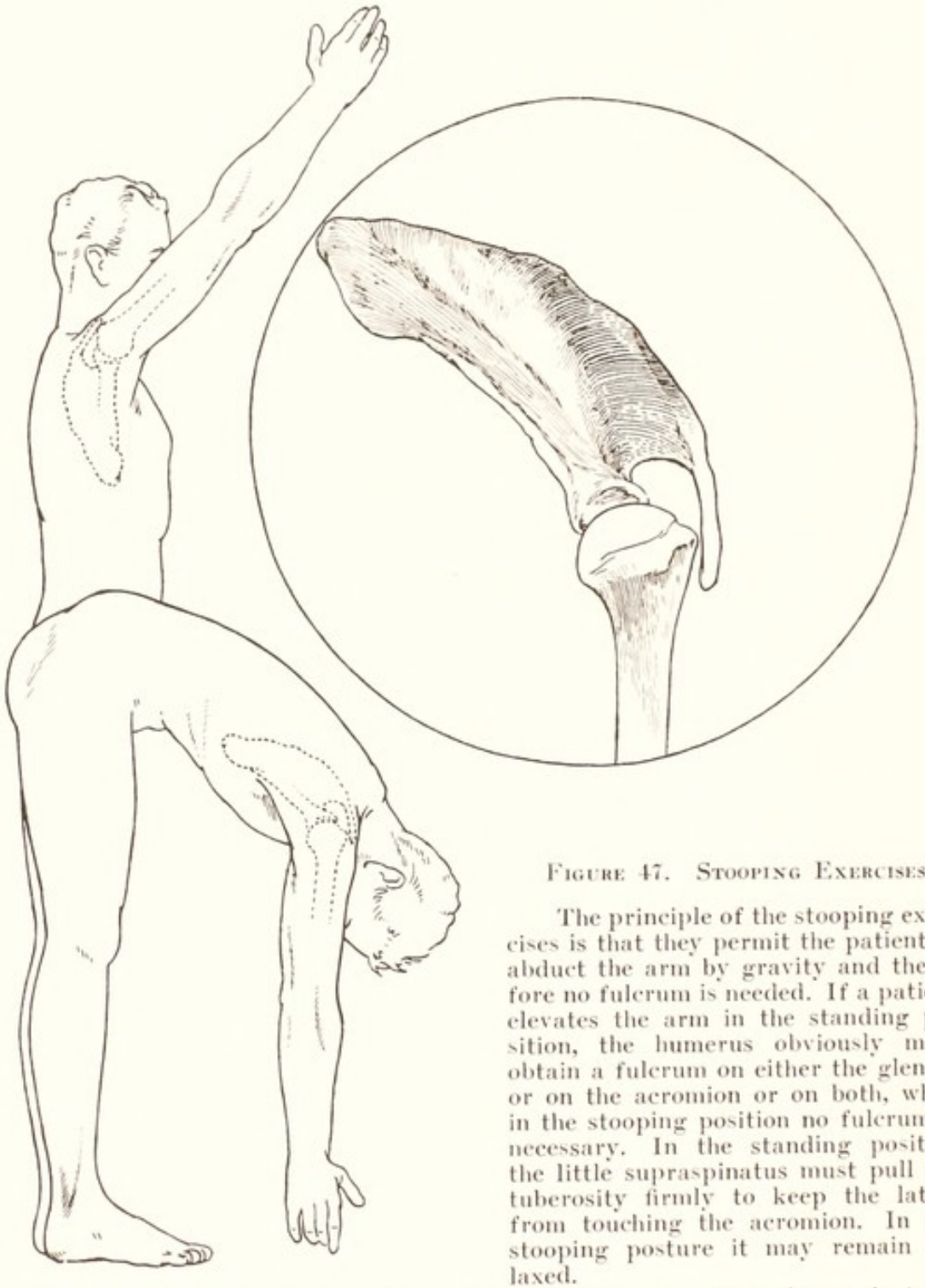


FIGURE 47. STOOPING EXERCISES

The principle of the stooping exercises is that they permit the patient to abduct the arm by gravity and therefore no fulcrum is needed. If a patient elevates the arm in the standing position, the humerus obviously must obtain a fulcrum on either the glenoid or on the acromion or on both, while in the stooping position no fulcrum is necessary. In the standing position the little supraspinatus must pull the tuberosity firmly to keep the latter from touching the acromion. In the stooping posture it may remain relaxed.

Since the sore point in most lame shoulders is on the tuberosity at just the region where it tends to obtain a fulcrum on the acromion, it is highly important that the patient with a damaged supraspinatus should not delay his recovery by frequently raising his arms. The exquisite tenderness which arises in these cases is nature's defensive effort to warn the patient not to use the arm in this way.

When the patient with the sore shoulder is in the stooping posture he can much more readily brush his hair, button the back of his collar or perform any other motions. He can, for instance, learn to put on his shirt and coat over the lame arm when stooping, and then after he has risen to the erect position put on the other sleeve.

When in the stooping position, either lateral or antero-posterior motions can be done with a pendulum-like movement without great muscular effort. In this position the humerus not only tends to avoid a fulcrum, but actually the weight of the arm helps to stretch the contracted tissue of the joint.

bandage. In my opinion many of these cases are made worse by this treatment, which encourages adhesion of the roof of the bursa to the inflamed tendon. All the periarticular structures become fixed, the muscles atrophy and become ischæmic and contracted. Many months may pass before the joint moves again, although ultimately it always does become mobile. The neuritic symptoms are particularly troublesome in such cases, although sometimes the ankylosis is entirely painless and the patients are only inconvenienced by the restriction. Spontaneous scapulo-humeral spasm is a sufficient guard for the inflamed joint and we should neither add to it with straps and bandages nor fight it with forced motions. Active "swinging exercises," with the patient in a stooping position, should be used in most cases. When the patient stoops the strain is taken off the supraspinatus and it is far easier to move the joint. I regard it as very unwise, until the convalescence is well advanced, to allow the patient to exercise the arm in a standing position because this forces the supraspinatus to coöperate with the deltoid in supporting the weight of the arm. The pain caused by this at once starts scapulo-humeral spasm and the joint ceases to move. It is better to let the patient stoop with the arm hanging relaxed. If the patient straightens up again as soon as spasm begins, he will in fact adduct the arm. In other words, when one stoops with the arm relaxed one abducts (elevates) the arm on the body; when one straightens up with the arm relaxed, one brings the arm into the anatomic position.

The principle of "stooping exercises" is useful in many ways. Gentle exercises of this kind prevent adhesions following the acute or subacute attack. Vigorous exercises loosen up adhesions already formed. Gentle postoperative exercises hasten convalescence; too vigorous ones start scapulo-humeral spasm and delay recovery. It is unwise to give passive exercise or permit active exercise which causes even moderate pain. Yet to fix the arm in bandages to relieve pain is also unwise. When one has seen the inside of these inflamed bursæ one can readily understand the danger of either fixation or over exercise. Gentle motion, unhampered by bandages or spastic muscles, is indicated to prevent adhesions. The patient should be allowed to change the position of the arm as freely as he cares to. He should be instructed to keep it in external rotation as much as possible, because an arm held in the sling position is in internal rotation, and if it adheres in this position, it requires a long time to gain normal external rotation. We should avoid scapulo-humeral spasm not only because it promotes adhesions, but because much of the actual pain

is due to the spasm pressing the inflamed parts together. Spastic muscles are not only painful but tender.

In two early cases with very small deposits and mild symptoms I have tried the experiment of keeping the patient in bed with the arm abducted, in the way in which I treat cases in the non-calcified type. (See p. 218.) This treatment seemed desirable for several reasons. It is the natural position assumed by many patients to obtain ease; *i.e.*, they sleep with the hand behind the head. In this position the supraspinatus is relaxed and its tendon is not stretched; this would permit an active blood supply to promote repair. This position would bring the inflamed portion of the tendon wholly under the acromion, where it could not adhere readily, and if it did adhere would tend to loosen from mere gravity. Both cases were soon relieved.

Another form of treatment remains to be mentioned—aspiration as suggested by Flint. After Flint's publication I tried this once with some success. I am satisfied that in this case I actually punctured the deposit and allowed it to leak into the bursa and thus relieved the pain. This was not exactly Flint's plan, but may also have been his achievement in his cases. I have opened so many bursæ without finding much fluid that I doubt the wisdom of trying this procedure. Like most needling in other tissues, it seems to me to carry all of the disadvantages of an exploratory incision and some serious ones of its own. It would be rational to thrust a large aspirating needle into the deposit under guidance of the fluoroscope, but I would prefer to be operated upon were I the patient.

As in the case of many other ailments which affect the human body, there is great variation in the degree and duration of the pain it produces. Even very acute cases may recover completely in a few weeks with the simplest palliative treatment. Therefore any form of therapy is deceptive in its results. I have often had a patient postpone accepting my advice for operation and seen recovery promptly ensue! No surgeon should feel as did Jack Horner and say, "What a big boy am I!" after removing one of these deposits. These rapid cures occasionally occur after any of the other forms of therapy, for nature does the work in spite of what we do at the same time. Yet at the present date, I can unhesitatingly say that unless there is some good reason to the contrary I should advise operation in all painful cases, and in all cases where the deposit is as large as a lima bean, even if there is no pain. I sincerely believe that operation is a far less serious matter than fixation, and no more dangerous than diathermy or intravenous therapy.

END RESULTS IN CASES OF CALCIFIED DEPOSITS

The following study is based on an analysis of twenty-nine replies to a questionnaire recently sent to patients on whom I had operated for this condition long enough ago to indicate whether there have been any unpleasant after effects, due to the operation, recurrence of the lesion or other complications. The period elapsed since the operation was ten to twenty-four years in nine cases, five to ten years in eight cases, and one to five years in eleven cases. All these patients stated that they had had no further trouble of any kind with the shoulder which had been operated upon, but ten had had similar trouble of minor degree in the other shoulder. In most of these cases there had been evidence of deposits in the other shoulder at the time of operation.

These data confirm my impression that when these patients have been operated upon their symptoms do not return in the same shoulder. Furthermore I have not known of any late complication which followed about forty other similar operations on patients who did not reply to this particular inquiry.

Two patients whose deposits were in the subscapularis, will be given separate consideration later, and are not included in the following *résumé* of the replies to the questionnaire. As in the case of most questionnaires, some of the answers were not satisfactory. For the sake of brevity, doubtful replies to individual questions will be omitted without making such notes as "answer equivocal," or "not stated." Consequently in the following analyses the sum of numbers in each statement will not always be twenty-nine.

Antecedent Trauma. Twelve stated that the attack followed a definite injury; thirteen stated that there had been no injury; three were uncertain. These figures are also in accord with my whole experience, which is that the injuries are usually trivial, and often uncertain. In compensation cases one is tempted to think that the history of injury may be purely an afterthought, although about half the cases may be said to give a definite enough history of trauma to account for rupture into the bursa. For instance, the twelve injuries were:

No. 79, business man, "Throwing baseball"; No. 81, iron worker, "Pulling on heavy bar"; No. 103, osteopath, "Fall"; No. 105, no occupation, "A bad fall down stone steps, and a sudden wrench later on"; No. 113, physician, "Fell on elbow with arm held rigidly against chest"; No. 121, surgeon, "Fall on stairs with violent swing of arms in attempt to preserve balance unsuccessfully"; No. 122, housework, "Wrenched shoulder in effort to avoid fall on staircase"; No. 138, osteopath, "Fall"; No. 140, business man, "Fall from a horse"; No. 142, a business man, "In making

motion of throwing spear felt a severe pain and was unable to raise the arm"; No. 143, waitress, "Raising tray."

The duration of symptoms prior to operation varied from a few days to ten years in one case. One man even stated twenty years, but the continuity was somewhat doubtful. There were eleven cases who stated that symptoms had existed from six months to five years, and two from one to six months. Ten were acute, that is, there had been no pain until within two weeks.

Relief of Pain. In acute cases pain was at once relieved by the operation and in chronic cases there was very little pain after the first few days. Eight patients reported no pain or actual relief from pain immediately after the operation. In other words, a really acute case is so much relieved by the incision that he may ignore the post-operative pain, but a chronic case, who has had no previous acute pain, is more aware of any later discomfort from the wound itself. Only seven mentioned pain after the first day and only one after the fourth day.

The subacute, annoying symptoms in convalescence varied from one to four weeks in most cases, but one case complained of some pain for seven weeks.

Use of Arm. Most patients began to use their arms within a few days and used them with more and more freedom from day to day. As a rule it took from one to three months before *perfectly* free use returned. By the end of three months all but two patients had ceased to be even annoyed by the shoulder. The symptoms of these two cleared up within seven months. One of these was an iron worker who continued to have pain on using a sledge hammer when he returned to work fourteen weeks after the operation.

Return to Work. In most cases the patients returned to work or to their regular avocations after a month or less, only three cases requiring more time, the longest being the above-mentioned iron worker. (An industrial case.)

Hospital Days. The time spent in bed averaged three and a half days. None were absolutely confined to bed. The time in the hospital averaged five and a half days; three patients remained only three hours; six remained ten days for convenience, not from necessity. All wounds united by first intention.

From the point of view of the operator, who does not endure the pain, the results of this study may be summarized as follows: In acute cases the severe pain is relieved at once by the operation, and even

in chronic cases the postoperative pain lasts only a few days. Within a few weeks the patient is about his usual pursuits, although he still has transient pain in extreme motions and cannot lie comfortably on the operated side. Then follows a period of a few months during which certain motions may cause annoying, sharp, brief twinges, and at night the arm may still cause a minor amount of discomfort. These symptoms become less obvious, and presently days together pass without a thought of the shoulder. This period is indefinite, but is to be measured in months, not in years.

In order to get the patients' points of view I included in the questionnaire a request for a statement on their general impression about the efficacy of the operation. The following answers were given:

Case No. 51, a physician, "Operation entirely relieved my acute pain which had been present for several weeks. The other shoulder has been involved since, and has cleared up with no treatment. It was never as acute as the operated one"; Case No. 55, a physician, ". . . as I suffered so intensely for so many months and was entirely relieved within a few days after the operation. If you remember my operation was performed under local anæsthesia. My recovery was a very quick one and as nearly as I can remember I was able to do my regular work in a very short time. In April (2½ mos.) I was playing golf with no discomfort whatsoever"; Case No. 56 (from patient's physician), "I feel quite sure he thought it a success. Patient died of angina" (9 yrs. after op.); No. 62, "To me the desirability of the operation is unquestioned, in competent hands, however. The pain is so wearing and causes such nerve irritability that the sooner it is over the better. I consider my operation a great success, and would advise any one to put up with the temporary inconvenience"; No. 63, "Grand in every way"; No. 65, "Am pleased to say that I have no pain whatsoever and have perfect use of my arm; in fact, I have had since about two weeks after the operation"; No. 66, "I have never been quite able to make up my mind as to the efficacy of the operation. On the one hand I have had no discomfort in that shoulder; on the other hand, the ease with which I am able to control any kick up in the other shoulder by avoiding gouty foods and drinks leads me to believe that it might have been possible to cure it without an operation"; No. 68 (See Plate II, Fig. 3), "Extremely successful, has never bothered me since"; No. 72, a surgeon, "I obtained immediate relief at the time of operation and aside from some soreness of the wound, I was perfectly well, and as I remember it, I was operating within six or seven days of operation, the exact date I do not remember"; No. 73, "85-90% improvement" (This patient, a physician, had had symptoms for ten years); No. 79, "100% successful"; No. 81, "To secure any lasting benefit, I think that an operation is the only thing"; No. 85, "Similar to the removal of an aching tooth, pain all gone, only the soreness which would follow any deep gash in the body, would say no operation could be more efficacious"; No. 93, "Excellent"; No. 103, a physician, "Decided success"; No. 105, "Unqualified approval. The X-ray showed a quiescent bursa in the other shoulder. I have been a bit careful and when

there has been the least twinge, have rested it. But even that has only happened three or four times in these years"; No. 113, a physician, "Absolutely perfect. The relief from pain and the conviction that there will be no recurrence are beyond price. Personally, I believe that any other procedure is a waste of time"; No. 114, "A wonderful operation. I walked home from the hospital three hours afterwards, and never had any pain or discomfort at any time except for a month when trying to use arm too strenuously. I recommend it heartily"; No. 121, a surgeon, "I was strong for it! At the end of one week I deliberately broke up adhesions by playing golf. After this the soreness subsided rapidly. The right shoulder bothered three months ago, no known trauma. Symptoms have now all subsided"; No. 122, "My impression is that the operation was very successful, and at times when my other shoulder troubles me I often wish I had both shoulders operated on"; No. 133, "Excellent results. I do not recall that I had any pain following my operation, with the exception of the few days I was in the hospital, and this was more from the soreness of the incision rather than actual pain from the shoulder. It was quite some time before I had the absolute free use of my arm, but I should say this was due to my lack of exercising it. However, it is absolutely perfect and I have used it a great deal"; No. 134, "I consider the operation entirely successful"; No. 140 (See Plate II, Figs. 1 and 2), "No question but that the operation, immediately, and apparently permanently, removes the trouble"; No. 141 (See Fig. 44), "Operation afforded me a very welcome relief after about four weeks of almost steady pain, and, although even now I do not put any more strain on my shoulder than I can help, it feels as strong as it ever did. I would not hesitate to undergo a like operation again to relieve like pain"; No. 142, "The result was most satisfactory. On only three or four occasions have I realized I had a shoulder and then only a slight soreness for a day or two after having used it perhaps a little too strenuously"; No. 143, "Good."

I am quite certain that most of my other patients to whom this questionnaire was not sent, or who did not reply to it, had as favorable results, because I have heard from most of them occasionally for many years after my operations. Details of some of these cases may be found in the Table on pp. 255-260.

I also have notes on about an equal number of cases who were not operated upon, usually because I thought the symptoms too trivial, but in some cases, because the patient refused operative treatment after having my assurance that in time they would recover at any rate. In most of these cases, especially in those in whom perforation had already occurred, the results have been as good as in those operated upon, although it seemed to me the patients suffered more. Many have recovered as promptly under some form of palliative treatment, as if I had operated upon them, but a few have had prolonged convalescences and suffered a great deal. My brother, a business man, had acute attacks in both shoulders for several months each, at different

times. He would not sacrifice the time to be operated upon, but obtained some relief from diathermy. On the whole it seemed to me he suffered more than any case has suffered after the operation, but he did not lose a day. This was a subacute case with no restriction. As a rule I have operated on only the worst cases. In fact, I seldom advise operation unless the pain is severe. The unoperated series is, therefore, in the main composed of much milder cases, for those with severe pain welcomed operation.

For the sake of my argument that operation is wise in those cases where the pain is hard to bear, it is not necessary to attempt to contrast the periods of disability in an unoperated series with those that were operated upon, because the operated series shows many cases where the symptoms prior to the operation had existed much longer than they persisted in any case after it.

It does not seem to me worth while at the present time to go into this question in greater detail, because it is evident to me from a study of the unoperated cases that these patients eventually recover under any form of treatment which is not actually detrimental. The question is wholly one of relief of symptoms, not of the removal of a condition which is in itself dangerous or likely to lead to permanent disability. Although I am inclined to think that those cases which are not operated upon may ultimately have weaker tendons than those in whom the deposit is removed, I have no statistical proof of this, nor have I ever known of complete rupture of the supraspinatus occurring as an aftermath of this lesion. My reason for thinking that the tendons are weakened is more on account of the frequency of finding, at autopsy, defects which might be accounted for on this hypothesis.

Deposits in the Subscapularis. It may be that deposits in the subscapularis are less responsive to surgery than when they occur in the other tendons. I have had experience with only two cases, but they were both instructive to me, for I criticize myself for being content with removing one large deposit and making no effort to be sure there was no deposit in the other tendons.

Case No. 84. This was an acute case of two weeks' duration in a single woman of 46. I operated February 2nd, 1926, and removed a deposit from the supraspinatus. The result was satisfactory. However, about two years later, very acute symptoms returned in the same shoulder. This time the X-ray clearly showed a large deposit in the subscapularis and the remains of a smaller one in the supraspinatus. (Plate II, Fig. 6.) I operated again and removed the subscapularis deposit. The immediate result was as good as before, but the convalescence was more protracted in the sub-

acute stage. She was an accountant and used the typewriter a good deal. She states that she went back to work two weeks after the first operation and four weeks after the second one, but her reply indicates some residual trouble: "Always occasional twinges since. Always 'cranky' as to normal use—have to turn certain way to put arm over and upward."

I am inclined to think that I might have saved this patient the second operation by a more careful X-ray study at the time of the first operation, for evidence of the other deposit may have been present at that time.

Case No. 117. A maiden lady of 64, who had a large calcified deposit in the subscapularis. (See Fig. 43.) She had had subacute symptoms for four weeks before the operation. According to her reply she had a longer and more stormy convalescence than any of my other cases have had; *i.e.*, the acute pain after the operation lasted six days; the subacute pain three and a half months, and the occasional twinges ten months. It was a year before she forgot about the arm entirely. There have been no after effects.

This patient did not answer the question about the general impression of the efficacy of the operation, but it is clear that she suffered somewhat more after the operation than she did before it. She was operated upon in another city, and I did not have direct care of her convalescence, as I have in almost all the other cases. I was, however, in telephone communication with her doctor, and saw her from time to time during the year following. There seemed to be no very distinct reason why she should have had a longer convalescence than the other cases, but there were several possible reasons, which it seems worth while to discuss because they may have some future importance. In the first place, the deposit was in the subscapularis, a rather unusual situation. The operation was not difficult, because outward rotation of the arm easily brought the deposit under the wound which was made in the usual situation in the deltoid. I did not, in her case, incise any of the other tendons, and there may have been a tendinitis in those tendons which was the cause of the delay. Also, she did have some hæmatoma in the wound, which might have accounted for the postoperative pain being longer than usual. It is my usual custom to lightly bandage the arm to the side for the first night, and next morning to remove the bandage and allow the patient to place the arm in any position he chooses. There was some delay in removing the bandage in this case, I am told, because the patient seemed to have so much pain. It is possible, also, that the patient was one who, in her anxiety to be conscientious in her statements, has given an impression in the answer that her pain was greater than it was.

As these two cases were the only unsatisfactory ones I have had, and as they were the only ones in which there were large deposits in the subscapularis, it would be logical to attribute the slow convalescences to the location of the deposit. Two cases do not give much opportunity for logic when complicated by the factors of the personalities of doctors and patients. It may be that I could have saved both patients a considerable amount of pain by more thoroughness, and it may be that the two ladies were not of a satisfactory type for surgery, and would have been better satisfied with diathermy. At any rate, they represent my two worst results from operations for calcified deposits. In spite of the somewhat stormy convalescences I believe both patients were saved much suffering, for they seemed to me to be cases of severe type. The operation is a trivial one from a surgical standpoint, but to some people an operation is an operation, and those who would be inclined to take a surgical experience too seriously may well be treated with palliative measures.

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Almost all of the above articles are brief reports of the first few cases of calcified deposit which were observed in each locality, and give illustrations of the X-ray findings. In my opinion the articles by Brickner, Carnett, Harbin and Stieda are the most instructive. The article by Küster in 1902 did not mention calcified deposits, but I am quite sure that the cases which he observed were such cases. His excellent and brief article was, so far as I know, the first rational description of subacromial bursitis. It was called to my attention some years after I had written my first paper, but was in fact written before mine.

Duplay's first article appeared in the *Archives Général de Médecin*, Nov., 1872, but it apparently was not at first appreciated. Another paper in 1896 received more notice and literal translations were at once printed in two English Journals (see ref. above). Most European authors credit to him the introduction of the term "Periarthritis of the Shoulder" (periarthrite scapulo-humérale, periarthritis humero-scapularis). The chief advances in our knowledge of the subject since Duplay's contribution have been due to the use of the X-ray (discovered in Dec., 1895), so that we have been able to study separately the cases in which there is a calcified deposit or in which a fracture of the tuberosity had taken place. Except for that of Dickson and Crosby, quoted on page 224, I have found no important study of the type of cases to be considered in the next chapter in which little if any calcification is found.

The articles by Baer and Painter both appeared in 1907 and I think were the first articles in this country to recommend surgical removal of the deposits. Bergemann and Stieda recommended surgery in Germany in 1908. Most of the articles advise some form of physiotherapy. It has seemed unnecessary to me to print the titles of all these papers on account of the large amount of space that this would require. Doubtless most of these authors would today be able to give as detailed a study as I have given in this chapter, but many of the articles show a lack of knowledge of minor points which have probably become plain to most of them during the intervening years. For instance, none of the articles seem to give proper consideration to the fact that nature's method of cure is by evacuation of the calcified material into the bursa. Many of the authors seem to think that the origin of the particles is in the bursa, and hence use the term "calcified bursitis." A good many attribute the onset of the trouble to recent trauma.

Most striking is the length of the bibliography of this chapter

as compared to that of the last one on complete rupture of the supra-spinatus, which, to my mind, is so much more important and disabling a lesion than that caused by calcified deposits. The reader should refer to the comparison of costs on the scheme on page 124. Even the brief bibliography that I have given at the end of that chapter is partly composed of articles which really do not refer to the subject, and are only related to it. In fact, my papers and that of Wilson are really the only ones devoted to the subject. It seems to me most important for the profession to learn to differentiate between these two common causes of shoulder disability. As a matter of fact it is essential on the patient's account that rupture of the supraspinatus tendon should be recognized and operated upon promptly, whereas, the treatment of calcified deposits is a matter of choice, although my personal opinion is in favor of prompt surgical removal of the deposit. There is so little difference between the results of surgery and the results of any form of palliative treatment that I do not feel in these cases, as I do in those of ruptured supraspinatus, that their treatment is essentially surgical.

My own articles have been extensively quoted (often incorrectly) in American and English literature. This has been one reason why I have taken so much pains in this chapter to enter into the minor details of this comparatively insignificant lesion, cases of which recover in due time by nature's own methods. This chapter may also be of some importance because I am able to report the results of many cases which were operated on a long time ago. However, I feel that in some of these patients, whether or not operated upon, the tendons have probably been weakened through destruction of the tendon fibers so that had they been subjected to unusual trauma they might have ruptured more easily than normal ones. As stated in the chapter on Pathology, there is some doubt as to whether the defects so frequently found by Dr. Akerson may not have been sometimes due to destruction of the tendon by these calcified deposits years before autopsy. In the cases which I have reported as operated on for complete rupture, there was no doubt in my mind from the pathology found that the lesion was purely traumatic, although I could not, of course, determine whether there may not have been a weakened tendon before the actual rupture. In no case did I get a definite history of a calcified deposit in years previous to the rupture. Autopsy specimens from cases known to have had these calcified deposits in earlier years, and in whom there had been no operation or history of later trouble, might clear up this point.

Chapter VII

TENDINITIS OF THE SHORT ROTATORS

Adherent Subacromial Bursitis—Frozen Shoulder

This is a class of cases which I find it difficult to define, difficult to treat and difficult to explain from the point of view of pathology. Yet these cases form a fairly distinct clinical entity, which I formerly described under the adherent type (Type II) of subacromial bursitis. The experience of the last fifteen years has led me to doubt the advisability of calling this entity "bursitis," for I now believe it is essentially a tendinitis, with only secondary involvement of the bursa. The typical case is one of "frozen shoulder," which shows no calcified deposit, and in which a history of trauma is absent, vague or not clearly associated with the onset of symptoms. These cases are usually diagnosed as "periarthritis" or "neuritis."

Figure 48. Frozen Shoulder

The term "Frozen Shoulder" covers the cases which are the subject of this chapter, but it also applies to many other conditions which cause spasm of the short rotators or adhesions about the joint or bursa. This figure shows the characteristic limitations of a severe case. The outline of the posterior edge of the scapula and the axis of the spine of the scapula and acromion are indicated. In the "frozen shoulder" the axis of the shaft of the humerus is nearly at right angles to the axis of the spine of the scapula, and remains so in whatever direction the arm is moved.

Although determining the relation of the axis of the shaft of the humerus to that of the spine of the scapula is a primary one for the understanding of the diagnosis or treatment of many shoulder lesions, it seems best to dwell on its importance here. Cases without distinct traumatic history which show a frozen shoulder, and in which the X-ray gives no information, are likely to be cases of the condition described in this chapter. Unless the other joints of the body show pronounced evidence of arthritis, you may be quite sure that you are dealing with a case of tendinitis.

Perhaps it is best to attempt to describe the course of these cases clinically and to point out the ways in which they differ from arthritis on the one hand and cases of calcified deposits on the other. Perhaps it is only theoretically that they differ from arthritis, which we have already reduced to tendinitis in a previous chapter. The great clinical differences are that they are not a part of a generalized arthritis and that they run a self-limited course and clear up

entirely without leaving the joint deformed or otherwise permanently damaged. They usually give the history of some slight trauma or overuse. Call some of them arthritis if you want to, but others will merge into the class of calcified deposits because the X-ray shows small flecks in the tendon. They might be called by whatever name is best for that unsatisfactorily named group and given the same name without the "calcified" part, just as the term aleukemic leukemia is used. I have removed bits of tendon from these cases which under the microscope show the same necrotic changes which we find in the tendon substance adjacent to the deposits, or adjacent to the rents in the rupture cases. In all three there is the same change in the tendon substance as is depicted in Plate VI. It is a degeneration of the collagen without signs of inflammation as invasion of lymphocytes. One is tempted to say, "Oh, well, all three are arthritis in different phases!" Undoubtedly all three may be fundamentally tendinitis, but clinically there is great difference to the patient in point of what he may expect in the way of pain and in success of treatment, according to whether there is a calcified deposit (weeks), a tendinitis without calcification (months), or a complete rupture (years). Practically they are three different clinical entities, although they might be spoken of as the calcified, the uncalcified, and the ruptured forms of tendinitis.

Last year I had four of these non-calcified cases to treat at about the same time, and it may be more instructive if I write of them together. They had in common:

The condition had come on slowly; pain usually felt near the insertion of the deltoid; inability to sleep on the affected side; painful and incomplete elevation and external rotation; restriction of both spasmodic and mildly adherent type; atrophy of the spinati; little local tenderness; X-rays negative except for bone atrophy. The pain was very trying to every one of them, but they were all able to continue their regular habits and daily routine.

One was a doctor of 62, an active man who takes good care of himself. Number two was a very vigorous, muscular, unmarried woman, who has been a champion golf player and still competes with much success at 46. Number three was a feeble old maid of 70. Number four was a married woman of about 50, apparently in perfect health. All of them were a little "run-down" without anything particular the matter. In each there was a possible history of some slight accident.

The golf player, who was also an extremely busy lady in charitable affairs, when told that she would get well anyway in time, took

no treatment and even played a little golf. The others submitted to the treatment I usually recommend to these cases; *i.e.*, rest in bed with the arm in elevation. I do not always give an anæsthetic and break up the adhesions as I used to do many years ago, nor do I open the bursa and break them up with my finger as I did for a period. I simply put the patient to bed, apply a splint to the back of the forearm and tie each end of the splint with a loop over the railing at the head of the bed. The position of the patient's body usually acts as sufficient extension, although placing blocks under the legs at the head of the bed assures it. A light dose of morphine is given the first night to procure relaxation, but not enough to absolutely remove the pain for fear of letting the arm "go to sleep." In twelve to twenty-four hours the spasm relaxes, adhesions yield, the tuberosity passes under the acromion, and the arm becomes abducted and externally rotated and is more comfortable. In a day or two I remove the splint and simply tie a bandage loosely around the wrist and to the head of the bed to remind the patient not to lower the arm. I get the patient up daily and begin the stooping exercises described on page 202. He stays in bed until he can freely move his arm about in any direction above his head. This is usually after one or two weeks. After discharge from the hospital he takes stooping exercises and for a few weeks sleeps at night with his arm in the hammock position.

Of the three patients thus treated, the feeble, elderly lady made the most prompt and satisfactory recovery. The doctor also did well. The other lady taught me a lesson. She was wilful and charming, and persuaded me to let up on the traction too soon. She was a very busy woman and rebelled against the restraint, for she was planning to go abroad. The result of my weakness in not insisting on keeping up the traction longer was that when she got up the arm came into the sling position again; the spasm recurred, and her continued restriction marred her trip. However, when she returned in two months the shoulder was almost well again.

This patient and another lady, whom I have since treated, exhibited a complication of this abduction treatment against which I wish to warn the reader. During the first few days they had intense abdominal discomfort which I now believe was due to acute dilatation of the stomach. A similar condition sometimes arises in cases where a plaster jacket is applied with the dorsal spine extended. The maintenance of the irritable shoulder joint in elevation has the effect of hyperextending the spine. I think this complication can be avoided by attention to the position of the patient's body and

by permitting them to occasionally rise and walk about with the hand of the affected side held by the other hand over the head. In these two cases the suffering from the abdominal condition was worse than that from the shoulder.

Another infrequent complication of the elevation treatment is difficulty in getting the arm to the side again after having kept it up for a week or two. In a few very sensitive patients this trouble has occurred, but in most cases if the patients get up each day and do the stooping exercises with my encouragement, they are able to bring the arm to the side from the first. Occasionally spasm persists to such an extent that even when the adhesions have been broken up under ether and the arm placed in elevation, the muscles will remain rigid and hold the arm in this erect position without yielding even when the patient stoops.

This condition of affairs need give no anxiety, however. Strangely enough, it is not very painful. One lady in whom this occurred used to get up and walk about with the arm as erect as that of the traditional Hindu fakir. She would even read quite contentedly, or write letters with the other hand, resting the erect arm against the wall. This painless spasm persisted for three weeks, when I gave her some nitrous oxide and gently lowered the arm. No adhesions were felt to give, and there was no pain afterward. A few weeks later, she had complete use of the arm.

Since this experience I encourage my patients to lower the arm at least once a day. Even with this precaution one of my recent cases, a very severe one, showed a similar degree of spasm and even at the end of six weeks could not completely attain the sling position, although she could easily place her hand behind her head. My effort is always toward keeping the ability to use the arm in elevation. If this end of the range of motion be maintained, the normal uses of the arm bring it soon enough into the sling position.

Breaking up the adhesions even under ether is always an unpleasant process, and especially so in cases where the joint has been "frozen" for several months. Sometimes great force is required, and as the adhesions yield there is often a loud snapping noise which vibrates down the humerus and gives the sensation to the operator that the bone has been broken or the ligaments at the elbow torn. I have never broken the humerus in doing one of these manipulations, but I believe it might readily be done. In one case in which I had opened the bursa and ruptured the adhesions there with my finger, the joint still would not yield. With my finger still in the bursa I forced external rotation with my other hand, and distinctly felt some of

the fibers of the subscapularis give way under my finger. I believe that unless great care is used such tearing in the fibers of the other rotators may thus occur. It would take but little force to bring this about when they are atrophied from months of nonuse, even if they are not actually partially necrotic.

For these reasons my present custom is to secure all the stretching I can by slow traction for forty-eight hours before I give an anæsthetic. In fact, the slow stretching usually accomplishes the purpose in this time unaided by manipulation, and, if an anæsthetic is necessary, little force is required. If much force must be used the bursa should be opened and the adhesions in it broken or cut before attempting to stretch the short rotators.

A surgeon who attempts to break adhesions should know the normal motions of the shoulder described in Chapter II. The manipulations should be thorough and the joint moved to its normal extremes in all directions but not a bit beyond them. I prefer to begin with external rotation, but not to carry it to an extreme position at first. Some of the adhesions preventing internal rotation are then broken. Then abduction in the mid-position to a right angle with the body; then external rotation in abduction; then anteroposterior motions in the sagittal plane, and finally elevation to the pivotal position. After breaking some adhesions in each of these directions, perform the whole set over again, making sure that in each position the extreme is reached *and not more than the extreme.*

During the thirty years in which I have tried to relieve these patients with frozen shoulders, I have tried and have seen tried many methods of manipulation and other forms of treatment. There have been few miracles performed and recovery is seldom rapid. A few cases already in a convalescent stage, with no pain but with restricted motion, have made prompt recoveries after snapping a few adhesions, but for most cases there have been many uncomfortable, restless nights and slow recoveries. However, recovery is always sure and may be confidently expected.

None of the patients I have ever had with this condition have had recurrences in the same arm, but some have had the same trouble in the other arm a year or two later. In one case I operated upon both shoulders and removed bits of tendon for examination which showed the same pathologic changes which I have always found in these cases or in the calcified or ruptured cases. I do not think the operations did either much good or harm, but the postoperative rest in elevation gave the desired effect both times.

Formerly before putting the arm in abduction I used to give ether and open the bursa. The appearances of the floors of the bursæ were always the same—a congestion over the supraspinatus tendon on the base of the bursa like that of a bloodshot eye. The congested area was usually circular with a whitish center much as in the calcified cases, but no macroscopic calcified material was present. Adhesions were often found. The congestion was in the synovia, not in the tendon beneath it. Although some cases made rapid recoveries, I gave up making an incision, not because it did any harm or caused any delay, but because I concluded that little was accomplished by it, and that the postoperative elevation was what really was doing the good by causing relaxation and therefore permitting the blood supply to accomplish its healing work more rapidly. In elevation also, the teres major, which has been constantly in spasm, is slowly stretched. Moreover, the old principle that adhesions will not form between the separated raw surfaces of the bursa also holds good. Perhaps, too, this position may approximate the synovial membrane at the edge of the cartilage. At any rate this treatment by rest in elevation is the best practical way to get these patients well with a minimum amount of pain so far as I know. This opinion was shared by the late Dr. Brickner of New York, who had great interest in shoulder cases. He used dumb-bell exercises afterward, while I prefer the stooping exercises as long as the joint is irritable.

If a patient cannot rest in bed for a week or two I advise diathermy, although I am not convinced that it does much good. A number of my patients have preferred, as did the golfing lady spoken of above, to do nothing except the stooping exercises. As a rule they have recovered almost as quickly as the cases treated by other methods. Some cases are so mild that they do not even consult a doctor. There is great variation in the degree of severity of symptoms and in the period of disability. Even the most severe cases recover with or without treatment in about two years. Although I have devoted much time to analysis of the one hundred cases shown in Chart I, on p. 124, I have not been able to work out any new facts which are worth recording, except those already mentioned on pages 135 to 140 in connection with the sex, the age, the occupation and the menopause. Even such analysis as I can make of the End Results is not fruitful. What knowledge I have acquired in forty years about this class of cases is not such that I can transmit it, but so far as my own practice is concerned I have diminished the relative number of cases of this kind, because I have subtracted from them the more pronounced calcified cases on the one hand and the more definite incom-

plete ruptures on the other. If the reader will refer again to page 124 he may understand that when we learn to define the borderlines more clearly, more of these puzzling tendinitis cases will be cured by the treatment suitable for calcified deposits and still more will be prevented by prompt and appropriate attention to cases of incomplete rupture.

I would be more exact if I could in regard to the period these cases require to recover, but since recovery is always by degrees, it is pretty hard for even the patients to say just when they are well. They require my care about six weeks, but as their symptoms clear up gradually, they always cease coming to me before they fully recover from the last twinge. The whole course with or without treatment, from the first symptoms to the period when they forget to think of their shoulder each day, is more apt to be a year than six months. Nine months' pain and annoyance is a fair guess. One rarely sees a case until it has progressed for three months, because the onset is slow and insidious and the pain at first seems bearable. It is the restless nights rather than pain in the day which eventually drive them to a doctor. Since most of these cases follow some minor injury, it is "my hunch" that they are often due to irritation caused by the tearing away of a few fibers of the supraspinatus on the joint side, thus separating the synovial membrane from the edge of the cartilage. The irritation creates protective spasm, and efforts to use the arm or limber it up maintain the spasm. Thus this group of cases is not clearly separated from the group I have called incomplete ruptures of the supraspinatus. The main difference is in regard to the lack of distinct traumatic history.

Although the type of cases spoken of under this heading of "Tendinitis" is pathologically still a little vague, it is a pretty definite clinical entity. It would require greater knowledge than is available at present to separate it pathologically from arthritis, bursitis, calcified deposits or from "rim rents" of the supraspinatus. I cannot too often warn the reader that complete rupture of the supraspinatus tendon is a very different clinical thing from these minor ruptures which set up a tendinitis or bursitis and recover after six months or so. In complete rupture cases the shoulders are not "frozen."

As a clinical entity, tendinitis is diagnosed rather by exclusion than by special symptoms. All severe cases present a "frozen shoulder," and thereby complete rupture of the supraspinatus is excluded. Careful X-rays rule out a calcified deposit. The lack of involvement of other joints excludes arthritis; so does complete recovery within a few months under appropriate treatment. These cases can

only be separated from incomplete ruptures by the absence of a distinct traumatic history, and this is practically a difficult line to draw. The discovery of abscessed teeth, suppurating tonsils or other possible portals for toxic absorption would tip the balance of judgment away from trauma as a cause. Even then we must not forget that a tendon made brittle by toxins may rupture.

The reader should not judge the relative frequency and importance of this group of cases by the length of this chapter compared to that of other chapters. He may, however, so judge any knowledge of the subject which I have to communicate to him. These are common cases, but it does not take a long chapter for me to tell all I know about them. I could write a whole book on my experiences with cases of this type, but it would have to deal more with human nature than with demonstrable pathology or remarkable success in treatment. The art rather than the science of medicine is called for in these cases, but in the cases of calcified deposit and in the complete rupture cases, cold, definite, prompt efficiency is more desirable.

Since the symptoms in these cases vary greatly in degree, prognosis is difficult at best, and since patients vary greatly in their ability to bear the same conditions, a great variety of puzzles are offered for solution. Some patients have little restriction and much pain; others have stiff, painless joints, and there are all degrees between. I am confident that I can shorten the convalescence of any case by the elevation treatment outlined above, but in most cases I do not advise it, and the mere assurance that they will recover in time seems to be of wonderful therapeutic value. If pain is severe enough to keep them awake much at night or to prevent them from earning their living, or from following their favorite sports, I advise the above treatment. Massage is a help. Light stooping exercises seem to me to be of great benefit. Attention to the general well-being of the patient is most important. For instance, a good vacation under pleasant, healthful circumstances seems to me more desirable than any form of therapy if the pain is bearable. If the arm is not well enough to make a vacation enjoyable, it can soon be made so by the elevation treatment.

Since this chapter was written a thoughtful article has appeared. (*Jour. A. M. A.*, Dec. 31, 1932, Vol. 99, No. 27, pages 2252-2257. "Periarthritis of the Shoulder, An Analysis of Two Hundred Cases," by James A. Dickson, M.D., and Edward H. Crosby, M.D., Cleveland.)

While there are many minor points on which I could not agree, I feel that this article is the most reasonable, recent attempt to cope

with the problem which I have tried to present in this chapter. It is an earnest, painstaking study of an unsolved question.

The especial point on which I cannot agree with the authors is that they state from a clinical point of view that there is little difference between the cases which show a calcified deposit and those which do not. My experience does not confirm this. I regard the calcified cases as very simple and easy to relieve as compared to the adherent, non-calcified cases. The results of operations on the calcified cases have, from the first, been as satisfactory as any surgical operations which I know of. Only in a few long-standing cases has the return of function been a matter of months. On the other hand, in the true non-calcified cases the convalescence is practically always a matter of months and sometimes of years.

Another fair criticism of this article is that the authors seem to have assumed that because they find infected foci in many patients, the condition of the shoulder is due to these foci. It is my belief that as large a percentage (38.5%) of infected teeth would be found among any two hundred persons of the same average age and class in life. The relation to the menopause is much more striking than that to the teeth. See p. 138, Chart III. Their last paragraph, which I quote verbatim, shows an admirable degree of intellectual honesty.

"The study of the cases reported in the literature and of our series of 200 cases has shown us most strikingly that an exact evaluation of the importance of any one of the factors which may be involved in the causation of the disease, or of the relative value of the various therapeutic measures, is practically impossible. For, in our series, it has been found that in all cases, whether the evidence pointed to infection, metabolic disturbances or trauma as the important etiologic factor; whether the treatment stressed was eradication of foci, physical measures, manipulation or operation, that the time required for recovery and the total duration of the disease was remarkably constant throughout. This suggests that in these cases there must be some general physiologic disturbance as a common denominator, which cannot be explained or accounted for at present. We reiterate this statement because this fact seems to have been lost sight of by many workers in their enthusiasm for one particular type of treatment, or in their zealous endeavor to attribute the symptoms to some particular etiologic factor."

I would be inclined to swallow this paragraph whole if it were salted with just a little optimism.

REFERENCES

Turn to those following Chapter VI, and note especially the remarks on Duplay's contributions.

Chapter VIII

OPERATIVE TREATMENT OF SHOULDER LESIONS

We cannot take the space in this book to describe the standard operations of amputation, excision, and arthrodesis of the shoulder joint, but we may offer the following brief suggestions for slight modifications of the usual methods.

In doing an amputation at the shoulder joint, if the pathology permits, the deltoid should be mobilized at the tubercle, pushed up as a unit preserving its axillary arterial and nerve supply, until the bursa is opened from below. The short rotators can then be divided and the head of the bone lifted off the axillary nerve. When the main vessels are tied and the rest of the arm removed, the intact deltoid can be sutured to the latissimus, teres major and pectoralis in such a way that much of the contour of the shoulder is preserved, since the nerve supply of the deltoid is not injured.

In the writer's opinion two of these operations can best be accomplished by a preliminary incision of the bursa. It is not difficult to do an excision through a bursal incision two inches long. Arthrodesis can also be done through this incision, although it is probably surer to result quickly by using a "sabre-cut" incision after the manner described by A. Bruce Gill (*Journ. of Bone and Joint Surgery*, Apr., 1931, Vol. XIII, No. 2, p. 287).

Operations on the shoulder joint may be done for the following pathologic conditions:

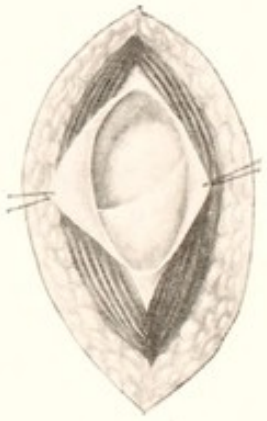
Tuberculosis of the head of the humerus.
Complicated fractures and dislocations.
Infantile dislocations.
Habitual dislocations.
Deformities due to paralyses.
Tumors of adjacent bones.
Tendinitis with or without calcified deposits.
Complete rupture of the supraspinatus tendon.
Rupture of the long head of the biceps tendon.
Adhesions, bands and villi in the subacromial bursa.

We must confine this chapter to the three chief operations which the writer is in a great measure responsible for suggesting to the profession. These are: first, the technique for the removal of calcified deposits; second, the suture of complete ruptures of the supraspinatus; and third, the "sabre-cut" incision for major operations about the shoulder joint.

THE TECHNIQUE OF THE OPERATION FOR REMOVAL OF CALCIFIED DEPOSITS

In most cases this operation can be done through an incision about one-half inch in length, but it will be perhaps safer for both patient and surgeon if an incision an inch and one-half is made to allow ample room to identify the anatomic landmarks. I will assume that the incision is of this size, although I have developed a technique of doing it through an incision just large enough to admit a nasal speculum with recurved tips. The only objects of this modification are to have less scar and less chance of intrabursal adhesions and perhaps less postoperative pain. Since even with the larger wound, *i.e.*, one inch and one-half, opening the bursa as widely as possible, the scar is small and postoperative pain slight, and adhesions more of theoretic than practical importance, one should not hesitate at any time to enlarge a half-inch wound.

Size of Exploratory Incision



Size of Incision for Suture of Tendon

FIGURE 49

On the left is a figure showing the size of the open exploratory incision, which is also sufficiently large for the removal of most calcified deposits. The figure on the right shows the size to which it is necessary to enlarge the incision in order to suture a rupture of the supraspinatus by the author's method. Both figures depict a normal bursa with the edges of the roof caught with sutures and held apart to show the smooth, white, convex base which covers the tuberosity and insertions of the tendons. In the lower portion of both figures a nictitating fold is represented. By rotation of the humerus, the whole base of the bursa may be inspected through the small left-hand incision. Lesions are seldom found at any other situation than on that part of this base which is just above the greater tuberosity. The writer believes that the little exploratory incision is almost harmless, provided the surgeon understands what he is looking for and does not attempt to explore further if the pathology is not at once evident when the bursa is opened.

Anæsthesia.

I prefer to do this operation under local anæsthesia in suitable cases, because it is easier for the patient. However, it is easier for the surgeon to have the patient under general anæsthesia, because

local anæsthesia does not relax the spasm, and renders it necessary to be more accurate in cutting down directly on the lesion. Even if no spasm is present before making the incision, it may be started up by touching the inflamed region with a retractor or other instrument. In a recent case the reflex spasm could be produced at will by touching the surface near the lesion, yet the touch of the instrument caused no sensation of pain. The patient was an intelligent doctor, and could describe his sensations accurately. When the retractors were out of the wound he could rotate the arm, but when they were in the wound he found himself unable to overcome the spasm by force of will, although he felt no pain except when I endeavored to force the rotation.

Although the operation can be done under such circumstances, it is unsatisfactory. I have found it so difficult to anæsthetize the base of the bursa where the deposit is, that now I do not attempt to do so. The tendon near its insertion is so dense that it is difficult to inject the novocaine, and this is as painful as incising the tendon; possibly more so.

Local anæsthesia does not permit a thorough exploration of the bursa, but when one is familiar with the local anatomy one does not need to see the whole bursa. When the patient is under ether, by a certain maneuver the arm can be pulled downward and forward, and the surgeon's forefinger introduced between the tuberosities and the acromion. When air once gets into this opening the atmospheric pressure is overcome, and the subacromial space remains open, permitting inspection and palpation of the whole bursa. This maneuver is more especially useful in cases of rupture of the tendon. In this case the joint is not held together either by atmospheric pressure or by the tension of muscles. Manipulation of this type is not necessary, and cannot, of course, often be done under local anæsthesia, but probably might be done under regional anæsthesia. I regard the latter as too serious a procedure, and would prefer a general anæsthetic.

From the patient's point of view, in spite of these drawbacks I favor a local anæsthetic, provided the surgeon knows where the deposit is, and can go directly to it through a small incision. It is then a trivial operation. But if the deposit cannot be accurately localized, and the anatomy is not clear in the surgeon's mind, general anæsthesia would be preferable in order to allow for some unnecessary manipulation and rough handling. I have operated on many doctors, and had a chance to hear their commendations or

objections. I am told that the actual curetting of the lesion is painful, but not more than can be readily borne. The incision of the skin and muscle, and of the bursal roof, is not felt at all.

Some patients are obviously poor subjects for local, and others for general anæsthesia. Surgical judgment on general principles is needed. I should advise any surgeon to do his first few cases under ether, for in spite of all the directions I can give, the anatomy is difficult to describe, and one is easily confused unless very familiar with it. The facts that the structures to the right or left of this small wound are reversed in relation to the surgeon's position as to whether he stands on the acromial or axillary side of the wound, and are also reversed in right and left shoulders, add to the puzzle.

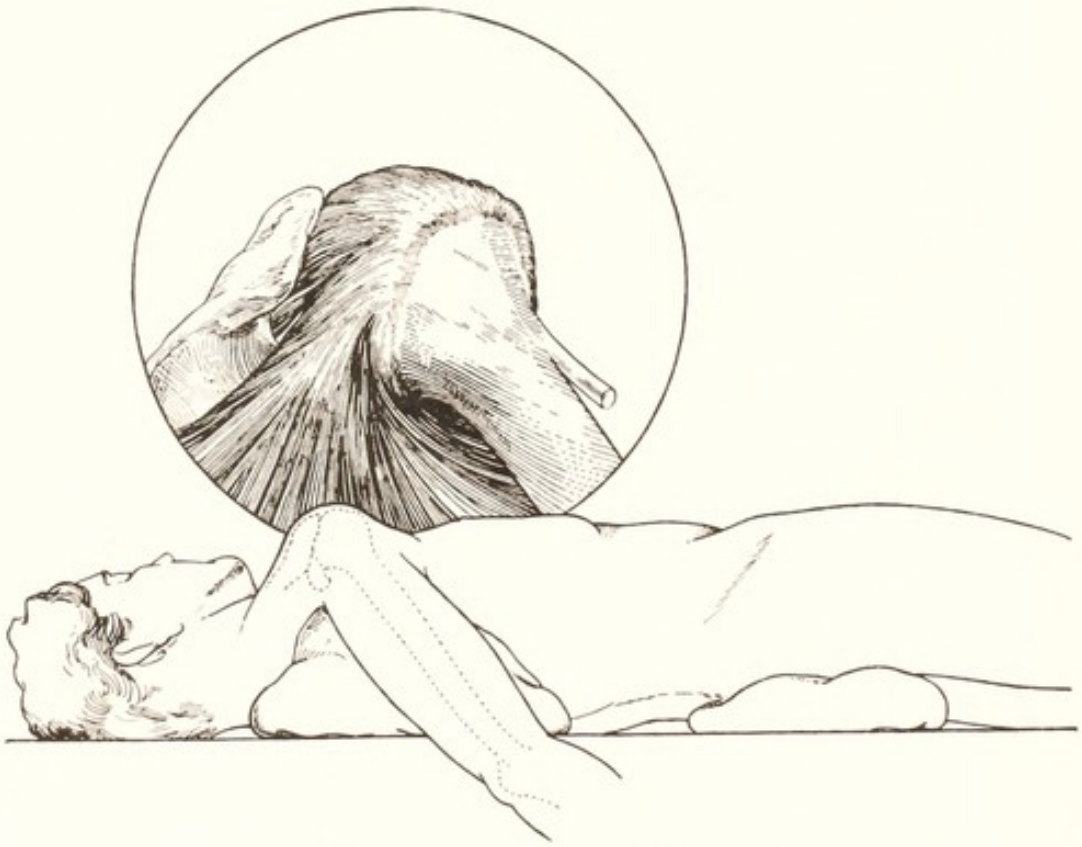


FIGURE 50. OPERATIVE POSITION

This is an important figure. The writer did not realize the advantage of placing the patient in this position, until he had done many of these operations, which had been attempted with the patient either in recumbency or in a sitting posture, strapped in a chair. The latter was a very satisfactory position after the strapping had been adjusted if the assistants could be trained to work in this unusual attitude. However, the cases were too infrequent for any routine to be established. Until one operates on these cases one does not realize how elusive is the shoulder on the operating table, and, since the field is entirely controlled by the position of the humerus in relation to the scapula, how any minute change in the position of either the arm or of the body is at once transmitted to the tiny wound. Gradually I worked out this position, and find that it gives the best possible exposure, and enables one, if the assistant is attentive, to perform a very satisfactory suture through the very small incision.

Preparation.

The patient is placed on his back on the table, and small sand bags or folded sheets are put under the shoulder blade and corresponding hip. This slightly raises the shoulder. It is well to draw the patient as close to the edge of the table as may be, so that when desired the elbow can be pushed backwards below the plane of the table—a maneuver which may be needed to expose the upper part of the bursa. The patient's face should be turned toward the opposite shoulder, and the anæsthetist and patient's head protected with a "goiter apron" tied around the patient's neck.

I always arrange a sterile dressing over the hand and forearm, so that during the operation the arm may be manipulated by the operator or assistants. A convenient way is to sterilize the shoulder and arm to below the elbow, and then to draw a sterile pillow slip over the hand, forearm and elbow, and attach it with sterile bandages as high on the upper arm as desired. The arm may then be drawn through the hole in a laparotomy sheet, and moved about at any stage of the operation.

Position of Incision.

In deciding on the point at which to incise, one must remember that the skin incision will remain stationary, and that by rotation of the humerus the point at which the calcified deposit lies may be brought beneath the incision. The deltoid muscle, which forms the bulk of the tissue which must be cut through, also remains nearly completely fixed. It moves a negligible amount when the humerus is rotated. One must remember also that the sides of the subdeltoid portion of the bursa move considerably in rotation of the humerus. For instance, having incised the skin and muscle, it is possible to continue the incision in such a manner that it would be entirely outside of the bursa; *i.e.*, it might not penetrate the bursa at all when the arm is in extreme internal rotation, but if the cut were made with the arm in external rotation, the bursa would be opened. To make the operation easy, one would desire to have the skin incision as nearly opposite the calcified deposit in the base of the bursa as possible. I have found that the incision best on the average is directly anterior to the head of the humerus, with its upper end near the acromio-clavicular joint, and its lower end at about the level of the top of the bicipital groove. In thin subjects one can feel the bicipital groove through the deltoid, and it is always well to do this if possible, because the acutely tender point will lie external or internal to the groove, according to whether the deposit is in the

supraspinatus or subscapularis. The expansions of these two tendons bridge the bicipital groove, but their real attachments are to the greater and lesser tuberosities on each side of the groove. I have sometimes seen the deposit apparently over the groove, but it had probably extended from the tendon on one side or the other.

If one cannot feel the groove before making the incision, one can always determine its position by palpation in the wound after the deltoid has been incised. It is well to do this in every case, for often the exact location of the deposit can be made previously by X-ray, and one can readily locate the subscapularis and supraspinatus if the groove is determined. Sometimes the floor of the bursa does not show sufficient indication of the deposit to guide the surgeon. In such a case the tip of either tuberosity may be readily determined with the point of the knife, and the tendon incised just above it. When a little of the white deposit appears, the incision can be enlarged in the line of the fibers of the supraspinatus or subscapularis, as the case may be.

In order to determine the position of the bicipital groove, one may take it as a good rule that when the elbow is flexed at a right angle, and the posterior part of the elbow put down on the table at the side of the patient so that the axis of the forearm stands directly vertical to the table, the bicipital groove will be at the most anterior portion of the prominence caused by the head of the humerus. From this point, rotation of the forearm either way brings the tendons into view according to the obvious anatomical arrangement. (Figs. 6 and 51.)

Incising the Deltoid Muscle.

Separating the fibers of the deltoid muscle is a little more difficult than parting those of the rectus in abdominal incisions, owing to the fact that the fibers of the deltoid are more or less "herring-bone" in arrangement. The incision seems to close right up on withdrawal of the knife, so that it is difficult to find the line again. I therefore usually put another instrument, as the point of a hemostat, in along the knife before withdrawing it, so that with my knife in my right hand and the hemostat in my left, I push the fibers apart as the assistant places two aneurism needles as retractors to take the places of my two instruments. These hold the line of incision, and the fibers of the deltoid are cut upward and downward to equal the length of the skin incision. Small, broader retractors are then introduced, exposing the upper surface of the roof of the bursa.

If the above has been understood, it will be seen that the skin incision and the deltoid incision are simple and standard. If correctly placed they would lie over the very top of the bicipital groove in the above-mentioned position, with the forearm vertical to the table. It is by no means necessary to hold the arm in this position while the skin and muscle are incised, but when the bursa is to be opened, the arm should be held in this position, the assistant being ready to rotate it one way or the other as the surgeon desires. Make sure that this point is understood.

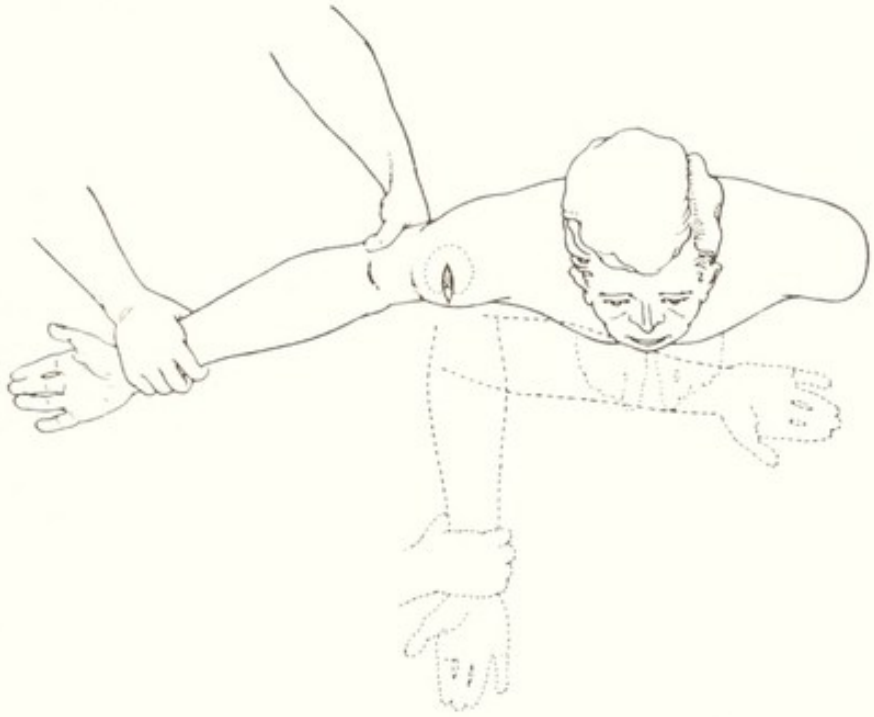


Figure 51*a*. Operative Position, Superior View

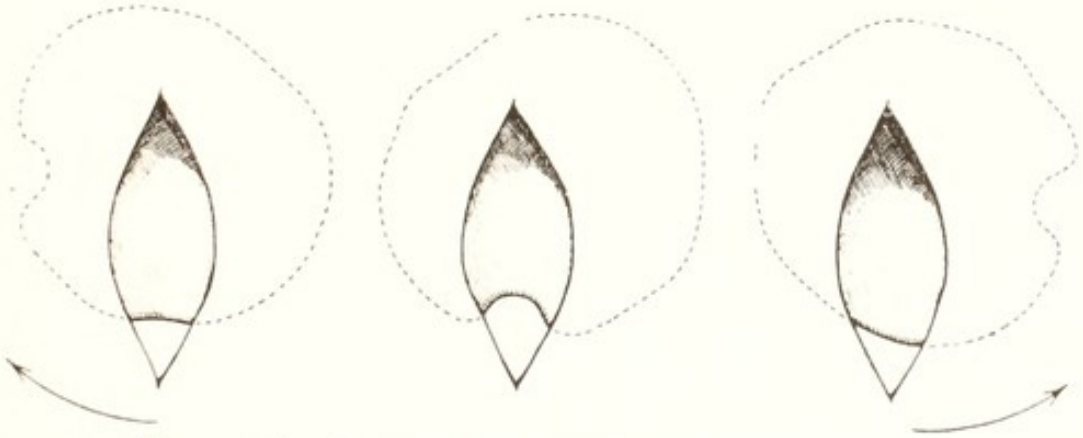


Figure 51*b*. Rotation of Humerus Beneath Incision

This figure should be studied with the last one, although for convenience the patient is here represented as standing up while the operator looks downward from above, and the assistant, holding the elbow at the side, rotates the forearm and consequently the humerus beneath the wound. The figures below represent the incision and its relation to the bicipital groove as the humerus is rotated. The incision remains stationary while the facets of insertion pass beneath the incision. A thorough understanding of these two diagrams is most important.

Incising the Bursa.

The incision in the deltoid having been made, and the fibers retracted, it is well for the assistant to rotate the arm, and in most cases the roof of the bursa is so transparent that the base beneath it can be seen to move as the arm is rotated. In cases in which the bursal wall is thickened from old inflammation, one cannot see through it, but one can palpate the tuberosities moving beneath it. Since it is important not to allow any blood to enter the bursa, small sponges should be ready.

It is well to tie up vessels before making the incision into the bursa, for if blood fills it, it is harder to determine the appearance of the base. At this stage the roof of the bursa is picked up with two pairs of forceps, as is customary when incising the peritoneum. A cut is made between the two forceps, and the air rushes into the bursa, so that the incision can then be enlarged upward and downward as desired. A normal bursa has a white, shiny floor, but a circular zone of deep red injected tissue is usually the guide to the area where a deposit lies. This deep red tissue, resembling that of a bloodshot eye, surrounds a white or pale area about one-half the size of a ten cent piece. Sometimes this area is obviously under tension and mounded up like a boil. At other times when the inflammation is less, the affected area is not raised, and there is only a barely perceptible whitening where the calcium deposit shows through the synovial floor of the bursa. Before incising this area it is well to look the rest of the bursa over, to make sure that there are no other lesions. The X-ray will probably have already determined whether there is more than one deposit, but even if the X-ray does not show more than one deposit, there may be a second one, so that in case of doubt, I believe it is better to prick any suspected area with the point of a knife. I have done this many times, and have seen no variation which could be attributed to this practice in the convalescence of these patients. Sometimes such exploratory punctures of the tendons have fortunately led to the finding of a second deposit, for the patient may suffer an attack of inflammation about one deposit, and then later have another attack due to inflammation about another deposit. While immediate attention to the inflamed deposit is the most important thing, it is desirable when possible to remove or free any other deposit. I recall few cases of deposits which were not surrounded by red zones.

Experience has shown that we rarely find any lesion of the bursa except at the points close to the attachments of the tendons, and these

points may all be inspected by simply rotating the arm through its full arc while the wound is held open. It is a little harder to see the extreme edges upward and downward, but practically the whole bursa may be inspected by merely rotating the arm. Pushing the elbow backward (dorsal flexion) tends to demonstrate the upper edge, and pushing it forward reveals the lower limits of the bursa. I have never recognized any lesion of the roof of the bursa. Lesions at the attachment of the supraspinatus are the most common, those at the attachment of the infraspinatus next, and those of the attachment of the subscapularis perhaps a little less common. This relative frequency also holds good in regard to traumatic rupture of these tendons. One must remember that the three tendons are closely incorporated at their attachments, and that these attachments to some extent overlap.

Removing the Deposit.

To return to the operative technique—we may assume that we have reached the point of incising a calcified deposit which we have found in the supraspinatus tendon. According to the stage of the pathologic process, whether acute or chronic, the calcified mass will be softer or harder; in the earlier stages it is little more than a milky fluid; in the older stages it is hard and gritty, coherent and sometimes encapsulated. In most cases it is of the consistency of ointment; often it resembles the contents of a wen. On incision this material usually escapes as if under tension. Sometimes this is very striking. A little nick is made, and there emerges a ribbon of whitish material just as one sees when a tube of zinc oxide ointment is squeezed. Sometimes, however, there is not much tension, particularly in the old cases, and the particles of calcareous matter seem to be incorporated in the substance of the tendon so that they cannot be curetted out without removing shreds of tendon with them. Occasionally the material occupies a well-formed pocket, and one gets the impression that it is wholly removed with the curette. I have oftentimes been content with merely incising one of these pockets, making no effort to curette out all of the material. The symptoms have disappeared quite as satisfactorily as in cases where a thorough curetting was done. Some surgeons, notably Brickner and Harbin, have stated that they excised all the calcified material. I am sure that if they do this, they will remove a considerable amount of tendinous substance which will be replaced with very little scar tissue, so that the tendon will be much weaker. I have sometimes thought that possibly cases of rupture of the supraspinatus tendon from

trauma may be particularly common in cases which have had previous attacks of this so-called calcified bursitis, which has weakened the tendon. At any rate, I feel that it is well to do as little damage to the tendons as possible. Attempts to "excise the bursa" are absurd and indicate an entire lack of knowledge of the local anatomy, physiology and pathology.

Closing Incision.

Having removed with the curette the major portion of the deposit, I wipe the cavity out, but do not attempt to sew up the incision in the tendon or in the bursa. Most other writers have recommended that the roof of the bursa be sutured. I have rarely done this, believing that it is better to allow the fluid formed by the synovial secretion of the bursal walls to seep into the areolar tissue. This would naturally wash out any particles of calcium and blood which remained in the bursa. If the bursa is closed these particles might cause adhesions, and perpetuate inflammation. Then, too, in a few cases in which I have closed the bursa, I think there has been more postoperative pain, due to distention of the bursa with blood and serum. I have not found that the adhesions due to the incision healing down to the tuberosity cause any permanent restriction of motion. It appears that the bursa itself reforms as a rule, perhaps not as a simple space, but as several small spaces.

I close the muscle with a few loose catgut stitches, because I have found that unless I do so the wound eventually heals with a depression between the fibers of the deltoid. Therefore, it is better to see that the deltoid muscle returns to its normal contour. Perhaps it is better to close the roof of the bursa, but it would need a considerable number of cases to prove it, for my experience is sufficient to show that it is certainly not necessary. It may add some slight shred of advantage, but I think not. The skin is closed as in any other surgical wound.

Postoperative Treatment.

After the operation I treat the patient just as I would a normal person who had a similar wound caused by an accident. The arm is carried in a sling most of the time for a few days. In about a week or ten days the patient can use the arm in a gingerly way, and can manage to dress himself, and use a knife and fork. Discarding the sling, I then encourage "swinging exercises" in a stooping position. When these become free the "standing exercises" are prescribed. In from three to six weeks the patient should be well, except for occasional twinges of pain and soreness and slight restriction in ex-

treme degrees of motion. These twinges of pain are not severe or incapacitating, and I suppose are due to the irregular contours of the bursal surfaces after the incision. They slowly disappear. My tendency is to allow any free use which does not cause pain.

Finer Points and Occasional Obstacles in the Operation.

In ladies it is well to have the patient try on a low-necked dress, and to make the incision at a point where the scar will be concealed by the shoulder strap. Since rotation of the humerus covers a considerable arc, the site of the skin incision can be varied a good deal. I have tried a horizontal incision once, but the scar seemed to have a tendency to stretch more than does a vertical one.

I want to repeat that it is important to tie all bleeding vessels before opening the bursa. The blood supply of the skin on the point of the shoulder is very active, and this is increased in cases of long-standing inflammation. Many small vessels sometimes have to be clamped to obtain a dry field. At the extreme upper angle of the wound there is one which is especially annoying, for it retracts into the muscle and readily drips into the open bursa, obscuring the field. As the wound is deep and small, even a little blood causes delay.

Incising the bursa itself may prove confusing, especially where there are adhesions. Normally the roof of the bursa is even thinner and more delicate than the normal peritoneum, but where there has been inflammation in the bursa, it may be as thick as blotting paper, and quite opaque, so that it is hard to see the floor rotate beneath it as the arm is moved. In such cases one usually finds straw-colored fluid in the bursa. In some cases where the deposit has broken through into the bursa, flecks of deposit in sheets of fibrin or milky fluid will be found in the bursa. Such cases would probably get well without operating.

It is not easy to open the bursa in some cases or even to know when your knife has passed through it into the areolar tissue in the nictitating folds. In case confusion arises, keep the arm in the position advised with the forearm vertical to the table, and cut down on the outer lip of the bicipital groove at its upper extremity. Do not cut directly on the bicipital groove, for you might unnecessarily open the sac about the tendon, if the bursal surfaces were adherent. This would probably do no harm, for in the cases of exploration for a ruptured biceps tendon I do not hesitate to do this and have had no unpleasant results. However, it does not help to locate the bursa, so it is best to recognize the groove by palpation before incising.

Adhesions of the surfaces of the bursa may make it difficult to open. In two cases I have opened directly into the space containing the deposit, passing through the two adherent surfaces of the bursa without separating them. Such an experience might give the impression that the deposit was in the bursa. In my experience adhesions are not the rule. They vary, when present, from recent film-like strands hardly more solid than fibrin to dense scar tissue which effectually prevents rotation. As a rule, one readily opens into a good, large bursa, and the white or yellowish deposit surrounded by the turgid, red zone is as conspicuous as it well can be. The confusion comes in long-standing cases when the acute inflammation has subsided, and is replaced by firm, relatively bloodless adhesions about a very small deposit.

It is quite possible that in cases where the deposit is in the subscapularis, a quicker and smoother convalescence might be obtained by pushing the bursa up from below, or from the inner side, and removing the deposit from beneath it without opening the bursa at all. This would leave a smooth bursa, and avoid the twinges of pain which the roughened surfaces produce if the bursa has been opened. I have done this in one case. However, it is surer and safer in most cases to incise directly into the bursa, and be guided by the red zone.

Remember that when the incision is once made, only *gentle* retraction is necessary. Rotation of the humerus, by using the forearm flexed at a right angle, should take the place of retraction of the lips of the wound. It would be unfair to a patient to attempt to open his bursa unless the meaning of the above sentence was clearly understood. (Fig. 51*b*.)

I have operated on a few cases after the calcified material has perforated into the bursa, but I think now that these operations were probably unnecessary. So many similar cases have promptly got well without any treatment, that I suspect that although my operated cases also promptly got well, the operations had little to do with the recovery. In opening the bursa in these cases a few days after the perforation, I have found a film of whitish fibrin between the two bursal surfaces. Under the microscope little particles of calcium were found thickly scattered through the fibrin, accounting for its whitish look. Recently I have seen two such cases demonstrated by X-ray, on which I did not operate, and the symptoms cleared up as promptly as if I had removed the fibrin.

It seems to me that the time when operation is most indicated is when the deposit has begun to cause subacute symptoms and has not

yet perforated. It is in such cases that the mound on the base of the bursa is found, and from which the white material exudes as soon as it is punctured.

I am inclined to think that spontaneous perforation will by nature relieve the symptoms in a few weeks, although it brings on the acute symptoms during the first few days. Operation, diathermy, puncture, injection of proteids, violet light or other treatment may easily get the credit for the good result. It seems a pity that comparative series of such cases treated by different methods cannot be arranged and controlled by a series of untreated cases. It is at least a comfort to know that all the cases under any treatment get well. The object of treatment is only to find the easiest, quickest, and most comfortable way.

My feeling at present is that when the X-ray shows that the deposit has become diffused in the bursa it is unnecessary to operate, unless the quantity is great, as in Fig. 44. One can tell that it has become diffused if the film shows that the deposit lies outside of the tuberosity and, *vice versa*, if it lies within the contour of one of the tendons it cannot be in the bursa. (See Plate II.)

Sometimes after perforation a portion of the deposit remains in the pocket in the tendon, and this gives a "shirt-stud" shadow in the X-ray.

Be on your guard in interpreting the shadow cast by the bursa with a film of fibrin in it containing the diffused particles. Remember that the bursa is a very thin concavo-convex space, and therefore the mass of material in a lateral view seems greater than it really is. (See Fig. 45.)

Operative Treatment of Rupture of the Supraspinatus Tendon

The best time to operate would be immediately after the injury. When in doubt of the diagnosis, exploratory incision of the bursa should be done. The technique of this incision is the same as that which has just been described for use in cases of calcified deposits. Practically the whole base of the bursa can be inspected through this incision and the exact extent of the rupture determined. The incision is then enlarged inward or outward at either end for a half-inch, depending on the direction of the tear. On account of the herringbone structure of the deltoid it makes little difference whether or not the enlargement of the incision is at an angle with the first one. A good exposure can be obtained with an incision one and one-half

to two inches in length. Do not enlarge upward farther than the coraco-acromial ligament.

Assuming that the operation is done soon after the accident, it would seem that no special directions would be needed. The surgeon knowing the normal relations would restore them by appropriate sutures and close the wound in his favorite manner. It seems to me that this immediate operation would be very easy, but I have not been able to operate on one of these cases in an early stage.

In general the operation has two main objects: the repair of the tendon to give power to the arm, and the making of a frictionless lower bursal surface to relieve inflammation and pain. Perhaps the latter is more important, for even a powerful arm, if painful, is not as useful as an arm which is rather weak in the power of abduction but not painful. It is important to keep these two objects in mind, for although in some cases both can be attained, it is sometimes necessary to take a choice between them, because the tissues may be so damaged and retracted that good approximation is impossible. In such a case we may wish to discard all hope of restoring power and devote our whole effort to trying to allay friction. For instance, the tuberosity could be excised wherever it is free from tendinous attachment, and hence is useless. This might diminish the pain by removing the eminence.

One must not feel too discouraged, however, about his repair work, for on several occasions I have opened a bursa a second time and found a smooth base and no visible sign of my suture, which, at the end of my previous operation, had appeared rough and clumsy with the ends of the tendons not even approximated but held "a distance." (See p. 245.) Even in a certain number of the delayed cases which I have operated upon, there has been little difficulty in making a satisfactory suture aiming for both objectives, but in other cases, there was little or no hope of making a smooth, even suture which would leave no rough eminence or sulcus. The latter is particularly likely to be the case where the tendon is evulsed from the tuberosity, leaving no stub to hold the stitches. In a few cases the retraction was so great that no suture could be attempted at all.

Special Points and Special Difficulties

I have found that in the old cases on which I have operated, it is seldom easy, often difficult and sometimes impossible to repair the tendon. It seems best to list the difficulties and then to discuss each.

1. Position on table.
2. Mobilizing the tendons.

3. The long head of the biceps.
4. Drilling the tuberosity or removing it.
5. Suturing the rent.
6. Formation of a new sulcus.
7. Frictionless surface.
8. Material of suture.
9. Shape of needles.
10. Closure of bursa. Disposal of fluid.
11. Postoperative treatment.

1. The arrangement of the position of the patient on the table to permit proper mobilization of the arm during the operation, is an important factor in technique. The point of the shoulder is a difficult region on which to work, for both the surgeon and the assistants. It slinks away and the patient's head and neck seem to wish to take its place. (See Fig. 50.) I should like to stress the importance of so placing a heavy sand bag under the shoulder and another under the corresponding hip that the patient is half turned on his side, while the head, with the face turned away, is at a lower level than the point of the shoulder. The shoulder should be slightly over the edge of the table toward the operator, so that the arm may be allowed to hang down in a position of dorsal flexion when desired. This position throws the distal portion of the supraspinatus tendon forward for the maximum distance from under the acromion.

The operator and assistant stand on the same side of the table, while the anæsthetist and nurse with the instrument table are on the other side. A second assistant is welcome, and often almost necessary, because the first assistant must at times give his entire attention to holding the arm and the nurse may be occupied with retractors. Much of the facility with which the operation is conducted depends on the assistant who holds the arm, for his ability to rotate just at the right time will enable the operator to put his needle at just the right point in the somewhat small field. Since the lips of the incision do not move appreciably, the operative field is really controlled by the assistant as he rotates the humerus, bringing this side of the rent or that into a position which the operator desires.

The maneuver already described, of letting air into the joint and bursa, is often a great help. The position in which to place the sutures is best illustrated by a diagram. (Fig. 52.) While this is the ideal, it is seldom possible to carry it out exactly, for too often the retracted, stiffened tissues cannot be worked into nice apposition.

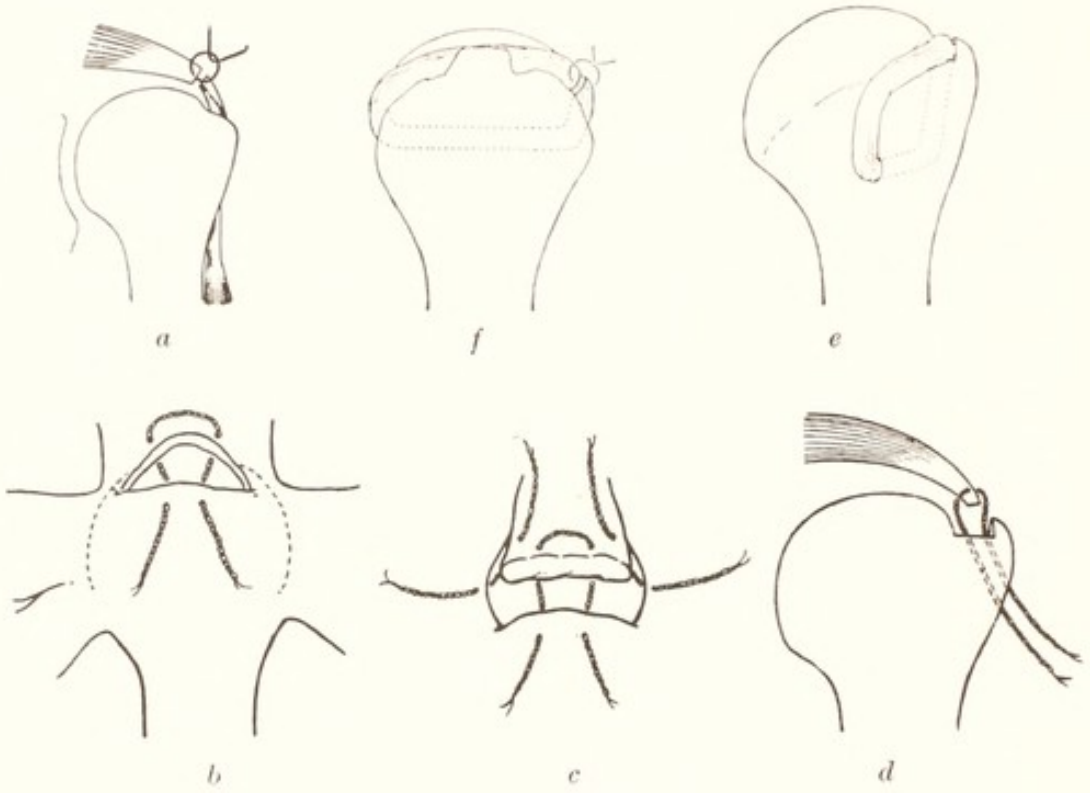


FIGURE 52. METHODS OF PLACING SUTURES

a illustrates the writer's suggestion that the biceps tendon may be sutured to the supraspinatus in some cases when the former has been already torn from the edge of the glenoid. *b*, *c*, and *d* suggest the method of placing the sutures in the ruptured supraspinatus and in the tuberosity. The ideal is *c*, for in this case the lines of incision have been carried up on each side of the supraspinatus to mobilize it. *d* illustrates Dr. Wilson's method of cutting a slot to receive the supraspinatus tendon. *e* and *f* offer a suggestion for operation in a case where the short rotators have been entirely evulsed from the head of the humerus. Fascia lata might be passed through a drill hole and through a slot over the tuberosity to form an anchorage for the tendons.

2. Mobilizing the tendons. When one considers that each one of the short rotators is separated from the other by a definite bony partition through most of its extent, and it is only the last three-quarter inch which is welded with the others into the terminal conjoined tendon or cuff (Fig. 10), it would seem easy to isolate any one tendon so that the more or less elastic muscle belly could be stretched enough to bring the tendon down again to the tuberosity and suture it there. However, if you try this on a normal shoulder at autopsy, you will find it is not easy, and when you try it on a ruptured tendon in which operation has been delayed for many months, you will find it impossible.

In the first place, you are cramped for room by the acromion and coraco-acromial ligament so that you cannot see the muscle bellies even in the normal shoulder. In the second place, if you dis-

sect back more than an inch on either the supraspinatus or infraspinatus, you run the risk of wounding the suprascapular nerve, and if you do, you may lose your power in those muscles forever.

In order to get at these tendons more effectively, I used to use the "sabre-cut incision," which gave a perfect exposure and every possible opportunity. (Plate VIII.) Even then the mobilization was only a little more satisfactory, so I have given up this incision. Practice has given me a little more confidence, and I believe now I can do almost as well through the simple routine incision. Dr. William Rogers has suggested removing the deltoid attachment with the periosteum from the acromion and suturing them back at the end of the operation. This seems rational, but I have not tried it and do not know whether one may rely on having the deltoid origin anchor again satisfactorily. I have sometimes thought that a subcutaneous osteotomy of the base of the acromion might mobilize it enough even without division of the coraco-acromial and acromioclavicular ligaments to allow easy access. The trouble with any incision which mobilizes the acromion is the long period which one must wait for union to occur before moving the joint. I am inclined at present to do all the mobilizing I can through the routine incision, and I find that I am constantly improving in my ability to do this.

It is probably best to remove the falciform edge of new tissue and to refresh the edges of the tendon itself. I attribute some of my imperfect results to my failure to do this. One learns by experience to put the suture back of the falciform edge, for the latter has no strength and the stitch at once tears out. One is tempted not to remove the edge because it is obviously difficult to close the rent without using it, and it seems folly not to save all the tissue one can. It might be contended that the falciform edge may have more tendency to unite than the real tendon substance, which has very little blood supply, so that perhaps I may be wrong in recommending the removal of this new tissue with which nature is attempting to repair the damage. The method of closure which seems to me the best is illustrated in Fig. 52.

3. The long head of the biceps. The problems connected with how to deal with the long head of the biceps when it is found exposed, owing to the retraction of the ruptured tendons, are not a few. I can only discuss them and do not pretend to solve them. Although I am not in agreement with some of Meyers' views on the importance of the rôle of the biceps tendon in shoulder injuries, I feel that his observations ought to be known to every one who operates on

these cases. To my mind, the rupture of the supraspinatus is the primary and important lesion which uncovers the biceps tendon, makes it slip a little at the top of the bicipital groove and to tend to be caught between the tuberosity and the acromion. At any rate, one often finds it a conspicuous, pink, inflamed-looking, swollen band lying across the joint cartilage at the bottom of the rent. (Plate VIII.) The portions exposed in the rent look inflamed; those covered by the remaining intact part of the capsule are white, glistening and normal. It is pretty obvious that our suture should cover up the biceps tendon without interfering with it otherwise. It usually lies just under the inner edge of the rent, but if any of the subscapularis fibers are involved, it lies entirely exposed. Sometimes it is not found at all, for it has been torn away from its glenoid attachment and has retracted down the bicipital groove. Sometimes it is split in two, longitudinally. Often it is flattened and frayed at the edges. Varying proportions of it may be ruptured. It may be composed of indefinitely separated longitudinal strands, some of which have become welded into the capsule. It may have little, rice-like tags on its edge. However, almost always the parts which do not become exposed in the gap left by the supraspinatus are normal in appearance.

When it has ruptured from the glenoid, it may be held high in the groove by a few remaining bands, and we can capture it and pull it up. What shall we do with it? We might try to suture it back on the glenoid, or rather on the fibrocartilage which surrounds the glenoid. Or we might attach it to the proximal portion of the supraspinatus, or to the capsule, or anchor it in the groove, or excise a part of it and use it to repair the supraspinatus. We might even take a relatively normal biceps tendon, clip its attachment off the glenoid, anchor the tendon in the groove, and then use the redundant portion to fill the gap in the supraspinatus. (Fig. 52*a*.) This would give the biceps muscle a fixed origin, and we would at the same time obtain a firm attachment for our supraspinatus. We should only have lost whatever function the long head of the biceps has from having its attachment on the glenoid rather than on the humeral head; *i.e.*, the outer head of the biceps would no longer be of use in motions of the humerus on the scapula, but could still apply its power in flexing the forearm on the humerus. What then is this function which we should lose so far as scapulo-humeral motion is concerned?

The function of the biceps muscle is fourfold. First, it is a flexor of the forearm on the humerus. Second, it is one of the flexors (or

extensors?) of the whole arm on the scapula; in a sense, therefore, it is a weak abductor or elevator of the arm. Third, the external insertion on the tubercle of the radius enables it to act as a supinator of the radius and hand. Fourth, the long head of the biceps passing through the intertubercular groove helps to retain the head of the bone on the glenoid, and stabilizes the head in the various degrees of rotation, as the arm is elevated. This function is well illustrated by the findings in two of my cases, which at operation showed that except for the subscapularis, the whole of the capsule with the tendons of the supraspinatus, infraspinatus and teres minor had been evulsed, yet the head did not tend to dislocate; apparently it was held in the joint by the long head of the biceps, and by that only. We lose nothing in the first function, little in the second, none in the third and but a problematic amount in the fourth, by using it in the way suggested in Fig. 52*a*.

So far as the action of the shoulder joint is concerned, particularly with reference to the functions of flexion of the arm on the scapula and of the forearm on the humerus, the origin of the short head of the biceps from the coracoid process is more important than that of the long head from the edge of the glenoid. The coracoid origin is sufficient to give power in these motions; the long head is chiefly a stabilizer and one of secondary use so far as the application of power is concerned. For instance, in cases in which the long head of the biceps is ruptured and no other lesion has occurred, the function of the shoulder remains almost normal.

The short rotators are sufficient to maintain the fulcrum on the glenoid in most positions of the arm, but where these short rotators are damaged, I am confident that the long head serves a very useful purpose in guiding the head of the humerus and restraining it from forging upward and getting its fulcrum on the acromion. I therefore regard it as important to keep the long head of the biceps intact if possible.

I have notes that in some of my operated cases, the biceps was torn away from its glenoid attachment. In such cases in future I intend to search for the distal end of the tendon and to anchor it with stitches in the bicipital groove, and also to the supraspinatus tendon, thus abandoning any idea of retaining its stabilizing function and being content with retaining its power as a flexor of the forearm. At present I see no good mechanical way of re-attaching it to the glenoid so as to make it function in guiding the head of the humerus as the latter is abducted. One is apt to think of the long head of

the biceps moving up and down in the intertubercular groove, but this is not what actually happens. The humerus moves up and down on the tendon; it is not the tendon which moves through the groove. (See Fig. 52.) On the whole, I should say that if the operator finds that the biceps is so damaged that he thinks it will not in future form a smooth cord on which the humerus can ride up and down, he had better use it, as described above, to replace the lost substance in the supraspinatus.

4. What shall we do if we find there is no stub of supraspinatus tendon left on the tuberosity to which we may suture the proximal portion? In long-standing cases we find a tuberosity completely bare of tendinous substance, and perhaps somewhat eroded. Since this tuberosity is useless unless we can suture the tendon to it, it might as well be removed. I have not hitherto excised the tuberosity in cases in which I could not suture, but it might be well to do so. Such an excision would make the surface which must ride under the acromion less apt to cause friction. Nature does exactly this by causing recession of the tuberosity. As a rule I have drilled two holes in the tuberosity with an ordinary shoemaker's awl, and passed a heavy silk suture through these holes and the tendon so as to draw the tendon as nearly as possible to the facet of insertion of the supraspinatus. This can usually be accomplished, but occasionally the supraspinatus is so retracted that I cannot quite draw it down to the bone.

I have on several occasions made a sort of plastic so that I covered the suture with part of the roof of the bursa, believing that the repair of the tendon comes not from the tendon itself, but from the adjacent synovial membrane which is much richer in vascular supply.

5. Another operative problem is how to repair the rent. As explained in the chapter on pathology, these rents are in a general way triangular, with the base on the humerus and the apex retracted, the apex being usually the center of the supraspinatus, and the sides the lateral expansions which are united to the neighboring tendons. The ideal way to close would be to bring the center of the apex to the center of the base, but if the retraction is great and the base is small, the triangle is so prolonged upwards that one is tempted to close the gap from side to side until very near the base, and then to make the last suture a triangular stitch. This method is easier, but it does not bring back the normal relations. However, it is a feasible method to use where there is much retraction. The exact way in which to put the sutures does not seem important, that is, whether they are mattress sutures or interrupted or continuous.

6. Formation of a new sulcus. If the reader will refer to Chapter IV, and especially to Plate VI, Figs. 3-4, and their legends concerning the remarkably effective method which nature has devised to attach the supraspinatus tendon to its facet, he will feel great doubt as to whether the surgeon will ever be able to imitate it with any degree of success. We need much study and experimental work before we can rely on being able to create a line of living cement such as the "blue line," with its pores for the finger-like processes. At present, from what we know of histology, it seems doubtful whether in adult life such a method of union of tendon to bone can ever be achieved. However, we know that tendon can form a fairly firm cicatricial attachment to raw bone. What is the best practical way to secure this?

If it were possible, we should wish to have the new tendon form on the raw surfaces of the sulcus and of the tuberosity down to the actual edge of the joint cartilage. When I drill the tuberosity I try to drill it as far as the cartilage edge, and I usually erode the bone of the sulcus with the point of a knife or curette, so that the tendon will have a little better chance to become attached by granulation. Dr. Philip Wilson has improved on my operation by cutting a slot around the cartilage edge and drilling through the base of the tuberosity. He then passes a slip of fascia lata through the drill holes to be attached above to the supraspinatus. He thus makes a more ideal suture, so that the tendon fills the entire sulcus and thus gains a firm hold on the tuberosity. It remains to be seen whether nature will tolerate such attachments indefinitely.

7. A frictionless surface for the base of the bursa is a most important point. Dr. Wilson's method has this advantage. It would be repetition to discuss this further, but I should like to repeat that even in those cases where the suture at the end of the operation has seemed rough, it may nevertheless be so changed by the healing process that a surface is produced which at a later operation appears smooth and normal.

8. I use silk sutures because I want them to endure long enough for new, strong, scar tissue or tendinous substance to form over them. I use a fairly heavy pedicle silk for the main suture, which passes through the holes in the tuberosity or between the proximal and distal portions of the tendon. I have on four occasions reopened the bursa later to remove these silk stitches because the patient complained of pain. The following are the findings in these four cases:

CASE 18

Mr. R. H. S. Age 60. M. G. H. No. 181765 E. S., Mar. 26, 1912. A typical case of complete rupture of the supraspinatus, one and one-quarter inches wide. Although much retracted, the tendon was caught and sutured in place with three mattress sutures. The functional result was good, but he continued to have more or less pain, apparently from the formation of a considerable amount of dense inflammatory tissue about the site of suture. On Feb. 13, 1913, under novocaine, the bursa was again opened and the tendon was found not only completely repaired, but there was a large amount of dense hypertrophic, callous-like tissue about the sutures. This mass impinged on the acromion in abduction; most of it was removed with the scissors and a new opening made through the supraspinatus into the joint, so that some of the synovial fluid could flow into the bursa and lubricate it. The result of this operation has been satisfactory. Twelve years later, on June 9, 1925, he called to see me because of a slight injury to his left shoulder. The right, on which I had operated, had given him no trouble in the intervening years, although he had worked steadily as a coachman.

CASE 29

Mr. M. M. W. Age 39. M. G. H. No. 184216 W. S., Aug. 5, 1912. A clear case of badly ruptured supraspinatus tendon. The tendon was sutured with heavy silk and function was restored. During the following year he had much pain on use of the arm in his work as a laborer. The bursa was again explored and the silk sutures and some of the chronic inflammatory tissues lying about them were removed. I also made a new opening into the true joint to permit the fluid to flow into the bursa. This was followed by improvement but not by complete relief. No late report. Note that entire repair of the rupture had taken place.

CASE 88

Mr. T. M. Age 50+. Operated on at Faulkner Hospital, July 24, 1926, six months after his injury. The supraspinatus, infraspinatus and part of the subscapularis were found to be torn away, exposing the biceps tendon, which was greatly inflamed. There was much fluid in the joint. A very unsatisfactory suture was made, and the tuberosity had to be drilled. The arm was put up in abduction. Mild sepsis occurred and there was much fluid drainage, so that the wound took several weeks to heal. Some of the deep sutures were taken out. In spite of this the result at first was good, and he returned to his work after five months. He worked for a year and

three months, although in some pain, and then had another slight injury. On July 2, 1928, I again explored the bursa and found that most of the sutures had pulled away, leaving the condition practically as bad as at the first operation. This was as bad a result as I have ever had. The patient was for a time benefited, but in the end gained nothing by the operation, for I did not attempt a second suture.

Case 112

Mr. A. C. Age 62. Operated on at the Trumbull Hospital on June 11, 1928, three months after his injury. A typical complete rupture of the supraspinatus was found and satisfactorily sutured. The immediate result appeared to be good. However, the patient would not go to work again, complained bitterly of pain on use of the arm and became very neurasthenic. On Feb. 7, 1929, I again explored the bursa, thinking that if I took out the deep sutures some of the irritation might be relieved. My notes say:

"I operated on him yesterday under novocaine anæsthesia. Dr. B. E. Wood was present and Dr. Stevenson assisted. Incision was made just inside the old scar and the bursa was opened. It was clearly shown that the former suture had been effective in restoring the insertion of the tendon. Moreover, the floor of the bursa was smooth and shiny, and there did not appear to be any cause for friction over the site of the suture. One heavy silk suture could be seen just below the transparent synovial lining of the base of the bursa; this was easily pulled out, but the other two sutures were buried deeply in the new-formed tendon and were found and removed with difficulty, as I was anxious not to weaken the tendon in so doing. In two of the sutures the knots were apparently untied; in one the knot was still present, but almost untied. At first I thought that the knots of the two untied ones had been left behind, but on reflection I think it is more reasonable to suppose that they had become untied as the tissues increased in amount and grew into the knots, which were cut very short. Yet it is possible that they broke off and remained in, although the total amount of silk in the untied ones appears greater than in the tied one by more than double. At any rate, very little silk could have been left behind.

"I did not feel satisfied that the silk was causing any trouble, for there appeared to be no inflammation about it, and the tender point of which the patient complained was nearly a half-inch away from the sutures, on the edge of the greater tuberosity close to the bicipital groove. That there was some inflammation at this point was made clear by finding a little crumbly, soft, cheesy tissue close to

the synovial sheath of the biceps tendon, which in certain positions bulged slightly. The repair of the tendon was weakest at this point, and I fear that my search for the sutures weakened it still more, although not to an extent sufficient to interfere with function, and recompensed by the finding of this suspicious tissue. Two tiny bits of this tissue were saved for pathologic examination. (Plate V, Fig. 5.)

The patient still claimed to be unable to work in January, 1931.

Since three out of four cases, which were explored a year or so after the first operation, showed not only firm tendons but hypertrophied ones, it seems to me that it is proved that suture may be effective. In each case I was surprised to see how well nature had restored the even convexity of the floors of the bursæ, which at the completions of the operations had been quite irregular and rough at the suture lines. All four cases, if operated on immediately after their injuries, might have had excellent results; as it was, although two of the four cases had good results, little was gained by the other two patients, unless they may take some satisfaction as demonstrators of the fact that these tendons even when badly broken may be repaired.

9. The shape of the needles is dictated by the shape of the field of operation and by the fact that a tremendous strain is put on them. They must be either fully curved or half curved, not over a half-inch long and with very strong shank and eye. One has to work between the acromion and the tuberosity, where there is very little room, so that even a curved needle such as is used in ordinary operations is too large to be turned about in this space.

10. Shall we close the roof of the bursa or shall we merely close the muscle, leaving the roof of the bursa free to allow the synovial secretions to seep into the areolar tissue?

As I have previously stated, there is usually in these cases a considerable synovitis with a large amount of fluid. If the bursa is closed tight, this fluid forms under tension and causes pain. Closure also tends to keep blood in the bursa which would otherwise be washed out by the fluid itself. I prefer the idea of leaving the roof of the bursa unsutured to allow this fluid to escape, but I am not prepared to say positively that it is not better to suture the bursa and allow free motion after the operation to pump fluid out between the stitches. The fact is, in cases where there is much fluid (and these cases are usually those that have continued to work in spite of the friction and pain), the fluid seeps into the soft tissues to an extent which causes marked swelling and sometimes induces an edema and

suggestion of sepsis. This used to be a frequent complication when I put the arm in elevation, permitting the lower side of the capsule to be held tense and therefore driving the fluid up toward the wound. Now that I treat them without restraint, I do not have this complication.

11. The postoperative treatment. I find that my tendency has been, as the years go by, to allow more motion and to allow it sooner. I usually pad the axilla with a small pillow and then let the arm lie on it in a position a little more abducted than that in which the arm rests in a sling, contriving as best I can to keep the hand away from the front of the abdomen, because the tendency of the patient after these operations is to get the arm in a strongly internally rotated position, and therefore the recovery of the power of external rotation is slow. After the first night is over, I remove the dressing and let the patient put the arm in any comfortable position which he can find. Each day I exercise it in a way which is difficult to describe, but which is a matter of personal touch. The general purpose of the exercises is to let the patient bend his body from the hips with the arm relaxed, as described under the stooping exercises (Fig. 47). As in treating fractures near joints, I try to make the patient do as much active and passive motion of the arm as I believe I can without displacing the fragments. It is impossible to lay down more definite directions, but I may say that by the end of the first week I expect the patient to be able to bend his body at the hips to a right angle, and to let both the injured and well arm fall in a relaxed position at right angles to his body. By twisting his body from side to side so as to make one shoulder higher than the other, alternately, he can also move the joint without contracting the shoulder muscles. During the second week he is encouraged to swing the arms a little in both directions in this stooping position. The wound should be soundly and completely healed and the patient discharged from the hospital in from ten days to two weeks. After that he is encouraged to take the stooping exercises.

If the patient is coöperative and understands the mechanics of the operation and can use common sense in taking his exercises, he gets on fairly smoothly, but there is pain of an annoying although not of a serious degree, not only for weeks but for months. I do not think this would be the case where the operation was done immediately after the accident. In convalescence it is a good rule to restrain the patient from exercising his arm in the erect position until he has learned to abduct it freely and strongly in the stooping

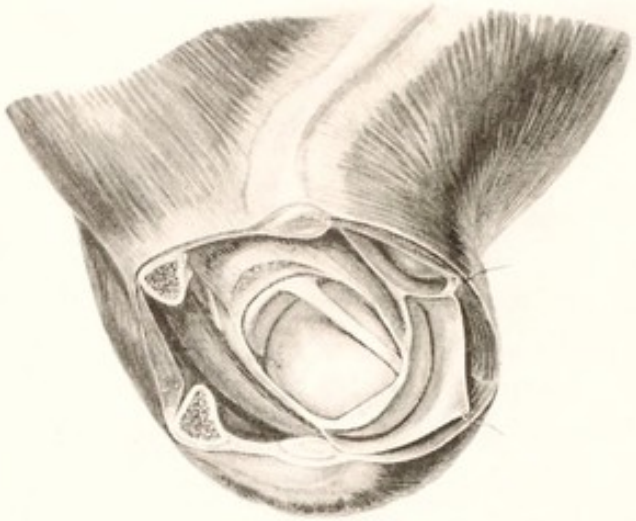


FIGURE 3

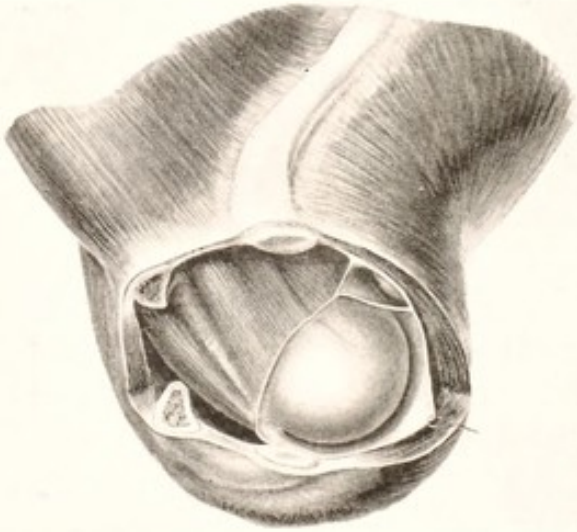


FIGURE 2

FIGURE 1

PLATE VIII. SABRE-CUT INCISION

THE SABRE-CUT INCISION

Reprinted from the *Bos. Med. & Surg. Jour.*, Mar. 10, 1927. It does not differ greatly from Kocher's posterior incision, but is more appropriate after a preliminary exploratory cut anterior to the joint.

FIGURE 1

"Sabre-cut" seemed an appropriate name for this incision, for it might well be made by the downward cut of a sabre on top of the shoulder. An incision is made through the acromio-clavicular joint and continued with a saw through the base of the acromion. The anterior point of the incision would be continuous with a previous routine bursal exploratory incision. When the acromion has been sawed through, an epulet of tissue, consisting of the deltoid muscle and the acromion process from which it arises, is formed to be pulled outward and downward. This step is accomplished with ease, for it is only held by a little areolar tissue and a few fibers of the trapezius attached to the upper margin of the detached portion of the acromion. The upper posterior fibers of the deltoid must be separated a little to gain mobility. In sawing the base of the acromion one must bear in mind the suprascapular nerve which supplies the supra- and infra-spinatus muscles and lies between them, a little below the saw-cut. It is deep enough to be out of the way of the saw but not of gross carelessness.

FIGURE 2

The second diagram shows the structures exposed when this epulet is pulled downward and outward. Even without dissection one can identify the subscapularis, supraspinatus and infraspinatus as they emerge to join together their tendinous expansions beneath the base of the bursa. To one unfamiliar with this dissection the smooth convex surface of this base appears to be the articular surface of the humerus. The subacromial and subcoracoid or coraco-humeral bursæ are nicely shown. As explained in previous papers, they are often intercommunicating and are always functionally one bursa although frequently, as in this instance, separated by one of the diaphanous nictitating folds. Notice the separated portion of the acromion and see how easily it will fit back into place.

FIGURE 3

The third diagram is identical with the last except that the supraspinatus and capsule have been cut across into the true joint and the ends of the supraspinatus depicted as retracted. The stub of the tendon is still attached to the tuberosity beneath the base of the bursa, while the muscular belly is retracting into the supraspinatus fossa. The glenoid and the articular surface of the humerus are exposed, with the long head of the biceps arising from the superior edge of the glenoid lying across the cartilaginous surface of the head of the humerus.

This is exactly the condition I have found at operation again and again in the living, except that there is seldom so much of a stub of tendon still attached to the tuberosity. Quite frequently it is entirely evulsed from the latter, requiring drilling of the tuberosity to resuture it. I have always found the base of the bursa to be torn across with the tendon. The point of least resistance appears to be about the subbursal portion of the tendon. In fact the tendon itself is very short, the muscle fibers beginning within a half-inch of the attachment.

In the long-standing cases on which I have operated the biceps tendon is found inflamed, swollen and bright pink in color, forming a striking contrast with the white articular surface of the humerus. Sometimes it is apparently absent entirely, having been evulsed and then retracted downward into its sheath.

To close this incision the parts are sutured back into place in the reverse order of these diagrams. It is probably safer to wire the acromion process, although catgut in the soft parts holds it well. I do not advise attempting to close the bursa even in the exploratory operation; a stitch or two in the muscle holds the edges in sufficient apposition and excess fluid may drain into the areolar tissue.

position. (See Fig. 47.) In long-standing cases the nerves of the region have already become sensitized and are slow in returning to a normal condition. Much of this postoperative soreness in the delayed cases is due to the sensitiveness and synovitis acquired between the date of the injury and that of the operation.

The pendulum will probably swing in future toward postoperative treatment in abduction and back again to adduction. Dr. Wilson now uses abduction after the sabre-cut incision and complete repair of the insertion into the bone by the use of fascia lata. It is possible that this method has the advantage of creating a larger gap between the head of the humerus and the acromion and the coraco-acromial ligament, because reunion of the mobilized acromion process would take place at a higher level, since it is pressed upward by the abducted humerus.

The Sabre-Cut Incision. Although I have personally given up the sabre-cut incision for cases of rupture of the supraspinatus, it is still used by others, especially by Dr. Wilson. It gives a splendid opportunity to repair the tendon or any other structure in the shoulder joint, but it is really a major operation, while the one I use is a minor one. The main reasons why I seldom use it are three. In the first place, I have learned to work through the routine incision in such a way that I can do the operation without cutting any ligaments or bone. This improvement has come about not only from doing the operation in dorsal flexion, but by using the method of rotating the humerus so that each desired point is placed in the middle of the small incision at the appropriate moment for a stitch. One assistant has to manipulate the arm in unison with the wishes of the surgeon. In the second place, I have found that after division and suture the acromio-clavicular joint may remain somewhat unstable.

A third reason is less technical and more in the domain of human nature. In Industrial Surgery there is not a frank understanding between surgeon and patient as in their ordinary professional relation. The patient is apt to have the element of compensation too strongly in mind, as compared to a coöperative tendency to make the best of the surgeon's attempt to better an injured limb, although both know it may never again be "as good as new." The extent of the sabre-cut incision exaggerates in the patient's mind the degree of the injury and the scar would certainly be impressive to a commission or jury.

Operations for correction of deformities due to paralysis of the shoulder muscles. There are many interesting operations of the above kind which have been devised and which in some cases in expert hands

have been successful, but none may be said to be of sufficiently standardized technique to be considered in this book, especially as I have had no personal experience in such cases. The most encouraging example is the operation recently devised by Dr. Frank R. Ober for the relief of paralysis of the deltoid muscle. (*Jour. A. M. A.*, Dec. 24, 1932, p. 2182.)

I have had the pleasure of seeing the case illustrated by Dr. Ober, and can testify to the ability of the child to raise the arms as indicated by his illustrations. Dr. Ober tells me he has since done a number of other cases with the same encouraging result. The operation consists in a transfer of the origins of the triceps and of the short head of the biceps to new attachments on the acromion process, thus using these muscles as abductors.

Non-Operative Treatment of Rupture of the Supraspinatus Tendon

I think every general surgeon who could see one of the complete cases at operation would agree that no form of physiotherapy could influence beneficially one of these lesions, except in a subjective way. The subjective symptoms might possibly be alleviated to a certain degree by massage or by diathermy, but as a matter of fact, nearly all patients say that these agents are of little help. From the very nature of the lesion, manipulations and forced exercises can do no good and might do some harm. If it is determined that a patient is not to have an operation, there are two rational plans of treatment: (1) Fixation in elevation to relax the tendon, improve the blood supply and approximate the torn ends; (2) Gentle "stooping exercises" to help nature smooth off the irregular surface of the lesion. This is merely an aid to nature's own partially successful method.

Fixation in elevation, to be effective, would necessarily have to be instituted soon after the injury, and would require at least three weeks. It would be useless in old cases, yet even in the old cases one can see, when the bursa is open on the operating table, that there is a tendency toward approximation of the torn ends of the tendon as the arm is abducted. Of course, in these old cases the proximal end is retracted and cicatrized in its retracted position. Even prolonged fixation in such cases could accomplish little except in possibly diminishing the size of the defect. This tendency to approximation observed at operation in old cases, when the arm is abducted, shows that it is probable that if the rupture were small, this treatment, if immediately instituted, would be worth while. If I were the patient, I would prefer incision and suture to this problematical and uncomfortable, even if reasonable, plan.

The "stooping exercise" method is founded on the pathologic findings described in Chapter III. Clearly nature has developed this method of absorption of the tuberosity to restore function after this accident, for as these patients, after a year or two of misery, improve somewhat, it is fairly clear that this method of smoothing off the eminence and sulcus which caused the jog is the best she can do. Nature's plan is the combination of this slow smoothing-off of the tuberosity and meanwhile increasing the supply of fluid, which, as the lower portion of the capsule becomes tense in any action, is forced up to the site of friction.

The idea of the stooping exercises is to begin this process with the weight taken off the arm so that the irregular surface is not forced up under the acromion by muscular tension. If the patient stands and elevates the arm, the very point which is sore becomes the fulcrum on the acromion for the deltoid to exert its power. When the patient stoops with the arm relaxed, the scapula can be abducted on the humerus without the need of the fulcrum either on the glenoid or acromion. Gravity takes the place of the power of the deltoid.

It is clear that until a patient can swing his arms freely in the stooping position without pain, he is not ready to use the arm when standing.

To patients who refuse operation I explain these mechanics and impress on them what I have said in the last paragraph. I also try to teach them to sleep with the arm abducted, with the hand behind the head. Palliatives in the form of drugs or physiotherapy are not often required in these cases. The pain is always bearable, if they do not increase it with work or exercise which make a fulcrum of the sore point. I think I can accomplish more with these patients by explanation of the mechanics of their trouble than I can with any palliative measures. Show a workman the normal function of the joint, the necessity of the supraspinatus to maintain the fulcrum on the glenoid, the tendency, without it, for the fulcrum to ride on the sore spot where it touches the acromion, the changed action of the weight of the arm when stooping or lying with the hand behind the head, and, as a rule, he will get your drift. He knows about levers and weights. When he understands his condition, he will get rid of the fear element and realize that though his arm is impaired, he has not really lost the use of it. He has learned that he has lost the use of certain motions unless he is willing to stand the pain. This education has more therapeutic value than drugs or electrical treatment. Massage, of course, may do good in stimulating the nutrition of the tissues, but it cannot unite the tendon.

TABLE OF AUTHOR'S OPERATIONS ON THE SHOULDER SINCE THE DEMONSTRATION OF THE FIRST CASE OF COMPLETE RUPTURE OF THE SUPRASPINATUS

Name	*Case No.*	*Occupation*	*Age*	*Date of Operation*	*Complete Rupture Supraspinatus*	*Fracture or Dislocation*	*Calcification*	*Partial Rupture or Tendinitis*	*Bands, Folds Villi*	*Other*	*Traced*	*Results*	*Comments*
Mrs. J. A.	1	Housewife	52	3–11–09	X						5 years	good	Page 126.
Mrs. A. V.	2	Housewife	73	3–24–09				X			. . . .	good	Immediate result good. Not traced.
Mrs. F. McI.	3	Housewife	44	3–29–09			X				24 years	good	Husband reports no further trouble with shoulder.
Dr. P.	4	Physician	. .	8–09				X			2 years	good	
Mrs. W. A. A.	5	Housewife	66	10–16–09		X					5 years	good	Similar Case 71. Page 288.
Mrs. F. A. B.	6	Housewife	62	11–27–09				X			5 years	good	SS 167122 M.G.H.
Mrs. A. R. N.	7	Domestic	27	12–1–09						X	13 mos.	poor	Page 112.
Mr. P. K.	8	. . . . .	62	1–3–11					X		3¼ yrs.	good	Pathologic Spec. No. 111–13. ES 173766 M.G.H.
Mr. D. R.	9	Hostler	40	1–10–11	X						10 years	good	Page 127.
Mr. M. McD.	10	. . . . .	23	1–20–11		X					. . . .	good	Page 318.
Mr. P. O.	11	Plasterer	62	1–21–11	X						3¼ years	good	Smallest rupture in series.
Mr. A. F. K.	12	Mule spinner	45	1–23–11		X					13 mos.	good	Page 287. WS 174082 M.G.H.
Miss L. E. B.	13	None	28	5–8–11			X				3 years	good	
Mr. P. S.	14	Laborer	39	12–28–11	X						3 years	good	Had only one arm; died of pneumonia in 3 years.
Mrs. C. V. McG.	15	Housewife	70	1–18–12	X						2 years	poor	Mild sepsis. Poor suture. Page 175.
Mr. W. H. R.	16	Business	59	3–6–12		X					20 years	good	Page 284. Fig. 57.
Mr. D. H.	17	Laborer	64	3–14–12				X			2 years	good	At work in a month. ES 181539 M.G.H.
Mr. R. H. S.	18	Coachman	60	3–28–12	X						13 years	good	Page 246. 181764 M.G.H.
Mr. P. A.	19	Laborer	54	3–28–12	X						2 years	good	No definite injury. Colored. Perfect result. P. 97.
Mr. J. D.	20	Paper cutter	54	4–15–12	X						2 years	good	(Cock's comb.) Page 89.
Mrs. M. F.	21	Housewife	50	4–20–12						X	Few mos.	fair	Complement fix. pos. g.c.? P. 112. ES 182228MGH
Mr. J. H. D.	22	Laborer	69	4–22–12		X					2 years	good	Page 288. 182238 M.G.H.

Name	Case No.	Occupation	Age	Date of Operation	Complete Rupture Supraspinatus	Fracture or Dislocation	Calcification	Partial Rupture or Tendonitis	Bands, Folds Villi	Other	Traced	Results	Comments
Mr. G. R.	23	Laborer	60	5–10–12	X						2 years	good	Sabre cut incision.
Mr. K. N.	24	Orderly	26	5–22–12		X					1 year	good	Sabre cut incision. P. 298. ES 182833 M.G.H.
Mr. C. L. D.	25	....	55	5–31–12				X			13 mos.	fair	Abduction somewhat restricted.
Mrs. N. B.	26	Housewife	51	6–3–12	X						8 mos.	good	Slight weakness in abduction.
Mr. H. H.	27	Marble worker	44	6–11–12	X						....	?	Villi removed. No suture.
Mrs. K. M. G.	28	Housewife	45	7–15–12				X			....	?	ES 183830 M.G.H.
Mr. M. M. W.	29	Laborer	39	8–8–12	X						....	?	New opening made into joint. Page 246.
Mr. A. E.	30	....	47	9–3–12						X	1½ yrs.	good	Page 384. WS 184726 M.G.H.
Dr. G. K.	31	Physician	36	9–3–12						X	20 years	good	WS 184847 M.G.H.
Mr. C. H. C.	32	Secretary	45	9–7–12				X			1 year	good	Recovery slow.
Mrs. C. B.	33	Housewife	38	9–7–12		X					1½ years	good	Page 291.
Mr. J. G.	34	Tailor	52	9–13–12					X		....	?	Opening in joint as in Case 29. P. 246. WS 184940 M.G.H.
Miss L. D. S.	35	....	52	12–9–12				X			Few mos.	good	Capsule torn. Page 219. ES 186452 M.G.H.
Miss F. B. M.	36	....	25	1–23–13				X			1⅓ years	fair	ES 187241 M.G.H.
Mr. C. J.	37	None	43	3–15–13						X	Few mos.	fair	Osteo Chondritis Dessicans. ES 188074 M.G.H.
Mr. J. H. J.	38	....	41	3–15–13				X			....	?	ES 188075 M.G.H.
Mr. E. E. C.	39	Harness maker	31	4–5–13					X		....	?	Opening made into joint. ES 188440 M.G.H. P. 246.
Mr. B. P.	40	....	39	4–24–13					X		....	?	Partial rupture. ES 188763 M.G.H.
Mr. C. T. W.	41	....	38	5–13–13						X	1 year	poor	Double ankylosis following fracture. Sabre cut. Unilateral excision of head of humerus. ES 189145 M.G.H.
Mrs. K. S.	42	Housewife	63	5–29–13	X						....	?	Immediate result good.
Mr. P. J.	43	Engineer	32	7–1–13	X						9 mos.	good	?
Mr. J. A. C.	44	....	30	7–7–13				X			9 mos.	poor	Tendons seemed to be adherent beneath. ES 190001 M.G.H.

Mr. J. R.	45	....	56	7–26–13		X					....	?	Probable fracture of tuberosity. ES 190348 M.G.H.
Mr. C. O. F.	46	Cooper	58	8–20–13	X						8 mos.	perf't	No definite accident. Used 6-lb. hammer at work. Page 97.
Mrs. E. A. B.	47	None	43	8–29–13			X				Sev. yrs.	good	
Mr. J. E. T.	48	....	50	11–19–13		X					....	?	Complicated by 2 previous ops. by others. ES 192342 M.G.H.
Mr. J. B.	49	Laborer	64	12–13–13	X						10 years	poor	Hopeless. April 24, 1914. Med. Wards? Page 385.
Dr. D. J. D.	50	Physician	36	3–24–14			X				5yrs.5mos.	good	Symptoms many years.
Dr. A. M. G.	51	Physician	39	6–5–14			X				19 years	good	Page 207.
Mr. R. B. E.	52	Chemist	38	6–29–14			X				11 years	good	No trouble at last note.
Dr. N. B. M.	53	Physician	52	11–8–14			X				5½ yrs.	good	No trouble at last note.
Mr. M. J. C.	54	Superintendent	52	2–13–15			X				1½ yrs.	good	No trouble at last note.
Dr. A. H. H.	55	Physician	53	1–31–16			X				17 years	good	Page 207.
Mr. A. C. B.	56	Business	50	3–29–16			X				17 years	good	Page 207.
Mr. M. C.	57	Laborer	63	3–29–16	X						16 years	fair	Very extensive; unsatisfactory suture. Pages 140, 163 and 175.
Mr. M. A.	58	Laborer	28	11–15–16					X		3 mos.	fair	Partial rupture.
Dr. J. F. C.	59	Physician	46	5–6–18			X				11 years	good	Page 194.
Mr. C. C. H.	60	Business	63	5–17–18			X				....	good	Immediate result good. Not traced.
Dr. H. J.	61	Physician	61	8–12–19				X			7 years	good	
Miss R. P.	62	None	45	12–14–19			X				14 years	good	Page 207.
Mr. A. P. T.	63	Business	40	5–18–20			X				13 years	good	Page 207.
Mrs. J. L.	64	None	54	9–18–20					X		Sev. yrs.	neg.	Died of cancer soon after. Page 416.
Mr. E. H.	65	Manufacturer	39	9–25–20			X				13 years	good	Page 207.
Mr. S. P.	66	Architect	39	1–31–21			X				12 years	fair	Complicated by alcoholism. Page 207.
Mr. D. S.	67	Farmer	50	2–19–21	X						12 years	good	Worked regularly on farm since a few months after operation.
Mr. G. B.	68	Business	53	3–1–21			X				12 years	good	Page 207. Plate II, Fig. 3.
Mrs. W. A. P.	69	Housewife	61	6–13–21			X				10 years	fair	Page 220, last paragraph.
Mr. J. McG.	70	Schoolboy	14	6–16–21		X					10 years	fair	Pages 305 and 306.
Mrs. E. C.	71	Housewife	68	4–5–22		X					4 mos.	fair	Pages 288 and 289. Fig. 58.
Dr. G. W. M.	72	Physician	40	9–19–22			X				11 years	good	Page 207.

Name	*Case No.*	*Occupation*	*Age*	*Date of Operation*	*Complete Rupture Supraspinatus*	*Fracture or Dislocation*	*Calcification*	*Partial Rupture or Tendinitis*	*Bands, Folds Villi*	*Other*	*Traced*	*Results*	*Comments*
Dr. C. P. S.	73	Physician	44	9–19–22			X				11 years	good	Calc. present for 10 years. Page 207.
Dr. H. L. T.	74	Physician	44	4–6–23			X				2 mos.	good	Immediate result good. Not traced.
Mr. J. D.	75	Longshoreman	49	3–14–24	X						6 years	good	Sabre cut incision. Later longshoreman. Pages 140 and 163.
Mr. J. S.	76	Milk delivery	32	5–7–24			X				....	good	Soon returned to work. Not traced since. Page 169.
Mr. C. S.	77	Carpenter	57	7–12–24		X					9 mos.	fair	Excision of tuberosity.
Mr. J. E. O.	78	Laborer	21	12–15–24					X		3 years	poor	Hypertrophy of deltoid (final result?).
Mr. F. I. W.	79	Supervisor	61	12–22–24			X				9 years	good	Pages 205 and 207.
Mr. H. W.	80	Laborer	77	3–6–25						X	2 mos.	fair	Courageous old man. Page 169.
Mr. J. J. S.	81	Ironworker	48	5–9–25			X				8 years	good	Pages 205 and 207.
Mrs. A. T.	82	Lab. Tech.	..	7–7–25			X				9 years	good	
Mr. C. D.	83	Teamster	48	1–23–26	X						4 years	poor	Calcification in coraco-acromial ligament. Pages 140, 163 and 175.
Miss G. A.	84	Stenographer	46	2–2–26			X				6 years	slow	Page 209.
Miss L. M. S.	85	Housewife	54	2–25–26			X				7 years	slow	Page 207.
Mr. F. F.	86	Laborer	33	4–16–26		X					6 mos.	good	Plastic on pectoralis major.
Mr. F. DeM.	87	Tel. Lineman	42	5–24–26					X		3 mos.	good	Page 71 (5).
Mr. T. McS.	88	Laborer	50	7–24–26	X						3 years	poor	Pages 140, 164, 175 and 246.
Mr. D. K.	89	Laborer	65	7–24–26	X						4 years	fair	Pages 141, 164 and 175.
Miss M. R. M.	90	Filing clerk	30	7–31–26			X				1 mo.	good	Rapid recovery. Not traced since.
Mr. J. A.	91	Mechanic	60	8–3–26				X			10 mos.	fair	Rapid recovery. Page 170.
Mr. E. G.	92	Plumber	26	8–12–26		X					1 year	fair	A case similar to that in Fig. 58.
Mrs. B.	93	Phys's wife	44	12–11–26			X				6 years	good	Page 207.
Mr. W. R.	94	Shipper	34	4–20–27			X				1 mo.	good	Soon returned to work. No late report.
Mr. M. E. T.	95	Machinist	36	4–22–27			X				3 wks.	good	Soon returned to work. No late report.

Mr. L. R.	96	Steamfitter	48	5–16–27	X						4 years	good	Worked afterward as steamfitter. PP. 141 and 164.
Mr. A. D.	97	Laborer	61	5–20–27	X						6 years	good	Heavy work in 3 mos.; worked steadily since. Pages 141 and 164.
Mr. A. C.	98	Laborer	40	5–28–27	X						6 years	good	Heavy work in 2 mos.; worked steadily since. Pages 141 and 165.
Dr. R. C.	99	Histologist	46	6–25–27			X				6 years	good	. . . .
Mr. R. W.	100	Electrician	. .	8–18–27				X			5 years	good	Previous operation by another surgeon.
Miss M. R.	101	Med. student	22	8–29–27					X		1 mo.	good	Good immediate result. Not traced since.
Mr. J. D.	102	Laborer	57	1–3–28	X						3 years	fair	Ulcer of stomach. Pages 141, 165 and 175.
Dr. G. L.	103	Physician	45	1–7–28			X				5 years	good	Pages 205 and 207.
Mr. H. B. W.	104	Engineer	51	1–9–28					X		2⅓ years	poor	Malingering 75%.
Mrs. G. S.	105	Housewife	60	2–14–28			X				5 years	good	Pages 170, 205 and 207.
Mr. F. A. F.	106	Truck driver	52	2–8–28	X						4 years	good	To work in 3 mos.; worked steadily since. Pages 141 and 165.
Mrs. L. B.	107	Cook	52	3–13–28	X						3 years	fair	Pages 141, 175 and 386.
Mr. T. C.	108	Stockfitter	69	3–16–28	X						14 mos.	fair	Cancer of stomach. Died in 14 mos. PP. 141, 165 and 175.
Mr. C. O. W.	109	Laborer	34	3–22–28						X	4 mos.	poor	Exploration of trapezoid bursa. Malingering?
Mr. C. B.	110	Lather	17	3–22–28				X			3 mos.	good	Ruptured biceps. Page 170.
Mr. M. G.	111	Laborer	46	4–12–28				X			6 mos.	fair	Page 171.
Mr. A. C.	112	Hostler	62	6–11–28	X						2½ yrs.	poor	Pages 141, 166, 175 and 247.
Dr. W. L.	113	Physician	55	8–18–28			X				5 years	good	Pages 205 and 208.
Mr. J. L. E.	114	Business	60	9–7–28			X				5 years	good	Page 208.
Mr. A. N.	115	Wrecker	48	9–24–28	X						4 years	neg.	Pages 141, 289 and 389.
Mrs. G. W. H.	116	Clergy's wife	47	9–27–28				X			4 years	good	Clear fluid discharge from tendon. Page 73.
Miss E. S. M.	117	None	64	10–26–28			X				5 years	good	Pages 183 and 210. Fig. 43. Slow.
Mrs. W. L. P.	118	Housewife	69	2–15–29				X			1 year	good	Slow. Page 220, last paragraph.
Mr. W. J. H.	119	Painter	63	3–12–29	X						2 years	good	Not industrial. Began work soon after op. Pages 141 and 166.
Mr. S. G.	120	Laborer	51	3–17–29				X			7 mos.	fair	Page 171.
Dr. R. B. G.	121	Physician	59	4–11–29			X				5 years	good	Pages 205 and 208.
Mrs. M. C.	122	Housework	. .	6–29			X				5 years	good	Pages 205 and 208.

Name	*Case No.*	*Occupation*	*Age*	*Date of Operation*	*Complete Rupture Supraspinatus*	*Fracture or Dislocation*	*Calcification*	*Partial Rupture or Tendinitis*	*Bursa, Folds Villi*	*Other*	*Traced*	*Results*	*Comments*
Mr. H. F.	123	Laborer	38	8–12–29	X						3 wks.	..	Drowned Sept. 3, 1929. PP. 141, 166 and 175.
Mr. L. A.	124	Grocer	38	8–17–29						X	3 mos.	good	Axillary paralysis. Exploration. Elevation. P. 384
Mrs. L. J. W.	125	None	40	8–17–29			X				1 year	good	....
Mr. J. C.	126	Carpenter	42	10–23–29				X			2 years	fair	Page 172.
Mr. S. H.	127	Laborer	60	12–3–29	X						1 year	fair	Pages 141, 167 and 175.
Mr. J. M. K.	128	Truck driver	39	12–28–29	X						2½ yrs.	good	Went to work in three months. Pages 141 and 168.
Mr. J. S.	129	Laborer	49	1–4–30	X						2½ yrs.	good	To work soon but had compensation for a second injury. Pages 141 and 168.
Mr. W. T.	130	Laborer	..	1–21–30						X	1 year	fair	Previous infection of joint. Page 498.
Mr. G. McG.	131	Loco. fireman	36	2–18–30				X			3 mos.	good	Page 173.
Mr. J. C.	132	Pipe fitter	49	4–28–30			X				....	good	Prompt recovery but not traced. P.B.H.
Miss A. M. D.	133	Stenographer	32	8–4–30			X				3 years	good	Page 208.
Mrs. E. C.	134	None	46	11–13–30			X				3 years	good	Page 208.
Mr. G. H.	135	Lineman	54	1–23–31	X						7 mos.	good	To work in a few months. Pages 141 and 168.
Mrs. P. W.	136	None	31	1–29–31					X		2½ yrs.	fair	Relief not complete. Complicated case.
Mr. W. F.	137	Lather	62	2–4–31	X						1 year	good	Compensation settled. Pages 141, 169 and 175.
Dr. H. M. L.	138	Physician	48	3–17–31			X				2 years	good	Died of cancer stomach in Dec. 1931. P. 205.
Mrs. L. B.	139	Housewife	..	6–8–31			X				2 mos.	good	Pages 69 and 76. Plate II, Fig. 4.
Mr. C. W.	140	Garage	38	6–27–31			X				2 years	good	Pages 205 and 208. Plate II, Figs. 1 and 2.
Mr. G. F. D.	141	Postman	41	7–3–31			X				2 years	good	Pages 192 and 208. Fig. 44A.
Mr. R. B.	142	Business	55	7–15–31			X				2 years	good	Pages 205 and 208.
Miss R. McC.	143	Waitress	31	9–19–31			X				2 years	good	Pages 206 and 208.
Mrs. E. B.	144	Musician	57	2–6–32			X				1½ yrs.	good	"Favors arm."

It has seemed to me best to present my operations on the shoulder tabulated in this way, rather than to give complete case histories of each patient. Many of the cases have been used in the text to illustrate different points, sometimes only the occupation being used and perhaps in another place the preoperative diagnosis given to compare with the operative findings, etc. Under the comments at the right of this table the page numbers are given when the individual cases are referred to in the text. Should any one desire to do so, he could, by referring to these pages, make up brief individual case histories. As a rule, the cases which have been spoken of in the text are those in which the result was poor or fair. Those which I have considered good, lack detail. On the whole, compared with the results of the cases in the rest of the table, the outcome in the cases of complete rupture has been relatively poor. The reader is urged to bear in mind that in all the cases on which I have operated my attempts were more or less delayed, for only cases 18, 27, 42, 49, 106, 115, 127, 128, 129 and 135 were done within two months of the accident. In cases 49 and 115 suture was impossible, and cases 27 and 42 could not be traced over a period of years, although the immediate results were good. I probably should not have written this book at all had the results all been good, for one of my chief objects is to urge prompt diagnosis and immediate operation.

Some of the cases marked "poor" actually did obtain some improvement. For instance, Case 88 worked over a year after the operation and then had another accident, so that his total compensation was the maximum allowed by law. Case 89 has worked steadily as a watchman after he had made a settlement with the insurer. I feel that I have certainly not exaggerated the benefits of surgery in these delayed cases, for I am sure something was accomplished in all of them, even in cases such as number 115 where no suture was attempted, for at least a definite diagnosis was made, as a basis for a settlement. If, as I hope, eventually the profession comes to realize the importance and frequency of this lesion, and the laboring class becomes educated to take it seriously, exploration through a small incision will be the rule in doubtful cases. When the insurance companies become interested in the problem, I shall be glad to coöperate in making a more extensive survey of the economic side than that which is presented on page 176.

REFERENCE

Wilson and Fowler have modified my operative methods. References to their articles will be found at the end of Chapter V.

Chapter IX

THE RÔLE OF THE SUPRASPINATUS IN DISLOCATIONS AND FRACTURES OF THE SHOULDER JOINT

This chapter does not aim to be an exhaustive treatise on fractures and dislocations of the shoulder, although a very large number of articles have been studied in order to compile it. During the summer of 1928, Dr. T. W. Stevenson reviewed the literature for me, with especial effort to find to what extent the bursa and the short rotators had been referred to. At about the same time, I took advantage of the service of the Library Bureau of the American College of Surgeons, with the request that they find for me all the references they could concerning rupture of the supraspinatus tendon. After a careful search they were unable to find any articles whatever which were devoted to the subject, although occasionally they found references to rupture or avulsion of the tendons in cases of dislocation on which operations had been done.

So far as I know there is no record in the literature of what I call the "Pivotal Paradox," described on page 43. I, myself, did not understand this paradox when I started to write the book, and only gradually puzzled out its meaning and significance. Certainly none of the authors, who have written on dislocations and fractures in this region, seem aware of the facts that in complete elevation of the arm almost no rotation can occur and that the extent of rotation diminishes as the arm is elevated. The lack of this knowledge of normal functions seems to me to render most of the previous explanations of the mechanism of dislocations and fractures in this region nearly worthless, for several important deductions can be made by the use of these facts. I have consequently eliminated much that I had previously written about the opinions of authorities.

In complete elevation there is only one possible geometric relation of the two units, *i.e.*, the head of the humerus and its tuberosities, with the scapula and its processes. No matter in what degree of rotation you start to raise your arm and no matter in what plane you raise it, the capacity for rotation will be less and less as the arm ascends until, in complete elevation, tuberosities and processes will be locked in a fixed position. In this fixed position the articular head of the humerus will be shown by X-ray in the antero-posterior view, to face nearly outward, and in the lateral view to face somewhat forward, *i.e.*, it actually faces obliquely outward and forward, ex-

actly reversed from its anatomic position. The axis of the condyles of the lower end of the humerus becomes antero-posterior, with the internal condyle pointing forward. The humerus has rotated inward 90° and has become inverted. You may say that the angle between the shaft and the axis of the neck gained 45° by *inversion* and then 45° by *external* rotation, or you may say, if you please, it first gained 45° by *internal* rotation and then another 45° by inversion. Do not proceed with this chapter until you are sure that you have understood this paragraph, even if it is necessary for you to take a humerus in your hand.

The reader must bear in mind that this pivotal position may be assumed by extension of the scapula on the humerus as well as by extension of the humerus on the scapula. For instance, the arm may be in this position when a boxer delivers a telling punch, "putting his body into it." However, the true pivotal position implies that the clavicle is elevated and carried backward to its extreme limit when the arm is elevated to its extreme degree. In other words, the scapula and humerus may move into their share of the pivotal position without the clavicle rising (subordinate pivotal positions), but the arm, to attain complete elevation and the true pivotal position, must be raised to the side of the head.

The scapulo-humeral joint will lock in different positions according to the degree of rotation of the humerus, in a very queer way. (See Figs. 25 and 26.) There can be very little scapulo-humeral abduction with the humerus in full internal rotation; *i.e.*, when the forearm is behind the back, even if clavicle is raised. Starting with your clavicle held down, try elevation of your arm in either internal or external rotation, and then compare the extent of motion when you start with it raised. You will see why authors have differed so much in their estimates of the degree of motion possible in the scapulo-humeral joint. To get the maximum you must perform elevation in internal rotation in the sagittal plane or external rotation in the coronal plane; you cannot interchange. These motions cannot be estimated by putting the scapula of a cadaver in a vise, because in life they vary with the relative positions of humerus, scapula and clavicle.

It is as if the secondary humeral joint, limited by the acromion and coracoid, were built with two long sloping sides meeting at an angle above the head, so that when the arm is raised in any plane it eventually comes to rest in the trough formed at the angle of the coronal and sagittal planes and cannot rotate any more. Nature's command is: "Make any combination of elevation and rota-

tion within this enclosure which you wish, but if you exceed these normal limits, you may dislocate or fracture your humerus."

A logical conclusion from these facts is that *in whatever way a forward or lateral fall occurs, no strain will come on the scapulo-humeral structures until they assume this locked position in complete elevation, provided rotation of the humerus is unhindered as the arm is being elevated.* In other words, when the arm is being elevated in mid-rotation in the sagittal plane and arrives at a little above a horizontal position, if inward rotation be prevented, some structure must give, provided the fall continues in this plane. This is exactly reversed in the case of falls in the coronal plane, for when outward rotation is prevented, some break in bone or soft parts must occur. The latter example is the usual one in dislocation and fracture of the head of the humerus.

Let us imagine the same forces applied in another way, *i.e.*, by leverage on the arm when the body is in a fixed position, for mechanically it is the same problem whether the falling patient exerts force on the outstretched arm, or the fixed patient has the same force applied to the arm. Let us imagine the arm thrust into a pipe and force applied to that pipe as in the following diagrams, by lifting its free end up from the plane of the paper. (Fig. 53.)

In making an effort to remember the combinations of rotation and elevation of the arm which may or may not be made, a simple formula is this: evolution has fitted the human animal to fall directly forward on his face with his arm and forearm in almost any position in relation to his body, but, once he has fallen, his arm and forearm cannot be brought much behind the plane of his body in the position in which he lies on his face on the ground, without in some way raising his body.

We may learn a good deal about the shoulder joint from watching the maneuvers of a wrestler endeavoring to turn his opponent from a face-downward position. These maneuvers are essentially the same as those represented in the diagrams above, that is, they depend on getting the flexed forearm in such a position that a little more leverage would break or dislocate the humerus, unless the prone wrestler does permit his body to be raised. Of all the possible positions in which his forearm and arm can be pulled backward, that in which the arm lies at the side will show the greatest backward mobility, *i.e.*, "dorsal flexion."

In lateral falls, when the abducted arm is held behind the plane of the body, the same dislocating or breaking strains that the wrestler

endeavors to secure may be produced, the force being supplied by gravity. Moreover, falls will be more violent and sudden, and perhaps catch the muscles when relaxed, instead of having the opposing muscles offer resistance by the voluntary tension which the prone wrestler can assume.

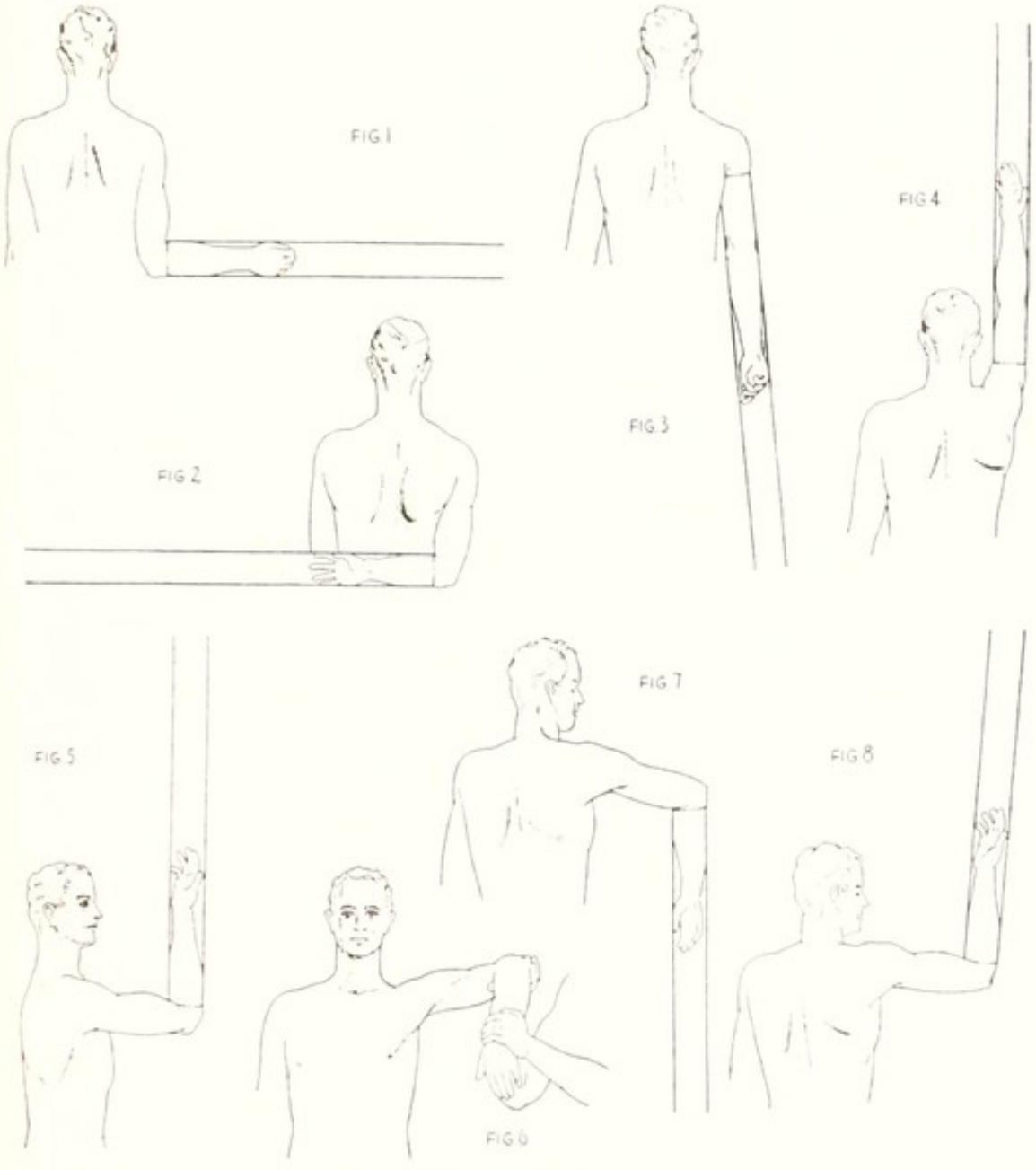


FIGURE 53. LEVERAGES CAUSING ANTERIOR AND POSTERIOR DISLOCATIONS

In interpreting this plate the reader must imagine the arm thrust into a pipe, and he must suppose that the patient's body remains in ironlike rigidity while the outer end of the pipe is raised directly upward from the plane of the paper. Provided the body remained rigid and the tube were raised in this manner, a dislocation of the head of the humerus or fracture must occur. Typical backward or subacromial dislocation would occur in Figures 2, 6 and 7, while subcoracoid dislocation would occur in the other positions.

As in simple frontal falls, so in simple dorsal falls no dislocation of the shoulder will occur, if the arms are free, for we may lie on our backs with our arms in any position anterior to the coronal plane of the body. Even when the dorsal fall occurs with the forearm behind the back or behind the head, dislocation is unlikely.

Thus we reach the conclusion that only headlong, lateral, or semi-lateral, sudden falls can produce the forced elevation combined with prevention of rotation which is necessary to cause anterior luxation of the shoulder joint.

It is possible to conjecture, from a more detailed study of the questions just considered, which forms of dislocation, or varied locations of the lines of fracture, will occur in the shoulder if the lines of force are known. For instance, it is safe to say that only the results of forces applied as in Cuts 2, 6 and 7, in Fig. 53, may result in subacromial dislocation, while all of the others produce subglenoid dislocation.

To express the writer's belief in another way: anterior dislocations of the humeral head occur after the arm has reached the fixed "pivotal position," or more often by prevention of rotation when the arm is at least above the level of the shoulder and the extent of possible rotation therefore greatly diminished; subacromial displacements occur when the arm is below the level of the shoulder and is rotated internally.

This belief is founded not only on the facts already mentioned, but on two others. The first is the remarkable ability of the arm to rotate quickly. Any one who has done any wrestling will remember how elusive were his opponent's arms, unless his elbows were bent, and how easily they slipped out of his grasp as he endeavored to obtain a "shoulder lock." The second is the utter unreliability of the histories given by patients of falls "on the shoulder." The reader is again referred to p. 10. Patients with these injuries almost always say that they struck their shoulders and do not realize that they fell with their arms elevated, *i.e.*, raised to fend off the ground. The inertia of the body is so great as compared to that of the arm, that even in a sudden fall the arm may be thrust forward or outward before the body reaches the ground. A man must already be hugging something, as a football, under his arm in order to fall on his shoulder.

The fact that the arm rotates readily in a safe direction as it is instinctively raised (*i.e.*, raised in relation to the scapula, although pointing downward) during a fall, means that subconsciously the arm

will usually reach the relatively safe pivotal position. Meantime the palm meets the ground and the force becomes disseminated to the various ligaments, muscles and bones of hand, wrist, elbow, shoulder and back, each in turn breaking the fall until the final bony lock in the pivotal position brings the stress to a limit. Even then the pectoralis, the teres major and latissimus, and finally the clavicle, may

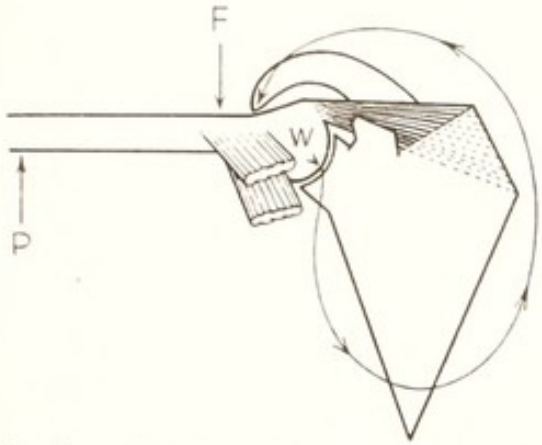


FIGURE 54. ACTION OF PECTORALIS MAJOR IN DISLOCATION

This diagram pertains to the discussion of whether the pectoralis major and latissimus dorsi should be regarded as furnishing a fulcrum in case of fracture of the upper end of the humerus. The contention is made that the pull of these muscles not only does not act as a fulcrum, but to some extent relieves the strain on the surgical neck, by pulling the tuberosities away from the true fulcrum which is the acromion. If these muscles acted as a fulcrum, the power would be applied on the glenoid and would immediately rotate the whole scapula, so that the acromion would become a fulcrum. The writer holds that in such accidents there can be no disruptive force in the region of the head of the humerus unless the acromion does act as a fulcrum.

The diagram further illustrates the fact that the power of the supraspinatus is applied in such a manner as to restrain dislocation, but that in forced elevation of the arm the upper edge of the glenoid acts as a wedge driven in between two points of application of strain. This idea is amplified in Plate IX.

still further disseminate the stresses. But notice that in this elevated position the lines of force of these muscles converge toward the glenoid, so that they are not, in this position, functioning as rotators as they do when the arm is at the side. The higher the axis of the humerus the more their power is exerted toward the face of the glenoid in their effort to avert the leverage of the shaft from gaining a fulcrum on the acromial edge. However, while they are in use in fending off the ground, the pectoralis and latissimus are acting also as strong internal rotators. This is the period of danger (Fig. 55), for the pivotal position, once reached, means safety, unless the downward factor in the fall is great. When the arm is in the pivotal position, the broad tendon of the latissimus actually covers the unprotected axillary portion of the capsule and tends to prevent the head from dislocating, although the pull of this muscle is in the main downward. In this position the long head of the triceps, the teres major and the latissimus actually form a support for the lower half of the capsule.

Many authors have contended that besides the obvious bony fulcrums which occur, there may be others momentarily maintained

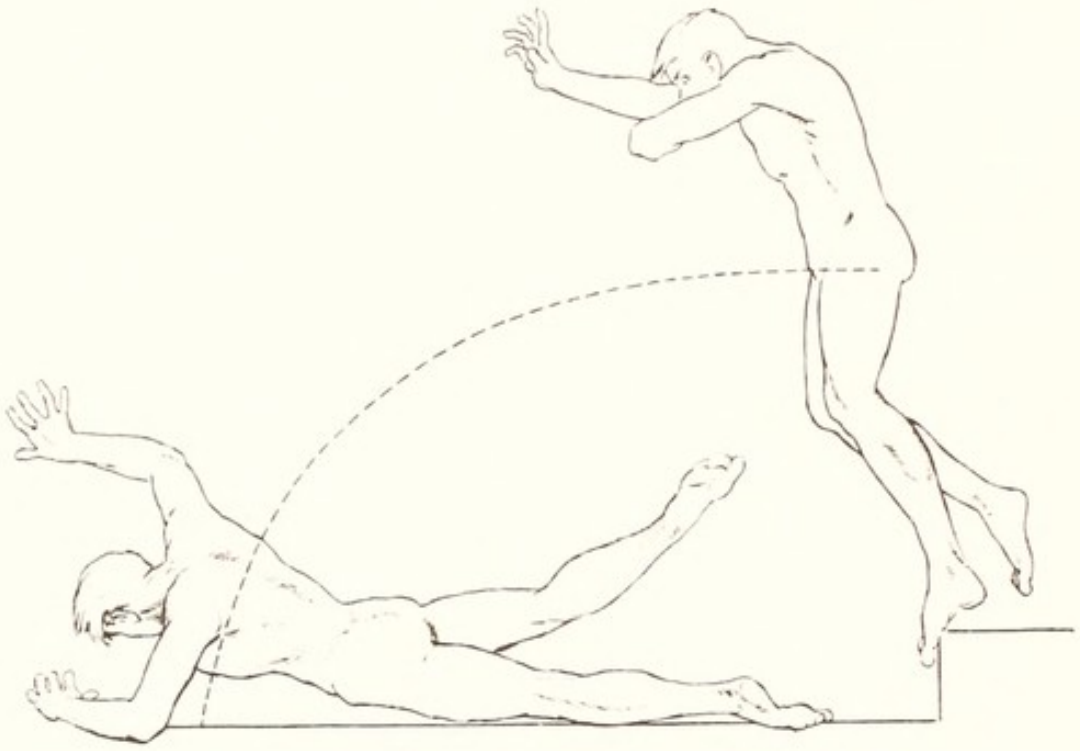


FIGURE 55. TRAJECTORY OF CENTER OF GRAVITY

The center of gravity of a person in falling necessarily always has a trajectory formed by the momentum with which he falls forward, combined with that with which he falls downward, and that which is exerted in relation to the median plane. The writer contends that in most instances, dislocations and fractures in the region of the upper end of the humerus take place when the trajectory meets the ground at or posterior to the point of impaction of the elbow, and also internal to this point. If the trajectory came far anterior to the point of the elbow, the arm would be folded to the side, or if it came far posterior to the point of the elbow, the arm would extend harmlessly beside the head. If it came near the median plane or to the opposite side of it, the arm would slip outward into the hammock position. It therefore seems highly probable that most of these fractures happen when the arm is internally rotated by the pectoralis, etc., and the fall is of such a sudden and violent character that the humerus does not have time to rotate.

If the vertical factor in the trajectory were great so that the patient was falling nearly straight, head downward, fracture or dislocation in the pivotal position would occur. The writer believes that in every case there is acromio-humeral contact, and therefore always a subordinate pivotal position (see Fig. 25).

by contracted muscles, which may lead to fracture or dislocation. Perhaps this is true, but I have been able to work out few such mechanical positions. For instance, the lever formed by the humerus when the arm is in the anatomic position, the pectoralis and latissimus contracted, and outward force applied at the elbow, may be considered either to have its fulcrum on the glenoid or to apply power there, with the pectoralis, etc., as a fulcrum. The latter might be the proper way to consider the problem if fracture occurred in the long arm of the lever, distal to these muscular attachments. It seems more logical to me to regard the applications of muscle pulls as forces acting on the obvious bony fulcrums. However, there can

be no doubt that when these muscles are contracted they would tend to prevent *outward rotation* of the humerus.

I am not in sympathy with the view of those authors who hold that the contracted pectoralis and latissimus act as a fulcrum to promote dislocation or fracture of the head of the humerus. I think the reverse is true, so far as their adductor action is concerned, for I am convinced that this action merely tends to prevent dislocation, since the force is applied to the long arm of the lever distal to the true fulcrum which is the acromion.

Lack of space prevents elaboration of the following additional reasons which support this conviction.

1. The directions of the majority of the fibers of the pectoralis major suggest that their contraction would bring the head toward the glenoid from the very start of elevation, *i.e.*, they would directly oppose dislocation.

In other words, the lower fibers of the pectoralis are inserted higher on the humerus than the upper fibers; thus all fibers of the muscle tend to pull in a line away from the acromion as a fulcrum while the arm is being raised.

2. The combined power of the adductors would not be enough to break the humerus, if both ends of the latter were fixed. Therefore, their power would not be enough to act as a fulcrum for a fracture in the short arm of the lever, although it might be sufficient in the long arm.

3. Even if the power on the long arm of the lever acting through the muscles as a fulcrum, became applied to the glenoid and to the supraspinatus and other opposing muscles, the result would merely tip the scapula so as to apply the acromion as a fulcrum.

4. Until the acromion became a fulcrum no disruptive force could take place between the scapula and humerus.

5. If the humerus touched the acromion at all and the pectoralis, etc., applied their power exactly opposite this point, the leverage exerted on the glenoid would not be changed in any way; nor would it be changed much if the point of application were moved a little away from the neutral point on the long arm of the lever. However, that slight change would diminish rather than enhance the tendency to dislocation.

6. The X-ray shows close apposition of acromion and humerus, when the arms are akimbo, in the salute position and in complete elevation.

7. The acromion is always the fulcrum, although in the above positions different parts of the acromial edge come in contact with different parts of the circumference of the humeral head. (Fig. 25.)

Although I believe that the pull of the pectoralis and latissimus actually help to prevent dislocation by their action as adductors, I am strongly of the opinion that as internal rotators they actually help to promote dislocation. The reasons may be briefly stated as follows: On account of the position of insertions of their tendons, to be adductors, they first have to be internal rotators. As adductors they do their best to fend off the ground until the last possible moment when the acromion has begun to be a fulcrum. Meanwhile, as internal rotators, they are drawing the arm into a subordinate pivotal position and thus prevent external rotation, which is the only method of escape for the arm if it must rise in the coronal plane. Thus we can conceive of their having power enough to keep the humerus internally rotated until it is too late for it to turn, although we cannot conceive of their being able to act as direct adductors strongly enough to withstand the falling weight of the body. If, in any case, they instinctively relax in time for rotation to occur, the arm will rise to the side of the head and no harm will have been done. Occasionally, however, the relaxation is too late, or *vice versa*, the violence is too sudden, and the arm will be caught in internal rotation in or near the coronal plane. It will then be too late for the adductors to relax in order to let rotation occur and thus permit the arm to ascend by the head.

The nearer the bent arm, in internal rotation, lies to the sagittal plane the safer it will be; the nearer the externally rotated arm lies to the coronal plane the safer it will be and *vice versa*. As a matter of fact, in healthy youth it is astounding how rapidly this instinctive rotation will occur. The football player may be hurled headlong by impact with other players in such a way that his body may be twisting laterally as it falls, yet his outstretched arms fend off the ground just long enough to prevent his breaking his neck, and in spite of the sudden, twisting violence, rotation at the last moment usually avoids dislocation of the shoulder.

Yet occasionally the resultant of all the forces of the fall makes the trajectory of the center of gravity strike posterior to the point of the elbow and to its inner side, while the humerus is internally rotated, and anterior dislocation will occur.

If the reader wishes to go into this in more detail he may force his mind to project a combination of Figures 26 and 55. It is

difficult enough to visualize the normal workings of the shoulder joint, but it is still more difficult to foretell the results, on this beautifully adjusted apparatus, of a fall downstairs. Yet I think these general principles usually apply.

On the supposition that our conclusion that the humerus must obtain a fulcrum on the acromion in order to exert a disruptive force to produce dislocation is correct, let us consider what occurs in the inner unit of the shoulder, Figure 8, Chapter I.

The fulcrum on the edge of the acromion obtains its skeletal support, whatever the position of the arm, directly through the clavicle to the sternum. As has been said, not only is upward dislocation of the acromio-clavicular joint prevented by the fact that the clavicular portion of it is superior, but by the strong coraco-clavicular ligaments. Thus the clavicle in any position furnishes a strong radius through which the pressure on the fulcrum is firmly sustained. Moreover, the S shape of the clavicle has been shown to not only withstand great pressure in a line between its two ends, but to have great elasticity when the pressure is released.

I wish to accent again three characteristics of the scapulo-humeral joint.

1. The capsule is necessarily loose.

2. The upper half is muscular and strong; the lower half is fibrous and weak.

3. Since the humerus can rotate many degrees (probably 100+) without moving the scapula, any one of the short rotators may receive the chief burden of the strain according to the degree of rotation when the acromion becomes the fulcrum. The following remarks will be based on the supposition that the supraspinatus is uppermost, as in Fig. 9, but would apply equally well when any of the other rotators were directly opposed to the force moving the elbow.

As the elbow rises upward not only is the supraspinatus contracted but the upper edge of the glenoid becomes a wedge in the reëntrant angle, between the articular cartilage and inner surface of the supraspinatus. This accounts for the frequency with which fracture of the tuberosity accompanies dislocation. In most cases not only the facet on the tuberosity for the supraspinatus will be carried away, but also a concavo-convex piece of bone comprised of the greater tuberosity and part of the lesser, and extending down to the point where the humerus touches the acromion, and including even the bicipital groove as a whole with its tendon intact. (See Plate IX.)

If the force goes no further we shall have a *false dislocation*, for the lower part of the capsule being loose and there being no sup-

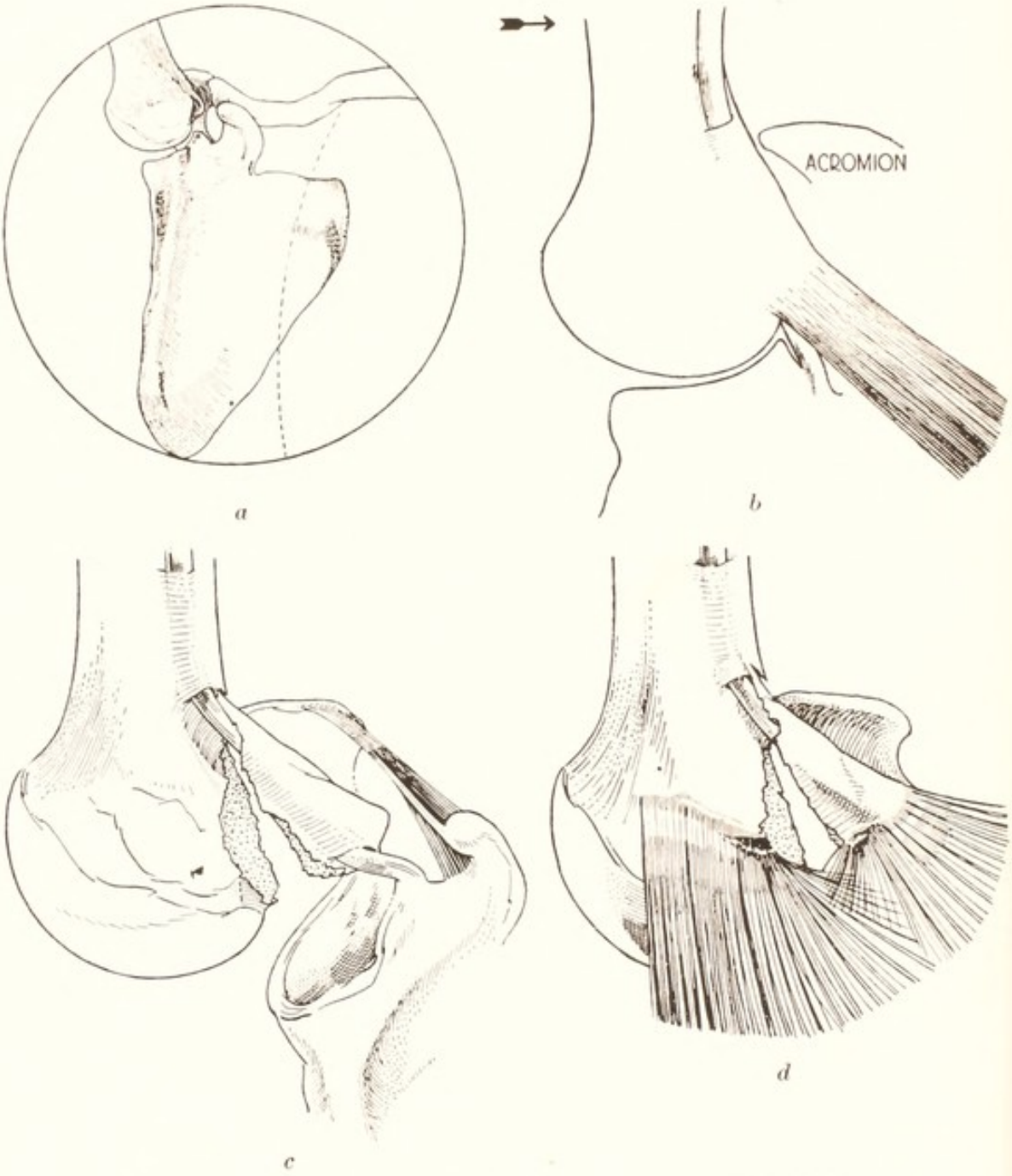


PLATE IX. FRACTURES OF THE TUBEROSITY AND FALSE DISLOCATION

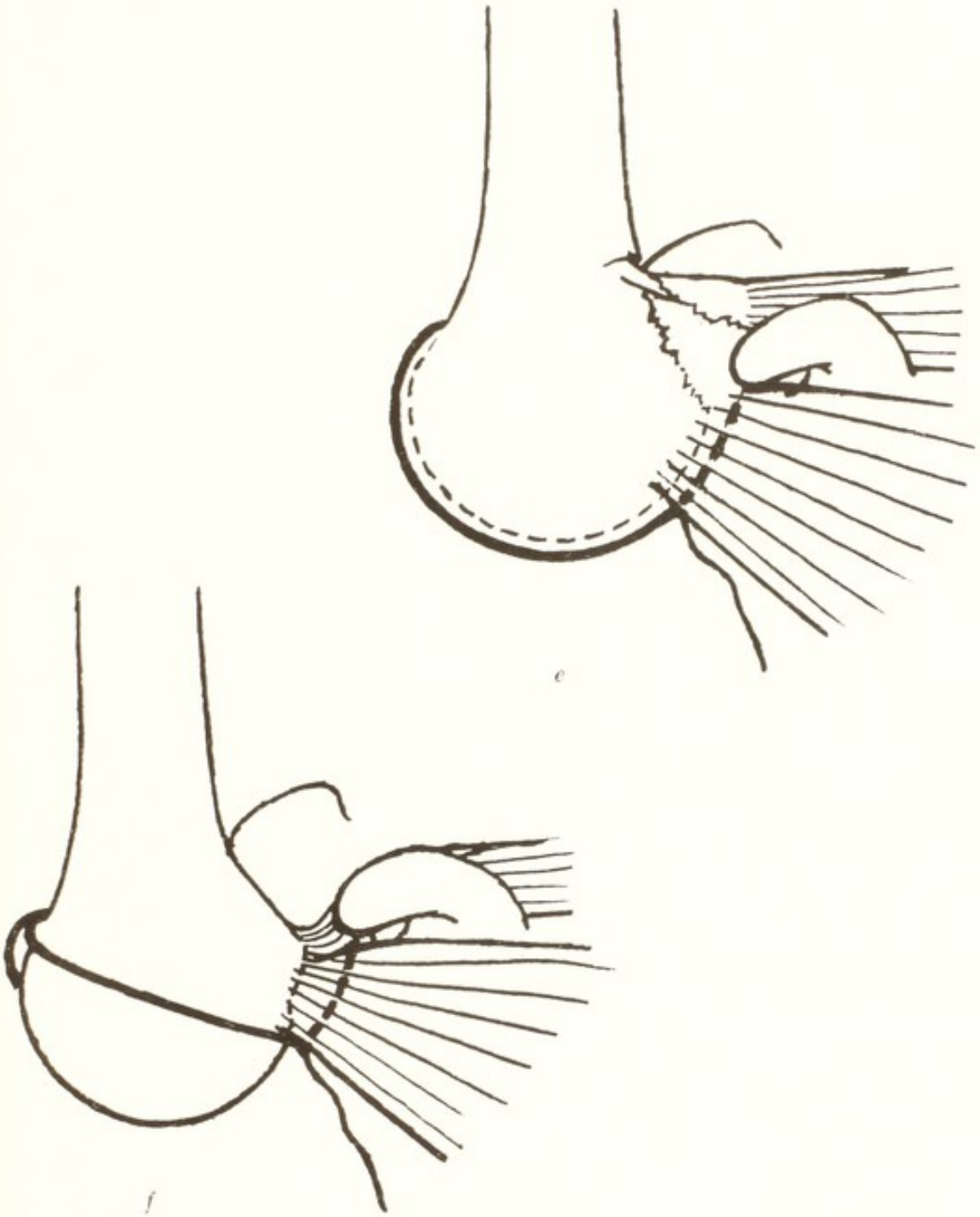
The mechanism of fracture of the greater tuberosity and its relation to false and true dislocations.

a. The pivotal position.

b. When leverage is exerted against the acromion as a fulcrum, the biceps tendon guides the upper edge of the glenoid to enter the sulcus as a wedge, thus tending to chip off the tuberosities. This wedge is not a point but the curved edge of the fibrocartilage backed by the rim of the glenoid.

c. The inferior extremity of the fragment is therefore at the point of impaction of the acromion. The biceps tendon, tuberosity and subacromial bursa remain in their normal relations. A false dislocation of the head may then occur without rupturing the lower part of the capsule because tension is relieved by the rent in the upper portion. The lower part of the capsule, which is normally capacious, will be merely carried beneath the glenoid, as the arm descends.

d. The same lesion with the muscles depicted. A portion of the subscapularis still remains attached to the lesser tuberosity, but most of the greater tuberosity, and part of the lesser, remain in continuity with the fragment.



e-f. Schematic drawings to illustrate the difference between a false and a true dislocation. False dislocation must necessarily be accompanied by rupture of the upper portion of the capsule, together with fracture of the greater tuberosity or rupture of the tendons. There is no structure except possibly the biceps tendon likely to interfere with its replacement, but in a case of true dislocation where the lower part of the capsule alone gives way, the sides of the capsular rent would tend to become tight around the neck of the bone, when efforts are made at reduction. In the worst cases where the two forms are combined an operation is required.

It seems as if some cases must occur in which the biceps tendon would be freed because the line of fracture might extend down the groove and the tendon would thus be separated from the greater tuberosity, and lie between the fragments. I think it usually remains in contact with both fragments if it is not evulsed from the glenoid by the same violence, in which case it retracts into the groove.

port above, the head will glide over the lower edge of the glenoid and fall into the lower part of the capsule, stretching it downward. Correspondingly, the dislocation will be at once reduced as the arm falls to the side, for it will not be pushed through a hole in the capsule and thus have an impediment to easy reduction.

This I believe to be the mechanism in most cases of fracture of the tuberosity, whether the accompanying dislocation is recognized or not. On the other hand, both the bone and the supraspinatus attachment may hold in whole or in part, and be stretched down over the glenoid, until the lower portion of the capsule is tensed and torn and permits the head to slip through it and remain subglenoid, with the torn capsule tense on each side of the surgical neck. This will be the ordinary uncomplicated *true dislocation.*

Violent falls may produce a combination, first wedging off the tuberosity and then driving the head through the lower capsule.

Other variations may be:

1. Instead of the whole tuberosity being wedged off, the supraspinatus may tear away only the facet of insertion.

2. Evulsion of the supraspinatus at the blue line may occur.

3. Rupture of the supraspinatus may take place just above the palisades.

4. Very rarely a lip may be pried away from the lower edge of the glenoid instead of having the capsular attachment give way. I suspect that this is more apt to occur when the forearm is very much rotated internally and the arm is akimbo.

5. Rupture of the long head of the biceps may accompany true or false dislocation, or any of the above variations.

In general, in types 1, 2 and 3, we may expect additional tearing either toward the side of the infraspinatus or toward the side of the subscapularis, according to the degree of rotation of the humerus on the scapula at the time of the fall.

These are the very obvious lesions which may occur, but I believe the most common complication to be the "rim rents" described in Chapter V, which occur not only with dislocation, but in many cases where these structures are just able to resist dislocation, although the synovia becomes separated from the articular margin and a few inner fibers of the tendon are torn.

Another factor which may resist dislocation remains to be considered—the atmospheric pressure which holds the bursal surfaces together. In the typical false dislocation with fracture of the tuberosity, I do not believe that the relations of roof and floor of the

bursa are destroyed. Until air is let into the bursa the surfaces tend to remain in contact. Many a time I have demonstrated this on the operating table. The same is true of the joint. To test in actual pounds the degree of pull required to separate these surfaces remains for some future observer. I feel confident that many pounds of direct pull would be required in the living to separate either bursa or joint to any great extent unless fluid is present. Even when the bursa is opened one cannot pull the joint surfaces apart without undue force unless the supraspinatus is torn, when they fall apart as soon as the air enters. The surface area of the bursa and that of the joint must be very nearly the same, roughly two inches in diameter, each. In X-ray tests one must remember that the cartilages do not show and that the presence of fluid permits separation.

A thorough understanding of what has been said in the preceding pages of this chapter is so important that a summary seems necessary at this point.

1. In spite of the usual histories which patients give of striking on the shoulder, the cause of dislocations or fractures is rarely, if ever, direct, but is usually a backward or downward (*i.e.*, backward and downward in relation to the body as the patient falls) force, acting in the pivotal position, or in a subordinated pivotal position, through the humerus as a lever, with the acromion as a fulcrum, and the weight represented below by the lower portion of the capsule supported by the triceps, latissimus and teres major, and above by the resistance of the supra- and infra-spinatus, the long head of the biceps, and the atmospheric pressure in joint and bursa.

2. During a fall, unless the elbow is maintained in flexion, rotation of the humerus readily occurs; but since no lateral motion at the elbow is possible, fixation of a flexed forearm in a given position may greatly alter the direction of force applied at the shoulder, so that dislocation might occur at a point in elevation short of the pivotal position, but usually above the horizontal. For example, a lateral fall in the coronal plane when the humerus is held in internal or mid-rotation, in such a manner that external rotation is prevented (as by contraction of the pectoralis major), or a somewhat headlong fall in the sagittal plane while internal rotation of the forearm is prevented, might result in fracture or dislocation.

3. It is very unlikely that forward dislocation ever takes place unless the fall is at least somewhat headlong, *i.e.*, one in which the elbow strikes a point anterior to the trajectory of the center of gravity.

4. In most instances subglenoid dislocation must be at first momentarily erect. The descent of the arm into the sling position, in which we usually find it, will be in internal rotation with the head of the humerus still displaced below and anterior to the glenoid, with the subscapularis relaxed and the other short rotators stretched over the glenoid. The long head of the triceps will be between the teres major below and the minor above, and the articular surface of the humerus will face backward on the origin of the long head of the triceps.

5. It seems probable that backward or subacromial dislocation never takes place from forces operating on the arm when it is being elevated, but must occur below the horizontal when the humerus is only abducted to a sufficient degree to permit the flexed forearm to be forced backward behind the body in internal rotation. *Vice versa*, anterior dislocation usually occurs only when the arm is above the horizontal, although theoretically, if the elbow were at the side and the humerus were rotated outward by the flexed forearm, anterior dislocation might occur from a sudden lateral fall which forced the forearm in external rotation behind the body. This would be a very unnatural way to fall, however.

If we accept the above explanation of the mechanics of dislocation, we may proceed to speculate on the reasons why fracture instead of dislocation often occurs from exactly similar falls. Age seems to be the determining factor, but this factor may be subdivided into two secondary ones, *i.e.*, the relative tensile strength of the structures at different ages, and the relative mobility of the bones in youth and in age.

Assuming stress in the pivotal position:

In early youth the humerus ascends high under the acromion, and as the epiphyseal line is relatively the weakest point, epiphyseal separation will probably occur.

As a rule, in youth and manhood after union of the epiphyses, tendon and muscle and bone are strong relatively to the lower portion of the capsule, so that dislocation will take place. If any fracture occurs it will usually be at the greater tuberosity. Occasionally it will occur at the surgical neck.

In old age the trabecular structure about the base of the tuberosities will be weak; therefore, comminuted fracture will readily occur. If the bone holds, dislocation will usually be accompanied by rupture of the supraspinatus, for the muscles and tendons will be weak and cannot disseminate the force.

The second factor, *i.e.*, the extent to which the head of the humerus may pass beneath the acromion, is also important in determining the seats of fracture in youth and in age.

In childhood the tip of the acromion is soft and cartilaginous, and thus the stress on the bony portion of the acromion would be met at about the epiphyseal line, although the head of the bone passes far beneath the cartilaginous acromion.

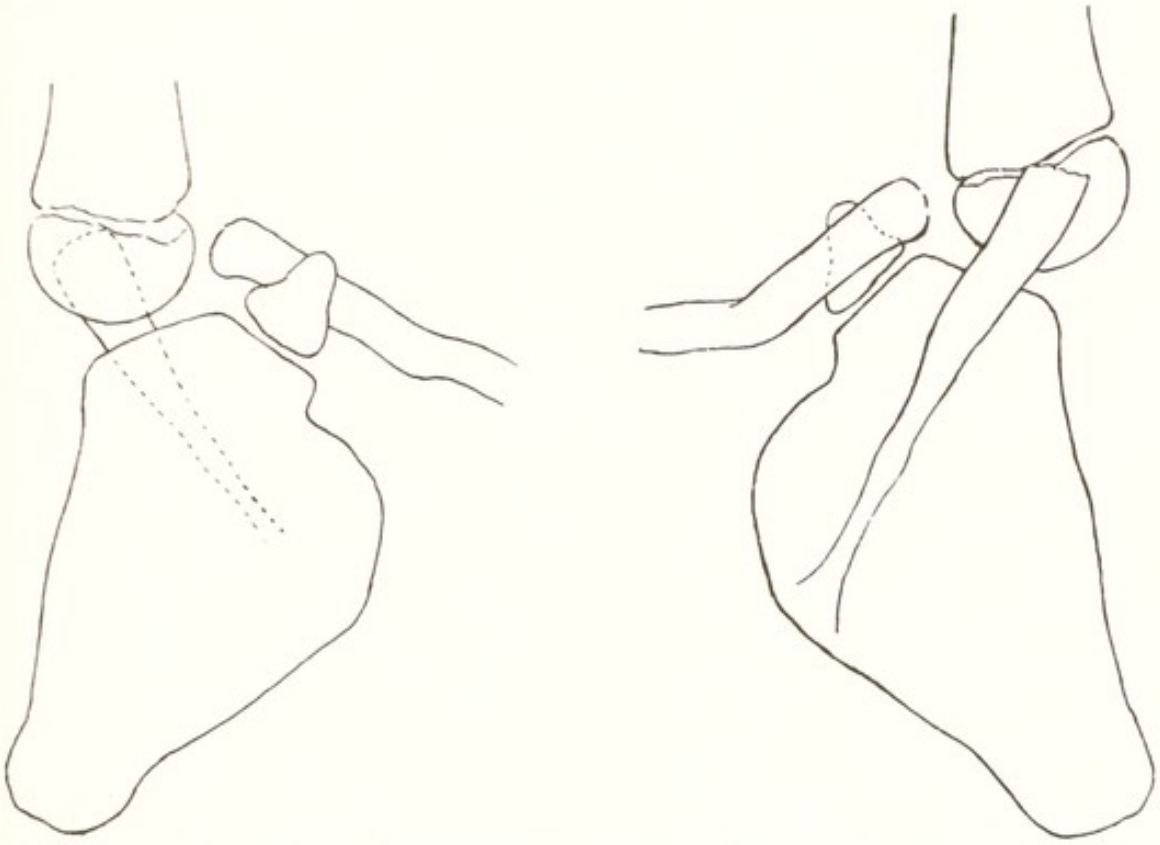


FIGURE 56. EPIPHYSES OF A CHILD'S SHOULDER BONES

Outline drawing from X-ray of child with both arms elevated. By use of a dotted line the figure on the left is made to appear as an anterior view while that on the right appears as a posterior. At this age the tip of the acromion is pure cartilage and does not appear in the film. Notice that the head of the humerus passes beneath the acromion just far enough so that the bony portion of the acromion would gain a point of impact very close to the epiphyseal line. The cartilaginous tip would bend and the breaking force would occur at the epiphyseal junction.

This figure also shows that the line of epiphyseal union of the coracoid is at its base. I have never recognized a separation of this epiphysis. My work has been such that I have seen comparatively few injured children, but I think that it is quite possible that this lesion does occur, and may be detected by characteristic symptoms. It is certainly one of those conditions which we should expect on purely mechanical grounds.

In youth the tuberosity also passes far beneath the acromial edge, and this will bring the fulcrum to bear low down on the surgical neck, just above the attachment of the powerful pectoralis major. Usually dislocation will occur with or without fracture of the tuberosity. Occasionally the surgical neck will give way at the fulcrum.

In the stiff, aged joint, the tuberosity will barely pass beneath the acromion, and impact of the latter will come at the point just below the tuberosities, where the cancellated bone is weak, so that comminuted, intracapsular fracture will usually occur.

It is likely that there may be some changes in the exact lines of these comminuted fractures according to the degree of rotation which the humerus attains at the time of fracture. That is, the acromion will be applied at quite different points on the tuberosity in external and in internal rotation.

Other factors may be important also. For instance, the rapidity of the application of force; the degrees of contraction of the various muscles; congenital or habitual variations in the structure or position of the bones and of other tissues; the weight of the body; many minor circumstances or unusual combinations of any of the above factors. However, the point I wish to make is that the pivotal position is to the human arm in its varied activities as his earth is to the fox. The elusive arm must be driven to its pivotal position to be caught, or tricked by preventing rotation on the way. With continued effort we may always dig the fox out, and with continued backward force we may always break or dislocate the head of the humerus, although the human arm is as clever in evasive rotation as the fox is in doubling.

There are many other points on which a consideration of the "Pivotal Paradox" is enlightening and which are worthy of study, for a thorough understanding of the mechanics of dislocations must be of help in their diagnosis and treatment. However, it will require many studies by many people before practical experience will cease to be our guide. Whether the deductions I have made above prove to be right or wrong, the following practical facts support them and are confirmed by all writers and by each surgeon in his own experience.

The causes of anterior dislocations are usually headlong or lateral falls with the arms thrust forward to fend off the ground.

The same kinds of falls may also produce the following lesions of the upper end of the humerus:

(1) Separation of the humeral epiphysis.

(2) Fracture of the surgical neck.

(3) Fracture of the tuberosities.

(4) Fracture of the anatomic neck.

(5) Comminuted fractures in which the typical form consists of four fragments; *i.e.*, the two tuberosities, the anatomic head, and the shaft. (See Fig. 60.)

Any of these forms may be complicated by concomitant dislocation of the articular head, whether or not it remains attached to the shaft.

The fractures will be discussed in the next chapter and dislocations in the remainder of this one. However, the two subjects are inseparable and one should realize that both fracture and dislocation occur in many cases which are classed as either lesion. I am inclined to think that a combination is the rule, especially in cases of fracture of the tuberosities, and that many supposedly simple fractures are accompanied by false dislocation of the head which immediately becomes spontaneously reduced. Previous dislocation is evident in cases of complete separation at the anatomic neck in which the head remains displaced, but unless the latter is completely separated, it will be dragged back by the tuberosities, which are rarely dislocated because they are sucked back by the bursa and almost invariably remain attached to the short rotators. In other words, these tendons may rupture without the occurrence of any bone lesion, but if one does occur, the tendons remain attached to the fragment. Only small crumbs of the tuberosities ever become really free even in the most comminuted fractures.

The number of shoulder injuries compared to the total number of industrial accidents reported in Massachusetts during eight years is shown in tabular form. This table is not perfectly accurate, so far as the figures on dislocations and fractures are concerned, because it was not arranged for the particular purpose for which I am using it. For instance, all fractures of the clavicle and all fractures of the humerus are included because no distinctions had been made as to the part of the bone injured. It is probable, therefore, that the number of dislocations is fairly exact, but that the number of fractures of the upper end of the humerus is considerably less than the figure given, and probably in every year less than the number of dislocations. Probably many of the cases which were classed as fractures also had dislocations and *vice versa*. Although there is a striking tendency toward uniformity of the numbers through the different years, the proportion of dislocations to fractures varies somewhat for the above reasons. It is fairly safe to say, however, that about one-fifth of all shoulder injuries occurring in industry are fractures or dislocations of the upper end of the humerus. It is likely that if we could get similar statistics from the population not engaged in industry, fractures would be more common than dislocations, because fractures in this region usually occur in elderly

people. Unfortunately, there can as yet be no statistics to determine how often the supraspinatus is injured as a complication of these so-called major (?) injuries, nor what proportion it forms of the other injuries which are unclassified. I believe that it costs our community more than all the other lesions together, not only because it is so common as the *major* lesion in fractures and dislocations, but because there are many unrecognized minor cases.

Year	1923	1924	1925	1926
All Injuries	64,890	60,439	59,079	59,488
Total Shoulder	1,466	1,350	1,288	1,351
Dislocations	180–12.3%	119–8.8%	117–9.0%	142–10.5%
Fractures	173–11.9%	163–12.0%	167–12.9%	149–11.0%

Year	1927	1928	1929	1930
All Injuries	64,167	60,330	60,195	61,741
Total Shoulder	1,438	1,382	1,479	1,470
Dislocations	121–8.4%	126–9.0%	122–8.2%	105–9.1%
Fractures	169–11.8%	148–10.7%	172–11.0%	183–12.5%

The shoulder is the most frequently dislocated joint in the body. This fact is quite properly generally ascribed to its relative instability. Dislocation of the humerus occurred 108 times in 528 cases of dislocation reported by Eliason, and it has been stated by various authors that forty to sixty per cent of all dislocations are of the shoulder. Many shoulders are probably dislocated and immediately reduced by the patient or his companions as they lift him after a fall, without knowledge that dislocation has occurred.

Dislocations of the head of the humerus have been classified in several ways, although for practical purposes only two classes are of importance, *i.e.*, the forward or subcoracoid, and the backward or subacromial forms. As the latter are rare, "dislocation of the shoulder" usually refers to the former.

Forward	Subcoracoid Subclavicular	All essentially the same; they vary chiefly in degree of mobility.
Downward	Subglenoid Subglenoid erect	
Backward	Subacromial Subspinous	Essentially the same.
Upward	Not subglenoid erect	Extremely rare.

The first four varieties in the table are essentially the same. If the tear of the capsule and stretching of the muscle bellies away from their beds are extensive, the dislocated head can be moved easily from a subglenoid to a subcoracoid or to even a subclavicular position. As has been shown, the erect subglenoid position probably occurs in every case momentarily, as the patient falls with the upraised arm, although depression and internal rotation at once ensue from muscular contraction, causing the arm to assume the position at the side in which we usually find it. Very rarely does the erect phase last long enough to be classified!

Subspinous is merely an extreme degree of subacromial dislocation. It occurs when the infraspinatus has been badly torn from its bed so that a large space under the spine of the scapula accommodates the displaced head. Upward dislocation implies an accompanying fracture of the acromion and is nothing more than a curiosity in extremely severe accidents. I have never seen it.

DIAGNOSIS OF ANTERIOR DISLOCATION

In simple subcoracoid dislocation of the humerus, the well-known signs are as follows:

1. History of a trauma to the shoulder in which something was felt to give way and a severe, acute, nauseating pain was experienced.
2. Active and passive movements are limited and painful.
3. The forearm is held flexed and internally rotated.
4. The elbow is away from the side and cannot be completely adducted, thus causing the long axis of the shaft to incline upward and inward.
5. Patient stands inclined to the affected side so as to bring the axis of the humerus to a vertical position.
6. Measuring from the acromion to external epicondyle, the upper arm appears lengthened.
7. The anterior axillary fold (on the affected side) is lower.
8. The shoulder is flattened.
9. The acromial process is prominent.
10. The head is palpable under the coracoid.
11. A soft crepitus can be elicited while manipulating the shoulder.

In other words, the facts to be learned from the history, inspection, palpation, motions and mensuration, plus the aid of the X-ray, will usually make the diagnosis beyond doubt. In fact, the diagnosis is generally so obvious that we must be on our guard not to overlook concomitant injuries and complications. While examining the pa-

tient one should take especial note of the circulation of the arm, and of the sensitivity of the skin over the arm and shoulder. The power of the deltoid, rhomboids, clavicular part of the pectoralis major, supraspinatus and infraspinatus, etc., should be carefully noted with a view to detecting paralysis. When the case is typical, the eleven signs are exceedingly plain, but this does not mean that when they are absent there is no dislocation.

It is very important for the reader to understand that all of the above signs, except the first and the last, fail when the articular head has been replaced on the tuberosity, which has itself been displaced into the glenoid. At this point the reader should refer to the history of Case 71, page 288, and study the accompanying diagrams. Our mistakes in diagnosis will occur in these cases where the head of the humerus can hardly be said to be out of place, but is certainly not in place, for the short rotators with the evulsed facets lie between it and the glenoid. (Fig. 58 and Case 115, p. 389.)

The second symptom was absent in Case 115 because the nerves were paralyzed or so retracted as not to be influenced when the arm was moved. The replacement of the head to nearly its normal position had completely removed the third to tenth symptoms and even the eleventh symptom, in this particular case, was absent or very difficult to detect.

Of course, such cases of pseudo-reduction are not common, but they occur now and then, so that we must never fail to bear in mind that cases of dislocation which are not quite satisfactory may become extremely unsatisfactory, if the supraspinatus or other short rotators are torn, or have dragged the facets toward the glenoid.

Treatment. The main therapeutic principles are: reduction, fixation and gradual return to function.

Reduction in most cases can be obtained by the Kocher or by the Astley Cooper traction methods. These procedures are too well known to describe in this text. The Kocher method is most generally used because much less force needs to be exerted, and hence less trauma should occur during the reduction. However, as Kocher himself said in his original article, it does not always work and it is sometimes necessary to use the Astley Cooper method.

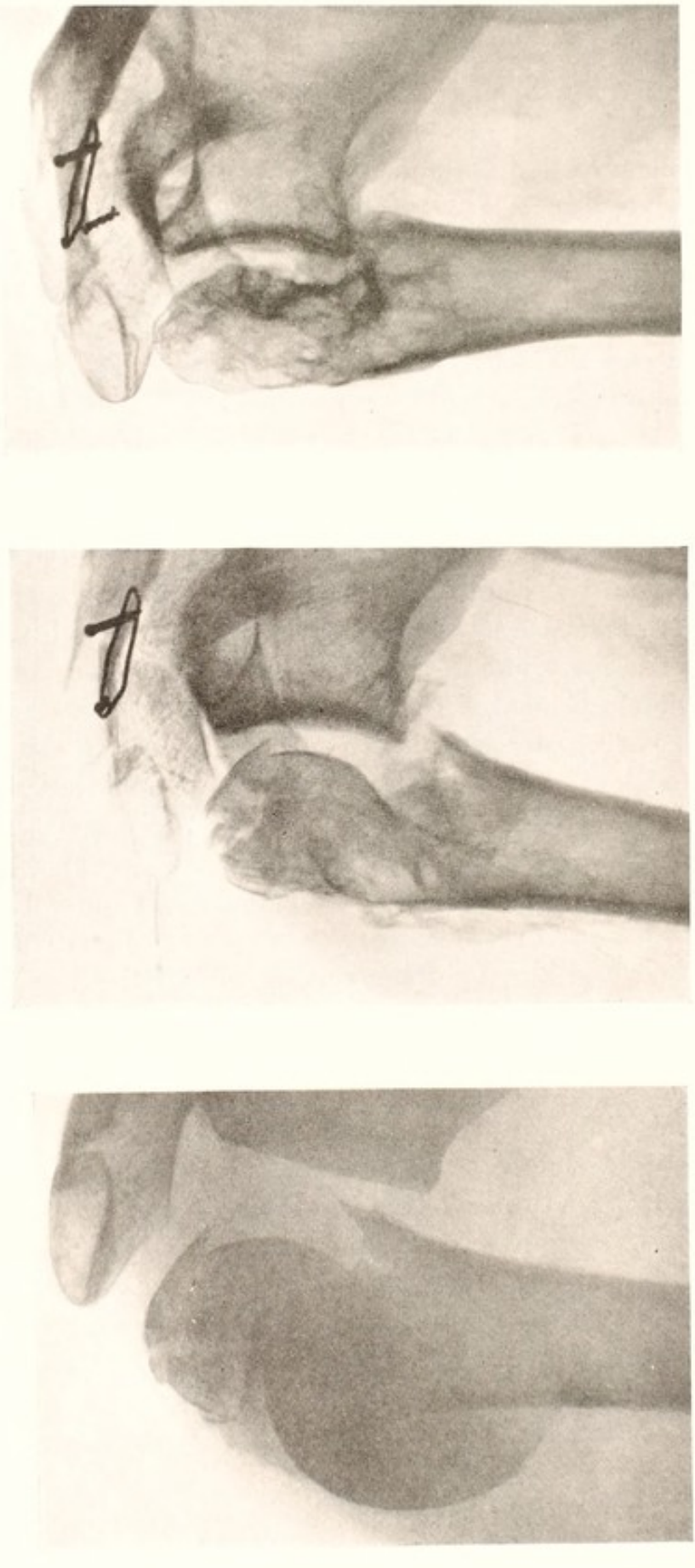
Much has been said concerning the optimum position in which to maintain reduction. This would seem to suggest that the ideal position had not been found. Some modern authors, notably Stevens, are of the opinion that the arm should be put up in abduction and external rotation.

A simple way of obtaining abduction and external rotation is to put the patient in recumbency, and fasten the hand to the head of the bed. A suitable ambulatory splint can be used, or both methods in conjunction. A splint maintaining abduction and external rotation is cumbersome, and attracts a great deal of attention. The ordinary airplane splint abducts, but internally rotates the arm. Abduction and external rotation put the patient in the position of a traffic officer stopping a line of cars. The writer does not advocate this abduction treatment, and prefers not to fix the arm at all.

The advocates for maintaining the arm in the sling position after reduction, contend that the torn capsule heals better because it is relaxed. This seems reasonable, unless the rent is longitudinal, in which case its edges would be approximated better in abduction. It has never been determined positively whether rents in the capsule are usually longitudinal or transverse. Some writers claim that the capsule is torn from the forward edge of the glenoid, and my opinion is that this is the usual condition, for this only means the tearing of the pillars which form the opening of the bursa subscapularis. More observations of cases where death has occurred from the same accident as that which dislocated the limb are much needed. If we knew in general how the capsule is usually torn, the answer to the question of whether to maintain adduction or abduction would be much more plain.

The writer believes that at present there is too great a tendency to confine the arm after reduction. It would seem more logical to let the patient use his arm a little—even to urge him to do so, in order that *débris* and blood clot may work out of the joint capsule into the areolar tissue, where they would be readily absorbed. If the capsule were emptied of the blood clot, it would seem that it would be more likely to have its edges fuse together again without leaving any distortion or undue irregularity. On the other hand, I believe that motion should not be forced for fear of tearing edges which are beginning to unite. For those who think this policy too radical, it would be safer to treat most cases in the sling position than in the abducted one, unless the surgeon were very well versed in the study of the shoulder joint. I think stooping exercises should be begun at once and continued daily in any case. We should use fixation only for comfort, and this means very little, for one may be sure that if there is severe pain after reduction some complication exists.

Prognosis. It would seem an easy thing to make a prognosis in cases of dislocation, but as will be seen shortly, complications are



c

b

a

FIGURE 57. FRACTURE DISLOCATION

FIGURE 57

X-rays of the case alluded to in the text in which the articular head had become displaced beneath the deltoid and was excised through a sabre-cut incision. This patient recovered with a useful arm, and was able to play excellent golf for many years afterwards. Undoubtedly fracture and dislocation occurred at the same time, and the head was left behind in the erect phase of dislocation, when the arm came to the side. The articular surface remained in nearly the position which it occupies in normal elevation. It seems possible that if this fact is recognized in similar cases, reduction might be accomplished by again placing the shaft in elevation, opposing the raw surface of the articular head to that of the tuberosity, and holding them together while the arm is brought to the side *in the coronal plane.*

Figure *a*, before operation.

Figure *b*, a few months after operation. The acromion, which had been sawed across for the sabre-cut incision, has been wired about two steel pegs.

Figure *c*, taken nineteen years later, when the patient was eighty. There had been no trouble from the wire or pegs in the intervening years. At this time motion in the joint still persisted, but was very limited in degree. However, it was sufficient to permit a slight amount of abduction and rotation, which permitted him to use his arm freely for ordinary purposes. Although this operation was successful in restoring a fair degree of usefulness, if I were obliged to operate again for such a condition I should not remove the articular head, and would endeavor to reconstruct a normal joint, perhaps employing Dr. Nicola's suggestion of anchoring the articular head by means of the biceps tendon. Before incising, I should hyperelevate the arm in internal rotation in order to appose the fractured surfaces.

frequent, often unrecognized, and greatly modify the period of disability. A fair prognosis in uncomplicated cases would seem to be a return to normal function in from four to eight weeks. However, complications are, I am sure, more frequent than is generally realized.

Gubler, in a study of insurance records of 252 workmen's compensation cases, states that recovery occurred in 94.1% of the cases after an average of thirty-eight days of treatment. Another investigator, Kuttner, reports a far different experience. He was able to trace fifty-four uncomplicated cases from among 168 treated during the previous five years. Only seven (13%) had regained full use of the limb without loss of strength or motion. In fourteen (26%) the range of motion was complete, but the strength was reduced one-half or more. The remaining thirty-two (61%) had some loss of motion, which in twenty-six amounted to inability to raise the elbow laterally above the level of the shoulder. He states that all of these cases were uncomplicated (which we doubt), and had been treated in the hospital by immobilization for a week, followed by massage and mechanotherapy for two to three months. Goebel reports similar unfavorable results on twenty-four patients. Twelve had full function, but subjective complaints. Twelve had limitation of motion. Lexer reports forty cases; ten with complete restoration, fifteen with some limitation, fifteen with pain and loss of strength.

These reports are self-explanatory, and coincide with my observation of results obtained in general hospitals. The conclusion is

that most of these patients received other injuries at the time of the accident or during treatment. I believe that uncomplicated cases tend to recover in about four weeks, unless interfered with by injudicious treatment, such as prolonged fixation.

Complications. Injuries to other structures about the joint are common and students should be most thoroughly warned of this fact. It is not so much that these complicating lesions are difficult to detect at the time of the first treatment, but that they are not suspected. It is a fact that these complicating lesions do often exist and do maintain disability and pain long after the joint itself has returned to normal, yet there is no organized effort of our profession to correct this condition. Industrial cases may be labelled malingerers, psychoneurotics, hysterics, or cases of traumatic neuroses, because of this prolonged disability with few detected physical signs. The five common complications of dislocation are the following:

1. Fracture of tuberosities.
2. Avulsion of the facets, or rupture of the tendons.
3. Fracture of the glenoid rim of the scapula.
4. Injury to the brachial plexus.
5. Rupture of the axillary artery or of other vessels.

Fracture of the greater tuberosity has hitherto been thought to be by far the most common injury accompanying dislocation. Graessner found the greater tuberosity broken twenty-four times in forty-eight dislocations, but in many of them the fragment was only a small scale of bone, indicating an avulsion of the tendon. Delbet found it in twenty-two out of one hundred and ten cases. Dollinger in five out of thirty-nine. Goebel twenty in forty-three cases. Schlaepfer found it eight times in one hundred and twenty cases, or 7.4%. Gubler reports it in eighteen out of two hundred and fifty-two cases, or 7.1%. Gubler also reports ten cases of other bone injury, and ten of nerve injury in his series. In my opinion, many of the cases where only a small facet is torn away should be classed with supraspinatus injuries rather than with fractures of the tuberosity, because they result in free communication between the joint and the bursa.

The bone injuries are generally easy to diagnose if looked for. The typical signs of fracture, especially ecchymosis down the arm along the biceps, are usually present and the X-ray is of great assistance. The real difficulty is too great a tendency to treat the major abnormality, the dislocation, and to overlook very important, but less obvious, injuries. Sometimes failure to recognize complications

is due to careless X-ray examination, but more often to the inexperience of the doctor who first treats the patient. Rupture or avulsion of tendons attached to the tuberosities should be suspected when the films are negative.

The injuries to nerves and arteries will be considered in Chapter XI.

References of Authorities to the Rôle of the Supraspinatus in Dislocation

Little is to be found in the literature to confirm my beliefs or to explain why the short rotators come to be so frequently injured. Only a few writers have realized the frequency of such injuries, and I do not think that any of them have appreciated the significance of the fact that the bursa and joint are thus put into communication.

Stimson says that the supraspinatus is sometimes, probably often, torn from its attachment to the humerus, and the same is true in less degree of the infraspinatus, and occasionally even of the teres minor. He states also that avulsion of the tuberosities may take the place of laceration of the tendons. Preston says that injury to the capsule is not infrequently accompanied by injury to the tendons which overlie and reinforce it, and when the violence producing the luxation is great, there may be a destruction of tendon continuity.

Stevens gives an excellent description of shoulder dislocation with especial reference to the short rotators. According to him, an anterior dislocation is an impossibility without putting a strain upon the tendons of the supraspinatus, infraspinatus and teres minor. With the humeral head in subcoracoid dislocation, the distance from origin to insertion of the supraspinatus is greatly increased, and in addition the tendon is angled over the rim of the empty glenoid. Similarly the posterior rotators are pulled over the posterior rim of the glenoid, and are almost always injured. "We may," he says, "assume that in every case of dislocation of the humerus, and especially in anterior dislocation, there is an injury to the tendon of the supraspinatus, and that often it is ruptured."

Very few other authors even allude to this tendon.

The following cases as well as the above quotations from the literature support the assertion that the facet of the supraspinatus may be torn off in dislocation.

Case Reports

No. 12. Mr. A. F. K. Age 45. Massachusetts General Hospital No. 174082 W. S., Jan. 23, 1911.

A subcoracoid dislocation of four months' duration. Open reduction

of dislocation. Supraspinatus was found retracted with facet of insertion. Heavy silk sutures to unite it with tuberosity. Feb. 24, 1912: An excellent functional result, but scar is ugly.

No. 22. Mr. J. H. D. Age 69. Massachusetts General Hospital No. 182238, April 22, 1912.

Five weeks ago fell, injuring right shoulder. Reduced by M.D. Went back to work. One week ago dislocated it again when drunk. I made an unsuccessful attempt at reduction in the Accident Room, and then carried the patient under ether to the operating room, and through the usual incision for the bursa I opened directly into the joint. A defect corresponding to the facet of the supraspinatus was found, the supraspinatus being retracted under the acromion. There was no piece of loose bone corresponding to the defect, although the whole joint was carefully searched. It must therefore be assumed that the loose piece had been absorbed. The dislocation was reduced, supraspinatus reinforced with silk and the wound closed. Two years later he wrote me: "I can do quite a lot with it, only when I reach overhead it gives way and causes some pain."

No. 71. Mrs. E. C. Age 68. On June 10, 1921, fell downstairs, broke left wrist and dislocated left shoulder. Ether was given by the local doctor, and he supposed he had reduced the dislocation. She consulted me on August 9, 1921, and after taking X-rays, I reported to the physician who had sent her to me that I thought that the shoulder joint had been reduced. The accompanying X-ray seemed to me to show that the bone was in place. I know now that this appearance is deceptive. In such cases as this, the tuberosity has been retracted into the glenoid and the head of the humerus rides on it. Therefore, there is not very much change of contour in the position of the shoulder, because the total amount of bony substance between the tip of the shoulder and the glenoid is the same, although the position of the tuberosity is reversed and lies in the glenoid. The following is an account of the operation which I did on April 5, 1922, nearly a year after the injury.

Sabre-cut incision. Prominent anterior mass proved to be head of humerus minus the tuberosity. The tuberosity had been retracted by the posterior short rotators and lay partly in the glenoid cavity and partly overlapping its posterior margin. The biceps tendon was displaced so as to lie between the head of the bone and the glenoid. It was excised. The retracted tuberosity was also excised. The synovial and tendinous capsule of the joint was completely gone, except at the anterior edge, namely, the portion formed by the subscapularis tendon. A good, practical result was obtained, *i.e.*, a movable, painless, weak shoulder, lacking power in abduction, but more useful than a painful joint.

It is my belief that in most of these cases of dislocation where the X-ray shows a portion of the tuberosity to be absent, careful pictures will show its presence in the glenoid or just below the glenoid. The capsule forms a pouch below the glenoid and this pouch catches the smaller fragments if they have become loose. The

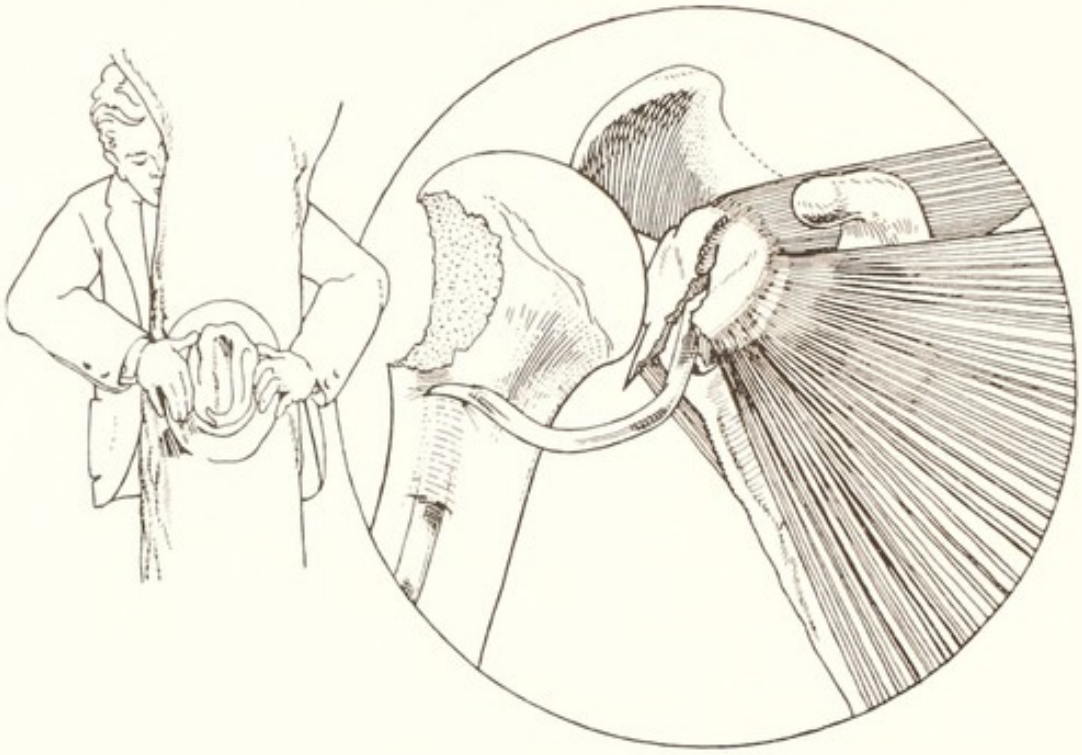
a *b*

FIGURE 58. THE USUAL CAUSE OF FAILURE TO REDUCE A DISLOCATION

Mrs. E. C.—X-ray (*a*) before the local doctor attempted reduction. The fragments of tuberosities may be seen external to the lower edge of the glenoid. The head of the bone is displaced far beneath the coracoid process; the form of dislocation might be classified as subclavicular. It is probable that in this case there was both a true and a false dislocation. After reduction (*b*) the tuberosities seem to be absent, though indications of their fragments are shown near the lower edge of the glenoid. I have operated upon a number of other similar cases (*e.g.*, Nos. 5 and 92), so that I have formed the opinion that when the X-ray after reduction shows the absence of the tuberosity, radical operation is indicated, even if the X-ray does not demonstrate the fragments. It is my belief that this form of displacement accounts for the great majority of instances, which the authors quoted in this chapter speak of, as unsatisfactory results.

cases are deceptive because a reasonable amount of motion may be found within the first few weeks and the position of the head is so nearly normal that it is not realized that the head does not actually touch the glenoid.

Case 115, to be reported later, was also an illustration of this condition. Two of the most alert industrial surgeons whom I know were fooled by the superficial appearance in this case, and I, myself, did not recognize it on my first visit. Even Stevens, whom I regard as having been particularly well informed about conditions in the shoulder, shows in his illustration, "Fig. 4," what I believe to be a case of this kind. He speaks of the fragments as having disappeared behind the head of the humerus. In cases showing X-rays similar to this, I would recommend exploration through the routine



c

FIGURE 58

(*c*) Explanation. Erect dislocation, as usual, preceded the subcoracoid position. The fragments of tuberosity, still held together by the musculo-tendinous cuff, slipped down on the glenoid, while the head was dislocated below and the arm fell to the side. Reduction was attempted and seemed successful, but the head of the humerus merely became superimposed on the fragments so that it seemed that the dislocation had been reduced, and the contour of the swollen shoulder became nearly normal. The biceps tendon was carried with the fragments on to the glenoid. In Case 115 there was scarcely any fracture except at the facets of insertion; the whole musculo-tendinous cuff had dropped back on the glenoid. In most cases the retracted tuberosities are held on the glenoid by the short rotators, as this man holds his hat on the further side of the tree. The biceps tendon may (Case 115) or may not (Case 71) be torn in such cases.

incision of the bursa. If it is obvious that the fragment has retracted into the glenoid, I recommend enlarging the incision to a "sabre-cut," and making an attempt to suture the structures in their normal position.

Before leaving the subject of anterior dislocation, I should like to italicize the following paragraph:

I believe that after the reduction of every case of dislocation of the humerus, the patient should be allowed to recover from the anæsthetic and be urged to move his arm freely, before any bandaging is applied. All motions may be safely performed except abduction in external rotation, and even this may be done with due care and using extension at the same time. If we find paralysis of any of the muscles, areas of skin anæsthesia, undue axillary swelling, gritting sensation in the joint, or a tendency for the joint to slip out of place, the patient should be at once hospitalized and consultation obtained.

(The author here advises the reader to study the next chapter on fractures and then to return to the remainder of this chapter, which discusses some of the more unusual forms of dislocation.)

Subacromial dislocation is rare. I can only recall one case in private practice. This was in a young man who was a personal friend. It is twenty years since I reduced his dislocation, immediately after the accident, and the shoulder has given him no trouble since. Probably most of these subacromial or subspinous cases run as smooth a course, but occasionally, as in the following instance, one proves to need operation.

No. 33. Mrs. C. B. Age 38. Massachusetts General Hospital No. 184814 W. S., Sept. 7, 1912.

A case of recurrent subspinous dislocation which had remained unreduced for three months. "Sabre-cut" incision, the joint carefully inspected and cleaned of old granulations and detritus. None of the short rotators had been ruptured. Dislocation reduced and acromion wired in place. April 17, 1914, she writes, "I am thankful to say my shoulder is all right. It does not seem to be as strong as the other one, otherwise it is fine."

This is the type of case in which the "sabre-cut" incision is particularly applicable, in fact, it is almost indispensable if one wishes to obtain a satisfactory cleaning out of the glenoid. In all these operations for old dislocations I have found the glenoid to be filled with old granulations and detritus. Satisfactory reduction could not have been done without thorough cleaning of the cartilaginous surface.

The second case is an illustration of what I believe to be the usual mechanism of subacromial dislocation. She gave the history that seven years previously the first dislocation had occurred, during a convulsion while she was in labor. Six months later she dislocated it again while "closing a door behind her." The last time it was dislocated by "turning quickly around in her chair."

In the case first mentioned, the young man sustained his injury while being thrown from a sitting posture on a toboggan which had struck a rock. He was thrown in the air—presumably still holding the railing of the toboggan. This might produce the same effect of internally rotating the arm, as in closing a door behind the back.

Old Dislocations

Speed considers a dislocation as old and irreducible after three months.

The obstacles to reduction are the same as for a simple dislocation plus such secondary changes as:

1. Cicatricial contraction from healing scars in the capsule and adhesions to surrounding structures.
2. Displaced bone fragments, osteophytic outgrowths, callus, etc.
3. Muscles shortened and atrophied.
4. Synovial space obliterated.

One can readily understand the difficulty of reducing an old dislocation if he will study the specimens to be seen in anatomic museums. The remarkable attempts of nature to derive some utility from a dislocated shoulder joint make reduction most difficult. At the point where the humeral head lies against the scapula, a tremendous bony proliferation takes place, and inevitably ankylosis or pseudo-arthrosis results. The glenoid becomes atrophied and filled with fibrous tissue.

The varying combinations of pain, deformity, and limitation of motion which these patients present have incited surgeons to attempt relief. Every surgeon of large experience has probably made a few attempts to help such patients, but before long finds that they form a class of cases in which he can be generous to his younger colleagues, who also in time learn by experience. As a matter of fact, these are serious cases and demand *expert* care, although this is not attainable at present, for there are no such experts, so far as I am aware.

There are five general lines of treatment from which we may choose.

(1) To leave nature to do the best she can.
(2) To attempt bloodless reduction under an anæsthetic.
(3) To attempt open reduction.
(4) To resect the head of the humerus.
(5) To perform arthrodesis.

Andrews concluded that an attempt at reduction by manipulation is extremely dangerous. In his words, "The bloodless method has a gory trail of accidents." He finds fifty cases of hemorrhage, mostly fatal, up to 1905. An interesting one was reported by J. C. Warren, in whose case a large aneurysmal tumor arose after attempts at reduction. The subclavian was tied and recovery ensued.

If a new joint can be produced at all, it should be possible to do it by the "sabre-cut" incision, for all the structures of the joint are readily visible and accessible. I have encountered two chief difficulties. One has been the fusion of the retracted tendons and tuberosities with the glenoid alluded to in Case No. 71. If many months have passed, there is little left of the cartilaginous surface of the

glenoid when the *débris* has been removed from it. However, this is not the chief obstacle—the real one being the absence of any synovial membrane to prevent adhesions of the cartilaginous head in its new bed. If the cartilaginous head is not too badly destroyed and seems likely to function, I should advise completing the attempt to make a new joint, but if the head has been eroded and there is no synovia left to surround it, adhesions will surely take place, and almost no motion will be secured. An irritable, painful joint with only a few degrees of mobility will result. In such a case, I think that one should deliberately perform excision or arthrodesis. The method of arthroplasty recently suggested by Dr. Laurence Jones of Kansas City offers a most hopeful solution of this problem.

Recurrent or Habitual Dislocation of the Shoulder Joint

Recurrent dislocation is not a common lesion, but it has interested a large number of investigators. Speed says that this lesion is peculiar to athletes and epileptics. This is more than a witticism, but not entirely true. Almost every author has put forward a somewhat different theory as to the cause of recurrent dislocation, and Speed has gathered together the most important ones. They are:

1. Defect in the head of the humerus acquired at first dislocation, or perhaps congenital.

2. Defect in the glenoid—acquired fracture of the edge, or congenital shallowness.

3. Rupture of the insertions of the external rotators of the head of the humerus.

4. Avulsion of tuberosities with or without rupture of the rotators.

5. Detachment of capsule from anterior lip of glenoid.

6. Enlarged joint from relaxed capsule following tears which have been given insufficient time for strong cicatrization, or repeated stretching without tears.

A seventh theory may be added. Since the shoulder joint is not a real joint, and is dependent for its integrity on a very complicated neuro-muscular coöperation, the essential feature in some of these interesting cases may be a failure of this coöperation. Reference to Plate I will show how easily incoördinated pulls from two opposing muscles might result in instability of the head on its fulcrum. Rupture or stretching of the tendon of the latissimus might thus make the joint unstable in the pivotal position. Certainly from an X-ray point of view the bony structures in most of these cases are normal.

The slight support furnished the joint by the bony structures has been considered, and it would seem more reasonable to suspect a defect in the major supporting structures, the muscles and their tendons, than in the bony structure. The importance of the supraspinatus and of the other short rotators has been emphasized, and it has been noted with what frequency their continuity is broken in ordinary dislocations. More authors have found occasion to mention lesions of the short rotators and tuberosities in recurrent dislocations than in simple dislocations, because the majority of the former cases are treated surgically, so that more opportunity is afforded for observation.

Considerable evidence is given in favor of each of the causes listed by Speed and probably any one of them could well account for a recurrent dislocation. Hildebrand found two cases of fracture of the anterior rim of the glenoid with laceration of the capsule, in which he obtained good results by reshaping and deepening the glenoid. In an X-ray study of twenty-one cases in Hildebrand's clinic in Berlin, Pilz found definite bony defects in the humeral head in fifteen. de Fourmestraux found a deformity of the head in four out of eighty cases. Henderson reported no bone injury found by X-ray in many cases. We have seen several cases in which no abnormality could be found with careful X-ray examination.

According to Stevens recurrent dislocations at the shoulder joint are always due to more or less tearing of the supraspinatus, infraspinatus, teres minor, and more rarely of the subscapularis, and their subsequent repair by scar tissue in a position of stretch. No definite cases to prove this were given.

Thomas concludes that habitual dislocation is due to a traumatic, cicatricial, anterior, hernial pouch of the capsule. The most constant lesion which he found was a tear in the anterior and lower part of the capsule. In an experimental luxation of the shoulder on a cadaver, he placed the head in complete subcoracoid dislocation, and found the supraspinatus, infraspinatus and teres minor not torn or greatly stretched. However, any assumption that such lesions do not occur in the living is unwarranted, for the conditions are so different. In such experiments lax atonic tissues replace the living contractile muscles, and the force is gradually and gently applied as compared to the sudden smashing force acting in the living. A force suddenly applied against active muscles would have much greater rupturing power than a much greater force evenly applied against dead muscles.

One of the earliest mentions of the rôle of the supraspinatus was by Duchenne, who wrote: "Recurrent dislocation cannot occur with a normal supraspinatus." Yet I do not entirely agree with this great authority, for in none of my cases of habitual dislocation have I demonstrated such a rupture, and in two I actually did demonstrate that there was no rupture. Furthermore, I have never seen habitual dislocation complicate a case of ruptured supraspinatus.

Speed states that the head of the humerus twists out of the glenoid through the inferior portion of the capsule, and he believes that to permit this dislocation there must be a great strain on the supraspinatus tendon, or even a tear in it.

It is in the treatment of these recurrent cases that the greatest variations of opinion are to be found. Almost every author has contributed a different technique. The surprising thing is that so many varied methods should produce such uniformly excellent results as are claimed for them, yet the number of methods suggests that none is highly successful. The essential points of some of these methods of treatment will be briefly given with their results, when obtainable. Many of the reports are based upon too few cases followed for too brief a period. It is noticeable that few authors have reported later series in a second paper, and this suggests that the late results and greater experience have not supported them in their early statements.

Treatment.

Two general principles of treatment have been applied to habitual dislocation. They are:

1. Prevention—control primary dislocation, and allow healing of capsule.
2. Reconstruction.
   - A. Suspending head of humerus from above.
   - B. Support from below.
     1. Reefing.
     2. Bone operation on glenoid.
   - C. Combinations of above.
   - D. Use of the long head of the biceps as a round ligament.

A shearing-off of the attachment of the capsule to the fibrocartilage of the glenoid is described by Bankart as the cause of recurrent dislocation. The defect is permanent and his operation aims to repair the rent. His incision runs from above the clavicle downward and outward over the coracoid for about five inches. The deltoid and pectoralis major are separated, not cut, and the coracoid divided and driven downward with the muscles attached. The subscapularis

tendon is divided and the capsule sutured to the glenoidal labium. The subscapularis and coracoid are sutured in place. Four weeks of rest is followed by active and passive motion. Four successful cases are reported.

Carrell, who gives the above classification of treatment, uses an ingenious combination of A. and B. An anterior incision exposes the long head of the biceps. The tendon is sectioned at its lowest level, and reflected from its sheath to where it emerges from the capsule. The distal end of the muscle is attached to the short head. To the free tendon is attached a piece of fascia about six inches long. A posterior incision running down four inches from the acromion separates the deltoid and exposes the teres minor. The fascia is passed under the neck, weaving in and out of the capsule. It emerges just above the teres minor and is passed through a drill hole in the acromion. The arm is immobilized at the side and motion begun in three weeks. Good results are reported in four cases. One wonders whether the posterior incision can be made without injury to the circumflex nerve. Recently Fowler has suggested a modification of this suspension operation. The biceps tendon is not interfered with, but a strip of fascia lata is passed through the capsule below the neck and anchored, both on the acromion and on the coracoid.

A bone transplant was placed by Eden under the raised periosteum of the neck of the scapula so that one-half to one centimeter stuck out in front of the joint. He also reefed the capsule and kept the arm abducted for three weeks.

Henderson's tenosuspension operation has been popular in America, but as seen by the discussion following Fowler's paper, it cannot be a thoroughly satisfactory procedure. Keller uses a crucial plication of the capsule through a posterior incision. Loeffler places a band of fascia from the greater tuberosity of the humerus to the acromion. Mandl used Finsterer's operation with success. The head of the humerus is held back by a band on the anterior surface of the joint, taking the place of the normal joint capsule. The band is composed of "part of the coraco-brachialis and part of the biceps." Oudard divides the subscapularis and overlaps it so as to shorten it about three centimeters. The tip of the coracoid is cut and a bone graft three to four centimeters long is inserted between base and tip. The coracoid may be slit and one-half slid down so as to lengthen the coracoid three centimeters. Nine cases were treated successfully. Six additional cases are reported. Perkins devised an operation in 1906 for suture of the torn capsule and

reports good results. A restraining ligament was made by Plummer and Potts using a fascial strip from the greater tuberosity to the acromion. They report two cases with good results. Nine cases of recurring dislocation were treated in a year by Riviere, who pleated the capsule by placing interrupted sutures through the subscapularis. Landes uses a fascia lata cord or several silk sutures and suspends the head of the humerus by passing this cord through a drill hole and slinging it over the clavicle just lateral to the coracoid. Selig, who considers atrophy or rupture of the external rotators the main cause of habitual dislocation, makes an incision through the supraspinatus fossa, separates the trapezius fibers, and shortens the supraspinatus tendon by plication.

It is Sever's belief that the subscapularis and supraspinatus prevent dislocation when the arm is elevated by opposing the pectoralis major, which adducts and draws the humerus forward, and pulls the upper part of the humerus inward and downward. He says that in all operations for recurrence, the good comes from cutting the pectoralis major and shortening the subscapularis. No results are stated. He notes that repair of the infraspinatus and supraspinatus can also be done at the same time. Other authors, notably Allis, have also held similar views.

The operation advocated by Speed is done through an incision just below the coracoid and extending down four inches. The pectoralis major is divided one and one-half inches from its insertion into the humerus, and the axillary structures put aside. The edge of the glenoid is next palpated, after which, a drill hole one inch deep is made diagonally into the neck of the scapula. A bone transplant from the tibia is driven in with three-fourths inch projecting. This is supposed to prevent the head of the humerus from slipping out forward into anterior dislocation, at the same time not interfering with the normal range of motion.

A heavy silk ligature reënforces the anterior part of the capsule in the operation of Spitzy. A curved incision exposes the deltoid insertion, which is cut and retracted. A heavy silk ligature is passed around the surgical neck and tied in front. The ends are left long so that they can pass upward anterior to the capsule, and be tied over the coracoid. The capsule is then folded over the ligature and sewed, thus shortening the capsule. The deltoid is re-attached, and after a rest period of four weeks exercise is begun.

Thomas was an exponent of the capsule pleating operation. He used a posterior axillary incision, and took a reef in the capsule.

He claimed exceedingly good results, as do the others. He performed capsulorrhaphy on eighteen epileptics suffering from recurrent dislocation. Twelve cases were successful, for a time at least. A capsule pleat and fascial transplant across the front of the joint is performed by Valtancoli, who reports eighty-six per cent cures.

It would seem that a patient stands some chance of cure by any of these methods. Therefore, it would be to the patient's advantage to choose the simplest, since his chances of recovery are as good as if he had the most complicated one. It is interesting to speculate why such a diversity of treatments should produce so uniform a result. It may be explained by the temporary or permanent limitation of joint function consequent to the operation. Some of the above procedures have as an object the limitation of motion, and all of them probably do limit motion. The trauma of the instrumentation, the bleeding and exudate, and the placing of sutures result in the formation of scar tissue. In addition the arm is immobilized for a time. Fixation and a sensitive scar produce pain on motion, and a certain mental impression which results in a patient's using his arm in a very gingerly way for a long time. Actual limitation or voluntary limitation are important reasons why the arm is not soon again moved into extreme elevation. It is highly probable that the late results of all these methods would not be entirely satisfactory.

Some authors have advocated and devised external apparatus to check abduction within a safe limit. These hobbles are a nuisance and offer no cure, but do prevent dislocation. The lesion is annoying and causes so much disability that many of these cases beg for operation. The treatment should be suited to the type of lesion and to the habits of the individual patient. Bony defects should be repaired or modified by plastic methods. Nicola's operation seems to be applicable, whatever the cause.

The writer has on two occasions done negative exploratory operations for this condition by opening the bursa but without entering the joint. The following case may be of interest to those who believe that deformities of the head of the bone may be the cause of recurrent dislocation, for in this patient a thorough exploration was done.

Case No. 24. Mr. K. N. Age 26. Massachusetts General Hospital No. 182833 E. S., May 22, 1912.

A large, powerful young man. First injury six months before while wrestling. Repeated dislocations after slight muscular efforts since then. "Sabre-cut" incision, supraspinatus divided and joint explored. The capsule was found to be torn, especially the portions at the lower and inner

edge of the glenoid. A sort of hernia of the synovial membrane existed, which evidently made a pouch for the head when dislocated. A plastic repair was done on this part of the capsule. Part of the articular surface was missing, as if it had been broken off and absorbed. The patient was seen by me about a year after the operation, and at that time had a perfect result. I have not been able to trace him since.

This particular condition of axillary tearing of the capsule has been described by Thomas of Philadelphia, and considered by him to be the most common lesion in shoulder injuries. I have little doubt that it is a factor common to all recurrent dislocations. One cannot imagine dislocations occurring without a tear of the capsule of the joint. In all these cases the pillars of capsule which bound the opening of the bursa subscapularis must be more or less torn to enable the head of the humerus to lie in the subcoracoid position.

I have seen numerous cases of habitual dislocation, but owing to my lack of faith in any particular technique I have done few operations. I have been content to teach the patient about the mechanism of dislocation and explain to him that he cannot dislocate his arm without combining rotation and abduction. He may abduct the arm as much as he is able to do so in internal rotation in the coronal plane with impunity. These cases are usually in young men, and as has been said before, in athletes or in epileptics. Athletes may avoid dislocation by giving up those forms of athletics which tend to produce it. I have known two young men, who were my personal friends, both of whom were subject to this annoying difficulty. Both patients eventually outgrew it or changed their habits so that it did not occur. One of them, for instance, had to give up boxing because the arm would at times dislocate if he gave a forward blow. In doing this, the relation of scapula and humerus are practically the same as when they are in the pivotal position. He was a skillful athlete at other forms of amusement. It seems to me that for this type of patient it is better to change habits than to submit to operation.

In the cases of epileptics, it is imperative that the patient should wear an apparatus which prevents the combination of external rotation and abduction or should be operated upon. It has not been my fortune to have the care of any such cases. Should I be compelled to operate for this condition, I should at present choose the operation of Nicola. I here reprint some remarks made by Dr. Nicola at the discussion of Dr. Fowler's paper.

"For three years I have been using my own operation, which does not utilize any foreign material. It is done through one incision, the convalescence is short, and so far as I know there have been no

recurrences on operations done by me as well as by about thirty other operators. It seems to me that it is getting more and more difficult for the student of orthopedics to decide which type of operation to use. In the end the operation that will become most popular is the one that is very simple to perform and can be done most frequently on almost any type of dislocation, no matter what the pathologic changes are (bony defects, muscle tears or capsular tears); and, finally, it is a matter of convalescence. In the operation which I have been doing, the line of incision begins just above the coracoid process and extends down and out in the line of the fibers of the deltoid. In my original description I stated that this should come between the pectoralis major and the deltoid, but I find that it is easy to approach it merely by going through the lines of the fibers of the deltoid. Before cutting the tendon, one should be sure to put transfixion sutures in both ends because frequently the arm may extend and the lower segment of the biceps tendon will slip behind the pectoralis and cause great anxiety. After the tendon is divided, a hole is drilled through the head of the humerus. I usually go anywhere along the bicipital groove and point the drill so that it comes out at the upper end of the angle of the head. The tendon is passed through the head and is sutured on itself, so that there is no muscle loss and the tendon has a tendency to restrict the movement and, therefore, keeps the head in the glenoid cavity. Some of the men have used this operation for fracture of the surgical neck of the humerus when there has been downward displacement of the head into the axilla, and there they replace the head in position and drill a hole so that they maintain the head in position."

This operation appeals to me as more likely to fulfill the conditions required than any of the others. Fowler's operation also seems to me a rational procedure, but not so simple as Nicola's.

Extract from a personal letter from Dr. Nicola, June 6, 1930:

"The cases reported in the reprint which you received, together with the rest, have been followed over a period of two years. The boxer has been back in the ring and has won two semifinal contests with no recurrence of dislocation. Case No. 3, which was an epileptic, was killed in an auto accident eight months after the operation. Through friendship with the coroner, I was able to examine the tendon of the long head of the biceps with special reference to the point which you made in your letter. I found that instead of thinning out of the long head of the biceps which extended to the glenoid cavity, the tendon above the humerus was thickened about the size of the little finger."

Extract from personal letter from Dr. Nicola, June 6, 1932:

"I have personally done twenty-four of these operations with one hundred per cent cures. I have not heard of any recurrences from the various men who are associated with me at the Hospital for the Ruptured and Crippled. I hope that you will soon find an opportunity to operate upon such a case and to convince yourself that this operation is very simple to perform. I am now doing it through a two and one-half inch incision taken just below the clavicle on the inner side of the coracoid process and extending downward through the fibers of the deltoid muscle. The hole through the head of the humerus is made with a one-fourth inch gauge, instead of a drill. This facilitates matters considerably."

This operation I am sure could be readily done through my routine bursal incision which separates the deltoid fibers directly over the bicipital groove.

INFANTILE DISLOCATION OF THE SHOULDER

Lesions of the bursa or of the supraspinatus tendon in childhood apparently do not occur, at least they have never fallen within my experience. However, a not very infrequent lesion in childhood, namely, birth palsy complicated by dislocation of the shoulder, sometimes causes confusion in the diagnosis of shoulder lesions in later years. We see the shoulder deformed from a lesion which occurred at or soon after birth.

There has been much discussion as to whether the term congenital dislocation of the shoulder joint should ever be used. It seems very probable that a large number of so-called congenital subluxations, if not a majority of the published cases, were instances of obstetric palsy in which the dislocation remained after the paralysis had recovered. Infantile dislocation may be discussed under three headings, according to whether it existed before birth, occurred at birth or resulted soon after from paralysis caused at birth.

(1) *Abnormality of development—true congenital dislocation.* The existence of such a clinical entity has been questioned, but it has been established without any doubt, although it must be very rare. R. W. Smith of Dublin, in 1839, was the first to bring this condition before the profession. He published an account of three cases in the Dublin *Journal* of that year. Two of these were males, age 20. The dislocation was subcoracoid. The muscles about the shoulder were wasted. One patient had club foot. The third case was an insane woman, age 29, in whom the condition was bilateral.

At post-mortem examination there were facets just under the coracoid, with a displacement of the capsule anteriorly to enclose the joint. The head of the humerus was flattened, and the acromion and coracoid elongated and hooked downward. In all the cases abduction was limited. Eleven years later Smith published two additional cases. In 1841 Guérin presented two cases and demonstrated a symmelian fœtus which showed this abnormality on both sides. Since these reports, numerous cases have appeared in the literature. Grieg analyzed the cases of fifty-eight authors in the Edinburgh *Medical Journal* of 1923. He makes the following statement. "So far I have only found twelve cases reported to date in which evidence brought before the profession fails to justify any other conclusion than that they are cases of true primary congenital dislocation of the shoulder."

(2) *Dislocation produced by obstetric manipulation or during birth.* This group is open to criticism because proof of its existence is not satisfactory. T. T. Thomas was firmly of the opinion that traumatic dislocation not only occurs, but is the primary factor with regard to paralysis. He believed that paralysis is secondary to a rent in the capsule due to a traumatic dislocation. Taylor, writing in 1921, states that neither he nor any of three obstetricians of a large Lying-In Hospital had ever seen a case of Erb's palsy in which the subluxation preceded paralysis. In 1866 Loignan wrote a thesis for a Paris doctorate on the subject. He found that he could not dislocate the shoulder by manipulation. The constant lesion which occurred, if sufficient force were used, was a fracture of the humeral shaft through the soft bone under the epiphysis. Other investigators, notably Sever, have been unable to produce traumatic dislocation or rupture of the capsule by manipulation in infant cadavers. The writer feels that dislocation occurring at birth is so rare as to be negligible in diagnosis.

(3) *Acquired subluxation due to injury of the plexus.* Obstetric paralysis was first described by Smellie in 1768. He thought the condition was due to prolonged pressure on the arm while the child was in the pelvis. It was brought before the profession, however, by Duchenne, who in 1872 described four cases in infants and believed it to be due to pressure on the nerve trunks. Erb described the palsy in adults in 1874. Since that time it has been known as Erb-Duchenne paralysis. Erb believed it to be due to pressure on the fifth cervical root, known as Erb's point. Fieux opposed this view and adopted the theory that traction is responsible. He demonstrated on infant

cadavers the fact that when the head is forcibly drawn away from the shoulder, the fifth cervical root is torn just proximal to its junction with the sixth nerve. With more force, the latter nerve may also be torn. Only the muscles of the upper arm are paralyzed. The palsy is usually flaccid.

Infants with Erb's palsy present a typical history and appearance. There is usually a difficult birth where traction on the head has been made under ether relaxation. The arm hangs limp and vertically at the side, the elbow is extended and the forearm pronated. There is inability to abduct, elevate, outwardly rotate, or supinate. There is extreme internal rotation so that the palm often faces outward. After a short time, there is wasting from disuse, and flattening of the shoulder soon appears. Passive motions are free at first, but the healthy muscles soon contract and produce limitation of motion—notably of external rotation. The muscles paralyzed are the deltoid, supra- and infra-spinatus, teres minor, biceps, brachialis and brachioradialis. There is no sensory disturbance.

Clark, Taylor and Prout estimated one case of palsy in 2,000 births. Most cases of birth palsy show some luxation of the head of the humerus. Fairbanks saw twenty-eight subluxations in thirty-seven cases of palsy, or seventy-six per cent; Thomas reported nine in twelve. Taylor has reported sixty-eight cases, forty-six of which showed subluxation. He has never seen the condition in patients less than six weeks old. Of those patients who had palsy, who were more than six weeks old, seventy-seven per cent showed subluxation. In 109 X-ray studies reported by Sever in 1916, sixty-four or fifty-nine per cent showed subluxation. The ages in this series varied from one day to eighteen years.

It should be noted that this form of dislocation is posterior, while the first two forms are anterior or inferior.

Mechanism of Posterior Subluxation. The appearance of the arm soon undergoes a change from the flaccid condition which follows the immediate injury for a few weeks after birth. The upper arm is brought forward and adducted by contraction of the well muscles. The elbow is usually a little flexed, the pronated forearm passing downward and inward across the front of the body. Abduction is checked before the arm comes to a right angle. Backward motion is usually impossible and external rotation is markedly limited. With the elbow at the side, it is often impossible to rotate the humerus out sufficiently to bring the forearm into the forward plane. The bones of the affected side—the humerus, scapula and clavicle—

are smaller than those on the opposite side. This is an important point in the diagnosis of late cases. The front of the shoulder is flattened, while there is a fullness behind, below the acromion, due to the head of the humerus. The muscles which tend to prevent posterior dislocation are the teres minor, supraspinatus, infraspinatus, and posterior part of the deltoid. The pectoralis major, the teres major and latissimus dorsi are rarely even partially paralyzed and rotate the humerus strongly inward. The subscapularis acts as a powerful internal rotator. These muscles become contracted while the paralyzed ones are stretched. The teres major and latissimus dorsi exert traction downward and posteriorly. The anterior portion of the capsule becomes shortened secondarily. Since the arm is held in an internally rotated position, there is a constant strain on the neck of the humerus, tending to twist it backward. In the plastic young bone, this twisting occurs and the plane of the head of the bone to the shaft may be changed ten or fifteen degrees. The dislocation is not truly posterior—it is rather a rotation of the head than a dislocation, so that the articular portion is rotated backward and the side of the head lies on the glenoid without having escaped from its capsule.

During the first year nothing can be seen in the way of bony deformity in the X-ray, except possibly a slight posterior subluxation and relatively small size of the head compared to that of the other side. As the child grows older, the subluxation increases. There is increased outward displacement and elevation of the scapula. The acromion and coracoid become hooked downward in front of the head of the humerus. The clavicle is shortened and its curves are more marked than in the normal. The coracoid process is usually elongated. The glenoid becomes shallow. These changes of the bones occur while the paralysis exists, and even if the paralysis clears up, they persist.

Treatment. In early cases the arm may be held in abduction, elevation, external rotation and supination by a light wire splint. Sever, however, prefers to let the child use the hand and arm freely with the risk of contractures. Passive motion and massage are carried out at frequent intervals. Operation is deferred until the child is three or four years of age. Once contractures have developed, it is best to cut the contracted muscles. Manipulation is of little value because of the tendency to recurrence, unless the paralyzed muscles have regained their tone. Sever has described his operation at length in a paper read before the Section on Orthopedic Surgery of the American Medical Association, 1925. He cuts the pectoralis and

subscapularis tendons and removes one-half inch to three-quarters inch of the tip of the coracoid with its muscle attachments. If necessary he removes the hooked acromion to allow reduction. The arm is put up in a light splint in abduction, elevation, external rotation and supination. Muscle reëducation by active and passive motion is begun after eight to ten days. The splint is worn night and day for three months, but is removed daily for exercises.

Prognosis. This depends largely on the recovery of the paralyzed muscles in early cases. If the child is seen early, contractures and subluxation may be minimized by passive motion until the nerves have regenerated. In late cases, however, where contractures have developed and there is no tendency of the nerves to regenerate, operation has to be resorted to and gives a good, but not brilliant, functional result. Sever records partial recovery in 297 of 394 cases which were operated upon. After operation there is great difficulty in inwardly rotating the arm. He states that this may be eventually overcome, however.

I have had very little personal experience in these cases, but the following case taught me such an important lesson that an account of it is presented in the hope that it may stimulate some one to follow up a series of such cases in which operations had been done in childhood.

CASE 70.

A strong boy, age 14, was referred to me for trouble with his shoulder, with the story that he had had difficulty since he was a baby, and that while he was a very strong, active boy otherwise, he was greatly handicapped by his shrunken, weak, deformed right arm. The diagnosis in the case puzzled me a good deal at first, for there was no paralysis, and the X-ray simply showed a rather small and deformed humeral head and glenoid cavity. In spite of this, by palpation, it was easy to feel that the head of the humerus was posterior to the glenoid under the acromion. Both the coracoid and the acromion were hooked down in an unnatural manner, which was easy to understand when one recognized the true character of the lesion, for the acromion process had nothing beneath it and was not performing its normal function. All parts of the shoulder, including the bones, were smaller than those of the opposite side. There was practically no motion in the scapulo-humeral joint and the arm was held in internal rotation. An extraordinary amount of compensatory mobility had developed in the motion of the scapula on the chest wall—not only in flexion and extension, but in abduction and adduction.

It was clearly a case of birth palsy in which the bones had remained out of place after the muscular paralyses had recovered, and therefore the patient now only suffered from the consequence of the luxation.

I operated on June 16, 1921, through a "sabre-cut" incision, and to my surprise, found that the cartilaginous surfaces of the glenoid and of the head of the humerus had remained in nearly a normal condition,

although they had been separated for so many years. The capsule was stretched and distorted, but not disrupted. The cartilaginous surface of the glenoid was rather puckered and the articular head of the humerus was small and rather misshapen, although less so than one would think from the appearance in the X-ray, which of course showed the surface of the bony centers of the epiphysis and not the cartilage. The contraction of the subscapularis and pectoralis major was so great that I had to divide their tendons in order to get the head of the bone in place. The coracoid process was so deformed that I had to excise about two-thirds of it subperiosteally in order to reduce the humerus. The "sabre-cut" incision, of course, mobilized the acromion. After reducing the head of the bone, I was able to put four silk sutures "à-distance" to reunite the cut ends of the subscapularis. I did not attempt to correct the torsion of the neck, although it caused some eversion of the arm when the head of the bone was in place. The whole wound was sutured, and the patient was put up in plaster in semi-abduction and semi-external rotation.

It was most remarkable to watch the boy's convalescence. There was a good surgical recovery, but the miraculous part was to see the promptness with which the bones and muscles tended to grow into normal condition. Within six months he had nearly normal use of his shoulder, and the condition of the muscles had greatly improved.

As the patient lived in another city I lost track of him about six months after the operation and did not see him again for ten years, when I looked him up in preparation for this book. It is fortunate that I did so, for the lesson which I learned was important and may be of help to others.

During the ten years that had passed, the boy had become a man of twenty-four, whose occupation was in moving buildings. He did much of the manual labor himself. When he came into my office he looked strong and vigorous, and I had to ask him which shoulder was the bad one. Then my disappointment came.

A skillfully made movable pad inside his coat concealed the small size of his right shoulder. His vigorous handshake and well-developed forearm gave no hint of the practically ankylosed joint which was displayed when he removed his clothes. On closer examination I found both coracoid and acromion clutching down on the head of the bone like crooked fingers grasping it and holding it fixed. It appeared as if the acromion process had bent downward from the point where I had divided it and had become fixed in this position.

After a time the explanation occurred to me. At fourteen, the acromion and coracoid are cartilaginous epiphyses, for they have not yet ossified. After my operation, although the head of the humerus was in place, it was small and underdeveloped because of imperfect function for fourteen years. Consequently the cartilaginous acromion and coracoid became bent down over it, grasped it, and then, at about twenty, turned to bone and held it fixed. I did not at the time of the operation realize that the greater part of the acromion does not unite with the spinous process until about twenty. I think now that if I had insisted that this boy should have slept with his arm elevated until he was twenty, and had kept up appropriate exercises, he might have obtained a more per-

feet shoulder. It is not a great hardship to form the habit of sleeping with the arm in the hammock position. Many people do this by preference. He had been able to hold his arm in this position when I last saw him six months after the operation. Later the strength of the divided internal rotators returned and tended to rotate the arm to its old position, and also to resist elevation in external rotation. The head of the bone being small, the cartilaginous acromion slowly yielded to fit over its convexity. I think that it is highly probable that if the late results of other operations which have been done for these cases were critically examined, the same disappointing condition might be found in later years. Operations should not be undertaken on these cases unless the parents are warned that the patient should be under the surgeon's care until his epiphyses are united; *i.e.*, when the patient is about twenty. I am inclined to think that nature's results at the end of ten years would compare very favorably with those of surgeons.

ACROMIO-CLAVICULAR DISLOCATION

A brief consideration of lesions of the acromio-clavicular joint seems advisable, although strictly speaking, neither the subacromial bursa nor the supraspinatus tendon are involved. One must remember that the coraco-acromial ligament intervenes between these structures. When this joint is dislocated the coraco-acromial ligament goes intact with the scapula. In severe cases the coraco-clavicular ligaments (conoid and trapezoid) are torn.

Mechanism. The acromio-clavicular joint, itself, is weak, but the conjunction of the two bones derives its strength from the conoid and trapezoid ligaments which are attached to the coracoid process of the scapula. Upward dislocation of the clavicle is favored by the upward and outward slope of the joint. The clavicular facet looks downward, outward, and backward. Dislocation is almost always caused by direct violence. A blow on the back of the acromion or a fall on the tip of the shoulder drives the acromion downward, inward, and forward, and the clavicle with the coracoid process as a fulcrum is torn away.

Dislocation is classed as complete or incomplete according to whether or not the facets clear each other. The ligaments of the joint itself are more or less torn, even in incomplete dislocation, but the conoid and trapezoid ligaments are usually torn in complete dislocation.

Diagnosis. The diagnosis is to be made from the history of trauma, pain and disability. The outer end of the clavicle is prominent and movable, and can be readily reduced, but reduction is hard to maintain if the coraco-clavicular ligaments are ruptured.

Treatment. Upward, outward, and backward traction on the scapula is indicated and can be applied by various braces or by

recumbency. The most favored method is the clavicular cross. A T-shaped splint is applied to the back, and the shoulders strapped to the cross arms.

Open reduction and fixation are occasionally necessary. Several operations have been devised, most of them using fascial strips. Representative among these may be cited Bunnell's ingenious method. He threads a cord of fascia through holes in the acromion and clavicle, and places a loop under the coracoid process.

Prognosis. If the reduction can be maintained, the chances are good that a patient will regain function in the course of several months, but soreness and some pain may persist for years. Arthritic changes may take place. I personally have never found it necessary to operate on acromio-clavicular dislocation, but in extreme cases I should recommend Bunnell's operation.

ACROMIO-CLAVICULAR ARTHRITIS

In contrast to the mechanism of the scapulo-humeral articulation, that of the acromio-clavicular joint is typical of the kind in which arthritis is prone to occur. It is a hinge joint with a very limited degree of motion. For its size, when in action, it carries an immense burden of weight. For instance, as one pushes open a heavy door, this little joint has to bear the equivalent weight of almost the whole power exerted. The same is true of the joint on the sternal end of the clavicle, but that joint has a much larger surface area. In laboring men who carry or lift heavy burdens, spurring from arthritis of the acromio-clavicular joint is so common as to form almost as normal a tissue as the great calluses in their hands. In many of the older individuals, the rims of new-formed bone actually fuse, so that the joint becomes obliterated. These changes may progress with very little pain and discomfort to the individual, or they may be accompanied by the usual signs of arthritis, which occasionally are so severe that they incapacitate the patient.

The acromio-clavicular joint holds an exposed position on the shoulder, and falling objects not infrequently strike the joint directly—perhaps breaking some of the small bony lips, causing hemorrhage about or in the joint. Such cases may be laid up for months on account of the local tenderness when they attempt to use the arm. I find it very difficult, in giving an opinion on compensation cases, to state when such individuals should be expected to return to work. The X-ray appearance is far from being a criterion. A man may have a very ragged and hypertrophied-looking joint and yet be conscious of no symptoms. Another man with barely perceptible changes may have much local tenderness. On the whole, it is

surprising how well these laboring men are able to bear acromio-clavicular arthritis. The diagnosis of these lesions rests entirely on the readily ascertained facts of localized swelling and tenderness, supported by the X-ray evidence of lipping of the joint. Confusion in diagnosis only arises when one allows one's self to center his attention on this joint and to ignore other really more important lesions. Never forget that this condition may attract your attention too readily, and by its presence conceal a lesion of the supraspinatus which is far more serious. Acromio-clavicular arthritis must be judged by the degree of symptoms, not by its X-ray appearance, for often there is much lipping of the edges of these joints and yet no symptoms at all.

Treatment. As a rule, these cases respond well to rest. A few weeks' confinement of the arm in a sling soon after a bruise on one of these joints is all that should be attempted. I am convinced that complete fixation of this joint is unfortunate. I do think that rest is very important in acute stages. Patients who have had prolonged symptoms have usually had either prolonged energetic treatment or prolonged fixation.

In a few subacute and prolonged cases, I have cut into the joint with the same idea that one has in cases of periostitis, where one incises to cause relief of tension. As a rule, however, I believe that simple rest is the only form of treatment which is important. The usual physiotherapy methods may be of some use, but my personal experience with them has been little.

Remember that the acromio-clavicular joint is not in immediate anatomic relation with the subacromial bursa. The coraco-acromial ligament intervenes. Therefore, acromio-clavicular arthritis does not prevent rotation or abduction in the scapulo-humeral joint, except in extreme positions. If these motions are not present, do not blame the acromio-clavicular joint entirely, even if it is swollen and tender.

Arthritic changes in this joint commonly occur after luxation or subluxation, and soreness may continue many months and sometimes a few years after such accidents. Men who do their own work generally continue at it in spite of this protracted soreness, but employees are apt to find their shoulders too sore to permit labor, if their compensation is paid. Thus this little lesion may be the cause of their never working again, for it is a commonplace that a year of loafing is a serious matter for an elderly laborer. Surgical obliteration of the joint should be considered, in some cases at least, as a mental stimulant. It is not an important joint.

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NOTE—Sinz (1932) gives a very extensive Bibliography. He recommends a bone transplant to the glenoid (after Perthes or Eden) but apparently was unaware of the work of Nicola.

Chapter X

FRACTURES IN RELATION TO THE SUBACROMIAL BURSA

I do not intend to describe in detail the diagnosis or treatment of fractures about the shoulder, but to state briefly certain points which are suggested from a study of the bursa and tendons, and their probable relations to the fragments in the various types of bone injury.

It is an interesting fact that the acromion process, although so obviously exposed to any blow in this region, nevertheless is rarely broken. One reason is because the scapula is loosely attached to the chest wall and has no direct connection with the rest of the skeleton, except through the articulation at the inner end of the clavicle. A direct downward force striking on the acromion process meets little resistance, for the whole shoulder yields unless the line of the force is transmitted in whole or in great part to the clavicle. As a rule, downward forces acting on the acromion tilt the lower angle of the scapula inward and the blow slides off the shoulder, leaving only an abrasion of the skin at the point of impact. The acromio-clavicular ligaments may be ruptured if the acromion does not thus elude the full drive of the blow.

In the case of most blows or falls on the shoulder the scapula will merely be knocked downward or to the side in one direction or the other, and the strain of the blow will be taken by the muscles which hold the scapula in place. If the line of force is nearly or directly in the line of the axis of the clavicle, the latter will be broken or its ends will be dislocated. The great exception to this rule that the whole shoulder will yield before the acromion will be broken, is when the nature of the downward force is direct and also sudden, as from some heavy object; *e.g.*, an iron bar striking the acromion. In such a case, the shoulder as a whole does not have time to move, so that the acromion or its base may be broken directly beneath the object with which the blow is struck. Such fractures, although often comminuted, are not usually of a complicated character or difficult to treat, because even though the inertia resists most of a sudden blow, a certain amount of yielding of the whole shoulder takes place and the fragments are not driven far out of position. Also there is much fibrous tissue to hold the fragments together. The bursa is in

direct relation with part of the under surface of the acromion and might fill with blood, but I have never opened the bursa in such a case and demonstrated hemorrhage.

Injuries affecting this region by indirect violence (as in hyper-abduction of the arm in falling) very rarely injure the acromion, or even dislocate the acromio-clavicular joint, although the latter is obviously weaker than the bone of the acromion. This is probably due to the fact that the clavicular part of the joint lies above the acromial part. When the violence is indirect it must be transmitted through the acromion as a fulcrum, yet the latter does not break. As Stevens says, the edge which receives the strain is built like a Roman arch.

The coracoid process is also a very firm piece of bone and rarely is affected by any kind of violence, direct or indirect. Fracture of its base in conjunction with fracture of the scapula is not very uncommon.

The outer end of the clavicle does not lie in direct relation to the subacromial bursa, for the coraco-acromial ligament is between them. This portion of the clavicle is seldom fractured, except by direct blows, although fractures often occur at the junction of the middle and outer thirds.

In all forms of violent injuries to the shoulder, the upper end of the humerus is the part most commonly injured, because its structure is much less firm than that of the acromion and coracoid, and because of the leverage which may be exerted by the shaft of the humerus. The long arm of the lever of the humerus applies the force with the acromion as a fulcrum in the neighborhood between the surgical and anatomic necks of the bone, which necessarily receive the strain when the humerus is abducted or elevated. The upper edge of the glenoid, to which the biceps tendon guides the tuberosities, acts like a wedge to separate the tuberosities from the articular head. (Plate IX.) The acromion acts as a fulcrum applied to the region of the neck at the base of the greater tuberosity. The articular head of the bone is then either dislocated or broken off at the sulcus. Often it suffers both injuries to some degree. (See Fig. 57.) If the articular head becomes free and remains displaced, the injury is called a fracture-dislocation.

Reference to Fig. 60 shows that the epiphyseal line is retained in adult life as a thin wedge-shaped subdivision, marking off the tuberosities and the anatomic head from the diaphysis. The lines of cleavage of most fractures in this region follow near these old

lines of epiphyseal union, and the head of the bone tends to become divided into four main fragments, or various combinations of these four fragments.

The tuberosities break off from the shaft at or near the transverse epiphyseal line, and the two tuberosities are also frequently partially separated by a line of fracture down the bicipital groove, this line representing very nearly their former vertical epiphyseal union. The fragments of the tuberosities usually remain in continuity with the tendinous insertions of the short rotators which are attached to them. The articular head forms the third unit and the shaft the fourth.

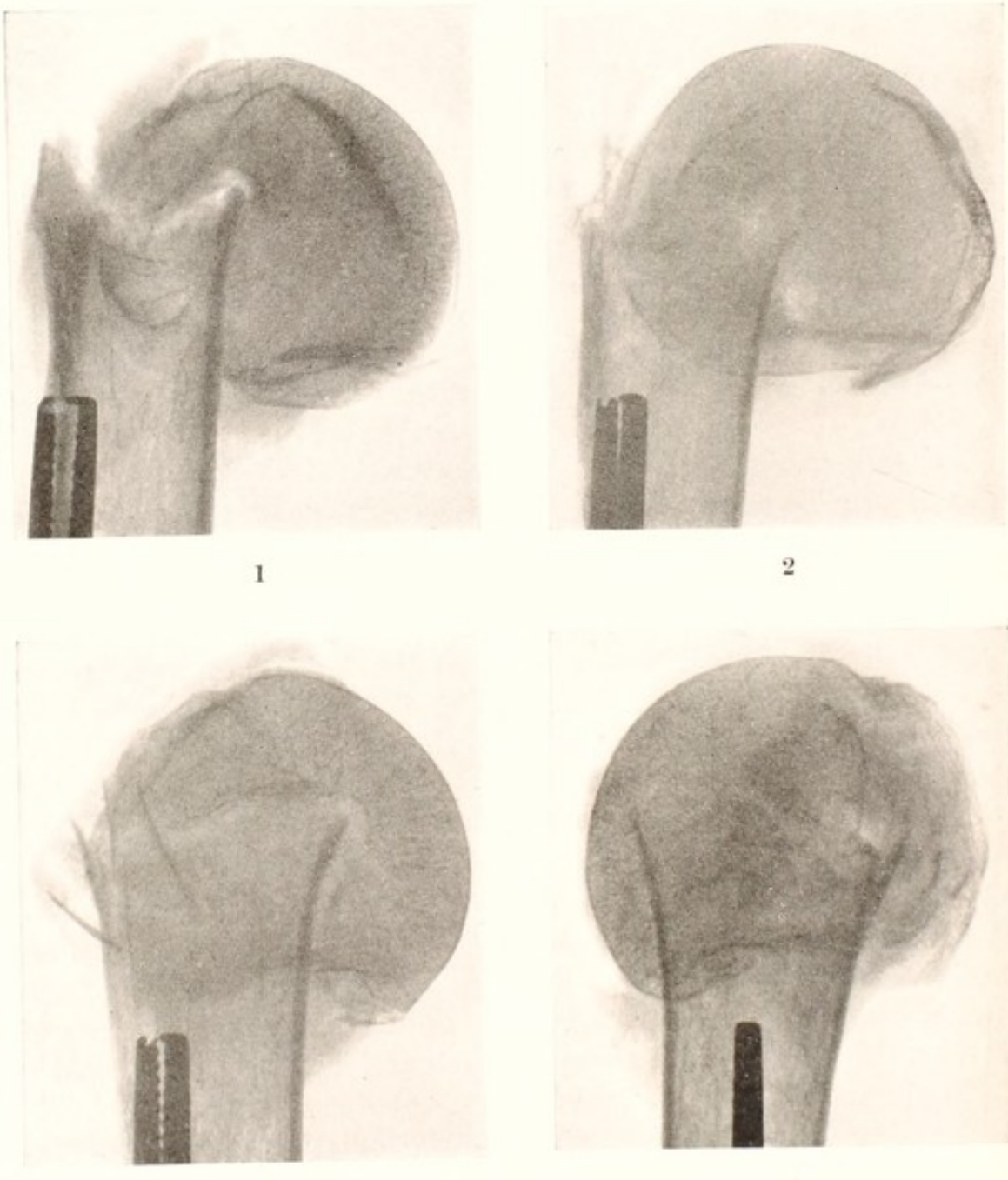
Since the base of the bursa is firmly attached to the periosteum of the greater tuberosity and the distal three-fourths inch of the supraspinatus tendon, this part of the bursa tends to remain in normal relations with the tendon and the tuberosity, while the subcoracoid portion may remain with the lesser tuberosity and the subscapularis.

The anatomic head completely covered with its cartilage may be separated from the bone at the line of the sulcus which surrounds it, and become entirely free. This fragment may be displaced in various directions. If the violence is great, and probably even if it is only of moderate degree, the inferior, weak part of the capsule of the joint is usually badly torn or peeled away from the anterior part of the neck of the scapula, so that the free head of the bone may escape through any rent in this capsule and lie in the soft tissue. It is perhaps better to say it may be "left behind," than that it "escapes." In one such case, I found the head with its surfaces reversed, lying beneath the deltoid (Fig. 57). In another such case, I removed it from the areolar tissue in the axilla and by a sabre-cut incision replaced it again in its proper position. In the first case the anatomic head was left behind where it had been displaced in the erect phase, while in the second case it was left behind in the later phase when the humerus had come to the side.

The tuberosities, on the other hand, being attached to the short rotators, do not tend to escape. When one operates on cases of this kind, one finds the fragments of the tuberosities held in on the glenoid by the short rotators as a man might take his hat and hold it on the further side of a tree which he clasped with his hands. The head of the bone, whether free or still attached to the shaft, may lie outside of these fragments. (See Fig. 58.)

In these complicated cases, the subdeltoid portion of the bursa is

of course often torn to some extent, but the portion beneath the acromion is scarcely affected. In other words, the under surface of the acromion, the supraspinatus tendon, and the external surface of the tuberosity are still covered with synovial membrane. When all the fragments are replaced, the major portion of the bursa may be still undamaged. Even when the bursa is torn on its periphery, it tends to heal readily and may be replaced by small bursal sacs, which together perform the function of the undamaged normal sac.



1 2

3 4

FIGURE 59. AN INSTRUCTIVE SPECIMEN

FIGURE 59

X-ray study of an instructive specimen of an intracapsular fracture, which was obtained by Dr. Akerson within a few days after the occurrence of the accident which caused the death of the patient. After dissecting away the deltoid, the fractured surfaces were not visible, for they were confined in the musculo-tendinous cuff and the superficial layer of the periosteum. The subacromial bursa was intact. The appearance at this stage of dissection was similar to that in Figure 8; *i.e.*, the normal inner muscular unit of the shoulder appeared to be intact.

The shaft of the humerus was sawed across, the short rotators and the capsule divided well back under the acromion, and the specimen removed. It was then grasped with toothed forceps, one blade of which was thrust into the medulla of the upper end of the shaft; then using the forceps as a handle, the above pictures were taken in different degrees of rotation without changing in the slightest degree the relations of the fragments. They show how deceptive are views taken from different angles, particularly after attempts at reduction. Slight changes in rotation of the shaft may cause an appearance which would deceive one into thinking that improvement had been effected in the position of the fragments. Contrary to the X-ray appearance, which shows that the shaft was entirely separated from the head of the bone and suggests that the fracture was extracapsular, it was in fact intracapsular, or at least covered by the periosteal extensions of the tendons.

When the specimen was dissected it was found that the superficial layers of the tendons of the short rotators which extend into the periosteum of the shaft had to be cut in order to display the fractured surfaces, and even then, it was far from easy to realign the fragments, although they were not, strictly speaking, impacted. It would be better to describe the condition by saying that the raw surface of the head was balanced on the posterior inner edge of the broken end of the shaft, and could readily be rocked but not easily freed. The fragments rotated as a whole and there was no real change of position between them except for a slight amount of rocking. It is highly probable that in most fractures in this region the conditions are very much the same as those found in this specimen, so that considerable traction and hyperelevation, as suggested in Figure 61, would be necessary to really correct the deformity. It is likely that unless this is done our clinical efforts are usually of little real importance, although the X-ray may encourage us if taken in a different degree of rotation. On the other hand it shows how tenaciously the short rotators with their periosteal prolongations cling to all the fragments, and tend to hold them together, even if in a jumbled mass. Fortunately nature thus procures in these cases a fairly good result in spite of our usually unsuccessful efforts to help. The study of this case makes one feel that early motion would be preferable to fixation, for nature supplies all that is necessary of the latter by reflex muscular spasm. If reduction is to be successful it must not be half-hearted; we must make a thorough logical effort on some such principle as that illustrated in Figure 61.

The first and second views are nearly lateral, *i.e.*, views which are difficult to take in a living patient whose arm is hugged to his side. The antero-posterior views below are the ones usually taken. The lesson is that each case should have at least oblique views, preferably stereoscopic, taken both before and after reduction. Antero-posterior views are deceptive.

In less complicated fractures where displacement has not taken place, the subacromial bursa is probably torn very little. I am inclined to think that in simple fractures, as of the tuberosity, it is not torn at all. The fact that the recovery from fractures of the tuberosity which are not much displaced is rapid, and experience in the two following cases make me feel quite certain of this position.

CASE REPORTS

Mr. E. A. F. Age 36. Massachusetts General Hospital No. 145677 E. S., Dec. 21, 1905.

The X-ray in this case showed a fracture of the tuberosity of the humerus. In the belief that the tuberosity had been pulled off by the supraspinatus and might be replaced accurately, the bursa was explored. It was found, however, that the whole bursa was normal. The line of fracture could be felt through the base of the bursa indistinctly, but it was thought best not to undertake any further exploration. Recovery was uneventful.

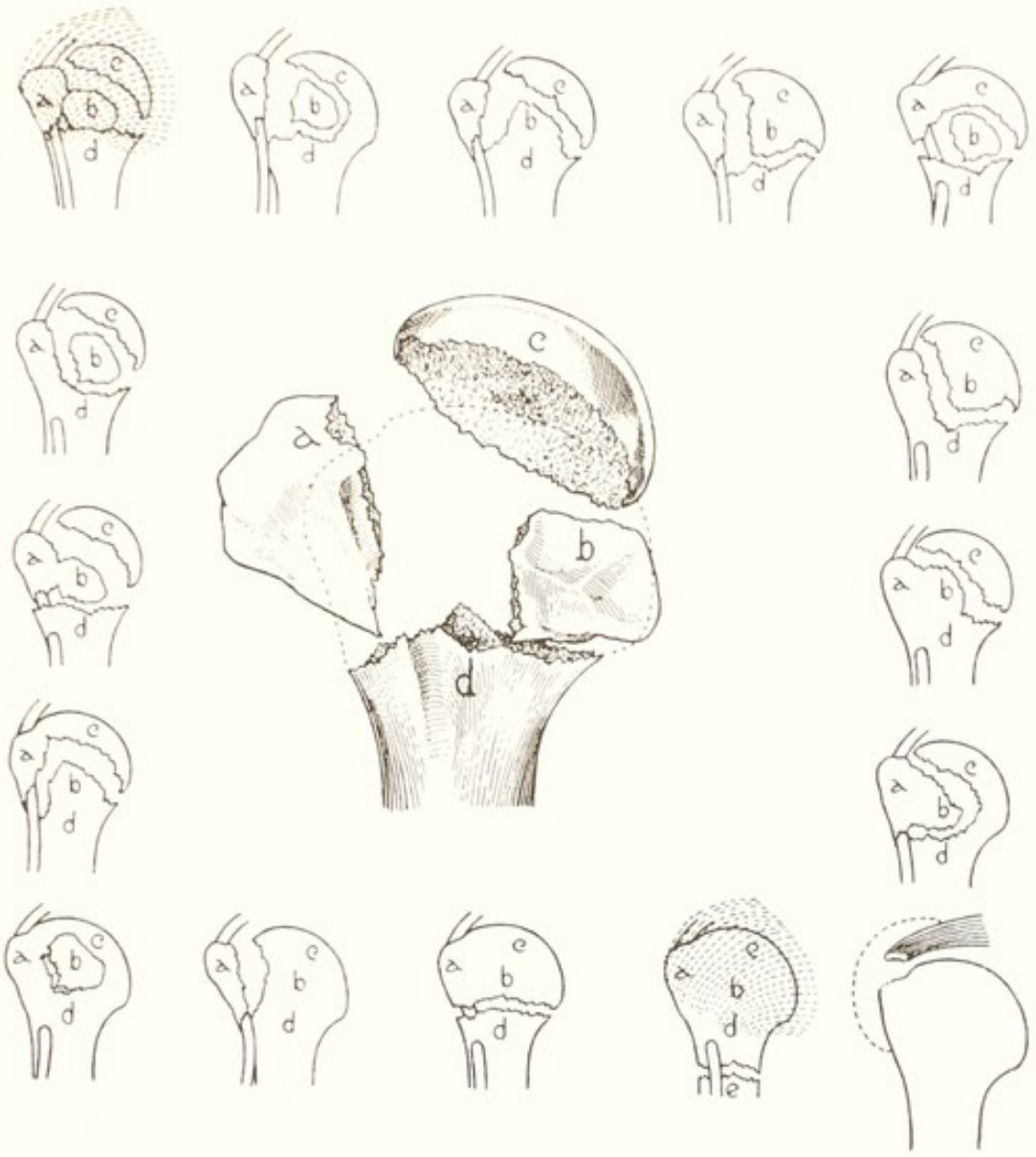
No. 10. Mr. M. McD. Age 23. Massachusetts General Hospital No. 174045 E. S., Jan. 20, 1911. In a similar case exploration showed that the bursa was intact and the tuberosity was satisfactorily uniting to the surface from which it was torn.

Figure 60 illustrates schematic drawings of the usual forms of fracture in this neighborhood. Naturally, I am unable to state that these are positive findings; they are only positive in the two cases referred to above and in a number of more complicated cases on which I have done operations. Most of these fractures are not complicated by disturbance of the function, and unite readily because the raw surfaces of bone are virtually in contact and are not bathed in joint fluid. Even if a little joint fluid can obtain access to them their own bleeding and clotting displaces it. This is not so with cases in which the evulsed fragment is small, and in which the bridging of the tissue must take place through a space constantly bathed in synovial fluid. To my mind in many other fractures such as those of the scaphoid in the wrist and of the femoral neck, the principle holds good that free access of synovial fluid to unapposed raw bone surfaces delays union.

FIGURE 60. TYPES OF FRACTURES OF THE HEAD OF THE HUMERUS

The lines of cleavage in fracture of the head of the humerus follow in a general way the former lines of epiphyseal union, although not exactly on these lines. A typical fracture is represented by the four fragments in the central figure, any one of which, in addition, is often more or less comminuted. In practically all of these fractures the musculo-tendinous cuff, merging, as it does, with the periosteum of the upper portion of the bone, holds the fragments together in a jumbled mass, as indicated in the upper left-hand figure. In most cases the lower line of fracture takes place through the cancellous bone just above the upper edge of the attachment of the pectoralis major to the edge of the bicipital groove, and therefore the firm tendon of the pectoralis holds the biceps tendon in the groove in the lower fragment. It is also firmly held by the tendons of the short rotators in the portion of the groove remaining in the upper fragment. Moreover, the superficial portion of the cuff extends over into the periosteum. Consequently all the different forms of fracture indicated in the above diagrams are essentially intracapsular until we come to the last two depicted in the lower right-hand corner; *i.e.*, fracture of the surgical neck and evulsion of a facet from the tuberosity.

In fracture of surgical neck all the structures which are really intracapsular are undamaged. The problem is not one, therefore, which involves the joint but one which merely requires realignment of the bone. The most serious fracture in my opinion is the last one depicted in the lower right-hand corner, which represents evulsion of the



facet of insertion of the supraspinatus (see Fig. 35), and is equivalent to rupture of the latter. This form of fracture is minute compared to the more extensive fractures shown in the other figures, but since it permits joint fluid to pass freely into the bursa, it will never unite by a natural means, and soreness and disability will continue from the pain induced by friction. It presents the same difficult mechanical problem as does rupture of the supraspinatus. Observe that in all these forms of fracture, the biceps tendon may be of help in realigning the fragments as illustrated in Figure 61.

If one realizes that the tuberosities have tendons firmly attached to their upper edges and that the base of the subacromial bursa is firmly attached to their superficial surfaces, and that they are usually concavo-convex pieces of cortex, frequently partially still attached to the periosteum on their lower edges, and more or less retained in place by the biceps tendon, it should be easier to reduce these fractures and to care for them intelligently.

The fact that the short rotators have firm holds on these frag-

ments should help us materially in replacing them. If the fragment has the position shown in Plate IX, which we may consider the usual position, we are likely to be able to reduce it by carrying the elbow backward and inward, thus exerting a pull on the supraspinatus. The fragment may be at the same time pushed into place by the surgeon's thumb. Occasionally the displacement is in the other direction, the tuberosity having been pulled too far inward by the subscapularis. In some cases, the fragment having been pulled by the supraspinatus too far under the acromion causes a decided obstacle, because in abduction it impinges between the humeral head and the acromion. If such cases cannot be corrected by manipulation, I believe the bursa should be incised and the fragment pulled back and retained in place by sutures, or even screwed or nailed down to its bed in the greater tuberosity.

If such retracted fragments are very small, as in cases where the supraspinatus tendon carries a portion of the facet of insertion under the acromion, they should be classed as ruptures of the tendon, and immediately operated upon. The sizes of the raw bone surfaces and the extent of the gap between them, through which synovial fluid may pass from joint to bursa, are important factors. If the raw surfaces are large enough so that bony contact can be secured and maintained, union will occur, but if the bit of bone is small and is dragged away by the tendon, the problem is the same as in rupture of the tendon. Separation of the superficial portion of the facet and rupture of the supraspinatus tendon are, therefore, essentially the same clinical entity, for they make a permanent communication between joint and bursa. Since this gap never heals and remains a cause of persistent irritation, the patient is usually worse off than if the lesion were a much more extensive fracture.

It is interesting to speculate as to the behavior of the biceps tendon and of its sheath in cases of fracture in this region. Anatomically in a normal patient, the long tendinous head of the biceps has its origin at the fibrous rim of the upper edge of the glenoid. Occasionally the whole tendon or a part of it may arise from the adjacent portion of the capsule as a developmental error, for normally the biceps tendon in the embryo is first a part of the capsule and then separates from it. In the normal case there is no mesentery, or any form of sheath or other attachment, covering the biceps tendon as it traverses the joint between the cartilaginous head and the under surface of the capsule, until it reaches the intertubercular notch and descends into the bicipital groove. Here there is a synovial

lined canal two or three inches long, which is a direct extension of the joint. The head of the humerus, using this tendon and its canal as a guide, runs up and down. It is not a fact, as may be superficially thought, that the long head of the biceps runs up and down in the groove when we move our forearms. To obtain motion between the biceps tendon and the bicipital groove in the humerus, one must move the scapulo-humeral joint. The expansion or contraction of the biceps muscle itself does not move the tendon in the groove, although it increases tension in that tendon. Extension of the elbow will also increase the tension in the tendon, although it will not move it. When the tendon is a little way down in the groove, it gets its blood supply through a mesentery-like fold in the lower portion of the sheath. That this tendon has a very superficial blood supply is sometimes beautifully demonstrated at operations for rupture of the supraspinatus. It may be seen crossing the gap, and is of a bright pink color, owing to the congestion of fine superficial blood vessels. This lower portion or sheath, where the synovial membrane ceases, necessarily has a certain amount of movement from the areolar tissue around it, which allows for play just above the belly of the biceps muscle. Fractures in this region, *e.g.*, the typical fractures of the greater tuberosity, do not tear the tendon out of the groove on the distal fragment or on the proximal; in fact, the periosteum usually is only bent at the lower point and is not entirely disrupted. The tendon of the pectoralis major holds the distal portion of the biceps tendon in contact with the shaft.

Since the biceps may function in any position of the glenohumeral joint, whether the latter is adducted, abducted, internally or externally rotated, the relation of the biceps tendon to the articular surface of the humerus is constantly changing. Bearing these functions in mind, let us consider what would happen when the typical fracture of the head of the bone occurs in three or four fragments. In a typical fracture in the pivotal position, the tendon would not be involved until the descent of the arm, when it might either be caught among the fragments, or might function in helping to realign them.

It seems to me highly probable that the lines of cleavage in these comminuted fractures take place near the bicipital groove, beneath the synovial and periosteal lining of this groove, so that when separation occurs, the biceps carries its sheath and the tuberosity with it. Unless this is so, it is very hard for me to account for the fact that in most cases the function of the shoulder joint returns so rap-

idly after fractures in this region. In the few cases in which I have had the opportunity to dissect specimens this actually was the case. (Fig. 59.) The biceps tendon would be caught between the fragments and its function destroyed if it failed to carry some of its sheath with it. At operation in badly displaced cases one often finds the tendon irrevocably injured, perhaps evulsed or caught behind the head next to the glenoid (Fig. 58), but in most cases where the fracture can be well set, the biceps somehow manages to regain its function.

Probably few surgeons consider these bursæ and tendon sheaths in treating fractures in this region. It has seemed to me that the consideration of these structures has helped me personally in handling these cases. If one bears in mind the typical fracture shown in Fig. 60 and the other fractures in this region as merely incomplete forms of this typical one, he will have a feeling of understanding as he treats each individual case.

Bearing in mind the four fragments which usually occur in the severe fractures, we may form subordinate types according to whether any two or three fragments remain united. Usually the four fragments are only partially separated, and either because held by periosteum and the musculo-tendinous cuff, or because prompt reduction has taken place, lie in mutually normal relations, even if as a group they are not in line with the shaft. If we could see the exact cracks in most fractures, I think we should find them more complicated than usually shown by the X-ray. In clear pictures there is much subordinate splintering of small bits of cortex. In many cases where the X-ray shows the head as one fragment, detailed study of clearly defined stereoscopic films would show the tuberosities partially separated from the head and from each other, but still held together by bits of fascia and periosteum, or by the cuff itself. The group is held together by the musculo-tendinous cuff in a jumbled mass, but still in mutual apposition, and if we can manage to cap this rather wobbly head on the top of the shaft we may obtain a good result. Usually this means disengaging the inner edge of the lower fragment from the head. The shaft is almost invariably in front of and somewhat mesad to the comminuted head.

The really important question is whether there has been escape of the articular head out of the capsule. So long as the articular head remains attached to a tuberosity, it cannot displace permanently, and so long as it remains between the tuberosities and the glenoid we may hope for a good result; but if it has escaped from the

capsule there should be no delay in deciding between operation and a stiff shoulder. Social and general conditions determine this decision, and the surgeon cannot promise much, for when he operates he will find a difficult task. My belief is that the head of the bone should be replaced through a sabre-cut incision, and that the chances of a good result would be far greater if the operation were done within a few days of the injury than if it should be delayed.

We have previously mentioned various deductions from the Pivotal Paradox, but there may be another and a very important one in connection with the reduction of fractures.

Since it is probable that most fractures of the head of the humerus occur in elevation, and since in this position the long head of the biceps is relaxed, it is likely that reduction may be best accomplished by returning the arm to this position, while traction is exerted on the elbow with the forearm flexed. The forearm should then be extended while still exerting traction. Next the arm and extended forearm should be brought to the side in external rotation via the coronal plane. The forearm could then be flexed and rotated inward into the sling position if desired, for the fragments would be engaged as soon as the traction was omitted. (Fig. 61.)

This maneuver would use the long head of the biceps as a guide to replacement of the fragments, since the tendon presumably still is held in its groove in both the upper fragment and in the shaft and neither fragment is capable of further outward rotation under normal circumstances. Theoretically, this maneuver, *provided the long head of the biceps has remained attached to the glenoid rim*, would disengage the impacted inner edge of the lower fragment and lock the fragments in proper position in most cases of epiphyseal separation, transverse fracture, and even in cases of comminuted fracture in which the tuberosities were still more or less held together by the musculo-tendinous cuff, by shreds of periosteum and by the intact base of the bursa. Even when the articular head has been displaced as in Fig. 57, it might be approximated to the shaft and tuberosities in the erect position and pushed back into the socket by the descent of the arm in the coronal plane! It is conceivable, also, that when the anatomic head has been dislocated in the axilla, it might be returned by the reverse motion.

Reference to the diagrams will give sufficient information on the minor fractures, except in two particular forms which are not generally understood.

One of these difficult types is simple impaction of the anatomic

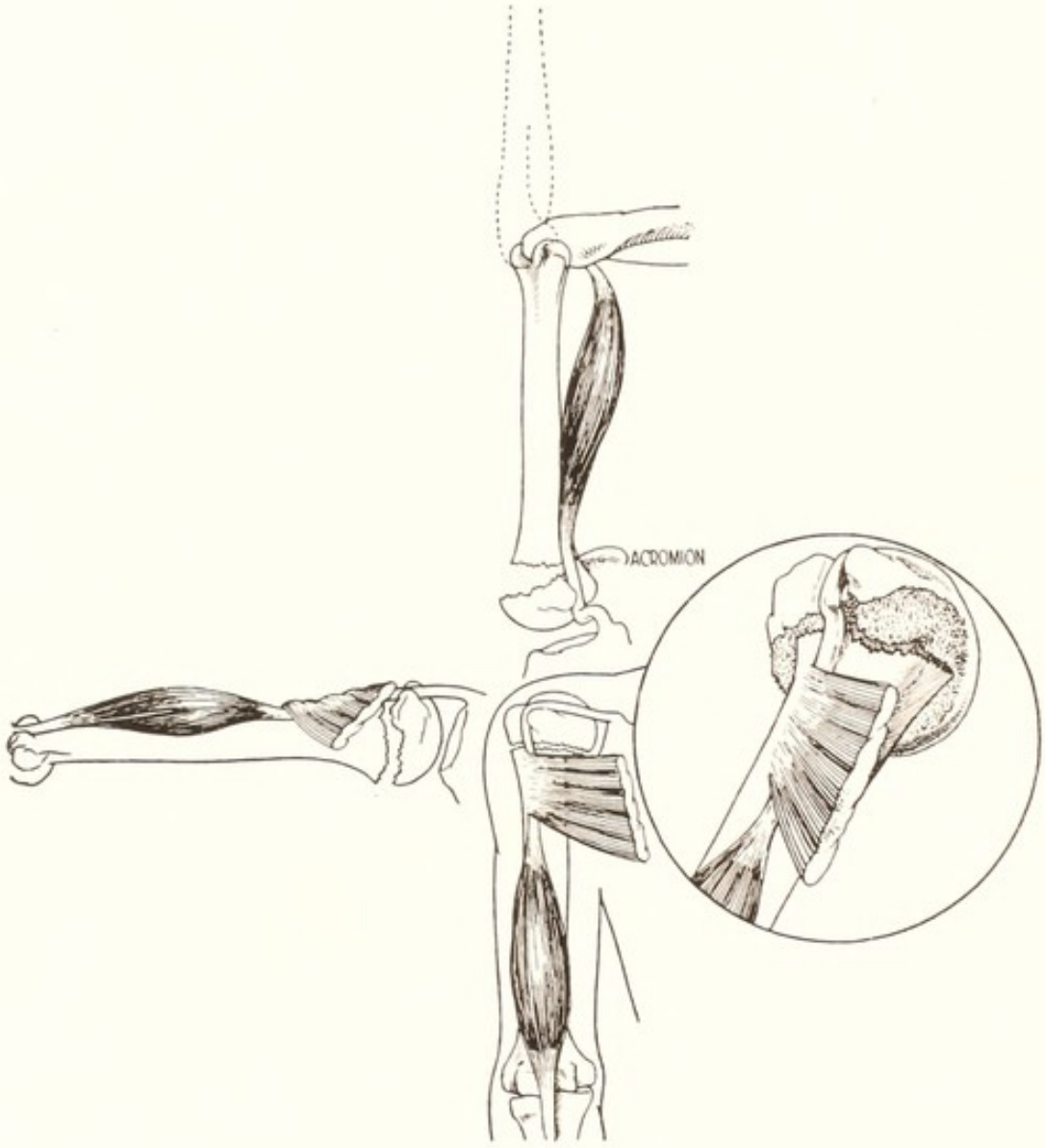
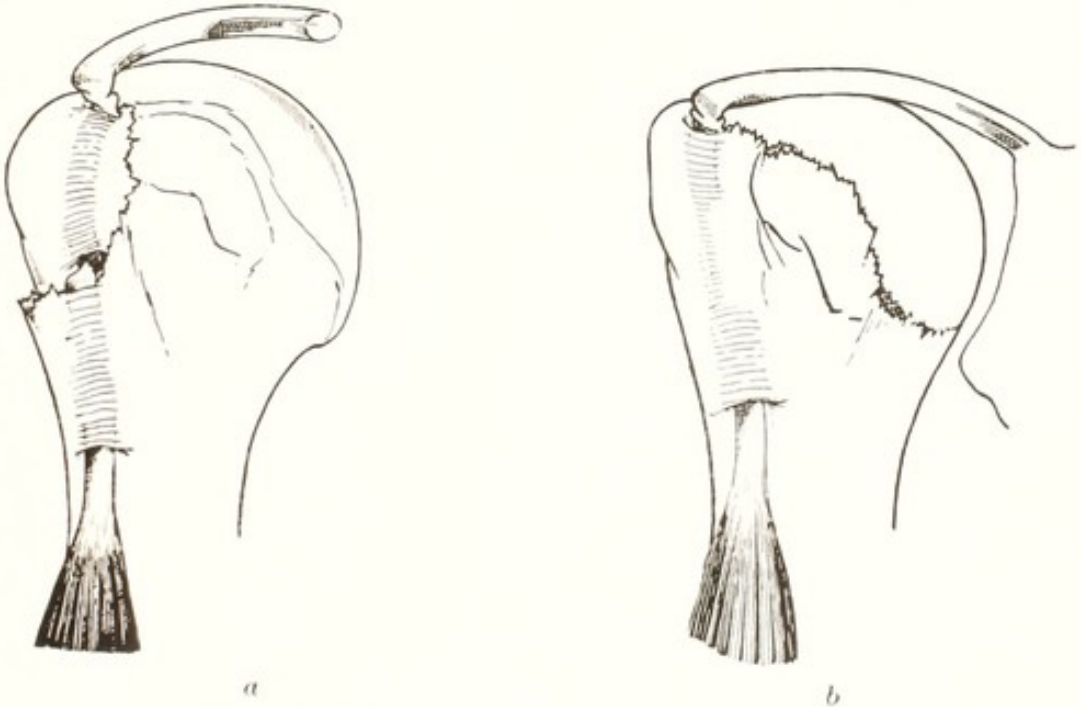


FIGURE 61. METHOD OF REDUCTION FOR FRACTURES OF THE HEAD OF THE HUMERUS

The circular insert illustrates the usual displacement of a fracture of the head of the humerus whether it is comminuted or not. The upper end of the shaft is anterior and its inner posterior edge is impacted in the spongy portion of the head of the bone (Fig. 59). The biceps tendon is held by the pectoralis tendon in its sheath in the lower fragment, and is also held by the expansion of the tendons of the short rotators in its groove in the upper fragment. The other figures illustrate the method of reduction, described in the text, which utilizes the biceps tendon to realign the fragments. The arm is first put in the pivotal position and the fragments are separated by using the acromion as a fulcrum. The biceps tendon is then made taut by extension of the elbow and traction upward. The arm is carried down in external rotation in the coronal plane, which still further tends to realign the fragments, for, normally, it represents the extreme degree of external rotation for both fragments. When the arm has come to the side in external rotation, and before the elbow is again allowed to flex, the humerus is put in internal rotation; then the elbow may be flexed, and the forearm will be in the sling position.

head into the tuberosity (Fig. 62*b*), which remains attached to the shaft. I do not mean the usual impaction of the shaft into the head. I have found that impaction of the head into the tuberosity is more disabling than some of the fractures with much wider separation of the fragments. The result of this impaction is that the length of the anatomic neck of the humerus is shortened, and the relation of the curve of the articular head is changed by rotation on the axis of the shaft. Furthermore, there is a scissor-like action on the articular edge as it obliterates the sulcus where the short rotators are attached. In other words, it cuts the attachments of the short rotators so that it leaves but a thin margin where they can secure their hold of the facets of insertion. There must necessarily be a considerable degree of traumatic tendinitis following such lesions. It is quite likely that the biceps tendon is pinched and held. Certainly from a clinical point of view, the convalescence in these cases is often delayed, and the clinical picture becomes similar to that of a severe bursitis.

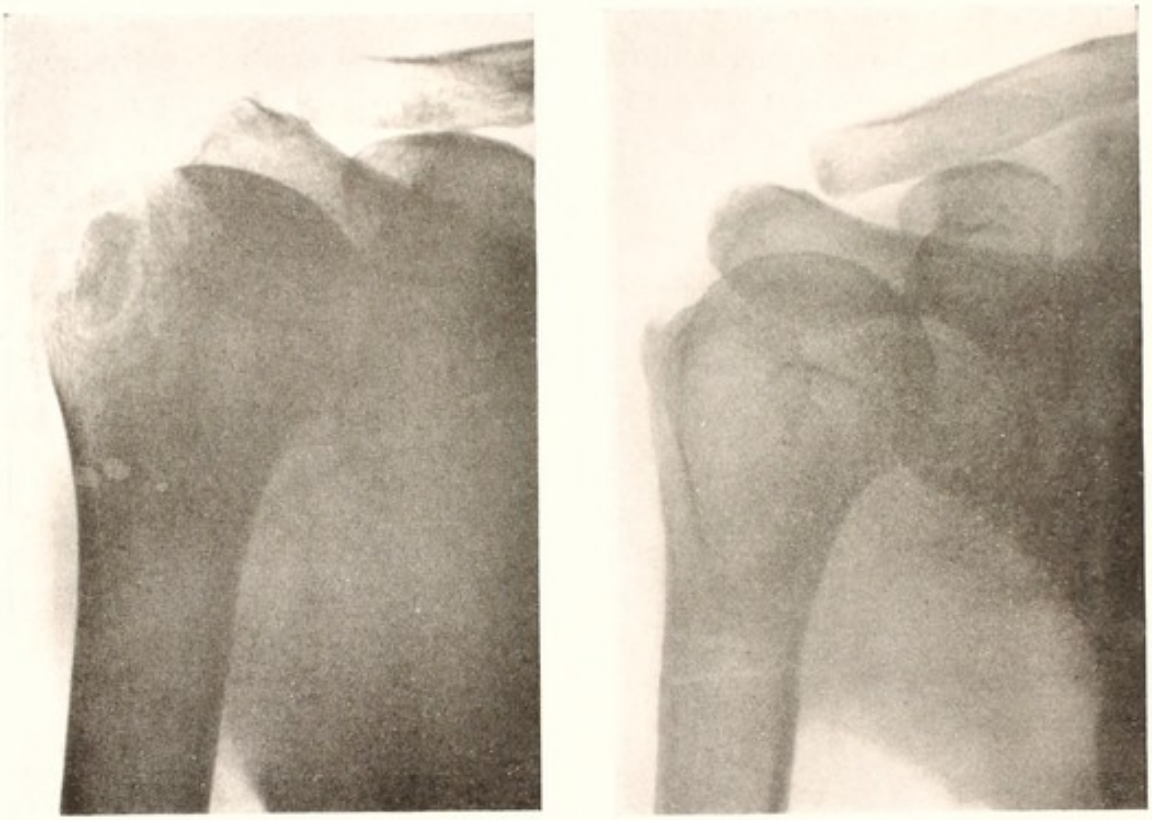
Another disabling form of fracture of slight extent and probably not very common, but usually unrecognized, is depression of the greater tuberosity (Fig. 62*a*). It is probably due to direct violence when the arm is in dorsal flexion, but perhaps it may occur from



a *b*

Figure 62 *a-b*. Two Disabling Forms of Fracture

Two very disabling fractures which cause but slight change in the X-ray projection but in their clinical course are more protracted than many cases of fracture which are apparently more extensive. An attempt is made to illustrate the involvement of the biceps tendon in these crushing fractures, but the reader must realize that the supraspinatus and other short rotators will also be involved in such cases.



c *d*

FIGURE 62 *c-d*. DEPRESSED FRACTURE OF THE TUBEROSITIES

c. X-ray three months after injury. Necrosis of the trabeculæ, surrounding the depressed fragment, has occurred.

d. A year later. A spur has formed at the site of the old fracture. There is a little calcification in the supraspinatus tendon.

impact on the glenoid in elevation. I have seen at least one such case. This form of fracture is easy to overlook, and one reason for this is that the patient is usually able to use the arm for a time after he has suffered the injury. Since the tuberosity is depressed, he may even be able to elevate the arm, and as it takes some time for the formation of a callus of sufficient degree to impinge under the acromion, there is a period of fair use of the arm before serious restriction begins. Neither patient nor physician realize the extent of the injury and the Röntgenologist also may overlook it.

The following is a report which I made to an insurance company on such a case, April 11, 1929:

"The patient is a well-developed, intelligent electrician of thirty-seven. He states that he has never had any serious accident, sickness or operation except a badly cut wrist as a boy, and diphtheria when still younger.

"Day before yesterday (April 9th) he fell from a ladder, and in falling struck his left shoulder on a marble slab. Although the shoulder felt

sore and bruised, he continued to work, thinking that the pain would soon go away. He has continued to work since, except that he went to your clinic yesterday and was referred here by Dr. L. He has been working this morning and is able to get along because he is the boss and can have his men do the manual work. He can help with one hand. On the first night after the accident he woke with much pain, but last night there was less trouble. He can use the arm a little, but it is sore.

"Examination. This patient has good muscles and good control over them. He appears to be a very intelligent, sensible person, not inclined to exaggerate his troubles. There is a conspicuous ecchymosis to the inner side of his left arm and some deep swelling beneath it. Both correspond to the regional course which hemorrhage takes when it runs down the bicipital groove, namely, along the tendon of the biceps and then down the supracondylar areolar tissue about the internal condyle, and then into the subcutaneous tissue of the forearm. This is almost a pathognomonic sign of involvement of the base of the bicipital groove by a fracture. It perhaps may occur with rupture of the long head of the biceps.

"The motions of the joint are not much restricted, but there is some very decided restriction in abduction. There is no atrophy and only slight tenderness over the greater tuberosity. The biceps cannot be exerted without causing some pain. The X-ray supports the diagnosis of a depressed fracture of the greater tuberosity, and this accords with the clinical symptoms.

"This type of fracture is rare and apparently trivial at first sight, but in the cases I have seen it has proved in the end to be more incapacitating than fractures of the surgical neck. It goes through two stages. In the first stage the symptoms are less severe, and are caused by the mere alteration caused by the depression of the tuberosity and the hemorrhage about it. The second stage causes more incapacity, because the callus about the fragment impinges on the acromion as the arm is raised. Then, too, the trabeculæ, which are crushed together, dissolve, and an osteitis is set up, so that in the X-ray you get the appearance of a sequestration of the depressed fragment. At this time—perhaps three to six months from now—there is much disability and pain. Since the fragment is pressed inward, the long head of the biceps may be damaged and become adherent in the flattened, narrowed groove. Also the supraspinatus tendon is partially pinched off at the edge of the articular head.

"Naturally, there is no standard treatment for such a lesion. The usual methods of treating fractures do not apply, where there is no need of reduction or immobilization. Even if you could raise the depressed fragment, the callus would impinge even more on the acromion. It seems to me it would be better to let nature take care of it than to interfere. If I had such an accident I would work as much as I could without too much pain.

"Fortunately, from what this man says, he is not obliged to do much actual work, and can hold his job without using the arm a great deal. He probably earned enough, that to be idle and lose the difference between his pay and his compensation would be a great hardship. I should think he would prefer to work as long as he can hold his job. The ques-

tion, naturally, came up as to whether physiotherapy or other palliative treatment would do him any good and hasten his recovery. I do not believe any form of treatment would help him, and almost any form of treatment would harm him as much. If pain or soreness is very severe, he could try massage or diathermy, if, practically, after a few trials he found that he obtained enough relief to pay for the trouble.

"He would naturally want to know whether free use of the arm would help or hinder his recovery, and on the other hand, if perfect rest would lead to shorter convalescence. I believe there might be a slight difference between complete rest and strenuous use, in favor of the former. The more he uses it the bigger the callus is likely to be, but the less he uses it, the less the change in circulation and the slower the rate of repair. I regard this as a toss-up, and if it were my arm would be guided solely by the discomfort caused by use. There are too few precedents to go by to offer any evidence of importance. I have never seen but a few cases, and do not know the exact efforts made at treatment, or the exact results. My general impression is of a slow return to normal function after not less than a year.

"A similar case which I saw for the Industrial Accident Board was incapacitated for over two years when I last heard from him, but the patient was an Italian laborer, and this man will work with soreness that would frighten an Italian. If I were in this man's place, I would stick to my job as long as I could stand it, using my arm a little, but not much. I would take no treatment and hope that the exercise of work would keep me generally fit and thus hasten my repair."

I saw the patient again on Sept. 30th, 1929. He had kept at work about three weeks before he quit, and then had baking and massage at the insurer's clinic. Rotation was free, but abduction was limited by the callus. There was atrophy of the spinati.

In November he went to work as an electrician, and worked off and on in spite of some pain for over a year. Even when I saw him last, on Dec. 3, 1930, he was still complaining of weakness and soreness in the shoulder, but at this time could do overhead work.

Fractures of the tuberosity which are not depressed sometimes also have this protracted course, and show late bursal symptoms from irregularities in the base of the bursa caused by callus, but in these cases where the fragment is at first depressed, there is an early, relatively painless stage. Fixation in any fracture of the tuberosity is likely to delay recovery rather than to hasten it.

It is remarkable in studying the literature of fractures in the upper end of the humerus to find how little detail is given to the probable relations which the muscles and bursæ bear to the fragments. Few authors even mention the subacromial bursa in this connection, and many do not even mention the tendons of the short rotators. Stevens has given by far the best brief account of the mechanics of the shoulder joint that I know of. In some minor

respects, I do not agree with what he has said, but as a whole, I regard his articles as very sound. I should like to quote many pages, did space permit.

Diagnosis. So far as positive diagnosis is concerned, we depend upon the X-ray. The clinical signs in the types delineated in Figs. 62*a* and 62*b* suggest those of rupture of the supraspinatus tendon, since either by partial crushing of the tissues at the time of the original injury, or by contiguity from hemorrhage about the fracture, a bursitis is started up. Later, the callus deforms the base of the bursa and makes it irregular, so that its smooth action is lost. Hence, we have the usual symptoms of bursitis, spasm, and restriction of motion preventing rotation and abduction. They are all present, but masked by the usual symptoms of fracture. There will also be localized tenderness in the same region as in cases of rupture of the tendon, and there may be, in certain stages, a jog in the scapulo-humeral motion as the callus passes under the acromion. The main clinical diagnostic point is the feeling to the examiner's finger, that the whole tuberosity is enlarged (at first by hemorrhage and later by callus). If the tuberosity is broken, an important sign often delayed for a few days, but seen within the first week, is the appearance of ecchymosis, as described in the last case history. This ecchymosis may also appear posteriorly to the deltoid in the post-axillary fold. If it is present below the lesion along the course of the biceps, one may feel fairly sure that there is a fracture near the tuberosities. I think it is present to some extent in most cases of fracture of the head of the humerus, but very frequently does not appear until after twenty-four hours from the time of the injury.

Prognosis and Treatment. One can by no means prophesy the length of the convalescence by the degree of fragmentation or displacement shown in the X-ray. Stevens is correct in his contention that it is the traumatic arthritis which needs attention more than the fractures. Traumatic arthritis, to my mind, means changes in the bursæ and musculo-tendinous cuff. The probable degree of communication which exists between the joint and the subacromial bursa through the gaps between the fragments is of great importance. Perhaps some day we shall determine such points by injecting non-radiable fluids between the fragments.

As a very obvious contrast in prognosis, it may be said that if the tip of the tuberosity is torn off by the supraspinatus and withdrawn a half-inch beneath the acromion, the prognosis of the period of disability would be much greater than in a case of fracture of the surgical neck, and considerably greater than in a case of a com-

minuted fracture in which the articular head and tuberosities were separated, but could be reasonably well returned to their normal position. Since apparently minor fractures may lay a patient up much longer than obviously severe ones, it would be a good rule for any industrial insurer to put every case of shoulder injury in a hospital for careful study and expert opinion. A few days with the arm elevated in every case might reduce the average period of disability by many weeks.

The use of an airplane splint is not satisfactory; because there is too much danger of nerve paralysis and of ischæmia. Stevens says: "External rotation in abduction as a treatment is almost an impossibility of accomplishment unless the patient remains in bed. In bed it is the simplest method and not an uncomfortable position for the patient."

As a matter of fact, it is impracticable to treat all cases in a hospital or even in bed at home. This is more because it is not customary to place these patients in a hospital than it is because such treatment would not be wise. Minor cases often occupy hospital beds which might well be used for the early stages of the treatment of these fractures of the head of the humerus.

Stevens used a special splint to maintain the arm in external rotation and abduction. My method is simply to apply light traction to the upper arm and to allow the weight of the forearm to sag backwards (recumbent position) in external rotation, using a fillet about the wrist which permits the patient to use the forearm, but restrains him from getting it in a completely internally or externally rotated position. As I treat these cases, I visualize the bursæ and try to give the patient enough active and passive motion to keep pumping blood or clot out of the bursæ into the areolar tissues, where it can be rapidly absorbed, leaving the joint and bursæ clean. This does not mean much motion, but a little is very much better than fixation. I would not hesitate to put the arm in elevation in a Balkan frame, or to permit the patient to move it anywhere above the level of the shoulder. I do think that it is important to keep a certain amount of pull on the humerus to prevent the biceps and triceps from driving the shaft upward on the fragments. Early stooping exercises are desirable.

Concerning the choice of cases which should be put in bed and those which may be treated in some ambulatory way, my judgment would rest largely on whether or not I thought the subacromial bursa was seriously torn, and as to whether there was enough gap between the fragments to delay union. As I have stated before, I believe that

many of the fractures of the tuberosity occur beneath the base of the bursa, without involving it at all. Such cases should not be confined, but the stooping exercises with the arm relaxed should be used to make sure that adhesions in the bursa do not take place. I believe that unless there is some very unusual condition, any case of fracture of the upper end of the humerus should have fairly good motion in six weeks, and the patient should certainly be at work in six months.

There is one very striking thing about fractures of the humerus, and that is that most cases eventually recover pretty good use of their shoulders *in spite of* any kind of treatment. Only those in which the displacement is very great or in which the treatment is neglected very grossly (perhaps by the patient) result in ankylosis. In cases in which the reduction is imperfect, consultation should not be postponed, because early operation is far more promising than if it is delayed for even a few weeks. Surgical skill in handling fractures of the head of the humerus will be displayed more in attaining rapid and comfortable recovery than in ultimately securing good results, for nature alone would produce them in most cases. Injudicious fixation is responsible for most delays and failures in the recovery of normal function. As in all pathologic conditions of the shoulder in which function is prevented, atrophy of the spinati occurs in a few weeks and usually does not disappear for many months.

REFERENCES

PHEMISTER, D. B., Fractures of the Greater Tuberosity of the Humerus, Ann. of Surgery, 1912, lvi, 440-449.

The above article by Phemister reviews the literature and discusses in an interesting way the methods of operation for fractures of the greater tuberosity.

ROBERTS, S. M., Fractures of the Upper End of the Humerus: End Result Study, which shows Advantages of Early Active Motion. J. A. M. A. 98; 367-373. Jan. 30, 1932.

This is the best article to which I can refer as to the present status of treatment of these fractures in progressive hospital clinics in America, for there is always some degree of progress that has not yet taken its place in the text books. It is a careful study of a series of cases treated at the Massachusetts General Hospital, where much attention has been devoted to fractures, during the last decade, by an active "fracture committee," so that the results may be assumed to be better than the average, even though they include a considerable number of instances where there was long delay in seeking hospital treatment. Nevertheless, it is evident that even in these carefully treated cases, the results are by no means perfect. Although appointed consulting surgeon to this hospital in 1929, I have had nothing to do with the treatment of these cases, nor have my principles of reduction, illustrated on page 324, Fig. 61, ever been tried out. The excellence of the results obtained by Dr. Roberts and his colleagues has in no way been dependent on the theories expounded in this chapter, which in many respects differ from those on which treatment was based in their cases. Are my theories sound enough to be tested? If so, by whom? At any rate, the economic loss in their series of fractures was evidently far less extravagant than would have been incurred in an equivalent number of cases of unrecognized complete ruptures of the supraspinatus.

Chapter XI

BRACHIAL PLEXUS PARALYSIS

By J. H. Stevens

[*Introduction by Dr. Codman*]

In 1929 when I had already spent a year on this book, I found, by mere chance, that my neighbor, Dr. Stevens, had for a long time made a hobby of studying the brachial plexus. I had often talked with him about other injuries of the shoulder, subjects on which he had made some important contributions, but he had never mentioned that he had been so much occupied with studying lesions of the brachial plexus. I found that he had never written anything on the subject because, after all the years of study he had devoted to it, he felt that little was really known about these lesions and that his personal views would interest very few readers.

I asked him if he would not review his notes and write a chapter for my book. Although he said that he could add very little to the ordinary textbook knowledge about the subject, I assured him that if he did give me the benefit of what study he had made, it would at least bring the subject up to date and show that it was open for still further investigation. I at length persuaded him to try. He had at that time dissected ninety-two plexuses through the courtesy of the Tufts Medical School, and since then, through the kindness of the anatomical department of the Harvard Medical School, he has been able to dissect a good many more, but owing to his illness during part of this time, he was only able to analyze sixteen of these later dissections for this chapter. However, these were done very carefully, with especial reference to confirming his previous observations.

On March 24, 1932, Dr. Stevens died suddenly from heart disease, leaving his work in preliminary manuscript form for me to use. The following chapter has been constructed from his manuscript, using largely his own words, but modified to some extent because I was obliged to abbreviate parts of it, and in other parts to add a few paragraphs to clarify his meaning. The substance of the chapter is his and he deserves the credit for all the originality which it contains.

I have used the portions of his manuscript which seemed to me original and enlightening, although in some cases I have not actually verified his observations or presumed to criticize his conclusions. For instance, I have made no dissections to verify the new nerve filament which he describes, nor can I take any stand in regard to his views about prefixed and postfixed plexuses. Neither could I review

and tabulate the 710 cases which he has collected and analyzed. I have merely set down his conclusions and can only say that they seem to me valid.

Dr. Stevens was one of the most independent-minded men I have ever known. He practiced surgery for many years in Boston, but was never associated with any of the large clinics. Nevertheless, he was a true student of medicine, and took the greatest interest in his own individual patients. Any case which he could not thoroughly understand led him to study the subjects involved most carefully, not only by searching the literature, but by experimental work in the machine shop or in the dissecting room. He had little respect for modern authority and always tried to trace his subject to the original writers who first described it. For these pioneers in medicine he had the greatest respect and admiration, and a corresponding scorn for plagiarists. As the years went by, his habit of giving intensive study to each obscure case which occurred in his practice, gave him a remarkable all-around knowledge of surgery and its problems. Few men in their fifties and sixties have the industry to dissect out the branches of the brachial plexus to satisfy themselves about mooted points which they have unearthed through finding differences of opinion among famous authors.

He seldom spoke at medical meetings for he was of a retiring disposition, although on superficial acquaintance he seemed assertive. I have never heard him "read a paper," but he published several papers on fractures, which to my mind show an unusually clear grasp of the subjects. It was very characteristic of him to have done all this work and more on the plexus and to have made no attempt to publish it. He enjoyed satisfying his own curiosity, but he hated the details of publication. I feel that I was very fortunate in being able to rescue from oblivion some of the thoughts suggested by his investigations. At any rate, I believe that the surgeon who is willing to give proper study to this chapter will agree with me on finishing it, that he has a clearer and better idea of brachial plexus injuries than he ever had before. One feels that the subject has been very carefully studied by the author, and that his views about the essential mechanics governing these injuries have been thoughtfully presented, and are not likely to be challenged.

E. A. C.

Causes of Brachial Plexus Paralysis

1. Traction.
   - *a.* Birth cases.
   - *b.* Blows on shoulder or neck depressing shoulder, or falls stretching head away from shoulder.

c. Falls when the arm catches, suspending the person at least momentarily, or where arm is twisted.
d. Being lifted by the arm as over a pulley, or dragged forcibly by the arm as in case of children jerked by parents leading them by the hand.
e. Accompanying dislocations or the reduction of dislocations.
f. Prolonged holding of arm in abduction and external rotation, as in operation.
g. Slipping while carrying a weight on shoulder.
h. Sudden movements, as in effort made in trying to lift weights.

2. Pressure.
a. Dislocation, where shoulder is out for a long time.
b. Exuberant callus, especially of the clavicle.
c. Saturday night paralysis, usually radial, terminal.
d. Crutch paralysis, terminal.
e. New growth, involving roots, trunks or terminal branches, secondarily, either from bony, cartilaginous or soft tissue pressure. 1. Benign. 2. Malignant.
f. Adventitious rib or bands.
g. Strait-jacket; several cases have been reported, but always where the patient freed the arm so as to cause constriction.
h. Following aneurism of the subclavian or axillary artery.
i. Esmarch bandage or tourniquet.
j. Degenerative bone lesions of the cervical spine, especially tuberculosis, causing pressure on roots.
k. Fractures; especially of vertebræ, by direct pressure.

3. Direct Injuries.
a. Cuts or wounds.
b. Fractures or dislocations causing direct injury.
c. Injection of the plexus by local anæsthetics.
d. Electric shock.

4. Toxic.
a. Following serum injections.
b. Infectious diseases, especially pneumonia.
c. Following anæsthesia, chloroform especially. (These are hard to separate from possible trauma from mal-position during the anæsthesia, but several cases have been reported where no mal-position could have occurred.)
d. Poisons—as lead—usually peripheral neuritis.

5. Spinal Cord Lesions.
   a. Infection.
   b. Degenerative processes.
   c. Tumors.
   d. Direct injuries to the cord itself.
   e. Hemorrhage within vertebral canal.
6. Psychic Paralysis. (Hysteria.)
7. Central.

   Monoplegia. Rare; never complete; spastic, not flaccid.

This list covers most of the causes of injury, but cannot indicate the relative frequency in which they occur; no such statistics have ever been collected. As a matter of fact, in a very large majority the injury is of the traction type. In a smaller number it may be of the pressure type. The lesions caused by the remaining five types are either too rare or too obvious to be discussed here. Most of the common injuries have to do with temporary, partial or complete displacement of the head of the humerus. The plexus may be injured by the same force acting concomitantly directly on the plexus, or by the prying effect of the displaced head on the cords themselves. If the head of the humerus remains out of position for some time, direct pressure on the plexus itself may be a factor.

As indicated in the diagram below, brachial plexus injuries may be classified for purposes of study in several ways besides the etiologic grouping given above.

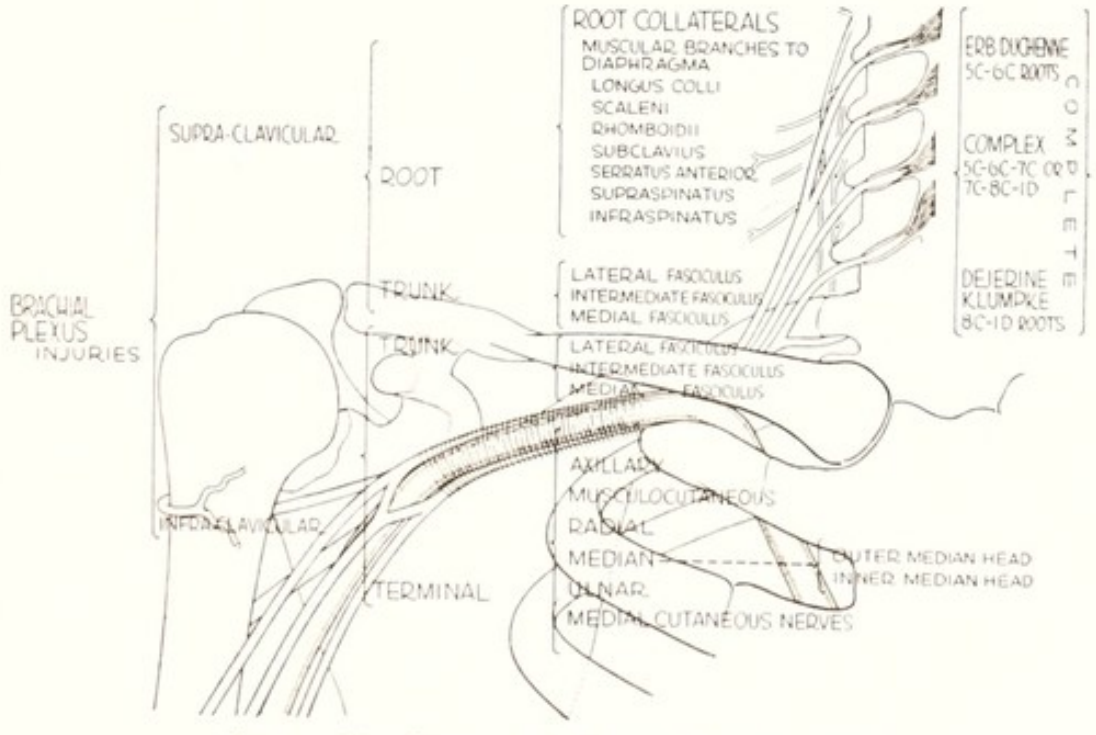


FIGURE 63. DIAGRAM OF NEUROVASCULAR CORD

The primary division into supra- and infra-clavicular lesions is important, not only because in the literature of the subject one finds endless discussion as to which group is the more common, but because when surgical exploration has been decided upon, one must choose between the upper or lower field for the incision. The secondary divisions obviously cannot be made strictly in subdivisions of the primary division, for a small part of the trunk lies above, and a large part below the clavicle. All root lesions are necessarily above the clavicle, and all lesions of the great terminal branches, including the axillary, are below the clavicle. However, some of the smaller but highly important upper terminal branches, usually spoken of as "the root collaterals," such as the dorsalis scapulæ, suprascapular, phrenic and long thoracic, are above the clavicle since they arise from the roots, or from the fasciculi not far from the roots.

In the text which follows, we shall frequently allude to that part of the plexus between the junction of the roots and the origins of the great terminal branches as the "trunk." This trunk when dissected is the "plexiform part." Since in most of its extent the "plexiform part" forms a compact bundle about the axillary artery, we also speak of it as the "neurovascular cord" or "integrated cord." This fascia-bound structure is a very real anatomic entity, although somewhat vague in all its limits, since each structure that enters or leaves it contributes or carries away accompanying strands of fascia. It is a single cord closely integrated by dense fascial investment from just beyond the interscalene segments to well below the shoulder, a single cord which flares out at the base into five roots of varying sizes, and, far below the apex of the axilla, flares out again into its terminal branches.

Root paralyses. The Erb-Duchenne, or upper type of brachial plexus palsy, is the most common form of paralysis from root injury. It was described in detail with only minor differences by both Duchenne (1872) and Erb (1874). It is accepted as a paralysis of the supra- and infra-spinatus, the deltoid, the biceps, the brachialis and the brachioradialis. If the lesion is far back on the roots, we are told that the paralysis usually includes the levator anguli scapulæ, the rhomboids, and sometimes the serratus anterior and the portion of the diaphragm supplied by the phrenic nerve. If these muscles are paralyzed it clinches the root diagnosis. If they are not, it does not exclude it, for anomalies occasionally exist. Accurate statements as to the condition of the muscles are too seldom included in most of the reports of examinations of cases of brachial

paralysis. Many cases of deltoid, biceps, brachialis and coraco-brachialis paralysis have been reported as Erb-Duchenne, but inclusion of the supra- and infra-spinatus and the brachioradialis is necessary for a diagnosis of this type. The coraco-brachialis, the supinator, and, as Harris pointed out years ago, the extensor carpi radialis longus and brevis are sometimes also included in this type, as will be explained later. The pronator teres may also be involved, for it frequently is supplied from the 6th cervical nerve. When the posterior part of the deltoid, which is also probably supplied by the 6C, and the pronator teres are involved, it raises the question of inclusion of the 6th at least. Probably the innervation of the pronator teres may sometimes come from the 7C. That of the coraco-brachialis and extensor carpi radialis longus and brevis may also more often come from the 7C than from the 6C.

Harris called attention to the fact that the inclusion of the 6C in a root lesion usually added nothing to the extent of the paralysis caused by a 5C involvement, and Sherren and other observers have agreed with him. So that the 6C is to be regarded as a sort of make-shift root, its inclusion adding little to the severity of the paralysis.

The clinical term "Erb-Duchenne paralysis" has come to be used very loosely. It no longer defines, as it should, a root paralysis of the 5C and 6C, but is extended to include cases which involve the 7C. The involvement of the 7C should place the case in the complex type. The term is also applied thoughtlessly to peripheral lesions of the axillary nerve, and even to cases of lead palsy in which the axillary is involved. Moreover, the term Aran-Duchenne paralysis is sometimes confused with it, and is erroneously used. This term indicates an entirely different pathologic condition, due to a degenerative lesion of the anterior column cells. It usually first affects the small muscles of the hand and is slowly progressive, and finally at least is bilateral. The careless use of these terms, without definite statements as to exactly which muscles are paralyzed in each reported case, renders many articles worthless for purposes of detailed study.

It is usually agreed that there is remarkably little sensory disturbance with the Erb-Duchenne type, and that this is confined to the axillary distribution on the external or postero-external surface of the shoulder; but all observers are by no means in accord with this, especially Rendu. André Thomas's two cases, of cut 5C and 6C, and of cut 6C root alone, both showed a well-marked sensory disturbance, not confined to the axillary distribution.

The lower or Déjerine-Klumpke type, due always to an involvement of the 8C and 1D roots, is a combined paralysis of the flexors and extensors of the forearm with integrity of the brachioradialis, supinator, pronator teres and the extensor carpi radialis longus and brevis. The intrinsic hand muscles are paralyzed and the upper arm extensors (triceps) are partially involved. In this type sensory disturbances are much more profound. Trophic and vasomotor symptoms are sometimes pronounced, although some observers believe that in a pure brachial plexus lesion, vasomotor and trophic symptoms are not pronounced. As few such injuries are pure, this is a fine distinction. Vasomotor and trophic fibers come via the autonomic to their somatic nerve distribution.

The syndrome of Poirier, or as the French call it today, the syndrome of Claude Bernard-Horner, is present when the 1D is involved far back on the roots, and is caused by involvement of the rami communicantes which pass to the stellate ganglion. It is shown by miosis, enophthalmos and ptosis on the side of the lesion, with preservation of the light reflex and accommodation; there is normal tension or slight hypotension and normal vision, but abolition of dilatation of pupil to cocaine.

Miss Klumpke's* contribution to the subject of brachial plexus injuries has given her name to this lower root type. Her report included three cases of her own which she followed in the Clinics of Vulpian and Lucas De Championiere, but only one was of this lower type. None of her own cases was proven by operation. The rest of her eighteen cases had been reported by other men and included the case of Flaubert. Her real contribution was her experimental work on animals, by which she proved that the syndrome of Poirier or Claude Bernard-Horner followed a section of the first dorsal root back of the ramus communicans to the stellate ganglion, and that it occurred only after section of the 1D or of the ramus itself.

*Miss Augusta Klumpke (1859-1927) was the eldest of four brilliant American sisters who were born in California and later educated in Switzerland. She studied medicine in Paris and achieved the distinction of being the first woman interne to serve in the Paris hospitals. In 1885, soon after her graduation, she published an article on paralyses due to injuries of the lower roots of the brachial plexus, a type which has since been designated either as "Klumpke paralysis," or as "Déjerine-Klumpke paralysis." This confusion has arisen because five years after the publication of this paper she married Professor Déjerine, a neurologist and also a pupil of Vulpian's. They were later joint authors of numerous books and papers considered among the foremost landmarks in the progress of modern neurology. The three younger sisters also became distinguished in their chosen fields, of astronomy, music, and painting. The youngest, Miss Anna Klumpke, is widely known as an artist and as the friend and biographer of Rosa Bonheur.—E. A. C.

An intermediate type of brachial plexus paralysis, due to involvement of the 7C root alone, is practically non-existent, except as an accompaniment of either the Erb-Duchenne or the Déjerine-Klumpke types, when it would be classified as "Complex." The pure 7C paralysis would be a partial paralysis of the extensors of the arm, forearm and wrist, including the abductor longus pollicis and the extensors of the thumb. As the coraco-brachialis and extensor carpi radialis longus and brevis and the pronator teres are supplied by the 7C at times, these would therefore sometimes be included in the paralysis, but as these last come also at times from the higher roots, they might not be involved. A knowledge of the condition of the root and trunk collaterals from the 7C and the intermediate fasciculus, would not be of as great help in diagnosis as in either of the other types. The lower part of the serratus anterior might show a paresis, but as it receives branches from the 5C and 6C, it would not be entirely paralyzed. The same applies to the subscapularis and the latissimus dorsi, which, while receiving fibers from the 7C, also receives at least as great an innervation from other roots.

Complex types. As a complication of the upper type, the inclusion of the 7C adds to the usual group of muscles the following: partial paralysis of the extensors of the arm, forearm and wrist, including the extensors of the thumb; a more profound involvement of the trunk collaterals to the latissimus dorsi and teres major; the entire paralysis of the subscapularis, the coraco-brachialis, and the epicondylar muscles.

As an accompaniment of the Déjerine-Klumpke type, the inclusion of 7C is indicated by the addition of absolute paralysis of the extensors of arm, forearm and thumb to the other symptoms of a lower type paralysis. In this type the subscapularis paralysis is never entire, but the latissimus dorsi is likely to be entirely paralyzed. The pectorals are seldom entirely paralyzed except in a complete lesion of all roots. The upper part of the pectoralis major, *i.e.*, the clavicular portion, is included in an upper type, and the lower part is involved in a lower type together with the pectoralis minor.

The complete type. In very rare cases where the arm has almost been pulled off, all, or nearly all, of the roots of the plexus may be injured.

In the period from 1870 to 1890, there was much discussion, particularly among the French, as to the exact location of root injuries, *i.e.*, whether the rupture occurred within the spinal canal and involved the cord (avulsion de la moelle épinière), or in the bony

gutters formed by the transverse processes, or between the point of exit from these gutters and the point where the roots join to form the fasciculi. The term "radiculo-médullaire" was also used in an almost synonymous sense with avulsion. It is probable that today the French neurologists may have dropped these terms or attached other shades of meaning to them than those which we have interpreted. However, we must present these terms because they have been so much used, although the writer is satisfied from his own observations on the cadaver that nearly all injuries must occur entirely outside of even the bony gutters, because careful dissection shows that the roots are always snubbed at the transverse processes, *i.e.*, attached to them by fascial connections. The reasons for believing that this snubbing will usually prevent injury to the roots within the canal, or even within the bony gutters, will be given later.

Some authors (*e.g.*, Shallow) have found at operation cysts containing cerebrospinal fluid close to the transverse processes, and have concluded that this was evidence of an evulsed root. This does not seem to me sound, because tubular processes from the dura not infrequently extend beyond the transverse processes and would be ruptured with the snubbing. (Fig. 63.)

Gross anatomy of the intact plexus. Before we take up in detail the study of the distribution of the nerves to the various muscles of the arm, it is necessary to consider the plexus as a gross unit, for we must always remember that in the body it has not been dissected and does not lie in the form of a diagram.

In the last sixteen particularly careful observations I found that each root of the brachial plexus was held firmly at the transverse processes and in the gutters of bone by invaginations of the prevertebral fascia. In the words of the mechanical engineer, each root is snubbed at the transverse processes. There it is firmly fixed. If it were not, and there was nothing to take the ordinary strain away from the spinal cord itself, a man might be paralyzed at any time by a strong pull upon either the arm or shoulder.

Below the clavicle, the cords of the plexus are firmly bound together and also to the surrounding structures, including the clavicle and the coracoid, by layers of the clavo-pectoral fascia. The three main cords join the subclavian artery as soon as it appears above the first rib, and remain integral with it in its course through the axilla. The clavo-pectoral fascia comes down from the back of the fascia which surrounds the subclavius muscle, forming the costo-coracoid membrane. This membrane goes to the coracoid and joins

the axillary fascia below. The clavo-pectoral fascia leaves the back of the costocoracoid membrane, passes between and invests each branch of the brachial plexus, surrounds the axillary artery and is continuous above with the prevertebral and below with the deep fascia over the first rib. Therefore, the integrated neurovascular cord is snubbed again, as well as at the transverse processes, by these fascial investments between the clavicle, the coracoid and the first rib; that is, in the apex of the axilla.

I should like to accent the facts in the preceding paragraphs because I believe that these points of attachment help to explain the different types of lesions resulting from a blow or stress above the clavicle, and also those resulting from stresses which come through the arm from below, either as straight tension, or as lateral stresses resulting from dislocation. I have been interested in the mechanics of the shoulder for many years, and have studied on the cadaver the probable relation of the plexus to the displaced bones in cases of fracture and of dislocation, and the conclusion has been reached that the explanations found in the literature of the mechanism of the involvement of the plexus in these injuries are not adequate.

I shall discuss the theories which have been offered to explain the mechanics of the more common lesions of the plexus from trauma, and then offer one which I believe shows why the plexus is either injured at its roots beyond their exit from the spine and before they join with others to form the plexus, or in its terminal branches, and never (except by cuts or wounds) in the plexiform part, which, nevertheless, is usually involved secondarily by hemorrhage and exudate between the nerve bundles.

Most observers, up to the time of Horsley, believed in a local injury to the brachial plexus, both in birth and adult cases. Erb believed that the injury was due to clavicular compression, but this idea was gradually destroyed by Gerdy, Fieux, and Horsley. The idea of root lesions originated with Seerétan, and thereafter there was a division of these cases into root, trunk and terminal lesions.

To Gerdy is given credit by the French of the traction theory, that is, straight pull as the cause of these paralyses, although Malgaigne knew it long before this, for he had paralyzed two cases himself in reducing dislocations by a direct pull of 200 to 250 kilos (more than 500 pounds). Flaubert must have known it, since he had thus caused paralysis not only in his celebrated case of 1827, but also in two others, by direct traction. Various experiments have been made, and a great amount of time has been spent on the me-

chanics of these lesions. The case of Flaubert was the starting point of the avulsion theory, that is, the separating or pulling out of the roots from the spinal marrow itself. This, we contend, is rare except in very violent injuries.

To Horsley, in England, is credited the idea that the lesions are caused by injuries which tend to spread the head and shoulder apart and thus to stretch the plexus. Today this is generally accepted as the most likely theory of production. Horsley's experiments are not given in detail, but in the *Practitioner*, of London, he said that by dropping cadavers on head and shoulder, he had been able to break the plexus.

Duval and Guillain, in 1898, made an extensive study of the plexus anatomically, carefully estimating the different angles formed by the individual cords from their origin at the spinal marrow to their emergence under the transverse processes of the vertebræ and across the neck to the arm. They assumed a transmission of stress by tension to the roots, which, because of their angulation, was more or less expended as *écrassurage* (crushing), at the points where the roots angle around the transverse processes. They held that this action was enhanced on the lower roots by elevation of the arm, and especially on the first dorsal root as it wound around the neck of the first rib. They believed also that the force was transmitted far back even to the spinal marrow in most cases.

In dislocation they believed that traction fell on the roots at the same time that it caused the dislocation, rather than that lateral stress was caused by the dislocating head. Most of the French and German profession accepted this explanation, and the idea that a supraclavicular, even radiculo-medullary lesion took place in most of these brachial plexus injuries became more and more engrafted in the minds of surgeons and neurologists.

It remained for Delbert and Cauchoix to point out in a very wonderful paper in the *Rev. de Chir.*, in 1910, that many of these supposedly supraclavicular lesions that had been diagnosed as radiculo-medullary, root or trunk lesions were in reality lesions of the axillary portion of the plexus; that is, terminal, and especially so when they accompanied dislocation of the shoulder. They reported three operated cases of their own, and the rest were interpretations of cases previously reported by others; but only a few were checked cases. They believed that the injuries were high up, *i.e.*, root lesions, or low down and terminal, but not of the plexiform part. In this last conclusion we agree. They studied thirty-six cases, all dislocations;

and they believed, as did Duval and Guillain, that the nerve lesions were due to the same stresses which caused the dislocation, rather than to lateral stress from the displaced head of the humerus. Their paper never has received the attention in America to which it is entitled. It is probably true today, when one speaks of a brachial plexus paralysis, that the majority of American surgeons visualize a lesion of the nerves of the plexus high up above the clavicle, either trunk or root or even radiculo-medullary, and give little thought to the possibility of infraclavicular peripheral injuries, especially to those of the axillary nerve.

There has been, we believe, a misconception in the minds of the profession regarding the frequency of brachial plexus rupture. True rupture, with complete separation of the torn ends, is a rare lesion either above or below the clavicle, whether of root or terminal branch, as compared to cases of injury without rupture. The cases of proved rupture, although few, are extremely important, both as to their mode of production and as to the exact point of the plexus which has suffered the separation, because from a study of these proven cases, it is easier to understand where the acme of stress is most likely to fall, in those less severe and much more common cases where paralysis supervenes without actual loss of continuity in any part of the plexus.

It has been said by various authors that Bowlby was able to collect nineteen cases of frankly supraclavicular rupture of the plexus, that Bristow added three more proven cases, and that Frazier and Skillern were able to collect records of twenty-one cases of actual supraclavicular rupture. Examination of all these original papers shows that Bowlby reported nineteen cases of brachial plexus paralysis, but only three of them were checked, namely, one case of his own, where operation was in the axilla and nothing was found; the case of Banks of actual proven rupture, which had already been reported; and the old case of Flaubert of actual avulsion, which has been reported so many times. Bristow reported one case of his own of actual rupture, and another case of actual rupture which had already been reported by Hartley. His third case was by no means proven; he attributed it to Senn because Senn had told him that he (Senn) had had a case. No details of this, whether proven or not, were included. Frazier and Skillern reported only one case of their own of actual rupture, and the celebrated case also reported by Mills; their others were cases from literature without detail, and again included the case of Flaubert. Frazier simply said that in the literature he had

found records of twenty-one cases with proven rupture of one or more roots, without skeletal injury. It may, therefore, be understood upon how little foundation theories as to the causation of brachial plexus ruptures now rest.

DR. STEVENS' THEORY OF MECHANISM OF PRODUCTION OF BRACHIAL PLEXUS INJURIES

A cord to be broken in tension, whether by a blow on its side transmitting the stress to both ends, or by direct pull, must be held firmly at the ends or there will be no tension. The brachial plexus is so held by the fascia, as we have shown in our dissections and by the description already given.

All cases of plexus injury of the type under discussion are due to tension, that is, traction. It makes no difference as to the basic mechanical stress whether a man slips carrying a heavy load; whether he is struck by a blow, depressing his shoulder; whether there is straight traction on the arm; whether his head is forced away from his shoulder; whether his face is rotated away from or toward the side undergoing stress; whether the arm is pulled in abduction and in external rotation; whether or not his arm is raised, lowered, supinated or pronated; if the stress is reflected on the cords of the plexus, it is tension. However, the different varieties of stress and the relative position of the arm and head at the time of stress, make tremendous differences in the kinds of lesion suffered, in the locality of the lesions and in prognosis. The nature, location, and seriousness of the injury, I believe, depend on whether or not the stress is received from above or is transmitted from below; whether or not the arm is above or below horizontal, and whether or not externally or internally rotated. These factors, together with velocity and magnitude of stress, determine, in most cases, the severity and the locality of the injury. Nevertheless, in all cases the stresses are of the same nature.

Consider the cords of the brachial plexus as a traction apparatus with its normal axis as a mechanical appliance on the 7C vertebra, with the arm at the horizontal, *i.e.*, a single cord with five separate points of attachment firmly snubbed at the transverse processes.

When tension is applied to this structure (the integrated cord), it falls on the offset roots. Any mechanical engineer will tell you that an arrangement of this kind will rarely transmit stress through five cords equally. If the force of the pull could fall exactly through the neutral axis at the exact center, and at an exact right angle to

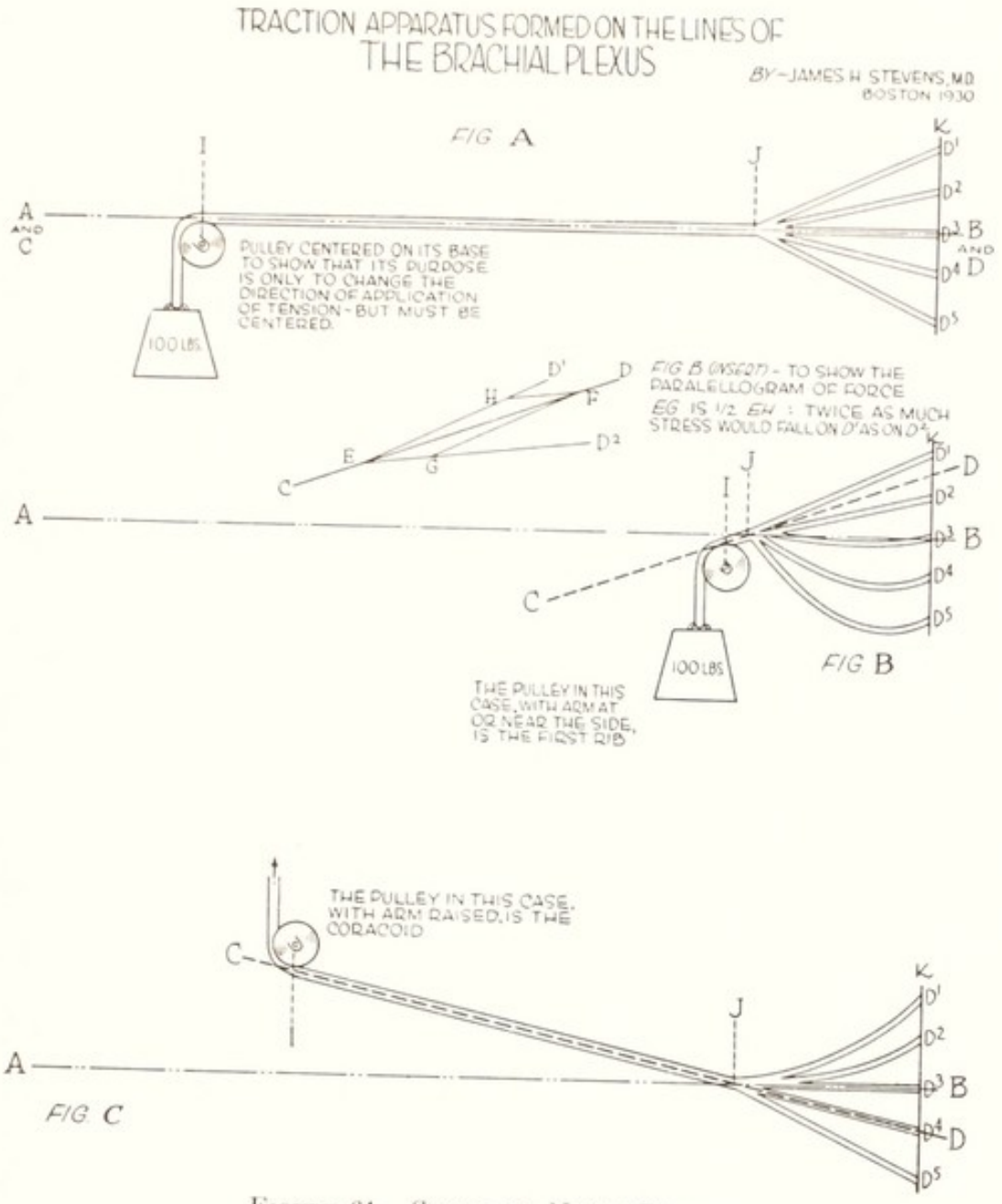


FIGURE 64. STRESS ON NERVE ROOTS

the base or plane of the structure to be lifted, the size of the cords being the same, it might be possible to lift a weight evenly, but he will also tell you that stress always tends to travel in straight lines, and depending on the position of the application of stress, the acme will usually fall to one side or the other of the neutral axis of such a structure.

A suspension apparatus is governed by much the same laws as a traction apparatus. In engineering a three-point suspension is more reliable than a suspension from a greater number of points. Perhaps this is the reason why three roots are injured in so many cases of brachial plexus paralysis, for either the two upper roots or the two lower ones may combine with the median root.

A traction apparatus must have a neutral axis and a line of resistance, and when the force of traction falls through this neutral center or axis, the traction is equally borne by all parts of the apparatus. Even a slight deviation from this neutral axis makes an offset pull to one side or the other, and in a structure of this kind, if the line of tension falls outside the neutral axis, as represented by the line of resistance, the entire force is transferred from that neutral axis; all tension is released on the cords on the other side, and a new neutral axis is instantly formed about the components which are now bearing the stress, to conform to the new line of resistance. All other components are out of the structure; they are lax and their influence is nil.

A pulley inserted as part of a traction apparatus is not placed so as to change the degree of pull on the structure to be lifted or moved. It is placed there in order to change the direction of the application of the force in order to make it more convenient or effectual, *i.e.*, to keep the neutral axis in the desired direction. The pulley must be so placed that a line from the pulley to the center of the structure to be raised or moved, falls through the neutral axis and the line of resistance. If this is not so, the force applied falls to one or the other side of the axis and the entire force of the pull may, therefore, fall to one side. If you elevate the pulley, the lines of tension and resistance will come below the neutral axis as it existed at first, and the acme of stress will be below. If you lower the pulley, the acme of stress will be above. Since the scapula is movable and the integrated cord passes under the arch formed by the coracoid and the pectoralis minor, a condition similar to a movable pulley exists in the shoulder.

There is no real pulley, but the cords of the plexus are held in this arch and, as the arm is raised and the clavicle and the coracoid rise, the latter acts much like a pulley, for it changes the direction of any force applied distal to it. In raising the arm, when the coracoid rises above the horizontal, the acme of stress would come on the lower roots. (Fig. 64*c*.) Lowering the arm lowers the coracoid, and the acme of stress is on the upper roots. At a horizontal, the neutral axis is near the 7C vertebra, and all roots might be stressed evenly, because the neutral axis, the line of tension and the line of resistance correspond. As a matter of fact, when the continuity of the bones is intact, the range of the coracoid is such that like a self-adjusting pulley it maintains the direction of pull very nearly in the neutral axis, as the arm moves.

As an exaggerated example of how the coracoid can change the direction of stress, suppose a man caught in a machine in such a manner that, while the arm is abducted in the scarecrow position, two parts of the machine travel in opposite directions, one pressing the elbow upward and the other part pressing the shoulder downward. The coracoid will be depressed, hooked over the taut plexus, and the force transmitted to the upper roots. The stress would be downward on the upper roots, even if the pressure above the shoulder remained fixed and only the elbow continued to be forced upward.

With the arm at the side and pulled downward, the pulley is not the coracoid, but the place where the plexus comes over the first rib anteriorly. This slight change of direction of the force would relieve, to some extent, the strain on the upper roots in a downward pull. Combined with this would be help from the clavo-pectoral fascia.

A breaking strain expended on the brachial plexus from above, as from a blow on the shoulder, or the stress suffered when one slips while carrying a weight, usually should cause a lesion of the 5C root. Five cords divided will not stand the strain as well as when they are combined in one.

The apparatus will break at the weakest point, *i.e.*, at one of the roots between the point where it is snubbed on the transverse process and the junction of that root with others. It breaks there, or it does not break at all in supraclavicular stresses. If it does not break, nevertheless the acme of stress is at the same point.

In this case of a blow from above on the neck or shoulder, or a slip while carrying a weight on the shoulder, the integrated cord is stretched, and the stress in both cases is transmitted to the points of firm attachment of the cord at both ends. Here is impact, and impact is infinitely greater than static load. A man carrying one hundred pounds on his shoulder slips or makes a false step, and falls even four to six inches. Instantly his static load, which he could carry with ease, becomes an impact trauma. The mechanical stress suffered here is exactly the same as it would be if he were struck on the shoulder by a hundred pound hammer falling through four to six inches and depressing the clavicle and the coracoid. The brachial plexus is instantly stretched between its two firm points of attachment, which are, as we have shown, at the transverse processes above, and at the clavo-pectoral fascial snubbing below, in the upper axilla. Again, the stress in tension is exactly the same as if the cords themselves received a side blow. Impact, as the mechanical engineer will tell you, so increases a stress over static load as to be almost unbelievable. (See Merriman, *Mechanics of Motion.*)

When surgeons contend that rupture of the roots would be impossible in the ordinary trauma, or in that due to dislocation, they fail to take into account the manner in which mechanical stresses may be magnified at the point of final application when they fall on structures of limited area. The reason that the roots do not always break, is that the stress does not fall on these limited structures alone in many cases. It is disseminated. In both cases cited above, the stress is transmitted to both points of attachment. One half is referred back to the roots, the other half falls on the place below, where the cords are held firmly by the clavo-pectoral fascia. So that sometimes, even in this type, we have an accompanying rupture or injury to the artery, because the fascial snubbing surrounding the cords gives way and the vessel is torn. Injury to the artery probably happens less often in case of blows from above than when the stress comes through the arm from below as pure tension, because in the latter case, the entire force of the pull is not divided. It falls first on the clavo-pectoral fascial snubbing, and is then transmitted back to the pulley at the first rib and the roots, but not until the fascial snubbing in the axilla has been injured. It is quite possible that we might have traumatic aneurism without rupture of the roots, because the stress might be disseminated after the fascia and the vessels have been injured, and then be too weak to break the roots.

In a very careful review of the literature the writer has been unable to find a single case of rupture in the plexiform part of the plexus, *i.e.*, in that part which has been alluded to as the neurovascular cord, although the artery itself may be torn. Even proved injuries of this portion of the plexus from bullets or cuts are rare, but we are speaking now of rupture from trauma without a penetrating wound. In reading the accounts of operations, on the other hand, one usually finds such statements as "the plexus seemed a mass of scar tissue," "the cords were welded together in an inflammatory mass," "on account of the scar tissue, nothing could be made out as to the exact location of the injury," etc., etc. Yet many such cases have recovered in whole or in part, indicating that no real rupture had occurred in the nerve fibers, and that the gross appearances were due to ecchymoses, exudate, or scar amongst the fascial envelopes and fibrous septa in the nerve trunks. In this region, as in others, intensive anatomic study of the mechanics of the structures reveals marvellous examples of architectural and mechanical designs, *e.g.*, the integrated cord when dissected is "plexiform," after the fashion of a complex design of parallelograms of forces. This is an admirable arrangement to disseminate stresses, for if the cord were pulled

at both ends, before rupturing the longitudinal strands, the force must break the little lateral bands of tissue which we cut when we dissect the plexus. It is probable that many little local hemorrhages, about, amongst, between, and within the individual trunks, cause the appearances so frequently described. Subsequent exudate and scar tissue complicate the picture, and choke the nerve fibers, usually temporarily only.

I believe the explanation of these injuries to the plexus is this simple mechanical one; in any case it has nothing whatever to do with the idea of *écrassurage* against either rib or transverse process. The stress will not be "radiculo-médullaire," except in very rare cases of sudden, violent injury, because to be so, the mechanical apparatus would have to break back of the snubbing, and that is contrary to the laws of mechanics. The snubbing is stronger than any individual root, but it is not stronger than the entire integrated cord.

If the stress comes from below through tension on the arm, there must be some separation of the bony framework at the shoulder, before stress can be transmitted to the soft tissues. For instance, the clavicle might be broken, or the humeral head might be pulled away from the glenoid cavity, or when dislocated, pried against the plexus. In postoperative paralysis in breast cases, the muscles are relaxed and the plexus assumes the stress. In cases of fracture of the clavicle when paralysis is a complication, we should think first of a root lesion, and only secondly of injury to the cords from actual contact with fragments.

Estimate of Comparative Strain on Integrated Cord and Root.

Some idea of the strain to which a root is subjected in one of these injuries, in comparison to that sustained by the integrated cord, may be obtained by reference to Fig. 64.

If the pull remains the same the stress per square inch becomes rapidly greater the smaller the cord. Engineers use the formula $\frac{\text{Pull}}{Pi\,R^2}$ = stress per square inch. By using this formula and assuming the integrated cord comprising the artery, fascia and nerve trunks to be one-half inch in diameter, we find that a pull of one hundred pounds would exert over five hundred pounds stress per square inch on the cord as a whole. If all the other components were removed and this stress fell on one nerve root one-tenth of an inch in diam-

eter, this root would have to receive over 12,000 pounds of stress per square inch of its cross section, which, of course, would rupture it. If the stress fell on two roots, it would be divided. In order to determine the comparative stress on these two roots, we use the parallelogram of force of the mechanical engineer, Fig. B. Therefore, if it is a breaking stress, the roots never break together; one gives, and then the entire stress falls on the next, etc., until dissemination of force stops the process. With stresses as above, why do these cords not always break? Simply because these stresses do not often fall on the cords in this way. Other structures, bones, ligaments, muscles, tendons, fascia, etc., receive some stress, but the bulk of the stress *may* thus fall on one root or two roots, and sometimes does.

Considerations such as these make us wonder at the remarkable arrangements which nature has supplied to prevent stress falling on any one root of the plexus. When a mother drags her child across the street by one hand, there are many anatomic structures which protect the plexus. The bones and ligaments form a chain which takes most of the stress. Should there be a loss of continuity in this chain, the coracoid process acts as a pulley to distribute the stress in the neutral axis of all the roots. Even when the stress arrives at the roots, the two lower ones join together and the two upper roots join together before they unite with the middle root; thus, the upper cord and the lower cord each form a two-point suspension, and the middle root is somewhat protected. When the two points formed by the upper and lower pairs unite with the 7th or middle root, we have a three-point suspension. These three-point suspensions then form the plexus, which is so arranged that each main cord and nerve is attached to its neighbors by fascial binding, uniting them into a trunk capable of sustaining much stress. Even then, when they pull apart, the fascial bindings between the cords must be torn before any individual cord may be torn. However, there are occasions when, owing to rupture in the continuity of the supporting structures, all stress will be thrown on either the uppermost or lowest root. Even then there remains the snubbing of the fibrous envelope of the nerves to the transverse processes to protect the spinal portions of the roots. Fainting, anæsthesia, or other forms of unconsciousness which relax the muscles remove a very important factor in protection.

The Theory Applied to Ruptures of the Terminal Branches.

Let us now consider the stresses which often result in terminal branch lesions. Imagine an arm in abduction and external rotation

subjected to still greater backward stress.* This is the position of dislocation, and lateral pressure on the taut plexus takes the place of pure tension on the whole arm structure. By this position of abduction and external rotation you have separated the terminal branches of your cord as much as possible, and if lateral pressure is applied, your apparatus is no longer a single cord with five lesser points of attachment where it is joined to the transverse processes; it now has six or eight separate smaller cords below, any one of which may have to take the major stress. In other words, you have added other factors to your mechanics. Offset may now fall, not alone on the roots, but at the other end of your apparatus. If it falls on any terminal branch that is smaller or weaker than any part of the integrated cord or roots above, it will break that terminal branch below its point of insertion into the integrated plexiform part. If, however, the main stress should fall through the radial nerve, which is greater in strength and size than most of the roots, it would probably be transmitted back to the roots as the weaker points. I have found in many dissections that the posterior or intermediate cord is as high in the neck as the lateral fasciculus. Force applied to the radial would be transmitted through the integrated cord back to the 5C and 6C to a greater degree than to the 7C, from which its motor portion arises originally. The weaker points are below and at the roots. This is why there have been ruptures reported of the musculocutaneous nerve, of the median heads and of the ulnar, at their origins in the axilla. We have many reports of injury involving each of these nerves in the axilla (forty-four operated cases), but there is no proven case of absolute rupture of the radial alone. Why? Because it is stronger than the clavo-pectoral fascia which binds it, and while it is weaker than the plexiform part of the plexus, and would break before that, it is stronger than the roots themselves on which individually, because of offset, the stress may also fall.

When we speak of a root, or a cord, or a nerve, as stronger or weaker, we mean that cord or nerve bound with connective tissue. No root or cord breaks until after its sheath has given way. No nerve can be stressed until its surrounding fascia gives.

The fact that fifteen cases of subclavian or axillary aneurism have been reported in conjunction with 135 injuries to the plexus, suggests that the clavo-pectoral fascia surrounding the integrated

*In this position the terminal branches tend to separate from one another, and in the opposite position (internal rotation with the arm at the side) to continue their course almost side by side.—E. A. C.

cord is usually stressed and often actually torn. In most of these cases there was only one incision, either a supra- or an infra-clavicular one, so that the conjunction of high and low lesions is probably greater than indicated in these figures. If in each case both incisions had been made, a still greater coincidence of fascial and arterial injury might have been shown.

Application of These Principles to Axillary (Circumflex) Nerve Injuries.

In dislocation of the head of the humerus, we have the cause of the greater number of axillary nerve paralyses. We have at times in addition injury to each of the other nerves in the axilla, and through the radial, for the reasons stated above, we may also have as a result of the stress of the rotating head, injury transmitted back to the roots. The axillary nerve is most often injured because in dislocation it is tensed across the head in the erect phase when the arm is elevated and externally rotated. The axillary is the one nerve which at this point has already separated, and is an individual cord. It is closely applied to the anterior surface of the subscapularis muscle, and is carried with the head when it turns out of the lower part of the glenoid. Since the subscapularis tendon is anterior, and firmly attached to the lesser tuberosity, this nerve must be more or less stretched in every case of anterior dislocation. The axillary is particularly vulnerable because it is firmly attached above to the main cord, and is snubbed below, where it passes back to wind around the neck of the humerus. Therefore, it is a short length of nerve subjected to a side stress, which is again nothing more than tension transmitted to both ends. The wonder is that it is not always paralyzed in cases of dislocation.

Confined solely to the axillary nerve would the stress be transmitted back to the roots? It would not, for the axillary is weaker than the plexiform part of the plexus, and it is weaker than any root. The axillary is stressed most often alone, not enough to rupture it, but enough to paralyze the deltoid. If it breaks, it will break between the place where it leaves the intermediate fasciculus and the place where it passes between the subscapularis and the teres major muscles, and nowhere else, because this is the weakest point. This accounts for the fact that simple deltoid paralysis is far the most common nerve lesion complicating dislocation.*

*In such cases the patient might still be able to elevate the arm with the supraspinatus.—E. A. C.

In our series from the literature, there were only three cases of actual rupture of the axillary nerve, aside from many cases of injury where the exact pathology was not determined by the operation. In addition, Weir-Mitchell reported nine cases of ruptured axillary, years ago, but they were not checked by operation.

The cases of *musculocutaneous paralysis* (aside from rupture), which have been reported, may be, and sometimes are, due to the mechanics already described. We believe they may be caused also by the pressure of a dislocated head which has been left out for a long time, although the nerve may be stressed by the rotating head in dislocation, especially in the first phase, exactly as all the others may be stressed. The idea that these nerves slide up and slip over the head of the rotating bone with any degree of facility is absurd. The only way they slip over the head is because the head of the bone is rotated under them, in spite of tension, and in spite of their being plastered in place by the tremendous contractions of surrounding muscles which are resisting injury. This nerve is also snubbed at its junction with the integrated cord above, and it is snubbed where it passes through the layers of fascia both internally to the biceps and beneath it, and where it usually passes through the coraco-brachialis muscle. Like the axillary it is a short cord which is being tensed by a side stress. It may be injured in this way or even ruptured, but we believe that many times as the dislocated arm falls back to the side, the musculocutaneous nerve, in this new position, is unable to free itself, and remains tensed over the dislocated head, until reduction frees it.

A case of subcoracoid dislocation of the right shoulder, caused by a fall, was observed by the writer in 1929. It was three hours before the dislocation was reduced, during which time the patient swung his arm, because he said that although it hurt him to do so, it felt better when he did it. He had a sensation of pins and needles during this time in arm and forearm. Reduction was not followed by abduction treatment, but the arm was supported by a swathe and sling. When he returned four days later, paralysis of the biceps, brachialis and coraco-brachialis was found. Two weeks later the reaction of degeneration was present in these muscles. No others were paralyzed. Epicritic loss was greater than protopathic in the domain of the musculocutaneous nerve. The absence of brachioradialis and supinator brevis paralysis, the absence of supraspinatus, infraspinatus, deltoid or teres minor paralysis, the presence of coraco-brachialis paralysis and the fact that paralysis did not occur at once, make it probable that this was an injury of the nerve in its axillary portion. It is best explained by compression from infiltrate which came as the result of long-continued pressure. First voluntary motion appeared at the end of eight weeks, and recovery was very rapid thereafter.

Although they are very common, very few cases of isolated deltoid paralyses, or combined paralyses of the deltoid, biceps and brachialis, appear in the literature, because many of them are mild and recover in a few months. Nevertheless, I believe that axillary injuries of the brachial plexus are much more numerous than supraclavicular injuries, and also, in the main, less serious. In this instance, ordinary practical experience is of more value than recorded literature. *Lesions of the plexus caused by, or at the time of, dislocation usually soon get well; cases in which recovery is prolonged or eventually imperfect are those in which the shoulder as a mass has been suddenly depressed.* Thus I believe that with a knowledge of the actual trauma which was sustained, it is possible to arrive at a better conclusion as to prognosis than it is from the clinical symptoms alone.

Avulsion of the nerve roots from the spinal cord ("radiculo-médullaire") must be very rare. There are only four cases of proven avulsion which I have been able to find in the literature and I shall speak of these later. In these four cases at least there can be no doubt of the lesion. How can we explain the mechanics of the avulsion cases, if, as we have said, the anatomical arrangements predispose toward more peripheral injuries? The simplest explanation is this: The mechanical engineer will tell you that in the face of tremendous stress, where velocity of stress is great, all rules governing stresses and strains fail. In the face of a tremendous stress with velocity, the snubbing of the roots at the transverse processes would give way and the stress be then applied to the nerve fibers within the vertebral canal. It is significant in these four cases that in not one was the 5C broken. It is evident in all that the stress fell through the 7C and the 8C, which are certainly the largest of the roots. The cause in each case was a direct pull in nearly the neutral axis. It was pure tension by pulling upon the arm in four cases, and in three of the four cases, at least, it was a tremendous stress, and probably so in the other. In all cases the arm was at or above a horizontal. In the case of Frazier, the arm was raised and the element of pure traction was evoked through the lower roots. In no other way can the escape of the 5C root be understood. In our opinion, there would always be in any real case of avulsion, mild or severe, cord symptoms which would be diagnostic. A modified syndrome of Brown-Sequard should be present below the spinal level of the lesion.

Detail of Nerve Paths in the Plexus.

Now that we have seen how traction may be distributed and, through the integrated cord, affect one root or another according to the direction of the pull, it is time to consider the detail of the nerve paths through each root and cord to individual muscles.

Such knowledge as we have of the distribution to the various muscles of the arm of the nerves which form the plexus, has been obtained with much labor in the past by those who approached the problem in three different ways, namely:

(1) Dissections on the cadaver to determine the exact anatomic pathways of nerve fibers from the spinal cord through the intricacies of the plexus to each muscle.

(2) Study of the cases recorded in the literature where definite paralyses of the different muscles have been recorded, and later were shown, by operation or autopsy, to be due to definite lesions of the plexus.

(3) Experimental studies in animals where lesions were made by operation and the resulting paralysis in the muscles carefully recorded, or where electric stimulation at different points in the plexus caused contraction of individual muscles.

Much of our knowledge has come from studies made by Paget, Jonathan Hutchinson, Déjerine-Klumpke, Herringham, Seeligmüller, Erb, Duchenne, Bardenheuer, *et al.*, in the era prior to the general adoption of antiseptic technique in surgery, a discovery which so broadened the interests of medicine that other fields more promising than plexus injuries were opened up. However, there is always some one interested in every problem, and to this one Harris, Sherren, Sherrington, Claude, Marie, Thomas, Henri Meige, *et al.*, have made notable contributions in more recent years. It has interested me to make a thorough review of the literature, to collect and study as a group all of the reported cases I could find, and to compare the various diagrams of the plexus presented by different authors, especially those of Kocher, Harris, Kerr, and the many anatomists. While the diagrams present a notable unanimity in general, there is much disagreement in detail, owing to the fact that there actually exists a considerable degree of anatomic variation.

To satisfy my own mind I have made ninety-two dissections of the plexus and have recorded my findings. Fig. 65 shows what I believe to be as accurate a diagram as can be made of a structure which is subject to so many minor variations and which, as those of us who have made dissections best know, may be so easily altered or distorted by our own manipulations.

FIGURE 65

This chart was made personally by Dr. Stevens, as well as the notes which accompany it.

I have not attempted to correct, or in any way change, the chart, although the terminology is not the one in use by anatomists at the present date. Dr. Stevens was accustomed to the old terminology, but in some instances used the more up-to-date one. I believe that any earnest student of this subject could change these terms as accurately as I might. If I attempted corrections I might make a mistake, for I am not as learned in the subject as was Dr. Stevens, although there are some obvious errors in minor details. As the notes were unfinished I have been obliged to correct them to some extent, although I have not checked them with the writings of the authorities quoted.—E. A. C.

In this scheme only dominant roots, or branches which have a large part in the innervation of the individual muscles, are given consideration.

NOTE No. 1.—The Coraco-brachialis, according to Piersol, is supplied from the 7C root; the Manual of Neuro Surgery, issued by the War Department, 1919, states from the 5C root. Harris in his "postfixed plexus" gives an 8C branch, but there is no record in the literature of a lower root injury accompanied by paralysis of the coraco-brachialis. I have never seen such a connection in the cadaver. Braus gives 6C and 7C. Spalteholz gives 6C and 7C, with 7C dominant. I believe 5C and 6C in many cases (and when so, the 5C and 6C are dominant), but more often it is from the 7C, in which case the 7C becomes the dominant root. Many times it comes from the 5C, but probably usually is from the 7C via the pathway to the lateral fasciculus. At times there is a branch from the intermediate fasciculus to the coraco-brachialis by another pathway, as given by Harris.

NOTE No. 2.—The Manual of the War Department gives the Adductor Pollicis as from the 6C and 7C root, which is, of course, quite impossible, except via a lateral ulnar head. Other anatomists give the 8C and 1D, which is probably right, and to this we adhere. Stewart gives 8C and 1D; Herringham, 8C; Harris, 1D; Braus and Spalteholz, 8C and 1D. The dominant root is the 1D. See Recaldoni and Pfeiffer and Ransom cases.

NOTE No. 3.—Pronator Quadratus. War Department gives a 6C in addition to the 8C and 1D, and Piersol gives the 7C in place of the 6C of the War Department. Herringham gives 7C, 8C and 1D. Braus, 7C-8C and 1D. I am convinced that it does not come from the 7C. 8C is dominant and the influence of 1D is little, if any; therefore it is not given on the chart.

NOTE No. 4.—The 1st and 2nd Lumbricales are given by Piersol as 6C and 7C. War Department only the 7C; Herringham, 8C; Stewart, 8C and 1D; Harris, 1D; Spalteholz, 7C-8C-1D; Braus, 8C and 1D. Our scheme is the 8C and the 1D, for all the lumbricales, and 1D is dominant for the 3rd and 4th lumbricales.

NOTE No. 5.—The War Department gives the Flexor Carpi Ulnaris as having a 7C branch. If so, it would have to go via the decussation to the lateral fasciculus, and could not possibly reach the ulnar, save by the lateral ulnar head. We have, therefore, in our scheme given the 8C and 1D (perhaps). Influence of 1D must be little, if any, so it is not included on the chart. The lateral ulnar head in our opinion is only sensory from the 7C. Braus gives 7C-8C and 1D; Stewart, 7C; Harris, 8C and 1D; Herringham, 7C-8C-1D; Spalteholz, 8C and 1D.

NOTE No. 6.—The Flexor Longus Pollicis is given by the War Department as 6C and 7C; as 8C and 1D by Piersol; Herringham, 7C-8C and 1D; Harris, 8C and 1D; Stewart, 7C; Spalteholz and Braus, both 6C-7C-8C. We have adhered here to the 8C. No cases of injury to the lateral fasciculus or upper roots have ever been reported as showing injury to the flexor longus pollicis; and where the 1D has been involved, the flexor longus pollicis has usually not been included.

Notes continued on back of chart.

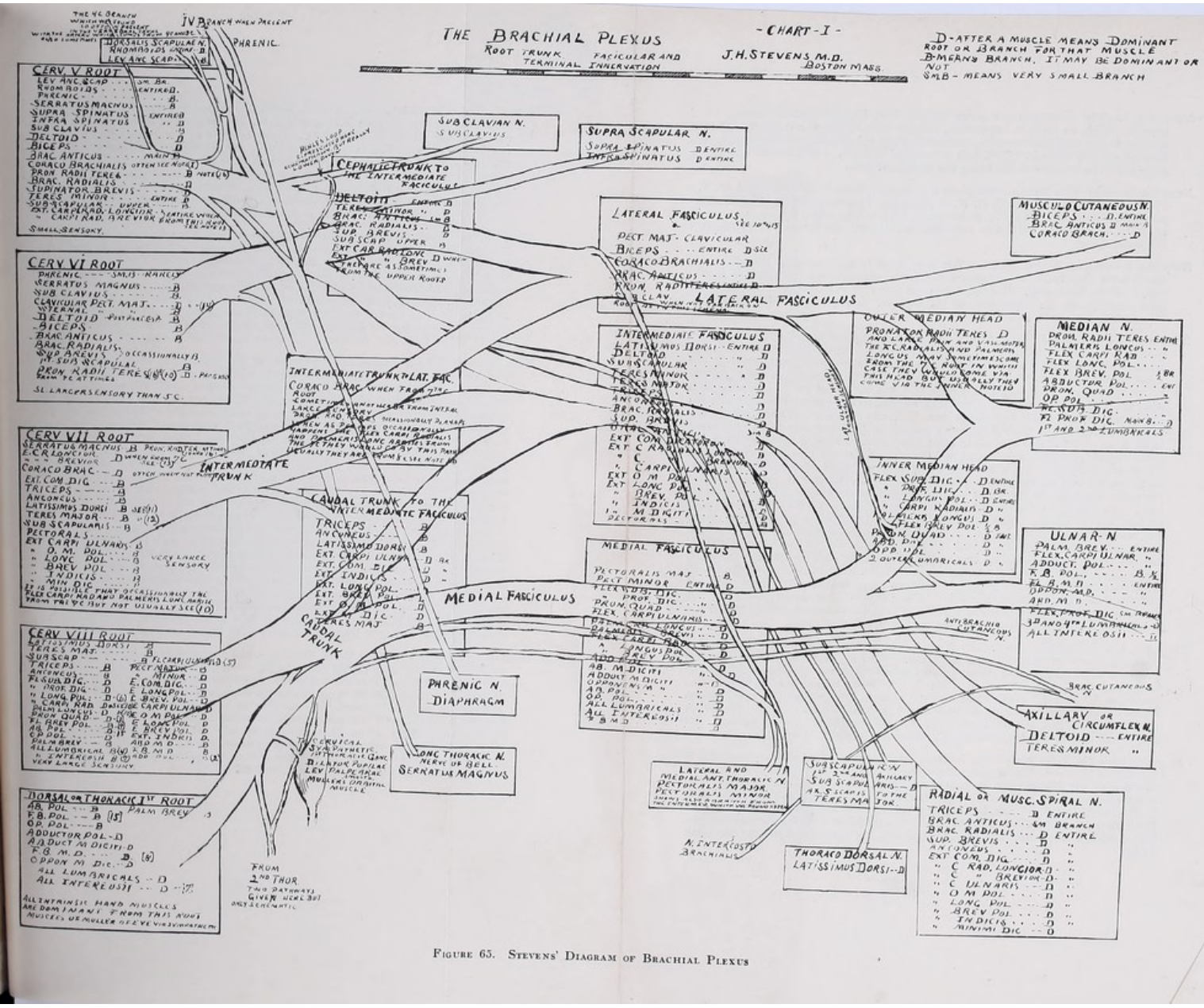


FIGURE 65. STEVENS' DIAGRAM OF BRACHIAL PLEXUS

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Note No. 7.—The Interossei are given by the War Department as having a 7C branch, which we believe impossible, save in rare cases where the 7C gives a branch to the medial fasciculus. Stewart gives 8C and 1D; Herringham, 8C; Harris, 1D; Spalteholz, 7C-8C-1D; Braus, 8C and 1D. I believe 8C and 1D is more likely to be correct, 1D being the dominant root.

Note No. 8.—The War Department gives the (Flexor Brevis Minimi Digiti, O. T.) Flexor Brevis Digiti Quinti, B. N. A., and the Opponens Digiti Quinti (Opponens Minimi Digiti, O. T.) as having a 7C branch, which it seems to me is unlikely. Spalteholz and Braus both give 7C-8C-1D. We favor 8C and 1D, and 1D is dominant

Note No. 9.—Flexor Brevis Pollicis. The inner head is innervated by the ulnar. In our scheme this head is considered as one of the interossei; therefore innervated as the interossei. The outer head is possibly 8C, and, if so, the nerve-path must go via the inner head of the median. Spalteholz, 7C-8C-1D; Braus, 6C and 7C; I favor 8C and 1D.

Note No. 10.—Palmaris Longus. The War Department gives 7C, 8C and 1D; Piersol gives 6C. Probably 8C takes a straight course to the median, and this is the scheme to which we have adhered. The flexor carpi radialis is given in the Tinel Syndrome as of the outer median head. The flexor carpi radialis and the palmaris longus are innervated probably from the same roots, although it is certainly not the 5C and 6C. They may at times be supplied from the 7C, via the outer median head, but Marie and Meige in electrical stimulation of the outer median head in sixty-four cases caused contraction only of the pronator radii teres (O. T.); of the inner head, they caused contraction of the flexor carpi radialis, the palmaris longus and the other flexors of the wrist and fingers. From our analysis of cases we believe the dominant root for these two muscles to be the 8C, and we so give it. If at times it is the 7C, then in those few cases the path might go via the outer median head, but we believe that this must be rare. The flexors and extensors of the wrist are given by Stewart as 6C, and of the fingers as 7C, which we believe incorrect. Harris gives flexor carpi radialis and palmaris longus as the 8C, as we do. Herringham, 7C-8C-1D, which is unlikely. Spalteholz, the 7C-8C-1D for palmaris longus, and the 6C-7C-8C for the flexor carpi radialis. Braus, the 7C and 8C and 1D for the palmaris longus, and the 6C-7C-8C for flexor carpi radialis. Our scheme gives the dominant root for the palmaris longus, the flexor carpi radialis, the flexor sublimis and the flexor profundus as the 8C. If there is a 7C branch, at times, it is not dominant usually, and neither is the 1D, which, nevertheless, probably gives a small connection. Therefore, only the 8C is given in our chart.

Note No. 11.—Latissimus Dorsi. The War Department gives 6C and 8C. Piersol is probably more correct, 7C and 8C roots. The only difference would be the presence of latissimus dorsi fibers in the cephalic division. Otherwise the course is the same, but the main nerve is the 7C. In many of our ninety-two dissections the 5C and 6C could have sent no branch to the thoraco-dorsal nerve. Purves Stewart gives 7C, which we know is not so, except in part; Harris as 8C-1D in postfixed, but he gives a 6C in prefixed; Herringham, 6C and 7C; Spalteholz and Braus both 6C-7C-8C. Our scheme gives 7C and 8C.

Note No. 12.—The Teres Major may be innervated by way of the axillary, and this is sometimes the case. The upper subscapular, from the 5C and 6C and posterior division of the 7C, usually goes to the upper part of the subscapularis. The middle subscapular goes to the subscapularis also. The thoraco-dorsalis goes to the latissimus dorsi, and the axillary subscapular supplies the teres major. Spalteholz gives 5C-6C-7C for subscapularis, and 6C and 7C for the teres major. Our scheme is the same for the subscapularis, but the 7C and 8C for the teres major.

Note No. 13.—The radial extensors of the wrist, it must not be forgotten, are supplied sometimes by the 5C (see Harris and Low, Fairbanks and Sherren), but more often probably from 5C-6C and 7C. In an analysis of the twenty-four cases of cut 5C, or 5C and 6C roots (not tension), the extensor carpi radialis longus and brevis were involved, or reported involved, only seven times, so they are not always from the 5C and 6C, therefore probably 7C; Herringham, 6C and 7C; Harris, 5C and 6C; Stewart, 6C; Spalteholz, 6C-7C-8C; and Braus, 6C and 7C. Our scheme is the 5C and 6C at times, and these are dominant. When from the 7C, as they often are, the latter is the dominant root.

Note No. 14.—While many muscles possibly have a small branch supply from the 6C, it is generally admitted that there is little extension of the paralysis produced by involvement of the 6C when the 5C is already involved. Direct stimulation of 6C causes contraction of the clavicular portion of pectoralis major, posterior part of the deltoid and some of the forearm muscles, probably pronator radii teres. Stewart gives the pectoralis major as 6C-7C; Harris as 5C-6C; which it certainly is at times. Spalteholz gives it as the 5C-6C-7C-8C; and the pectoralis minor as the 7C and the 8C. Our scheme gives the pectoralis major as the 6C-7C and 8C, and the pectoralis minor as the 7C and the 8C.

Note No. 15.—All the flexors and abductors of the thumb, and all the extensors of the thumb, are given by various authors as having a 6C branch. Both Piersol and the War Department give the abductor pollicis as 6C-7C, as does Braus. Spalteholz gives the thenar muscles as the 6C-7C and 8C, and even 1D for the opponens pollicis. Tinel specifically says paralysis of the abductor pollicis, the opponens pollicis and the flexor brevis pollicis is evidence of injury of the inner median head and therefore from the caudal roots. Very rarely have any injuries to the lateral fasciculus or cephalic roots been accompanied by disturbance of thumb action, and for that reason we have not given a 6C origin to any of these nerves. The flexors and abductors of the thumb are innervated by the 8C and 1D, through the inner head of the median, and the extensors of the thumb by the 7C and 8C, dominantly at any rate. André Thomas and Leverty are the only authors mentioning an involvement of the thenar muscles in an upper root lesion. Braus gives the opponens pollicis and flexor brevis pollicis as 6C and 7C, and the adductor pollicis as 8C and 1D. Our scheme is 8C and 1D for flexors, abductors and adductors of thumb; for extensors of the thumb 7C and 8C, with a small connection from 1D at times, which is not dominant, and therefore not included in our chart. The abductor pollicis longus (B. N. A.), it must be remembered, is the extensor ossis metacarpi pollicis of the old terminology as it is called in this chart. Its innervation is via the radial from the 7C and 8C, and it is considered here as an extensor. Nevertheless, in a pure median paralysis through its action, the thumb may still be abducted.

Note No. 16.—The Pronator Radii Teres is often supplied from the 5C or the 5C and 6C. Many times probably from the 7C, when not from the 5C or 6C. Sherren says that in his Erb-Duchenne type he never saw the pronator radii teres involved. In two of Harris' cases it was involved, and in two of André Thomas'. These were skilled observers. In our series it was involved with a 5C, a 5C and 6C, and a 6C cut, so it certainly comes from the upper roots, at times, but that it does not always do so, is evidenced by our list. In Apert's case with a 7C-8C-1D involved, all the epitrochlear muscles were a mass of fibrous tissue, while the epicondylar were in good condition. So probably the pronator radii teres is at times from the 7C. Braus gives 6C and 7C.

Animal experimentation has been of some value, especially on monkeys, but the differences from the human have prevented very much advance from this direction. I do not feel that I have myself derived enough benefit from articles on this phase of the subject to review them here. The anatomic method giving the best results has probably been the examination of the macerated fetal structure and the tracing of individual nerve fibers through the intricacies of the plexus. This was the method used by Herringham. The anatomic charts which we have incorporated here have many points of difference from those which have been published by various authors and anatomists. The root scheme which we have found helpful, differs quite markedly from most of those published, including those of Tinel and Benisty, Henri Meige and Purves-Stewart among the neurologists, and of Piersol, Gray, Spaltcholz and other anatomists. Some of the reasons for these differences will be found in the notes attached to the charts, or in the text. Figure 65 will give the reader our idea of the root innervation and of the ordinary arrangements of the plexus better than a detailed description, bearing in mind always that this representation is only schematic, and does not resemble in the least what the surgeon sees in dissection as he comes down upon it from the supra- or infra-clavicular incision.

Prefixed and Postfixed Plexuses.

There have been several classifications of the varieties of the brachial plexus. Harris's division into prefixed and postfixed types has been very generally accepted and used by anatomists and neurologists. Briefly, the prefixed type has a 4C connection to the 5C and no 2D to the 1D, while the postfixed plexus has a 2D connection to the 1D and no 4C connection to the 5C, therefore making them essentially different types, which do not overlap. This would be of little practical importance, if true, since even its authors do not claim that it raises or lowers the individual root constituents of the plexus one whole vertebral segment. There are other minor individual nerve differences in this classification, but these are the essentials. I doubt the importance of this classification and give my reasons as follows.

In our 1930 series of sixteen plexuses there were four with small connections from the 4C to the 5C root, as usually described. In three of these prefixed plexuses there was also a 2D connection making them, at the same time, postfixed plexuses; but both of these connections were small. This was not all. By careful dissection I found in eight plexuses another small connection, apparently from the 4C,

which ran down through the foramen transversarium, beside the vertebral artery, to join the 5C root outside the transverse process. I was unable to find any record of such a nerve in the literature. I traced it into the spinal canal at the 4C. In no case did it come from the sympathetic trunk, so far as I could determine, and I cannot believe it to be a fiber which comes out with the 5C root beyond the transverse process and then turns back to become part of the sympathetic nerve, which constitutes the so-called vertebral nerve of the sympathetic, described years ago by Vulpius and Francke. I regard it as another small connection from the 4C to the 5C. In six of the eight, it was present with a 2D to the 1D. In the two others, the 2D to the 1D connection, it was probable, although it could not be determined absolutely, as against a connection with the sympathetic alone, since the relation was too close to permit of a decision. The 2D connection to the 1D was sometimes separate, but usually connected with the sympathetic branches to the first thoracic ganglion. In several it was impossible to tell whether or not it was a 2D connection to the 1D, or a branch to the ganglion. The thorax always has to be opened in order to be sure of a connection from the 2D to the 1D. In the sixteen dissections a 2D connection was present in ten cases, and impossible to determine in four others.

I believe, if carefully looked for, there is in the majority of cases a small 4C connection to the 5C, and also a small 2D connection to the 1D. This is of importance, especially since the operation of stellectomy has come so prominently to the front. Injury during removal of the stellate ganglion to this 2D connection to the 1D, when it exists, may be accompanied by some minor paralytic involvement in the ulnar distribution in the intrinsic muscles of the hand.

SUMMARY OF THE 1930 SERIES OF SIXTEEN DISSECTIONS

4C to the 5C (The ordinary connection)	4
4C to the 5C (By the vertebral filament which we have described)	8
Without any 4C to the 5C	6
5C to the cervical plexus	2
2D to the 1D	10
Impossible to determine as between the 2D to the 1D and a sympathetic connection	4
No 2D to the 1D	2

I submit that with these findings, the idea of a prefixed and postfixed plexus must be rejected, because if a plexus is prefixed it certainly cannot be postfixed, even as a distinctive academic type. If we accept the presence of this vertebral nerve filament which we have described, we have ten plexuses out of sixteen dissections with a 4C connection of some kind to the 5C, and also ten with a 2D to the 1D and eight with both. With this proportion, any classification based on the presence or absence of a 4C connection, or the presence or absence of a 2D connection to the 1D, would seem to us artificial, unnecessary and misleading.

The posterior connections to form the intermediate fasciculus usually come from the roots individually, but the amount of fascia left unremoved may account for different opinions. In the sixteen cases of the 1930 series freed from enough fascia, we found that they not only come from the roots behind their junction in the case of the 5C and 6C, but the same arrangement holds good for the 8C and the 1D. However, in sixteen plexuses the 1D failed to send a connection to the intermediate fasciculus six times. It was nearly always small, even when present. Cunningham originally denied any such connection, as did Testut and Quenu. Herringham states that it is seldom present, yet Harris found such a connection in eighty-two per cent of his cases, and Kerr found it in 169 of his 175 plexuses.

Classification of Anatomic Variations in the Brachial Plexus.

Obviously no such single diagram as Figure 65 can be utilized for every instance of brachial plexus injury, for almost every individual plexus would differ in some detail from any standard we might set. However, we may attempt to classify the most common and important variations in the hope that in time we may be able to understand exceptional cases of paralysis. These variations have been classified especially elaborately by Kerr, and for the sake of brevity, I present his study of the plexus here in a tabulated form, which I have myself arranged from his written descriptions.

KERR'S ANATOMIC CLASSIFICATION OF VARIATIONS OF THE BRACHIAL PLEXUS

(From a Dissection of 175 Plexuses)

ARRANGED SCHEMATICALLY BY DR. STEVENS

CLASS I Having a 4C root 110 plexuses 63%	TYPE A Differs from the normal by 7C giving off a ventral division which joins the medial fasciculus or inner trunk 5 plexuses 3%	TYPE B Normal type 5-6-7C ventral divisions form the lateral fasciculus or outer cord. 8C-1D form the caudal or inner trunk. 7C forms the intermediate trunk. All trunks give off a posterior branch to form the intermediate fasciculus or posterior secondary cord 101 plexuses 58%	TYPE C Differs from the normal by having a branch from the medial fasciculus or inner trunk which goes to the lateral fasciculus or outer 4 plexuses 2%
CLASS II No 4C connection 52 plexuses 30%	TYPE D Same as Type A except for class 1 plexus 1%	TYPE E Normal—same as Type B except for class 51 plexuses 29%	Kerr had no type in Class II to correspond to Type C
CLASS III Sending a branch to the cervical plexus 13 plexuses 7%	No type here to correspond to A and D above in his dissections	TYPE F Normal—same as Type B or E except for class 12 plexuses 7%	TYPE G Same as C above except for class 1 plexus 1%
Total 175	6 plexuses 3%	164 plexuses 94%	5 plexuses 3%

This chart gives as comprehensive an idea of these different types as I can find, but we must assume, as he does, that the types do not overlap, although this is evidently not strictly true. His divisions are perhaps useful for anatomic study, but must be greatly simplified to be of use to the practical surgeon.

The normal type (Types B, E, and F of the chart belonging to Classes I-III) includes 93.7 per cent of all his dissections, and

Classes I and II include 91.58 per cent, or all but thirteen cases in which there was a branch to the cervical plexus. This last is an artificial distinction, and to me it seems more simple to classify the plexuses as I have below. Certainly the normal type should be Type A to facilitate remembering the different types.

Whether or not there is a small ascending connection, consisting of one or two filaments at most, to the cervical plexus above, on which Kerr bases his Class III, is a matter of no particular importance, even anatomically. In our ninety-two plexuses it was present in only thirteen per cent. In our 1930 series of sixteen plexuses, very carefully done, it was present in two cases, one a fair-sized nerve. Kerr found it in only seven per cent of his series.

In ninety-two dissections, twenty-two of them superficial observations, but nevertheless including the roots, we have never seen a 4C connection to the 5C which was of the size described by Harris, and most of the connections were small indeed. I have never seen a brachial plexus with a 4C connection as large as that represented by Kuntz in his wonderful work on the Autonomic Nervous System, but that is simply a representation. I have seldom seen this connection of a size which would permit of a suture, as has been reported in the literature. In such cases we should gravely question the reports. Especially with scar and infiltrate present, we do not believe that any surgeon could suture this connection in most of the cases as we have found them.

Kerr altogether ignores the presence or absence of a 2D connection to the 1D and therefore makes no claims of special types depending on this connection. All his classes are subdivided into types which he designates by letters which do not correspond for the various classes and consequently tend to confuse. His types A and D, depending on a 7C connection to the medial fasciculus, ignore those plexuses which contain a lateral ulnar head, which we contend is probably normal or at least common, and which probably comes from the 7C root, although via the lateral fasciculus where this exists, and certainly goes to the inner median head and to the ulnar, which are the terminal branches of the medial fasciculus. If it goes to the terminal branches of the medial fasciculus, it would seem to be much the same thing as if it went to the medial fasciculus itself. In sixteen dissections we found it present eight times.

DR. STEVENS' SCHEME OF SIMPLIFICATION OF KERR'S CLASSIFICATION AS CHECKED BY DISSECTION OF NINETY-TWO PLEXUSES

CLASS I A 4C connection to the plexus. In 92 dissections we found a 4C connection in 55. It is rarely anything but a few filaments, but sizable when covered by fascia. This is the connection described usually and not the filament described in the text as the vertebral canal connection 55 plexuses 60%	TYPE A Normal type, same as Kerr's type B. Lateral fasciculus formed by 5-6-7C ventral divisions. Medial fasciculus formed by 8C-1D. Both trunks give a post branch to join with the posterior trunk of the 7C to form the intermediate fasciculus 52 plexuses 57%	TYPE B This type has a 7C branch to the inner or medial fasciculus 3 plexuses 3% No account was taken here of those with a lateral ulnar head, which we contend belong here and are common. See text. Two others went to the inner median head instead of the medial fasciculus, and are therefore excluded, although practically they are the same	TYPE C A branch from the caudal trunk or medial fasciculus to the lateral fasciculus. We had no plexuses in this type Kerr has 2% in this type
CLASS II No 4C connection to the plexus 37 plexuses 40%	TYPE A Same as A except for class 34 plexuses 37%	TYPE B Same as B except for class 2 plexuses 2%	TYPE C Same as C except for class Kerr has none in this type 1 plexus 1%
Totals	94%	5%	1%

In this chart no account is taken of the vertebral connection from the 4C to the 5C as described by us in the text because there were only sixteen observations in which it was sought.

The individual variations of terminal branches which might be the cause of unusual paralyses are many. Of variations in the larger anastomoses in the upper portion, that of the musculocutaneous to the median is the most common. In seventy-five plexuses Kerr reports a connection from the musculocutaneous to the median eighteen times. In two of our last sixteen dissections there was a fused musculocutaneous and median as far as the elbow, and in six others a less

extensive connection. In none of the entire ninety-two did we find a connection from the median to the musculocutaneous. Ignoring the smaller connections, in ninety-two plexuses we had eight large connections from the musculocutaneous to the median, where this connection was of such a size as to appear to be the main terminal branch of the musculocutaneous. In three, the junction of the musculocutaneous and the median was just above the elbow. In one, the median outer and inner heads were separate to the elbow, and at this point were joined by a branch of the musculocutaneous, all three being of equal size; therefore, three separate pathways for the median innervation. It may be seen that a peripheral nerve injury involving only one or two of these branches would be difficult to locate. In one dissection the entire musculocutaneous joined the median four and one-half inches below the clavicle. In only one case did we have a connection from the radial to the ulnar high up in the axilla, and in only one was there a connection from the median to the ulnar at the elbow. In the hand and forearm, the median and ulnar often sent small branches of anastomoses. A knowledge of these anomalies may be of use in any case of unusual muscle or sensory paralysis.

The types of brachial plexus, so far as stress is concerned, are three.

Type 1—where the suprascapular comes frankly from the 5C root or by two roots from the 5C and 6C before they join to form the lateral fasciculus. Obviously, a stress here could fall on the lateral fasciculus with a greater chance for the escape of supraspinatus and infraspinatus. There were fourteen such cases in our series of ninety-two, and by removing a little more fascia, it is found that the suprascapular goes back to the 5C root in nearly all cases, sometimes alone and sometimes with a filament or two from the 6C. In one dissection we found a suprascapular entirely separate from the lateral fasciculus, with three branches of origin—4C, 5C and 6C—these branches coming from far back on the roots. The origins from the 5C and 6C were at the exits from the gutters of bone at the transverse processes. In two cases of the entire series, but with a good deal of fascia removed, the suprascapular came directly from the 5C, and the 5C sent no ventral branch to the lateral fasciculus, but only a posterior which joined the posterior of the 6C and 7C. In this case the lateral fasciculus was clearly 6C and 7C, and the musculocutaneous nerve could contain no fibers from the 5C.

In the second type stress might well be transmitted so as to fall strongly or entirely on the 7C root. It is characterized by the fact

that the 7C ventral branch passes directly to the outer median head. In fact, the ventral connection of the 7C is the outer median head, although it may receive a filament from the 5C or 6C.

In the third type the 7C ventral division went to the medial fasciculus without any branch to the lateral fasciculus. In such cases any ventral 7C root fibers would have to go via the inner median head or the ulnar, and therefore it is clear that a wound of the medial fasciculus would cause more damage to the median innervation than would be usual, and might even involve the coraco-brachialis when this muscle is innervated by the 7C. There were only five cases of this type in our entire series of ninety-two. In no case of this type did we have a lateral ulnar head. So we assume, as stated above, that the lateral ulnar head is probably from the 7C.

The last two of these three types were so small in number as to be negligible, except in an intensive anatomic study of the plexus. Therefore, in the majority of injuries, stress might not fall heavily on the suprascapular nerve.*

Recorded Cases of Rupture or Division of Parts of Plexus.

The diagram of the plexus which I present as possibly an improvement on those of other authors has also been checked or modified from a study of 710 cases of brachial plexus injury from the literature. In arranging the root charts of muscle innervation, we have utilized the information obtained by an examination of the reports of paralyzed cases in which the exact pathology was known. There were some cases where, with cut or ruptured upper roots, it was possible to draw fairly accurate conclusions as to the innervation of muscles from those roots. A careful examination of the table, representing muscle paralysis, with cut or ruptured 5C, or 5C and 6C, or 6C roots alone, will, we believe, bear out our statement. Although these observations were mostly recorded by trained men, many were not neurologists trained in electrical reactions, and it is likely that reports as to the condition of certain muscles are not included. Such a conclusion cannot apply to men like André Thomas, Harris, Sherren, Thorburn, Head, *et al.*, whose cases are included.

The degree of involvement of the muscles supplied by the collaterals arising near the roots, should be considered in the differential diagnosis of a root lesion. We believe that this is most important, and that a report should state their condition. Many times this information will clear up a seemingly complex problem. Nevertheless,

* In such cases the arm might be abducted by the action of the supraspinatus alone.—E. A. C.

even the reports in this selected series of twenty-four cases, many of them from masters of neurosurgery, show how few times the muscles supplied by these collaterals were definitely recorded as injured or as not injured. It seems highly probable that their condition was ignored in some cases, and that if their condition had been carefully examined, paralyses would have been found more often.

In the case of the lower roots the same method was pursued, but here it was more difficult, as there was no case of frankly cut or even injured 7C *alone*, except Mingazzini's case. The Déjerine-Klumpke type has been seldom checked by operation, except in the four cases where it formed part of a complete paralysis, and no root conclusion could be drawn.

The cases of Recaldoni, Pfeiffer, Ransom, Gosset and Quenu, *et al.*, which, while not of cut roots, were at least involvements of the 8C and 1D in pathological processes, were also utilized, together with many others, in arriving at what we believe to be at least an approximation of the muscle innervation of the lower roots. The rest was by exclusion, *e.g.*, many cases were reported as injuries of the 5C, 6C and 7C. Not one of these included the paralysis of either the flexor carpi radialis or the flexor longus pollicis. Therefore, we assume that these muscles do not receive their nerve supply from the upper roots.

The table of twenty-four cases where the lesion was known and the paralyses were of Erb-Duchenne type is interesting as a diagnostic study. It tends to prove that no distinction can be drawn between lesions of a 5C, a 5C and 6C, or a 6C lesion alone. It shows how little weight is to be placed on the lack of the report of involvement of the root collaterals even in a frankly Erb-Duchenne type. On the other hand, paralyses of the supraspinatus, infraspinatus, deltoid, biceps, brachialis, and brachioradialis may be confidently expected, as shown here, always to occur in this form of paralysis. If the muscles supplied by the root collaterals are paralyzed, it helps the diagnosis, but this table clearly shows that if they are not included, it just as certainly does not rule out a complete lesion of these upper roots.

There were twenty-four cases involving the 5C or 6C, where we could localize the injury sufficiently for our purpose. In two of these the 7C was also slightly involved, but not enough to be of much importance, so they are included. One of these had a slight involvement of the triceps, but the other was purely Erb-Duchenne. One other case not included in the twenty-four, Winnen's case, involved a 4C connection to the 5C, at the point of junction, it was said.

INCIDENCE OF MUSCLE PARALYSIS IN TWENTY-FOUR CASES OF UPPER ROOT TYPE

Levator scapulæ	2	
Rhomboidei	5	4 complete, 1 partial.
Diaphragma	0	The nerve to the subclavius gives a branch to the phrenic usually (Henle's loop).
Subclavius	1	
Pectoralis major—pars clavicularis	3	
—pars sterno costalis	0	All partial. The clavicular portion receives not only a 5C, but another branch usually from lower down.
Pectoralis minor	1	Partial.
Supraspinatus	24	
Infraspinatus	24	
Deltoideus	24	In one of these with cut 6C, anterior portion reacted slightly.
Teres minor	6	Only specifically mentioned 6 times.
Biceps	24	
Brachialis	19	
Coraco-brachialis	5	Specifically mentioned.
Brachioradialis (Sup. longus)	20	One partial with a cut 6.
Supinator (Sup. brevis)	8	
Extensor carpi radialis longus	8	
Extensor carpi radialis brevis	8	
Triceps	3	Only mentioned as "slight" in any. Jacobson and Sargent. Sicard with a slightly injured 7C.
Latissimus dorsi	0	Not once mentioned.
Subscapularis	0	
Serratus anterior	1	
Teres major	3	Two complete (5C, and 5C and 6C), one partial (5C).
Pronator teres	5	With a 5C cut, a 5C and 6C, and a 6C alone.
Flexor carpi radialis Flexor pollicis longus Palmaris longus	0	Not once mentioned.
Flexor pollicis brevis (one head) Opponens pollicis Abductor pollicis	1	This was a case where the 7C was slightly involved. André Thomas case which with Leverty's case (unchecked of E. D. type), are the only two which report an involvement of these muscles in an E. D. type.

In only seven cases was there specific mention of any particular sensory disturbance.

Conclusions from Analysis of 710 Reported Cases.

In the series of cases from the literature, beginning with Flaubert in 1827, I found that many were of little importance, since the details were too meager to give us much information as to the methods of production, or even the exact muscle paralyses, and in most of them there was no operation or autopsy by which to check. With all the World War wounds, it would seem that there should have been details in an immense number of cases. Such, unfortunately, was not true. Thousands of cases of peripheral nerve wounds, and some in great detail, were reported, but where the brachial plexus was concerned, the description of the operation in most cases was confined to the wounds, with no detailed account of work on the plexus itself.

These cases were reported by many men. Gosset—fourteen. Moynihan—eleven cases of brachial plexus; four upper, seven lower. Tuffier—280 operations on nerves, but no brachial plexus. Boinet—twenty-five cases of brachial plexus injuries. Tinel—in 639 nerve lesion reports, twenty-seven brachial plexus. Price, Feiss and Terhune—sixty cases of brachial plexus injuries; five complete. Mauclaire—four cases, one axillary, none of them complete. Chiray and Roger, Leclerc, Benisty, Féré, Wiart, *et al.*, reported others. In all, there is a lack of detail, so that we can get very little help from their analyses for our particular purpose.

In 710 case reports examined, there were 135 cases in which an operation or autopsy had been done.

TABLE

Total case reports studied			710
Total cases operated or autopsied	supraclavicular	86	
	infraclavicular	49	135
Supraclavicular injuries			86
Nothing found			6
Avulsion			4
Ruptured or cut roots or traumatic neuroma of such a nature as to show rupture in the opinion of the operator			53
Scar tissue only			23
Total			86
Infraclavicular injuries			49
Rupture of one or more roots or reported as total disintegration of cords, or neuroma of such a nature as to cause the operator to report rupture			23
Scar only			21
Nothing found			5
Total			49
			135

Let us analyze the eighty-six supraclavicular injuries in order to avoid the fallacious conclusions which we think other students of the subject have come to. Eighty-six cases of supraclavicular rupture would seem a large proportion of the 135 cases on which operation or autopsy had been done. Doubtless among the remaining 575 which were unchecked, and in which no conclusions could be drawn other than the incidence of paralysis, the proportion of supra- to infra-clavicular injuries might have been the same, but as to that we can only theorize. To determine the cases of actual rupture, we must exclude the following fifty-four cases from the eighty-six supraclavicular injuries.

Cuts of the 5C and 6C without other trauma	16
Wounds or cuts of other roots	4
Roots cut for other reasons than trauma	3
Reported as avulsed because faradization of the roots failed to cause contraction (a fallacy)	2
Nothing found at operation	6
Reported by the operator as scar only (some of these may have been ruptures, but it is not likely)	23
Total to be excluded	54

This leaves only thirty-two cases which represent instances of trauma other than cuts or wounds, where roots were reported as separated either because the two ends were found, or because the traumatic neuroma or scar was of such a nature as to lead the operator to believe that such was the case.

Of the cases of Tubby, Tuffier, Bardenheuer, Bristow, Hartley, Banks, Thorburn, Sherren, Kalb, Fisk, Ginsburg, and Thomas, there can be no doubt; we may include also the four cases of avulsion; but of some cases where the report was made of a neuroma or a disintegration of the plexus, or of scar of such a nature as to prevent actual knowledge, there is grave doubt, not of injury, but of an actual rupture of any part of the plexus.

The German and Austrian literature has been examined less thoroughly, but including Bernhardt, Wölfler, Winnen, Erb, Kalb, Kramer, Bardenheuer, *et al.*, and excluding all the cut roots or wounds, we have been able to accept only thirty-two cases as definite supraclavicular ruptures!

I have found only four proven cases of *rupture de la moelle épinière* (avulsion) in all the history of medicine.

1. Flaubert of Rouen, France, 1827. Reduction of a dislocation by the combined force of eight men. All roots except the 5C

ruptured from the spinal marrow and recovered below the clavicle at autopsy. Death on the fourth day.

2. Apert, France. Boy, arm caught in the wheel of a heavy cart; thrown over and over. Autopsy thirty-two and one-half years later. Complete rupture at the spinal cord of the 7C and 8C and the 1D. No ascending degeneration of spinal tracts, but the brain area was smaller.

3. Boyer, Toronto, Canada. Birth case. Arm pulled. Rupture more pronounced of the 7C, but 5C-6C-8C and 1D are included as injured. Ascending degeneration marked. Brain area involved smaller. The autopsy was made forty-one years after the injury.

4. Charles Frazier, Philadelphia. Man struck by another man falling three stories; 6C, 7C and 8C ruptured, from the spinal marrow. Laminectomy, for intolerable pain, afforded the opportunity to study the exact seat of the injury. Dr. Frazier cut the 1D and the 5C sensory roots. In this case there was a suspicion of a fractured vertebra.

These are the only four cases I can find in the literature with *proven* avulsion, but these cases have been responsible for the misconception that the usual locations of injuries to the plexus are within the spinal canal or in the bony gutters proximal to the snubbings on the transverse processes.

Treatment.

Of the non-operative treatment little need be said except that a paralyzed muscle recovers quicker when not in a condition of stretch. One must know and analyze the individual muscle paralyses in order to meet this indication. If one learns to prevent contractures which follow in these paralyzed cases from over-action of muscles not paralyzed, he will have gone a long way in the intelligent treatment of brachial plexus injuries. In cases of complete interruption of the nerve paths, contractures are usually soft and reducible. Only in irritations do we get the hard, irreducible griffs, and these must be prevented by treatment. It is easier than to correct them after they have occurred—in the domain of the median, the *accoucheur's* hand; in the domain of the ulnar, the claw hand; in the domain of the radial, the wrist drop.

Operation.

Immediate operation is indicated in all cases of penetrating wounds with paralysis involving the branches of the plexus, and in all cases showing the complication of subclavian or axillary arterial

injuries. The presumption should be against operating in cases of tension or traction injuries. Were I to select the factor of most importance in determining the question of operation, I should wish to know the manner of the trauma. Lesions of the plexus accompanying dislocation and near dislocation will rarely cause actual rupture, and, therefore, may recover as well without operation as with it. The chance of suture of a ruptured nerve trunk is a forlorn hope. The main indication for operation is the release of pressure from exudate. The resection of a nerve simply because it has a traumatic neuroma or local enlargement is unwarranted; there must be clear evidence of entire anatomic separation. False neuroma is often hard to distinguish from true, and it will many times interrupt the electric current. See Henri Claude, Déjerine, *et al.* Lysis, simple longitudinal incision, as taught by Bardenheuer long ago, will give the greater percentage of cures, and resection should be reserved for those cases showing actual separation. The time to operate in secondary cases is of importance. Some say three months, but there are many cases that will not begin to show first motor return in that time, and therefore, if operation were to be the rule, it would be better to operate at once. In expert hands immediate operation is justifiable in any doubtful case, but a surgeon without a knowledge of the local anatomy and general history of the subject might easily do harm. In most cases recovery of function begins within seven or eight months, but many cases have recovered first motion at a later period. In one of my cases, a root lesion due probably to abduction and external rotation during a breast amputation, the first voluntary motion appeared at the end of five and one-half months. Faradic contractility and voluntary motion were noticed on the same day.

The best argument for not operating in tension injuries is that actual rupture of the brachial plexus is a rare condition, although temporary palsy is common. The reason is that the stresses do not fall entirely on the brachial plexus as in our theoretical traction apparatus. After the bones and ligaments are separated, stresses are distributed to all the fascial investments of the axilla and neck and so disseminated. Much of the strain, especially when transmitted from below, is taken up by the tendons as evidenced by rupture of the long head of the biceps, so often observed in dislocations and in fractures of the upper end of the humerus. Always, as explained on p. 348, there are small tears and injuries of the fascia about the nerves and vessels, and probably also far removed along the fascial planes. There are petechial hemorrhages and exudate which surround not only the roots but the cords of the plexus in both the supraclavicular

and axillary regions. But if rupture occurs, it is at the roots or of terminal branches and not of the plexiform part, which, nevertheless, may be swollen by pressure of the exudate within the fascia surrounding it.

Some Practical Rules in Diagnosis for Use with Chart.

There are many exceptions, but the following points will usually hold good.

If in an upper type lesion the brachioradialis is involved, it is usually a lesion of the 5C or 6C roots.

If the brachioradialis is not involved in a purely radial or extensor paralysis, often entire at first but later only partial, it is a lesion of the 7C. In such a case the coraco-brachialis, when it comes from the 7C as it often does, would be the only flexor in the upper arm paralyzed and would become the key muscle.

It must not be forgotten that in the wrist drop from paralyzed forearm extensors due to lead poisoning, the integrity of the brachioradialis and supinator is almost diagnostic, and this condition must be excluded.

If all the extensor muscles of the arm and wrist and fingers, and the brachioradialis and supinator also are paralyzed, it is a peripheral lesion of the radial nerve.

If only the deltoid and teres minor of the upper group in addition to the above are involved, it is a trunk and not a root lesion of the intermediate cord or a peripheral lesion of two nerves—radial and axillary.

If it is a combined paralysis of the flexors of wrist, thumb and fingers, except the brachioradialis and the pronator teres, and all extensors are involved, it is a lesion of the 7C and 8C and 1D.

If the lesion is as above but only partial of the triceps, the latissimus dorsi and partial of all the extensors of the wrist and fingers, with escape of the brachioradialis and extensor carpi radialis longus and brevis, it is a lesion of the 8C and 1D roots.

If the paralysis is of all the flexors of wrist, thumb and fingers with the exception of the brachioradialis and the pronator teres with escape of the triceps, anconeus and all extensors, it is a lesion of the medial fasciculus.

If only the ulnar flexors of the forearm are involved and all the hand muscles except the opponens pollicis and the abductor pollicis brevis and one-half the flexor pollicis brevis, it is a lesion of the ulnar.

DIFFERENTIAL DIAGNOSTIC CHART

Between Root Lesions of the Brachial Plexus of the Erb-Duchenne Type and Terminal Lesions Resulting in Similar Paralyses.

By James H. Stevens, M. D., Boston, Mass.

Muscles Involved	Erb-Duchenne Paralyses due to Root Lesions		Paralyses due to Lesions of Terminal Nerves in the Axilla		
	5th or 6th Cervical Roots, singly or together	With Additional Lesion of the 7th Cervical Root	Musculocutaneous and Axillary Nerves	Musculocutaneous, Axillary and Radial Nerves	Musculocutaneous, Axillary and Median Nerves
1. Levator scapulæ	Partial sometimes	Partial sometimes	Never	Never	Never
2. Rhomboidei	Often	Often	Never	Never	Never
3. Serratus anterior	Upper sometimes	Often	Never	Never	Never
4. Diaphragma	Slight or rare	Rare, slight only	Never	Never	Never
5. Subclavius	Often, but hard to determine	Often	Never	Never	Never
6. Pectoralis major (clavicular)	Often or always. Seldom reported	Often or always	Never	Never	Never
7. Subscapularis upper	Often. Hard to determine	Always. But partial at times	Never	Never	Never
8. Supraspinatus	Always	Always	Never	Never	Never
9. Infraspinatus	Always	Always	Never	Never	Never
10. Deltoideus	Always	Always	Always	Always	Always
11. Teres minor	Always	Always	Always	Always	Always
12. Biceps brachii	Always	Always	Always	Always	Always
13. Brachialis	Always	Always	Always	Always	Always
14. Coracobrachialis	Sometimes	Always	Always	Always	Always
15. Brachioradialis	Nearly always	Always	Never	Always	Never
16. Supinator brevis	Nearly always	Always	Never	Always	Never
17. Extensor carpi radialis longus	Often	Always	Never	Always	Never
18. Extensor carpi radialis brevis	Often	Always	Never	Always	Never
19. Triceps	Almost never	Always—partial	Never	Always entire	Never
20. Anconæus	Never	Always—partial	Never	Always	Never
				Always	Never

23. Extensor pollicis brevis	Never	Always	Never	Always	Never	Never
24. Latissimus dorsi	Never	Partial usually	Never	Never	Never	Never
25. Flexor carpi radialis	Never	Never	Never	Never	Never	Always
26. Pronator teres	Sometimes	Always	Never	Never	Never	Always
27. Flexor pollicis longus	Never	Never	Never	Never	Never	Always
28. Flexor digitorum sublimis	Never	Never	Never	Never	Never	Always
29. Flexor digitorum profundus	Never	Never	Never	Never	Never	Always except ulnar part
30. Flexor pollicis brevis	Never	Never	Never	Never	Never	Always one-half muscle
31. Pollicis opponens	Never	Never	Never	Never	Never	Always
32. Abductor pollicis brevis	Never	Never	Never	Never	Never	Always
33. Sensory anæsthesia and analgesia	The sensory loss to both cotton wool and pin prick is remarkably little, and usually is limited to the cutaneous area just below the shoulder externally and posteriorly. At times it extends over the lower two-thirds of the deltoid. The anæsthesia and analgesia sometimes extend on to the anterior surface of the forearm to the wrist (5th and 6th cervical). If the 4th cervical is involved the cutaneous loss extends higher over the shoulder.	The anæsthesia or analgesia in this type is more pronounced, including the deltoid region as given in the lesion of the 5th and 6th cervical but includes the antero-external and postero-external surface of the arm and forearm and both surfaces of the thumb and index finger (7th cervical).	In this lesion there is rather well-defined loss of both forms of cutaneous sensation over the lower two thirds of the deltoid, extending more posteriorly (axillary); of the front of the arm at the elbow and of the anterior surface of the forearm on the external side, including the base of the thumb, as well as the postero-external surface of the forearm on the lower third, extending to the base of the thumb.	In this type of injury cutaneous sensory loss is pronounced, over the lower deltoid region (axillary); over the antero-external and postero-external surfaces of the arm (radial), and even of the posterior surface entire (radial); over the antero-external and postero-external surfaces of the forearm (musculocutaneous) and of the thumb, except its flexor surface and the tip (radial); half the dorsal surface of the hand and the dorsal surface of the index and the middle, and one-half of the ring finger, except the two terminal phalanges (radial). Radial injury, it must be remembered, is often not accompanied by any great cutaneous disturbance.	In this type there is profound sensory disturbance over the lower two-thirds of the deltoid (axillary); over the anterior surface of the forearm to the wrist and extending somewhat to the postero-external surface of the forearm, as well as in the lower third (musculocutaneous); anæsthesia and analgesia of the external palmar surface of the hand, ball and flexor surface of the thumb and of all the flexor surfaces of the outer two and one-half fingers, and of the dorsal surfaces of the two end phalanges of same fingers, usually. (Median.) Rarely; this last is covered by the radial.

DIFFERENTIAL DIAGNOSTIC CHART

Between Root Lesions of the Brachial Plexus of the Déjerine-Klumpke Type and Terminal Lesions Resulting in Similar Paralyses.

By James H. Stevens, M. D., Boston, Mass.

Muscles Involved	Déjerine-Klumpke Paralyses due to Root Lesions		Paralyses due to Lesions of Terminal Nerves in the Axilla	
	8th Cervical and 1st Dorsal Roots	With Additional Lesion of the 7th Cervical Root	Median and Ulnar Nerves	Median, Ulnar and Radial Nerves
1. Eye muscles of Müller	Often; always if lesion is far back on root	Often	Never	Never
2. Latissimus dorsi	Partial	Always complete	Never	Never
3. Teres major	Partial	Always complete	Never	Never
4. Subscapularis	Partial rarely	Partial	Never	Never
5. Pectoralis major	Not usually	Partial	Never	Never
6. Pectoralis minor	Many times	Usual	Never	Never
7. Triceps	Partial	Always	Never	Always
8. Anconæus	Partial	Always	Never	Always
9. Brachioradialis	Never	Never	Never	Always
10. Supinator	Never	Never	Never	Always
11. Extensor carpi radialis longus	Never	Sometimes	Never	Always
12. Extensor carpi radialis brevis	Never	Sometimes	Never	Always
13. Extensor digatorum communis	Always	Always	Never	Always
14. Extensor carpi ulnaris	Always	Always	Always	Always
15. Extensor pollicis longus	Always	Always	Never	Always
16. Extensor pollicis brevis	Always	Always	Never	Always

17.	Pronator teres	Never	Never or very rarely	Always
18.	Palmaris longus	Not usually reported. We believe usually involved	Usually	Always
19.	Flexor carpi radialis	Many times or always	Usually or always	Always
20.	Flexor pollicis longus	Usually or always	Always	Always
21.	Coracobrachialis	Never	Sometimes	Never
22.	Adductor pollicis caput obliquum	Always	Always	Always
23.	Adductor brevis pollicis	Always	Always	Always
24.	Flexor pollicis brevis	Always	Always	Always
25.	Hypothenar muscles	Always	Always	Always
26.	Interossei volares	Always	Always	Always
27.	Lumbricales	Always	Always	Always
28.	Pron. Quad.	Always	Always	Always
29.	Sensory	In this type the sensory disturbance is apt to be pronounced, especially in the forearm extends from the axilla to the hand along the ulnar side, both anteriorly and posteriorly, often leaves unaffected the area of the axillary inner surface of the arm, which corresponds to the intercostobrachialis nerve. In the hand there is anesthesia of the palmar and dorsal surfaces on the ulnar side.	The sensory disturbances in this type are more accentuated than in the pure DK type. area. Loss of sensation usually is confined to the hand volved, except a small strip anteriorly, which receives its supply from the 6th cervical nerve. The anesthesia is usually confined to the palmar and dorsal surfaces on the ulnar side, the thenar and hypothenar eminences, the dorsal surfaces of the thumb and fingers. The arm is involved in its entirety except for the region of the axillary and the intercostobrachialis and part of the domain of the musculocutaneous.	Sensory disturbances in this type are more accentuated than in the pure DK type. All brachii cutaneous and cutaneous antebrachii medialis are involved, are apt to be entirely confined to the hand and fingers, even the palmar side, the thenar and hypothenar regions, the dorsal surfaces of the thumb and fingers, are involved, on the dorsal surface there is anesthesia of the ulnar area of the hand, but the index and middle fingers are not involved except at the tips.	In this type the sensory loss involves the posterior surface of the lower upper arm; the posterior surface of the forearm on the outer side; the entire hand, fingers and thumb.

If it is a paralysis of the flexors of the wrist and fingers, including the flexor longus pollicis and the pronator teres, with escape of the brachioradialis and supinator, the flexor carpi ulnaris, part of the flexor profundus digitorum and all interossei, it is a peripheral lesion of the median. In this case the abductor pollicis brevis and opponens pollicis and part of the flexor pollicis brevis are also paralyzed, but the hand muscles and adductors of the thumb are not involved.

The abductor pollicis longus, it must be remembered (the extensor ossis metacarpi pollicis of the old terminology), is an extensor innervated by the radial. Nevertheless, it may act as an abductor of the thumb. It is not involved in a pure median paralysis.

Care must be taken not to confuse a root lesion with a peripheral paralysis which involves two or more nerves. In such a case diagnosis is more often difficult and will tax the knowledge of the observer.

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Postscript by Dr. Codman.

The late Dr. C. A. Porter was greatly interested in peripheral nerve injuries, especially in those of the brachial plexus. Through the kindness of Mrs. Porter I have been able to study the manuscript and notes of an unpublished paper in which he reviewed the results of all nerve injuries operated upon at the Massachusetts General Hospital up to 1919. This work, as a whole, supports by the details of cases, many of the conclusions reached by Dr. Stevens, especially those in regard to the advisability of refraining from operation in post-dislocation injuries, and the importance of prompt operation in cases of paralysis following those types of sudden and violent injury, which depress the whole shoulder or which drag the whole body by the arm. The futility of belated operations is shown by the fact that in ten cases of attempted suture of the brachial plexus, improvement, and this of slight degree, was found in only two, a ratio no greater than might be expected in untreated cases. Dr. Porter's rough notes will be placed in the Hospital Library in the hope that they may be of use to some future student of this discouraging branch of surgery. It is evident that this lesion is one among the many rare ones which need immediate attention from a surgeon who has given especial study to the subject.

Chapter XII

LESIONS OF THE BRACHIAL PLEXUS COMPLICATED BY RUPTURE OF THE SUPRASPINATUS TENDON

Instances will arise in industrial clinics where it will be difficult to differentiate between a direct injury to the supraspinatus tendon and a circumflex paralysis. The most important point I have in mind to accent in this chapter is that one should never be misled by the fact that the patient's deltoid is paralyzed, into thinking that the supraspinatus is undamaged. As a matter of fact, the combination of these two injuries is not infrequent, and the supraspinatus injury remains undetected because the deltoid paralysis seems to be accountable for the fact that the patient cannot raise his arm. I want to present arguments for my conviction that in case a patient has deltoid paralysis and is unable to elevate his arm, we should make an exploratory incision in the bursa to determine whether the supraspinatus tendon is ruptured. This dictum is supported by my observation that if the supraspinatus is intact, *i.e.*, neither ruptured nor paralyzed, the patient *can feebly raise his arm in spite of paralysis of the deltoid.* I believe that the industrial surgeon who bears this in mind may be able to save his company some trouble and expense.

In the previous chapter, Dr. Stevens has made it very clear that most of the postdislocation paralyses are due to local, peripheral injuries of the axillary and musculocutaneous nerves, caused by actual stretching of these nerves by the head of the humerus in the course of its process of dislocation. He has also shown that the suprascapular nerve is subject to a considerable amount of variation in its association with the 5th root, so that many times it may escape paralysis, even in cases otherwise of Erb-Duchenne type.

Although I have never studied the plexus in regard to the mechanical stresses caused by different forms of injury, as Dr. Stevens has done, I had come to somewhat the same conclusions from clinical study, for I had observed cases in which it was very clear that the site of the lesion was below the nerve supply of the supraspinatus and above that of the deltoid. My observations had brought me to the conclusion that one should always be suspicious that the supraspinatus may be ruptured, rather than paralyzed, in all cases of postdislocation palsies.

For the purposes of our particular study concerning the diagnosis of paralysis of the supraspinatus *versus* rupture of its tendon,

either of which combined with deltoid paralysis would cause inability to perform elevation, observation of the conditions of the rhomboids and pectorals is important, as the nerves for the rhomboids and those for the clavicular part of the pectoralis major leave the plexus above the circumflex, and also above the suprascapular nerve which supplies the spinati.

One can, by observation alone, determine the question of whether the pectorals or rhomboids are paralyzed, but the spinati under cover of the acromion are not readily palpable, although in a perfectly normal shoulder it is easy to feel (through the trapezius) the contraction of the supraspinatus when appropriate effort is made by the patient. When the patient folds his arms across his chest, and then shrugs the shoulders, the contours of the rhomboids appear. However, in cases of injury where the whole shoulder is tender, it is difficult to make sure that the patient is really making an effort to contract his supraspinatus. Thus the positive is pretty positive, but it is difficult to say that you are sure that the supraspinatus is out of commission. Later atrophy of these muscles does not signify paralyses, for it occurs after almost all shoulder injuries.

Nevertheless, if the rhomboids are paralyzed and the pectoralis major is partly paralyzed (the clavicular portion), we may assume that the supraspinatus is, also. Since the nerves to the pectorals have an additional supply from the lower cords of the plexus, paralysis of the pectorals need not necessarily be found. It is especially important to observe the clavicular portion of the pectoralis major, for this is probably always supplied by the 5th and 6th roots and perhaps by these roots only. Of course, proof of paralysis of the supraspinatus does not necessarily mean that there has been no rupture, for this might have occurred simultaneously with the same injury which dislocated the humerus and injured the plexus. If the rhomboids are paralyzed it makes it more probable that the supraspinatus is merely paralyzed and is not ruptured, but it does not absolutely exclude rupture.

As a matter of fact, in injuries to the plexus from dislocation or near-dislocation, we commonly find only the axillary nerve involved and consequently only the deltoid and teres minor paralyzed. The teres minor is naturally overlooked. Often the biceps and coraco-brachialis and brachialis anticus are simultaneously involved, and not infrequently the supinator longus. Yet all these may be involved without the spinati, for the suprascapular nerve may be almost independent of the 5th root.

Of course, testing the activity of these muscles implies coöperation on the part of the patient, who must exert his will to raise the arm in spite of the soreness from the recent injury. Fortunately, in cases of paralysis the sensation of the joint is usually absent owing to the sensory paralysis of the axillary. In a recent severe case in which all the tendons had also been evulsed and the axillary paralyzed, I could move the joint about in any direction, without causing the least pain.

Taking all these considerations into account, I should lay it down as a dictum that if, following such injuries, one finds the deltoid paralyzed and the patient is unable to slowly raise his arm with the supraspinatus alone, the probability of a diagnosis of a ruptured supraspinatus tendon is so likely that exploration of the bursa should be done, unless there is a coincident paralysis of the rhomboids or the clavicular portion of the pectoralis major.

The following cases are illustrative of some of the points considered:

CASE 30

On Sept. 3rd, 1912, I operated on a man of 47, who had a circumflex paralysis which followed a dislocation two months previously. I was in doubt as to whether the supraspinatus was torn. The bursa, on exploration, proved to be normal, and the supraspinatus tendon was intact. It occurred to me that as the patient must wait for his incision to heal, he might as well wait in abduction and external rotation and give his deltoid a rest, on the general principle that all paralyzed muscles recover their tone better if they are maintained in a relaxed position. After a few days this man began to be very complimentary about the success of my wonderful operation, and said he could move his arm better than he had since the accident. In two weeks the power of the deltoid had almost entirely returned and the patient was greatly pleased.

Of course, my operation being simply a small incision through the deltoid fibers and the roof of the bursa had nothing to do with the matter. His improvement was due to rest in elevation. Since this experience I have treated deltoid paralysis in the same way, sometimes without exploratory incision, with invariably gratifying results. Most of the improvement takes place in the first two weeks, so that the patient is usually very coöperative, for he realizes his progress. Part of the rapid progress in the first two weeks is due to readjustment of the swelling and edema in the joint and bursa. If the patient has been up and about for several months and the shoulder has "dropped," the space between the acromion and the head of the bone must become filled with fluid or with edema of the joint and bursal tissue. The elevated position gives this edema

an opportunity to be absorbed, the circulation becomes more active and the muscle fibers, not being stretched, soon respond. It may be months, however, before the muscle becomes fully developed again.

The principle of relaxing paralyzed muscles is well accepted, but so far as traumatic circumflex lesions are concerned, it has certainly not become an appreciated routine practice, in this community at least. Previous to the above experience I had never seen it applied to a case of circumflex paralysis, and even since the above experience, my own cases have been the only ones I have observed, although I have no doubt the principle is constantly gaining in the extent of its recognition and perhaps practice. (See p. 482 on relaxed capsule.) It is well recognized now in the treatment of infantile paralysis. I feel sure that there are many industrial cases of axillary and other paralyses, for which our insurance companies might well pay for the use of a hospital bed for the sake of the time which the patient would gain in returning to work. As a rule, the exploration of the bursa should be done with local anæsthesia at the same time, for it is a trivial operation and causes no delay. The wound is healed long before the use of the arm returns. If a rupture of the supraspinatus is found and repaired, permanent disability may be prevented.

Case 49.

On Dec. 13th, 1913, I operated on a man of 64, who had an axillary paralysis following a dislocation three weeks previously. On this case I made a preoperative diagnosis of a rupture of the supraspinatus as well as of deltoid paralysis. Exploration showed that not only the supraspinatus but the infraspinatus, teres minor and long head of the biceps were evulsed. The destruction was too great to make any suture possible, so the wound was closed and the arm put up in abduction. Within two weeks there was a fair return of the power of the deltoid and months later the deltoid even became hypertrophied, but the full power of abduction never returned. I was called to see this patient ten years later, on April 23rd, 1923, because he had an ununited fracture of the neck of the femur. The deltoid of the injured arm was very large and powerful, but he still could not abduct the arm when standing.

Undoubtedly if this patient had been operated on immediately after the accident, the capsule and short rotators could have been sutured in place. Increased experience in these cases makes me feel confident that at the present time I could suture the tendons in a similar case even after three weeks, but the longer the period of time that elapses between the injury and the operation, the more retracted the tissues and the greater the difficulty. Quite recently I had to abandon suture in a case of seven months' duration in which both spinati and the teres minor had been evulsed.

A more encouraging instance was Case 107, a strong woman of 53, who had been employed as a cook. On July 18th, 1927, she fell on the floor of her kitchen and dislocated her left shoulder. She was taken to an emergency hospital, and the dislocation was reduced. The shoulder remained sore, almost useless, and did not progress. After seven months she was referred to the office of the insurer and an X-ray was taken showing that the dislocation had been reduced. Next day, Feb. 16th, 1928, she was referred to me. I reported as follows:

"Examination of the left shoulder reveals that there is a sensory and muscular paralysis of the circumflex and little power in the biceps. The head of the humerus falls away from the glenoid, as is usual in these cases of circumflex paralysis.

"I would recommend that the patient enter a hospital and have the arm retained comfortably in abduction. I have had a number of cases which responded promptly to this treatment, probably because the nerve trunks are put at rest and their circulation therefore made better for repair. Also the drag of the weight of the arm is taken from the deltoid and its fibers have a chance to recover their tone.

"With this treatment the prognosis is good. Two weeks in this position with occasional appropriate exercises produces marked improvement. If it does not, the prognosis is poor, but I have never yet failed to see demonstrable improvement recognized by the patient in this time."

* * * * *

"In my report of Feb. 16th concerning this patient, I stated that Dr. ——— reported to me by telephone that the films showed normal bones. I have since called at his office and inspected the films. They are not wholly negative for they show, faintly, crumbs of bone in a position suggesting that they were torn off the greater tuberosity by the supraspinatus tendon. The films were not clear, for the patient moved, but the suggestion is strong that the damage is not only to the brachial plexus but that a certain amount of evulsion of the superficial part of the tuberosity occurred at the accident.

"At your suggestion, the patient entered the Trumbull Hospital on Feb. 19th to be under my care, and I put her to bed with the arm held loosely in abduction, so far as it was possible. I have seen her yesterday and today. She is not very coöperative and I find her a hard patient to handle, as she is rather stupid about getting the idea of keeping her arm up and when the nurse's back is turned, gets it down again.

"There is spasm of the groups of muscles on each side of the axilla and this tends to make her arm return to her side, as there is no opposition in the deltoid. It appears to abduct fairly well up to about half the normal distance and then there is a block, as if the above-mentioned fragments impeded further progress. It may be that there is some callous formation about them. However, I shall try for a few days longer to cope with her and endeavor to carry out the treatment. Unless she becomes more coöperative I fear that I may have to discharge her, for I do not think she would be willing to have an exploration of the bursa to see if the tendon is evulsed with a bit of tuberosity attached."

* * * * *

"I reported on this patient last on Feb. 21st. Since then she has remained in bed at the Trumbull Hospital with the arm in abduction. I finally succeeded in getting it into her head that she should help, and after the first week she, herself, could notice the daily improvement and has been more enthusiastic with her coöperation. It took about five days to overcome the spasm of the latissimus and pectorals, etc., but when they once yielded the arm could be comfortably retained in a semi-abducted and externally rotated position without other apparatus than a bandage, about her wrist tied to the head of the bed, to remind her.

"Her progress has been good, and there is distinct improvement in sensation and muscular activity in the deltoid region. I give her exercises in the stooping posture and she can do them quite freely, the arcs of motion being normal and the pendulum movement taking the place of the muscular power of the deltoid. There is still no real power in the latter, but I am confident I can feel a beginning contraction of the fibers, especially when a counter effort is being made.

"In my opinion it is well worth while to keep this patient in the hospital as long as she is clearly improving and she is willing to stay. The result of the two weeks makes me confident that progress will continue and the result will be good. I wish I could also explore the bursa to see whether the tendon is torn or whether the atrophy and loss of power in the supraspinatus is wholly from the nerve paralysis. I suspect the tendon is torn, but as it is masked by the paralysis, I am not certain.

"Would Dr. ——— approve of my trying to persuade the patient to have this done? The bursa and tendon could be inspected through a very small incision, not over a half-inch long. It would save the patient much time to have this done now rather than to wait until the paralysis would disappear. Then, too, if the tendon is torn, it would be easier and less painful to mend it while the paralysis is present than after the power of the deltoid has returned. If I were in her place I would beg to have this done, but I can see that she is not a patient to be easily persuaded."

Consent being obtained, the operation was done.

"Operation on Mrs. B., March 13, 1928—ether anæsthesia. An incision was made (not over one-half inch long) over the bursa and the new instrument I have had made was introduced. It is a modified nasal speculum and worked perfectly. On incising the bursa, free fluid escaped. The diagnosis of rupture of the supraspinatus and infraspinatus could be made and the extent of the rupture determined. The incision was then enlarged into the routine bursal incision, about two inches in length.

"Considerable difficulty was encountered in pulling forward the retracted ends of the tendons. The biceps tendon was intact and covered by the edge of the capsule and the tendon of the subscapularis. I eventually succeeded in drawing the tendons together, and, as no stub was left on part of the tuberosity, I drilled a hole and passed a braided silk ligature through it and the heaviest portion of the supraspinatus tendon. I also curetted lightly the raw bone on the tuberosity to stimulate attachment of the tendon. Some crumbs of bone were found in the retracted portion. One bit was removed and another sewed to the tuberosity with

the tendon still attached. The muscle was closed with catgut, but the bursa was not sutured. A folded pillow was placed in the axilla.

"N. B. In this case an unusually accurate closure was made, perhaps because the retracted muscles were paralyzed and yielded more when pulled forward. A good result should be obtained if the wound heals normally, and, as I fully expect, the muscles regain their power."

The wound healed well and the muscles redeveloped. The patient has now a good strong arm with considerable power in abduction. I saw her last on Nov. 10th, 1930. She still complains of soreness and pain and weakness after using it, although it is a year and a half since the operation. She still receives compensation and feels unable to go to work. The X-ray shows a defect in the tuberosity where the small fragments were removed. The patient admits great improvement, but it must be put down as one of those cases of which the surgeon is proud, but in which the patient is not wholly satisfied. Of course the question of compensation enters the problem. If I could obtain as good a result as this, operating seven months after the accident, it seems to me very convincing that similar operations, done immediately, would be very successful.

I have on two occasions explored the bursa in other cases of axillary paralysis and found evidence of partial rupture of the tendon not extensive enough to demand suture. The wound did not in any way interfere with the convalescence, and the power of the deltoids returned satisfactorily.

It may well be asked how we are to know immediately after a shoulder dislocation has been reduced, whether the deltoid is paralyzed or whether there is crepitus in the joint from fragments of chipped tuberosities? Do I recommend churning the joint about and risking redislocation and further traumatization? Might not a deltoid paralysis disappear within a few days? Would I make it a rule to explore every bursa after every dislocation because the supraspinatus may be ruptured?

Let me say emphatically that I believe little if any harm could be done by permitting the patient to move his arm immediately after the reduction. Uncomplicated dislocation cannot recur unless the arm is abducted *and externally rotated*, but to test whether paralysis is present in the deltoid, it is not even necessary to manipulate the arm. One can hold the elbow at the side and ask the patient to make an effort to abduct. If the deltoid can be felt to contract, that settles the question. This test should be applied both before and after reduction, and can do no possible harm. In case of doubt, I should not hesitate to move the elbow away from the side in internal rotation,

and then to let the patient hold it there if he can. Unless the arm is externally rotated or carried to the pivotal position, it cannot dislocate. A case that readily redislocated should certainly be explored.

Electrical testing of the muscle for the reaction of degeneration (*i.e.*, lack of faradic response and persistence of a slow galvanic response) is of no help in the first ten days, for it takes about this time for degeneration to occur. Even later on, the simple test of palpation of the muscle during voluntary effort seems to me nearly as reliable and more practical.

Chips of bone from the facets should be found by the X-ray either before or after the reduction. In at least two of the above cases they were present, as shown by the X-ray and confirmed by operation. Their very presence indicates that exploration is advisable, unless the fragment is large enough to suggest that the base of the bursa is not torn as discussed under fractures of the tuberosity.

The following case will be reported in some detail, not only because it is an illustration of the point we are at present discussing of the desirability of exploration of the bursa in cases of circumflex paralysis following dislocation, but because it illustrates many other points spoken of elsewhere in this book.

CASE 115

On Sept. 10th, 1928, a man of 48 was referred to me by an industrial insurance company. He was a well-built, wiry individual who had never been sick or had any serious accidents, although for most of his life he had been employed as an adzeman in a building and wrecking company, a rather hazardous occupation. On August 21st, 1928, he fell from a second-story roof, and as he fell, caught the edge of the roof of a shed on the first story with his bent and half-abducted right arm, thus sustaining him for a moment while the full weight of his descending body, in an almost upright position, plunged downward. A tremendous force was therefore brought to bear just at the shoulder joint. He was taken to Hospital A, one of our best institutions, where X-rays demonstrated a subcoracoid dislocation. Ether was given, the dislocation reduced and the patient sent home with his arm bandaged. He was attended by a doctor at his home, who readjusted the bandages a few times. On August 29th, this doctor, feeling that there was something still wrong with the shoulder, called the medical clinic of the insurer and requested that they take over the case. On August 30th, a representative of the medical staff of the insurer called to see the patient at his home and found "arm markedly swollen, shoulder black and blue, the entire arm edematous. The appearance of the shoulder at that time looked as though the joint were still out of place. He accomplished none of the usual motions of the shoulder, etc." The doctor then took the patient to Hospital B, where more X-rays were taken, which "conclusively prove that the head of the humerus is in proper relation to the glenoid." The man

received palliative treatment at this hospital for a few days, during which the swelling subsided a little, and he then attended the insurer's clinic, where I saw him on Sept. 10th, and found that his deltoid was paralyzed, that he had "dropping shoulder" and fluid in the joint. I advised having him enter another hospital to be treated with his arm in abduction for a week or two. He entered Hospital C, Sept. 11th, and I took charge of him.

On Sept. 17th, after having reviewed the X-rays taken at all three hospitals, I reported to the insurer's medical department:

"At the first visit I put him up in as near abduction (elevation) as I could, and on each occasion since I have managed to get the arm into still greater abduction. The patient has much improved. The deltoid fibers have contracted so that they are no longer flaccid, and I believe that I can feel a little contraction in them, particularly in the anterior portion.

"When the arm is almost completely abducted (elevated), it is quite evident from my examination and from the patient's feeling that the head of the humerus is in approximation with the glenoid, but when the patient stands or sits up, letting the arm fall, the head of the bone is thrust forward and downward, presenting the peculiar resemblance to dislocation which had been noticed by Dr. ———. On thinking the case over I have become satisfied that this patient has five different lesions.

"First, he had a rupture of the axillary vein (P. S. I think now that this was probably the axillary artery itself.), or one of the other large veins, causing a hemorrhage which infiltrated the whole upper arm and upper part of the forearm, and also the subcutaneous tissue on the adjacent side of the chest.

"Second, he had a direct trauma to the circumflex nerve, or to the cord higher in the neck, causing paralysis of his deltoid.

"Third, a fracture of the greater tuberosity, which resulted practically in a rupture of the supraspinatus, because the fragment of bone (P. S. A tiny one.) has been left down under the deltoid, *and the supraspinatus has retracted under the acromion.*

"Fourth, a dislocation of the humerus which allowed the head of the bone to be pushed down into the axilla, while at the same time the fragment of the tuberosity was pushed below under the deltoid by the acromion.

"Fifth, it is possible also that the long head of the biceps is torn; certainly there is something which tends to slip in between the head of the bone and the glenoid as soon as the arm is adducted.

"In my opinion, it would be best for this patient, to do an exploratory operation under local anæsthesia, and open the bursa to see whether the supraspinatus tendon is torn, and if so, whether it can be replaced. It probably would be best to leave the displaced fragment to absorb."

Soon after this I did operate.

"A routine bursal incision was made, and after opening the roof of the bursa, the articular cartilages and joint cavity were directly visible. There was no sign of the torn supraspinatus, which had retracted. The incision was lengthened downwards and upwards, so that it extended from the coraco-acromial ligament downward about two inches; still none

of the short rotators were seen. A sheet of tissue resembling joint capsule was cut through in making the incision, and this was not true joint capsule, but light scar tissue, which had formed beneath the deltoid. A large, bare area of bone was exposed at the position of the greater tuberosity. The bicipital groove was visible, but the biceps tendon was not to be seen. The subscapularis was detached from the lesser tuberosity.

"By pulling down upon the arm a space could be made between the head of the humerus and the glenoid fossa. The finger could be passed completely around the head of the humerus without encountering any attachment of the short rotators. The joint cavity was washed out with warm saline, and the glenoid inspected. Some soft tissue (P. S. The musculo-tendinous cuff.) covered over the glenoid, and the normal glenoid fossa could be palpated beneath it. This tissue was in all probability composed of the short rotators. It was thought inadvisable, if not impossible, to pull out and readjust these rotators. At no time during the manipulation, although only the skin was anæsthetized, did the patient complain of pain.

"This case has been remarkable in many respects. Nothing was accomplished by the operation except to determine the extent of the injury. When the wound was opened it was quite apparent that the nerve supply of all the tissues involved beneath the skin was destroyed. For instance, I could put my finger in between the glenoid and the head of the humerus, and feel all around the head without giving the patient any apparent pain. I could raise the arm in abduction and internal rotation, and move it about at will without starting any pain or any spasm. All the short rotators were evulsed from the bone, and the head could easily have been pushed out through the wound as far as the surgical neck, as we do in excision of the head of the humerus. When the joint was apparently reduced the short rotators and capsule lay across the glenoid in such a way that the head rested on them; therefore, accurately speaking, the dislocation was entirely unreduced, and yet only a film of tissue one-eighth to one-quarter inch thick separated the articular surface of the humerus from the articular surface of the glenoid. The condition was similar to that represented in Fig. 58, except that the fragments of bone were much smaller."

I feel very certain that the force which caused this injury resulted in pushing the head of the humerus straight down into the axilla, with such violence that the whole capsule together with the short rotators and biceps was evulsed entirely, probably in one piece from the attachments on the tuberosities. The infraspinatus and teres minor were torn as well as the subscapularis and biceps tendon. As the head of the bone descended into the axilla, it ruptured an axillary vessel, or probably a large artery. At the same time a chip from the greater tuberosity was pushed by the acromion into the space beneath the deltoid, and when the dislocation reduced, this chip still stayed down there as shown in the X-ray. It was the same

force and the same leverage which usually results in fracture or dislocation, but in this case the bone, except for a bit of facet, held, and all the soft parts gave way.

The problem of surgical treatment after I had exposed the condition was too difficult to solve. I could have cleaned out the glenoid, but if I had done so, I could not have gathered the ends of the short rotators and resutured them to the tuberosity, even by drilling holes in it, for the tuberosity itself was too much damaged.

The only operation that I can conceive of doing in this case, and this might still be done, would be to use a sabre-cut incision, dissect out the short rotators separately, clean out the glenoid, pass a strip of fascia lata through the head of the humerus, and attach the short rotators to it. (Fig. 52.)

I did not feel that I could perform such a difficult operation under the circumstances. It seemed to me better to leave the arm in the condition that it was, hoping that a false joint would develop which would permit him to use the other motions of his arm without abduction.

The patient was put to bed with the arm in an abducted position, and I let him remain in this position until the wound was healed and the power of the deltoid had returned. This resulted in the patient's feeling much encouraged, so that he would not consent to having another operation performed, feeling that eventually he would recover the remaining use of his arm. Finally, believing that he could get a more useful arm, I urged him to have an arthrodesis performed, but he would not consent to this and insisted on returning to his birthplace in Newfoundland, where he could live cheaply on his savings. He settled with the insurer and left. On May 23, 1932, he wrote me that there was no improvement but that there was no sign of axillary aneurysm. At any time he could have an arthrodesis done which would give him a painless, useful arm, although with the arc of motion limited fifty per cent. I should have done this operation in the first place. He may still develop an axillary aneurysm.

Among the instructive things about this case were:

That there was no anæsthetic skin area larger than a fifty cent piece, and this was down at about the middle of the deltoid. Yet when the skin was anæsthetized, the whole joint could be manipulated without any feeling on the part of the patient. This is to be explained by the fact that the nerve supply of the joint is from the deep branches of the axillary and also largely from the suprascapular, which was removed from the field by its retraction with the

capsule over the glenoid. The seat of incision in the skin was supplied by the cervical plexus, which was not injured. Although this case showed all the common complications of dislocation, I feel that many cases have the main one of rupture of the supraspinatus.

As a matter of fact, after satisfactory reduction of an uncomplicated dislocation, the patient does not need immobilization, and a certain amount of active and passive motion is desirable, and neither painful nor harmful in any way. I believe that fixation in the sling position for ten days or two weeks after dislocation is actually harmful, and that routine exploration of the bursa in every case would be preferable. *Yet I do not advocate routine exploration, although I believe it should be done in all cases of deltoid paralysis that cannot voluntarily abduct the arm, in all cases where evulsed facets are demonstrated by the X-ray, and in all cases where the surgeon is uneasy as to whether the dislocation is properly reduced.* Any such rule may have exceptions.

I have never known or seen a case of deltoid paralysis which *completely* disappeared in a few weeks, let alone in a few days; it always takes weeks or months for the complete recovery. If the patient is to be laid up some time, why not make the exploratory incision at once, and if no rupture is found, get the benefit of the rest with the arm elevated?

The reader may question my dictum that when the supraspinatus is not injured the patient can abduct his arm by the use of this muscle alone. My evidence comprises a number of cases personally observed and a few cases from the literature.

Perhaps the most convincing case which I can report was that of a young Italian laborer who had absolutely no deltoid muscle, but who could elevate his arm, not only easily, but with great power, with an hypertrophied supraspinatus. This patient had been seen by a number of other doctors, and the question of the existence of a bone sarcoma had been brought up, because the shoulder was so misshapen that the appearance suggested a tumor just above the scapula. Some one also had made the diagnosis of progressive muscular atrophy. The following is a copy of my report to the insurance company.

Sept. 30, 1926. This patient is a rather small Italian laborer, age 26, whose face is distorted and scarred. He claims that this condition of his face is due to wounds received during the war in Italy. Otherwise than that, he says he has been well, until April 29, 1926, when he was climbing a ladder, holding a pail in one hand, and a rung broke and he fell. He does not remember how he fell, nor on what he landed, but the records

submitted with him state that it was three stories. He was taken to a hospital and treated for fracture of two of the left lower ribs and an abrasion of the hand. He was soon after discharged from the hospital, but within a week after this developed pneumonia and had to return.

With the request for the examination of this patient you were kind enough to submit a folder containing a portion of his record. This folder does not contain records of his case prior to August, so that I am unable to ascertain either from the record or from the patient just what was the condition of his shoulder during the first few weeks after the accident. It appears from your record that the tumor in the supraclavicular fossa was not noticed until August. I can only give my impression from the examination at this date without a real history. The X-ray, which you also kindly sent, rules out any lesion of the shoulder bones and indicates that the tumor is subfascial.

Examination: The scars on the left forehead and cheek indicate that he had an old wound resulting in entire disappearance of the left masseter muscle and perhaps some loss of bone. The bulging side of his face is the normal one, and the disparity of the two sides is due to the contrast of the normal with the atrophied side, rather than *vice versa,* as one would guess at first sight. The deformity of the face is so great as to suggest a "facial hemiatrophy." One must consider the possibility that this was a congenital condition and that it was not related to the wound and scars.

The examination of the shoulder discloses a most unusual condition, namely, a complete atrophy of the whole deltoid muscle without the involvement of the neighboring muscles, except as shown by a mild fibrillation. It is very interesting that the patient can perform complete abduction of the arm with the supraspinatus alone without the deltoid. He does this so easily that it suggests that the present condition has existed much longer than since last April. Another point which suggests this conclusion is that the patient's arms are covered with tattoo marks, which are not misshapen in any way, as they would be if they had been done before the atrophy of the deltoid occurred. He states that these tattooings were done in 1923.

Referring to the tumor in the supraclavicular fossa, there is certainly a hard mass at this point, but I do not feel sure that it is a tumor in the sense of a new growth. It is subfascial, firmly fixed to the upper ribs, but not to the scapula. It is not tender to any great extent. It is immovable, smooth, hard and not elastic. It is covered more or less with muscle and is difficult to describe accurately. I am not sure that it is not a peculiar curvature of the ribs due to the unusual shoulder condition which may have existed since infancy.

There is one other finding which may have some bearing, and that is a partial if not complete paralysis of the left serratus magnus, causing a slight angel wing appearance of the posterior edge of the scapula.

Discussion: This case has puzzled me a great deal, but I have come to the conclusion that it is an instance of an old infantile paralysis, and that the recent injury in April had little or nothing to do with the condition of the shoulder. I do not think that the diagnosis of progressive

muscular atrophy is correct, although I would admit that it might be if good proof could be obtained that the patient possessed a deltoid within the last few years. The extraordinary development of the supraspinatus muscle is very much against this hypothesis. I am even inclined to doubt that the facial condition is due, as the patient says, to wounds received in the army. I would be much more inclined to think that the man "got by" in a physical examination and entered the army, as many of our own men did, without a thorough looking over. On the whole, I am of the opinion that no treatment will do this patient any good, and that he, himself, knows that he is trying to put over conditions which he previously had knowledge of, as results of the recent accident. I can feel slight irregularities on some of the lower ribs which suggest that they have been fractured, but nothing that I would be willing to declare were evidence of fracture.

The patient had noticed no tumor in the supraclavicular fossa, nor does he complain of symptoms from it, nor does the X-ray show any definite indication of a true tumor. From the patient's point of view, exploration of this tumor might be worth while. The possibility of its being a neurogenic fibroma or sarcoma has to be considered, but I am inclined to think that exploration would show it to be a compensatory deformity of the rib, due to the use of the arm without the deltoid since childhood. I cannot, however, believe that it is a part of the duty of any insurance company to have this done for the patient.

Notice the similarity of development of the two hands and forearms. This is consistent with a deformity of the shoulder in infancy, and not with one beginning April last.

I finally convinced the neurologist by an X-ray comparing the two scapulæ which showed that of the affected side to be much smaller than the other, and he withdrew his tentative diagnosis of progressive muscular atrophy. The upper three ribs were bowed outward and gave the appearance of tumor. As a matter of fact, the cause of the atrophy of the deltoid in this case is unimportant for my argument. Whatever its cause, the deltoid muscle was gone, and the supraspinatus was observed to function as a powerful abductor.

I have from time to time seen other cases in which the supraspinatus was able to perform abduction in spite of a paralyzed deltoid. Quite recently I saw such an instance in a patient of my own, who was riding a motor cycle and collided with a truck. Besides minor cuts and bruises, he had a definite paralysis of the deltoid, but in spite of this he could elevate the arm to an upright position. After several months the deltoid returned to normal, and a year after the injury was as well as ever. In this case, also, the sensory area corresponding to the circumflex distribution was no larger than a half dollar, if we may judge from the skin anæsthesia.

SENSORY LESIONS OF THE SHOULDER

Before leaving the subject of injuries to the brachial plexus, something should be said in regard to whether the sensory roots may be affected without involvement of the motor roots. So far as I know, such cases have not been described, and it is even very rare to find any mention of sensory involvement in the descriptions of cases of motor paralysis of the upper type. In the lower type the skin anæsthesia is in the hand and arm, and not in the shoulder. Probably most neurologists would at once suspect hysteria, if anæsthesia over the shoulder without motor involvement followed an accident, yet I am convinced that such symptoms may arise from *bona fide* organic causes.

The fact that anæsthesia so seldom accompanies motor injuries is an additional argument to support Dr. Stevens' contention that ruptures of the plexus are usually external to the snubbing on the transverse processes. If the injury extended within the spinal canal, the posterior sensory roots should be involved. But may these roots, or their posterior sensory branches, not be involved separately? I am inclined to think that they may, perhaps not within the spinal canal, but after they have emerged from the bone and are perforating the deep layers of fascia on their way to the skin.

In certain cases I have found areas of extensive paresthesia in the regions supplied by the posterior branches of the cervical and brachial plexus over the dorsum of the shoulder. There was localized anæsthesia or an intense hyperesthesia in this area. There was no definite muscular paralysis, yet the individuals were incapacitated on account of pain and soreness on the use of the shoulder. I am not able to state exactly what this condition is, but I am inclined to interpret it as an injury to the posterior sensory roots of the brachial plexus at a point between the dura and the place where they penetrate the deep fascia in the back near the vertebræ. It seems possible that a tearing of the heavy fascia at this point might stretch or pull off a number of these roots, without doing any damage to the motor roots.

Case 1. W. J. A muscular, strongly-built teamster of 37. On Feb. 7th, 1929, he jumped up several inches from the floor to pull down a lever in a factory and felt a sudden, sharp pain in his neck and was immediately unable to use his left arm. He saw many doctors and had various forms of palliative therapy. I saw him on July 31st, 1929. He stated that there had been no great change in his condition since he was injured.

Examination showed a powerfully built man in apparent health, who held his head and shoulders in a peculiar manner as if dreading any twist or turn of his body. The posterior muscles of the right side of the neck were very prominent, as contrasted to the depressed condition of those on the left. It seemed to me that this lack of symmetry was due to lack of function of some of the left muscles rather than to spasm of the ones on the right, yet the trapezius could be contracted to hunch the shoulders so that the muscles affected, if any, must be the deep group. No special muscle could be identified as paralyzed. I found an area of intense hyperesthesia to light stroking of the skin of the back of the left side of the neck and chest, corresponding to the distribution of the dorsal branches of the cervical and brachial plexus; yet this area was not sensitive to a pin prick.

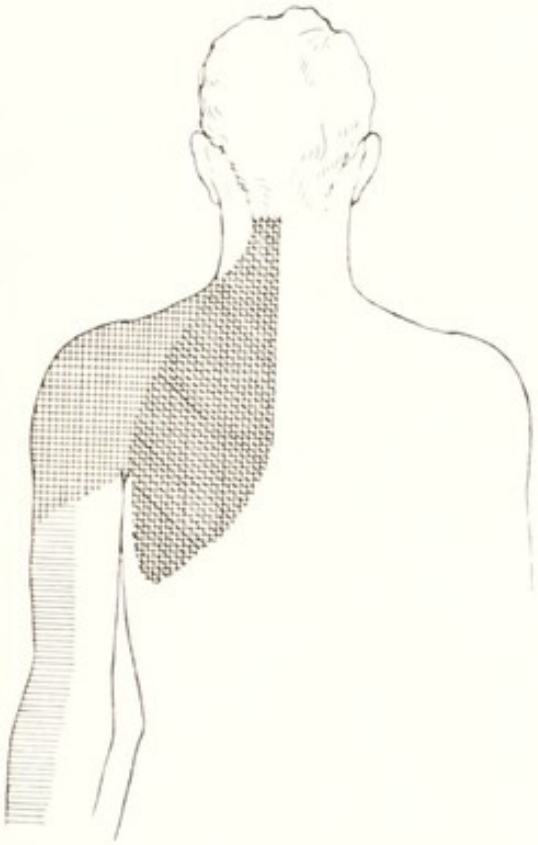


FIGURE 66. AREAS OF SENSORY DISTURBANCE UNACCOMPANIED BY PARALYSIS

The disturbed sensation area in Case 1 involved only the back. In Case 2 it involved the back and shoulder. In Case 3 it involved the back, shoulder and outer and anterior portions of the arm. There was no definite evidence of paralysis of any of the muscles, although the writer could not absolutely determine that there were no paralyses of the deep muscles of the neck. The curved posterior margin on the back was identical in all three cases.

A second examination was made Feb. 3rd, 1930. There had been slight improvement, but the condition was essentially the same. Motions of the joint and the development of the muscles were normal.

Case 2. J. M. A red-haired, strong-looking Irish laborer of 24 was struck unexpectedly from behind by a huge roll of leather, which was immediately followed by another roll which also struck him. He was knocked to the floor unconscious and taken to a hospital. He sustained injuries to the hip and ribs and to his left shoulder. I saw him on June 27th, 1929, nearly six months after the accident. He stated that the symptoms had been the same ever since the accident, so far as the shoulder was concerned. He had completely recovered from his other injuries.

Examination showed that the left shoulder joint and the muscles about the shoulder were normal. He, too, held his neck in the same peculiar position. There was tenderness about the upper cords of the plexus and pain in the rhomboid region. There was anæsthesia to a pin prick over the whole area supplied by the posterior branches of the left cervical plexus, and by the circumflex and posterior branches of the upper five or six thoracic nerves. The posterior outline of the anæsthesia was similar to the outline in Case No. 1, but the area of anæsthesia extended over the circumflex area also. I saw this patient again on December 10th, 1929, and the anæsthesia remained the same. This case was considered to be hysterical by a neurologist, but I feel sure it was not.

Case 3. J. D. On October 14, 1929, a longshoreman of 42 was struck on his left shoulder and left side of his neck by a heavy load swinging on a hoist, and was knocked about fifteen feet. I saw him on March 3rd, 1930, nearly six months later. He stated that his condition had been about the same since the accident. He complained about inability to use his left arm without pain.

Examination showed a similar paresthesia to that in Case No. 2, except that in addition to the area involved in Case No. 2, the areas supplied by the median and radial nerves in the forearm and hand were also involved. In this case also there was no paralysis or wasting of the muscles in the arm. They were, however, somewhat weak. There was some delayed sensation in the ulnar supply of the little and ring fingers and ulnar region of the forearm. This would seem unlikely in an hysterical case.

Several other similar cases have come under my observation. They are mentioned in this connection because my belief is that this group of cases constitute a real clinical entity, although the peculiar character of their sensory symptoms, without motor symptoms, suggests hysteria. I am sure that similar cases will be met by other surgeons who see industrial cases, and feel that they merit study and discussion. In the three cases the posterior outline of the paresthesia was the same. Unfortunately I have no treatment to suggest.

Within six months after writing the above, I saw two more very similar cases of this puzzling type. They would have escaped my notice had I not had the previous experience. I feel that it is important to test the dorsal region of the neck and shoulder for anæsthesia or hyperesthesia, particularly in strong, muscular individuals, such as all these men were. They were not at all of hysterical type, and yet the only findings to account for their great and continued complaint of pain were these unusual areas of paresthesia.

There is a possibility that these areas of paresthesia represent overuse of physiotherapy. All these cases had had prolonged treatment. I am convinced that "Baking," whether by hot water bottles or electric pads and lamps, may produce similar disorders of the

nerve terminations in the skin. However, these patients all stated that their symptoms were the same or worse soon after their accidents. Although I cannot explain the exact mechanism of the injury in the cases described, I am loath to believe that previously sound, muscular men, after admittedly serious injuries, should succumb to hysteria and exhibit its symptoms by skin paresthesia in this region with a definite posterior border arching away from the spine. The fact that the posterior branches of so many different nerves are involved suggests a long, vertical rent in the deep fascia of the neck.

REFERENCES

I know of no writings on the subjects considered in this chapter.

Chapter XIII

HYSTERIA, NEURASTHENIA, NEUROSIS, TRAUMATIC NEURITIS, MALINGERING

The line between organic and functional lesions is difficult to draw in cases such as those alluded to near the end of the last chapter. Yet the distinction should be made. In a book it is easy to do this by changing the title of a chapter, but in practice there are cases in which wise men disagree.

I have nothing particularly original to contribute to the subject suggested in the heading of this chapter. My work has been that of a surgeon, and while I have always been interested in the phenomena of surgical conditions involving the various distributions of the motor and sensory nerves, I have had no particular experience in handling cases of functional nervous disorders. Every surgeon and practitioner unavoidably sees cases in which the hysterical or nervous element is involved, although it is rare for any one not engaged in neurological work, particularly, to see and to *treat* cases which are purely functional in character. Unavoidably, the question of the nervous element is frequently presented in cases of lesions in the shoulder. In fact, it seems to me that it is normal in the average individual to present a certain percentage of this complication after injuries of any kind, especially if the question of justice or of compensation is under consideration. It would seem to me that a twenty-five per cent allowance for exaggeration might be fairly given to any normal patient when there are Medico-Legal questions involved. Furthermore, it is also true that the longer a patient is laid up as the result of an injury, the more his mental state is involved in relation to the physical side. His mind becomes riveted on the injured part; he notices every little point of soreness, and marks it deeper and deeper on his mentality in order to use it to strengthen his case. He goes to bed thinking of his troubles and thinks of them when he wakes—perhaps he dreams of them. Often in industrial cases, he is insufficiently fed; his general condition deteriorates; he talks to every one who will listen, and describes the details of the accident, the character of his suffering, and his feeling that injustice has been done him. From a life in which his whole mind has been filled by his occupation, he is thrown into idleness and has nothing to do but dwell on his misfortunes. Such a man is not abnormal, in my opinion, and cannot be considered hysterical or malingering, until

he passes from the twenty-five per cent class into the fifty or seventy-five per cent class. One of the strongest arguments against our Workman's Compensation Laws is, that they so often result in producing this state of mind.

After all, what are the conditions named in the title of this chapter? The following definitions are given in Dorland's Medical Dictionary:

Hysteria: Disease mainly of women, characterized by lack of control over emotions and acts.

Neurasthenia: Depression due to exhausted nerve energy.

Neurosis: A nervous disease, especially a functional disease.

Traumatic neuritis: Inflammation of a nerve caused by an injury.

Malingering: Feigning illness.

Doubtless, by consulting textbooks on Neurology, these definitions might be made more satisfactory—certainly much greater detail would be given and therefore more confusion. To the ordinary surgeon or practitioner, these definitions would suffice, and yet when one comes to apply them to individual cases, they are not so far apart that it is easy to label our patients. All five diagnoses might be applied correctly to one case at different times. Even the two extremes, hysteria and malingering, are not easy to separate. We can readily picture an hysterical woman quite unconsciously simulating any kind of disease, or we can picture a husky, shrewd, healthy man feigning an illness merely to get a few hundred dollars compensation. Between these extremes, we find the ordinary man I have described above, who takes a trivial lesion too seriously, will not endure a degree of pain with which a brave man would work, and prefers to associate himself with those who blame the rich for all the misfortunes of the poor.

In industrial work, one often sees that a quarrel with a foreman tends to make a healthy man exaggerate his injury. His sense of injustice from the behavior of the foreman predominates over his common sense, and he uses his illness as a method of discrediting the foreman. He perhaps has more zeal to have a doctor support his contentions, in order that the foreman will be put in wrong with his employer, than he does to obtain compensation for an injury, the pain of which he would have endured if there had been no quarrel about it.

It sometimes seems to me that it might be easier for an Industrial Accident Board, in questioning experts about the relative amount of the nervous to the physical element after a given injury, to ask for a statement as to whether, in the doctor's opinion, the

hysterical or neurasthenic, or malingering element formed a twenty-five, fifty or seventy-five per cent basis in the case—freely allowing twenty-five per cent for the average man. The individual who is one hundred per cent physically injured, could only be a dead one. There are men who will endure physical injuries and continue to work in spite of pain, sleepless nights and overbearing foremen, but we cannot judge the average by these truly brave individuals. When it comes to the patient in whom we believe the nervous element is one hundred per cent, it will have to be shown that prior to the injury he had always been one who complained in an exaggerated way about other trivial experiences in his life. It is hardly possible that a sound-minded, sound-bodied man will be turned by an injury into a malicious, cowardly individual, who would allow his self-respect to fall so low as to present extreme hysterical symptoms. In those cases in which the nervous element and the physical element combine in such degree that they fall into the twenty-five to seventy-five per cent class, we are more likely to make mistakes. The same individual, as the months pass, may descend from the twenty-five to the seventy-five per cent class.

I have the feeling that some neurologists, who have seen many mental cases and the extreme forms of hysteria, are too ready to pin the label of hysteria to symptoms which may possibly be due to physical conditions of which they are ignorant. It is no easy matter for a neurologist to keep accurately in his mind, the areas of peripheral distribution of the sensory nerves, nor the paths of these nerves as they pass through the different portions of the brain, through the spinal cord and through the canals of exit between the vertebræ and outward through the planes of fascia. Physicians having had experience with many mental cases, and having seen that hysteria in extreme cases can mimic almost any physical condition, are ready to believe that the phenomena, in an individual case in which the symptoms consist in areas of unexplained anæsthesia, are due entirely to the mental condition. Surgeons, on the other hand, dealing with concrete anatomic structures, rather than with functions, err on the opposite side. Not infrequently they even neglect to test for anæsthesia at all. If they can conceive a possible anatomic basis to explain the areas of anæsthesia, they prefer to accept that explanation, rather than to assume a functional disorder. The neurologist and surgeon will, therefore, come to agree on those cases in which their combined knowledge of anatomy and pathology is unable to interpret a group of symptoms.

Yet they both may be ignorant of the true cause, for it has been my experience to have seen patients with rupture of the supraspinatus who have had the misfortune to be classed as neurasthenics, malingerers, or hysterics, because the signs and symptoms which I have alluded to in previous chapters have not been recognized by the doctors in attendance.

I am afraid there are still many doctors who have never even heard of rupture of the supraspinatus, and many of our teachers in our medical schools will be astonished at the statistics of Dr. Akerson, that this lesion is as common as demonstrated by his finding evidence of it in nearly forty per cent of his autopsies. As I look back on my own experience, I find that even for years after I had recognized the existence of this lesion, I made the mistake in some cases of attributing the patient's unwillingness to raise the arm to his tendency to malinger.

Before one makes a diagnosis of hysteria or its allied conditions, the following four points should be taken into consideration especially.

(1) The age of the patient. It is generally admitted that if a patient is to have serious hysterical phenomena, these signs appear in youth. It is most unusual for a healthy individual in adult life who has previously been normal to exhibit a sudden attack of hysteria.

(2) The industrial record of the patient. If a suspected patient has been in continuous employment and given satisfaction at his work for a long period of years prior to his injury, it strongly influences me to consider that the neurotic element in his case is not large.

(3) The length of the period, from the time of the accident to the time of the examination, must be taken into account. I have already explained that a normal individual, forced out of employment for many months by a painful injury, may present a pitiable mental condition. The wolf is at his door and his family's door, and the elements of worry and sleeplessness, insufficient food, and self-concentration, bring about what I should consider a normal high percentage of the nervous element.

(4) The degree of fright received at the time of the accident, especially as evidenced by the behavior of the patient within the first few weeks after the accident. There is no question in my mind that a healthy man may be reduced to a pitiable condition of "nerves" by a single accident, and I am inclined to believe that the brain may register a local fear complex, following an accident to any given

part of his body. It seems to me possible that the brain may receive such a shock from the supposed loss of the use of one limb, that the corresponding portion of the brain may be in a helpless condition, while the rest of the patient, so far as his fear for the rest of his body goes, may be normal. Such a condition may be a "traumatic neurosis."

The three cases alluded to at the end of the last chapter as possible instances of trauma to the sensory roots of the brachial plexus were all in healthy, physically strong, adult males, who had worked steadily and had exhibited their symptoms immediately after the accident—and persisted in their claims of the same symptoms for many months afterwards. To my mind, these facts are strong evidence that the lesions were of a true physical nature. That the anatomic injury is hard to explain, from what we know of the connections of the sensory nerves at the ganglia near the nerve roots, may be attributed to our ignorance. It seems to me side-stepping to assume either a conscious or a subconscious effort to imitate such peculiar conditions, especially as the three cases do offer a pretty close resemblance in the areas of distribution to the known areas supplied by certain peripheral nerves.

The diagnosis of true hysteria should not be made lightly in cases of cutaneous anæsthesia about the shoulder. One should be very sure that the limits of the field do not correspond to the known anatomic distributions of the different nerves and their branches. Even this is not enough, however, for the areas of anæsthesia must be looked at also from the segmental point of view.

One of my cases, for instance, had an area of sensation on the inner side of the upper arm corresponding with the area supplied by the nerve of Wrisberg, which comes from the third and fourth dorsal segments, yet the skin all about this area was anæsthetic down to the ulnar distribution in the hand, which also comes from about the fourth dorsal segment.

It is out of place here to go into the fine diagnosis of peripheral nerve lesions. It would take too large a proportion in a book which is intended to have its focus on the subacromial bursa, which gets its sensory nerve supply from only two nerves. These are the suprascapular and the circumflex in the deep tissues. The skin nerve supply comes from the cervical plexus.

A true hysteric anæsthesia has the boundaries which the patient thinks should be there, as, for instance, the classic "glove anæsthesia," bounded by a ring around the arm entirely at variance with the

anatomic nerve supply. But complete hemianæsthesia is usually considered a very sure proof of hysteria, although it is anatomic in distribution.

While I was writing this chapter (March, 1930), an industrial accident case was referred to me. He was a dull-looking Portuguese laborer of 43, who had fallen twenty-five feet from a staging on December 21, 1927, and had been unable to work since. His main symptoms were in the back and sacroiliac region. His case had been under discussion for over two years, and four doctors had given their opinions without agreement being reached. No one had suggested hysteria. On examining the shoulder, I found that he was anæsthetic over the whole right shoulder and arm, but further investigation proved that he also had a *complete* hemianæsthesia. The muscles were well developed, and there was no asymmetry of his body. The diagnosis of hysteria was made and was later confirmed by a neurologist.

The following case illustrates the difficulty of diagnosis between traumatic neuritis, traumatic neurosis and hysteria, in an injury to the shoulder. Possibly the case furnishes an example of all three diagnoses.

Case Report

R. S. An Italian of 43 was helping to lift a huge rock on Sept. 15, 1922. The rock slipped and the man injured his shoulder. His arm became useless and swollen. A local doctor bandaged his arm to his side with a Velpeau bandage. He had been incapacitated ever since. I saw him nearly two years after the injury. The following is my report in full:

"I examined this employee at my office on July 24, 1924. He is an Italian laborer, undersized, quick, nervous and voluble, but very difficult to understand.

"With the request from the Board to examine this patient, I also received copies of the reports of the following physicians who had previously examined him: Dr. ———, Dr. ———, Dr. ———, Dr. ———, Dr. ———, Dr. ———. There seems to be a fair unanimity of opinion that the condition is hysteria.

"I hesitate very much to offer a contrary opinion, but my belief, which amounts to a conviction, is that this condition is not hysterical, certainly not wholly hysterical. I will grant that this man presents a picture of imperfect self-control and is evidently obsessed with the idea that it is impossible to use the arm. To him that arm and shoulder are two-thirds or more of his whole mental horizon. Yet I believe that extreme physical pain from a true injury is the basis of his trouble.

"My explanation of the condition is this. He had an injury of more or less severity to his shoulder—perhaps a dislocation, perhaps a ruptured capsule or tendon. Following this a bandage was misapplied—too tight—the circulation in the extremity, particularly in the nerve trunks,

was impeded. The arm 'went to sleep' in the bandage. Brachial neuritis of a degree just short of total paralysis resulted. Instead of a complete 'drunkard's paralysis,' with a flaccid condition of the muscles, a condition of paresthesia and imperfect muscular control resulted. The pain, which would have been destroyed by a little greater pressure, was on the contrary aggravated, because a little circulation persisted in the nerve trunks. The paresthesia and hyperesthesia caused by the neuritis extended up and down the brachial plexus and even to the subsidiary anastomosing nerves. Pain and hypersensitiveness are after all protective processes. Nature demanded rest for that brachial plexus and obtained it in this old, hereditary way. I believe that in this case she has succeeded in saving a set of damaged nerves which were all but permanently destroyed.

"I am aware that this explanation has little of direct proof to support it, being founded more on personal opinion than on demonstrable points. However, there are certain facts that support it and others that might be obtained. *First:* Sudden onset of hysteria in a man of this age is very unusual, but it is not at all unusual for doctors to diagnose hysteria when a real lesion is present. The burden of proof is on the one diagnosing hysteria. *Second:* Atrophy has been a striking feature since the first examination. I have never seen hysterical paralysis produce marked atrophy. *Third:* This patient presents a peculiar spasm of the muscles of the forearm, which is inimitable in hysteria, but is the rule in the 'fish fin hand' accompanying injuries of the nerve trunks of the arm. It is felt on the tendons of the wrist while one attempts to extend the fingers.

"Great light would be thrown on this case if we could obtain a reliable record of the first few days after the injury. What record has the ——— Hospital of the first aid? Did he have extreme pain after the first bandage was applied? How long was it left on? Was he given opiates? Did the hand swell after the bandage was applied? Was it purple? I think an Italian interpreter should see the family and get a matter-of-fact answer to these questions. The ultimate prognosis I believe to be good. He has, I think, passed the turning point now. He is clearly still disabled."

One of the doctors who saw this case was a prominent neurologist, and he believed it was a case of hysteria. It was easy for him to believe that an Italian laborer of 43, in good condition, from merely straining his arm while helping to lift a large stone could have an hysterical attack which resulted in such a serious condition two years later. If the injury had come from a runaway railroad engine, from which the patient in great fright barely escaped, I would be more likely to agree to hysteria; but to suppose an Italian laboring man in lifting a rock would receive adequate emotional cause for a true hysteric attack lasting two years, is more than my mind can follow. It is easier for me to believe that the patient really did strain his arm—perhaps ruptured his supraspi-

natus or biceps—had hemorrhage in the tissues (there was note of immediate swelling). After this he was put up in a tight dressing by his local doctor. It is easy for me to believe that a swollen arm, bandaged tightly to the chest with a Velpeau bandage, can cause all the symptoms shown by this man. Under these conditions I have several times seen the hand swell, the fingers become crippled, the shoulder joint become fixed, the forearm flattened where it lay in contact with the chest distally, and swollen near the elbow where it was not in contact. Indications of disturbance of the median and ulnar nerves in the hand from pressure from bandages are only too common. Two years after the injury, this patient presented symptoms in accordance with the later effects of such restriction. I believe that if we could have had sections of different parts of the nerves of the shoulder and arm at times during the course of this trouble, we should have found evidence of anæmia, necrosis, and inflammation at various points in the course of the nerves. We should have probably found an anæmic necrosis of the muscles of the forearm of mild degree. The diagnosis in this case would have been traumatic neuritis. If the question had been one of neurosis, should we have found decided atrophy two years after the injury? I think not. Yet the neurologist who made the diagnosis of hysteria accepted atrophy, for hysterical contractures, after several years, have been alleged to bring about atrophy. I have never seen such a case.

Strongly in favor of the hysterical origin was a test made by the neurologist. He gave an anæsthetic and says in his report: "Under an anæsthetic, rigidity completely disappeared and he was able to move the arm in every direction. When he recovered from the anæsthetic, however, he immediately assumed his former attitude and would not perform any movement of the arm." The neurologist was unable to effect any improvement by suggestive treatment.

To my mind, even this strong statement does not counterbalance the fact that the X-ray expert reported that the bones of the affected side were markedly atrophied. It is easier for me to believe that the neurologist's examination of the patient under ether was inexact in stating that the rigidity *completely* disappeared, than it is to believe that atrophy of muscle and of bone of such duration (a year) would not be accompanied by some rigidity in the shoulder joint.

This case is presented more because it illustrates the definitions of the words which head our chapter, than because it is a case in which a conclusion has been reached. I wanted to illustrate the point

of view which the neurologist will usually take, compared to the point of view of a surgeon. I had no opportunity to treat this patient, but I may say now that my own mind is so occupied with rupture of the supraspinatus tendon as the most common cause of obscure shoulder injuries, that I am prejudiced in favor of its being the primary factor in this case, and I should have explored his bursa. The case offers a text for preaching that the application of a Velpeau bandage, after an acute injury to the shoulder or arm, is a serious procedure. Medical students enjoy the triumph of applying their first Velpeau. It is a beautiful bandage which the teacher enjoys teaching, and the pupil enjoys learning, but applied in acute injuries, it is as dangerous as it is beautiful.

Neurasthenia. We use the words hysterical and neurasthenic too loosely. Neurasthenia does not imply loss of function without consciousness of the patient. It implies a relative loss of nervous energy, such as comes from worry and overwork in a normal individual whose resistance becomes lowered, or from congenital or nutritional lack of proportionate control over mind and body. It does not seem necessary to me to discuss this condition, which is so apparent in every sort of post-traumatic lesion. The term should not be used as a definite diagnosis after a given shoulder injury. It is a word to be applied to most long-standing post-traumatic conditions if the nervous element is somewhat in excess. Hysteria, on the other hand, is a definite diagnosis, the name of a subconscious, functional disease. The terms traumatic neuritis and traumatic neurosis, also loosely used, should be distinguished. I think that neurologists feel that traumatic neurosis is a local form of hysteria; that is, a local functional disease, due to a traumatic origin, accompanied by no demonstrable—even microscopic—signs of inflammation in the nerves. Traumatic neuritis, on the other hand, would show under the microscope signs of inflammation somewhere in the course of the nerve. A typical instance might be the adherent nerve caught in the callus of a spiral fracture of the humerus. Such forms of neuritis are, in my mind, very common, and associated with most cases of subacromial bursitis and rupture of the tendons of the short rotators. The myositis in the substance of the supraspinatus muscle, and the anæmic necrosis in its tendon, must have an effect on the finer branches of the suprascapular nerve. Every case of periarthritis following an injury would, therefore, be complicated by some traumatic neuritis.

Traumatic neurosis is a term which I think the neurologists use with much the same meaning as I have alluded to as a local fear complex.

I believe *malingering per se* in Industrial Accident cases must be rare, but I do not think it is unusual for a case, which originally was a disability from *bona fide* trauma, to become one of typical malingering. The most important reason that this sequence is rare, is, of course, that the benefits received are small.

If we take the five terms used in the heading of this chapter as a whole, including even the malicious element of malingering and the wholly involuntary phases of hysteria, we may at least differentiate this whole group of "nervous conditions" from true cases of injury to the bursa and its adjacent structures. I would certainly not be likely to use any of the above diagnoses in cases which presented atrophy of the supra- and infra-spinatus muscles. Practically every long-standing case of rupture of the tendons of these muscles results in atrophy, and this atrophy persists for many, many months—even after the local lesion in the tendon has been repaired by surgery. It is noteworthy, however, that the deltoid as contrasted to the spinati may be hypertrophied, or at least equivalent to the muscle on the opposite side, because the deltoid is overworked to replace the supraspinatus.

True lesions in this neighborhood also almost invariably present some limitation of motion in the extremes of the arcs of external rotation and abduction. After a little experience, one can tell at once whether these limitations are due to voluntary efforts on the part of a patient. There is a peculiar feeling about the limitation due to actual adhesions. Limitation due to the patient's unwillingness to allow the arm to be lifted up is quite different. He exerts a voluntary downward pull on one's hand. One may lift the patient's arm with one hand, and feel his opposing muscles contract, with the other.

If the patient has no limitation of motion, and has a jog in that motion, with a disturbance of the scapulo-humeral rhythm, it is very unlikely that the nervous element is predominant. It is scarcely possible for those who have not given attention to lesions in this region to appreciate the amount of pain and disability which may be present in a patient, who, to a superficial examiner, appears to have no decided signs of any lesion. I cannot too strongly recommend a search for atrophy, limitation, and irregular scapulo-humeral rhythm. It is due to the fact that these symptoms have been ignored that the

diagnosis of rupture of the supraspinatus has been so long delayed in attaining publicity. Do not laugh at a display of "nerves" when these signs are present. Instruction about them does not receive its fair share of a medical student's time. We were taught in detail the rarer forms of fracture and dislocation which are obvious to any one who has learned his anatomy, but nothing is said of this truly more important lesion. This chapter may seem to have been written to belittle its title, but I have tried to give due proportion to the ever-interesting effects of the mind on the body, so far as the shoulder is concerned. I do think that there is usually a physical basis for complaints of pain after shoulder injuries, and that injuries of the supraspinatus should be carefully considered in every case.

REFERENCES

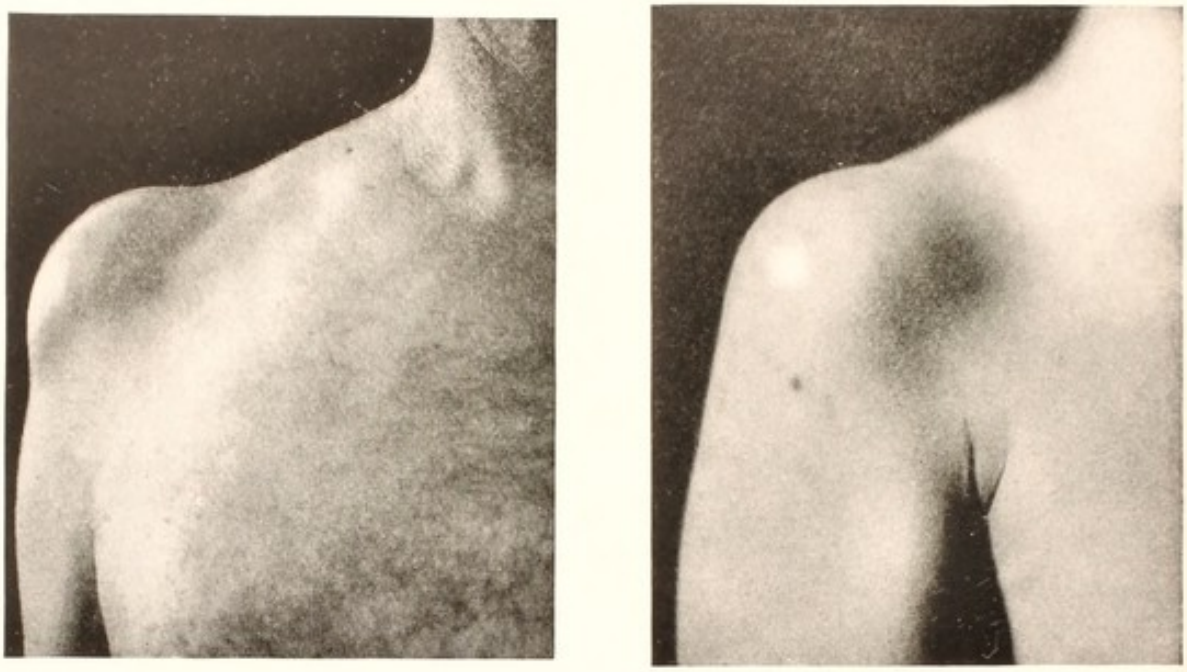
I know of no articles which relate to hysterical manifestations in lesions of the shoulder.

Chapter XIV

TUMORS IN THE REGION OF THE SUBACROMIAL BURSA

Any reference to skin lesions over the region of the shoulder will be omitted, although it would be appropriate to mention them if I knew of any particular types which might be confused with a bursal lesion or which were especially common in this region, or of any ordinary tumor of the skin which had peculiar characteristics when located on the shoulder. The shoulder may be attacked by skin lesions of any form, the number of which is not as great as the number of the names applied to them. It would be a boon to the general practitioner if the names for lesions of the skin could be codified on the basis of clinical entities, as has been done for bone sarcomas. We should find that the number of skin diseases of importance as true clinical entities would be few in number, compared to the total of their complex mystifying names. I will merely mention that Herpes Zoster ("Shingles") may affect the nerves on the dorsum of the shoulder and be the cause of obscure pain.

Lipomas of the subcutaneous tissue do have a peculiar relation to this region, for this form of tumor seems to occur with great frequency about the shoulders. They are of actual importance in diagnosis in relation to lesions of the bursa, because not infrequently the pain caused by a shoulder injury is referred to a lipoma which may have been there unnoticed for some years. The accident perhaps served to call the patient's attention to it in his search for the cause of the local pain. I noted the presence of lipomas in a little over one per cent of my cases. I have several times seen one on the tip of the shoulder exactly over the subacromial bursa, so that its external appearance simulated a swelling due to distention of the bursa itself. Usually these tumors are posterior rather than anterior, but they may overlie the acromion. They may assume great size, but are usually small, varying in size about as much as does an oyster, and they are often of a similar shape. Owing to the tough skin on the dorsum of the shoulder, they remain quite flattened, and if an attempt is made to remove them, they do not shell out as easily as do similar tumors in other parts of the body. Several patients were very emphatic in saying that the tumors had not been there before the accident, although of course they probably had been there for a long time. Theoretically, it is possible that a local bruising of the subcutaneous fat might initiate the onset of a lipoma. Personally, I think this is



a *b*

FIGURE 67. SIMILARITY OF APPEARANCES OF LIPOMA AND DISTENDED BURSA

a. A lipoma, the situation of which is almost exactly over the subacromial bursa. *b* shows the shoulder of the patient from whom the X-ray in Figure 44 was made. The greatly distended bursa makes the contour of the shoulder appear swollen, although the swelling is wholly beneath the deltoid.

very unlikely; the reader is referred to the discussion of the traumatic origin of bone tumors at the end of this chapter.

Fascial tumors are not common about the shoulder except in close relation to the bones, when they would be called periosteal fibromas or fibro-sarcomas, according to the nomenclature of the Registry of Bone Sarcoma. I have the impression, however, that this rare form of tumor is relatively more common about the scapula than about other bones.

The subject of bone tumors in relation to the shoulder joint will be taken up in some detail, partly because I have been interested in the Registry of Bone Sarcoma, and partly because I believe that any surgeon who is studying lesions of the bursa should be prepared to encounter lesions of those structures which are adjacent to it.

Before taking up the diagnosis of the different tumors which occur in the shoulder, let me present some theories which have occurred to me in regard to Bone Tumors in general. The shoulder joint is a region in which all the different forms of bone tumors are found. The bones of the shoulder do not play favorites as regards the different kinds as do many of the other bones. For instance, osteogenic sarcoma very rarely, if ever, affects any of the small bones

of the fingers or toes. A tumor of the lower end of the radius is almost always a giant cell tumor. Osteogenic sarcoma seems to have an aversion for the lower end of the tibia. Myeloma prefers a rib, and ribs prefer myelomas. Ewing's sarcomas select the shaft of a bone, while giant cell tumors prefer the epiphyses. A tumor of the shaft of the bone, with a metastasis in the skull, would probably be an instance of Ewing's sarcoma.

I am convinced that the various forms of tumors differ somewhat in detail according to the bone that they attack, and that they, to some extent, mimic the form or characteristics of the part of the bone that they are in. Thus in the future, I believe that it may be possible for a pathologist expert in bone tumors to guess, from the microscopic appearance, not only what bone the tumor is probably in, but what part of that bone. At present there are vague indications that such detailed diagnoses may be made; an instance is a form of giant cell tumor involving the tuberosities of the humerus, which is more common in this region than in any other.

It would seem to me that not only does the character of the tumor vary with the individual bones of the skeleton, but that some forms of tumors have abortive morphologic plans, as do the peculiar growths called "galls" formed on trees and plants, some of which resemble leaves, some flowers, and some bark. For instance, some osteogenic tumors, both benign and malignant, tend to form cartilage on their surfaces and to have bony bases, thus resembling roughly in form the condyles of a joint. Some of the lesions, such as benign osteogenic tumors of the fingers, resemble a twin of the phalanx itself. If in an osteogenic tumor there is a portion bulging into the popliteal space, this portion will generally be composed of cartilage, resembling in shape the big knobs of the joint cartilage. Osteogenic sarcomas, as a whole, resemble a normal callus about a fracture in their fusiform contour, and in their activities, which are always both intra- and extra-cortical. I have come to believe that tumors exhibit an architectural plan (to be sure, a very irregular one), but resembling the same mysterious morphologic design which makes the normal cells form the definite part in which the tumor arises. According to this idea the malignant impulse must be given rather to the plan than to the tissue in which the potentiality of repair lies. The normal repair process knows when its specifications are completed, but the malignant repair process has lost its growth or repair restraint and goes on unchecked, but still vaguely follows the plan of the part of the bone involved.

SARCOMA TABLE NO. 1 (Cases 1 to 1059, inclusive)

	HUMERUS			CLAVICLE			SCAPULA		Totals
	Upper	Middle	Lower	Outer	Middle	Inner	Acromion	Body	
Osteogenic Sarcoma	49, **112, 140, 282, 335, 371, 382, 436, 484, 505, 544, 570, 613, 643, 669, 809,** 812, **851, 852, 911, 1000, 1002, 1003, 1058**	**146,** 147, **751,** 156, 1016	**289,** 299, **354,** 357	**278, 492, 504**		**486**	759	**11, 197, 250, 419, 530, 612, 622,** 867, **1004**	47
Ewing's Sarcoma		**164, 241, 245,** 246, 326, **330, 561,** 576, **618, 786, 790, 876, 916**		28, **162**		**196, 629**	137, 919	**366, 515**	21
Multiple Myeloma		**242, 461, 676**	**829**	**773, 958**		594, **680**			8
Angioma								941	1
Typical Giant Cell Tumors	4, 117, 556		107, 791, 913				54, 392, 649	200, 481	11
Epiphyseal Chondromatous Giant Cell Tumors	5, 86, 174, 391, 603, 815, 816, 902, 1031								9
Atypical Sarcoma	**104,** 409, **421, 655, 779, 886, 998**	83, **297**	564	**6**		1059		**63,** 111, 447, **746**	16
Metastases (Cancer)		**66, 148, 150, 347, 782**		**926, 972**		**94**			8
Benign Osteogenic Tumors	31			993				253	3
Inflammation (Including Cysts and Callus)	166, 169, 455, 724, 732, 868	199	207, 652		18, 438, 823				12
Discarded for Poor Data	412, 413, 575, **735,** 827	631			596, 597				8
Totals	55	30	11	11	5	7	6	19	144

SARCOMA TABLE NO. 2 (Cases 1059 to 1335, inclusive)

	HUMERUS			CLAVICLE			SCAPULA		
	Upper	Middle	Lower	Outer	Middle	Inner	Acromion	Body	Totals
Osteogenic Sarcoma	**1068, 1071P, 1080, 1087P,** 1107, **1127,** 1138P, **1141P, 1149, 1215, 1237, 1238, 1276,** 1285, **1295, 1322, 1323**	**1079, 1096,** 1254	1207 1268 **1277**			1150		1073 1217	26
Ewing's Sarcoma		**1123**						**1314**	2
Multiple Myeloma		**1163**		**1158 1226**					3
Angioma								1209	1
Typical Giant Cell Tumors									0
Epiphyseal Chondromatous Giant Cell Tumors									0
Atypical Sarcoma									0
Metastases (Cancer)	**1145, 1182**							**1088**	3
Benign Osteogenic Tumors	1171								1
Inflammation (Including Cysts and Callus)									0
Discarded for Poor Data		1243							1
Totals	20	6	3	2		1		5	37

Analysis of Registered Cases of Tumors of the Shoulder Bones.

In order to write this chapter advantage has been taken of the opportunity offered by the Registry of Bone Sarcoma, and in the summer of 1930 the following study of the cases registered up to No. 1,059 was made. Table I shows the relative frequency and the sites at which the different forms of bone tumors occur in relation to the shoulder joint as far as may be judged from the evidence of the 144 cases which involved the humerus, clavicle, and scapula. Notice that the figures are the Registry numbers of the cases, except for the marginal ones, which indicate the totals in each lateral or vertical column.

In April, 1932, a second study was made and the mortality, as indicated by the heavy type on the charts, brought up-to-date, and the text revised accordingly. Table II was prepared from the cases registered since the previous study from No. 1,059 to No. 1,335, but was not used in the text because there was nothing found in this second study except confirmatory evidence for some of the observations already made. The chief value of Table II is to indicate in a general way that the relative figures are much the same as in the first study, *e.g.*, in both tables thirteen and one-half per cent of all bone tumors were in the shoulder bones, and osteogenic sarcoma in the head of the humerus is the most common shoulder tumor. However, in Table II there is a notable lack of giant cell tumors. Furthermore, Table II might be of value to any future student of this subject, for it gives him the Registry numbers for ready reference so that he can send for their envelopes for his own study. The cases in Table II are too recent for the results to be of statistical value.

A large proportion, seventy-two of the one hundred and forty-four tumors of the shoulder, lay in relation to the subacromial bursa, for they involved the acromion process, the outer end of the clavicle and the upper end of the humerus. They probably rarely actually invade the bursa. Nevertheless, we must consider them if we are to be prepared to make detailed differential diagnoses of conditions affecting the shoulder. Even after I had studied shoulder conditions for many years, I operated on a patient under the diagnosis of subacromial bursitis, and later found that this had been an unnecessary operation, for although there was a bursitis, the pain had been really due to early metastatic cancer in the upper end of the humerus. At the time of the operation the X-ray showed no evidence of invasion of the bone. Shoulder pain is the first symptom of most tumors of the head of the humerus, and while it is quite true that hitherto no *early*

cases of bone tumors have been diagnosed, there is, nevertheless, hope that in the future we may learn, either by careful X-ray or clinical findings, to discover bone tumors in their infancy. We must keep the question in mind in the case of every painful shoulder. Of the 1,335 tumors in the Registry collection on April 7, 1932, not one can be said to be in an early stage. Some were discovered soon after symptoms began, but the X-ray showed the disease well advanced.

It is sometimes well to stop to consider relative proportions, and as I am a general surgeon who has had among other hobbies the studies of bone sarcoma and of obscure shoulder lesions, the reader may be interested in my comparison of their frequency. Bone sarcoma, though more serious, is not as common as rupture of the supraspinatus tendon in ordinary everyday life. The relative frequency of the two lesions may be contrasted by the fact that Dr. Akerson was able to find lesions of the supraspinatus in thirty-nine of one hundred consecutive shoulders at autopsy, as compared to the fact that the Registry of Bone Sarcoma, sponsored by the American College of Surgeons, whose membership is over 7,000, has in ten years been able to collect records of only 144 cases of tumors in the shoulder bones. To be sure, rupture of the supraspinatus tendon is usually not recognized, while tumors of the bone present such dramatic, pathetic and fatal pictures, that if one case occurs in a hospital, every doctor connected with the institution will probably see and know about that case. The rapid advance, the grotesque appearance, the fatal ending (which is the usual history in bone sarcoma), attract attention, whereas rupture of the supraspinatus never causes death, and is difficult to demonstrate, even when the diagnosis is made. Many surgeons have not yet even heard of it, much less can they diagnose it. I therefore enter as a preliminary statement that I know that this chapter is disproportionate in size to the rest of the book, yet not so disproportionate as is the literature of bone sarcoma to that of the supraspinatus.

Before speaking in detail of bone tumors, let me say something of the Registry, and in order to do this, I should like to present a facsimile of the face of a ten by twelve inch manilla paper envelope which the Registry uses to contain the data of each case. The reading matter on this envelope has been given a great deal of thought, and I beg the reader to take a magnifying glass and to study it in detail. The central, vertical division is apparently the most important, because it contains a list of bone tumors and purports to be a

standard classification for common use for surgeons, röntgenologists and pathologists. This has been the working classification for the American literature of this subject for the last decade, but experience shows that it needs improvement. *At the bottom of the Registry envelope two sentences occur which are of great significance.*

"It is believed that this list covers all bone tumors which are known to have a natural history distinct enough to justify prognosis or to indicate special treatment. If you believe there are others, please register illustrative cases." This rule should exclude serious discussion of other old or new terms, unless definite data are filed on which the committee may open discussion.

If a patient has a tumor of one of the shoulder bones, it should be one of the tumors on this list, which was agreed on by two committees of pathologists. At that time our ideas of the relative frequency in which these tumors might occur were very vague. As experience comes we find that we have now little use for some of the terms. For instance, we all find fault with "periosteal fibro-sarcoma." I could not point today to a single Registry case which typifies what we meant by this term, nor has any article appeared in the literature of the last decade which presents a study of a group of such tumors. We did mean something by the term, and it still has some value, but most of us would prefer to use parosteal or extra-periosteal fibrosarcoma for one of these rare cases. We meant a tumor which had nothing to do with the bone, which was fibrous, and arose from the outer layer of periosteum, or adjacent fibrous tissue. Experience has shown that such tumors are rare.

In fact, our whole understanding of the term "periosteal," in relation to sarcomas, has gradually changed. For instance, we find that practically every case of osteogenic sarcoma is both "medullary and subperiosteal." Since the term applies to *all* cases, it is of no special significance. We also made what I consider a mistake, of using the term "periosteal" to qualify a subdivision of osteogenic sarcoma. We thought then that there was a periosteal sarcoma pure and simple, as opposed to a central sarcoma pure and simple. Some of the committee still cling to this idea, but personally I have never yet seen an osteogenic sarcoma which was confined to the periosteum, and was not intracortical as well. They are always, in my experience, both extra- and intra-cortical. To be sure, one sarcoma may have more than nine-tenths of its bulk extracortical, and only one-tenth intracortical, and another may have more than nine-tenths of its bulk intracortical and less than one-tenth extracortical. Essen-

Facsimile of a 10 × 12 manila envelope used by the Registry of Bone Sarcoma to contain the data about registered cases. The reader is urged to examine carefully every paragraph. The particular case illustrated caused a great deal of discussion in 1920-23. Today a similar case, I feel sure, would receive the unanimous opinion of all the pathologists that it should be classed as a case of giant cell tumor.

CASE NO. 38

Registered by Dr R. D. McClure of Detroit, Mich. on Oct 5-1920 Name Fred Kaiser

The Registry of Bone Sarcoma is an activity of the American College of Surgeons to stimulate the study of cases of Bone Sarcoma and to keep before the Medical Profession the End Result Idea which is the basis of the minimum standard of the College. If the members of the College are in earnest it is practicable to register with complete data every case which occurs in the United States, and to study these cases intensively until diagnosis and treatment are reasonably standardized. Experience shows that these cases as a rule suffer from incorrect diagnosis or from unsatisfactory treatment. Every surgeon, roentgenologist or pathologist, and especially every member of the American College of Surgeons, is urged to register any case that comes to his knowledge. Since delay in treatment due to incorrect diagnosis in the clinic, in the X-ray room or in the laboratory is the rule in these rare cases, it may be said that the registration of a case under treatment, even though it is a record of diagnostic and therapeutic mistakes, is nevertheless an evidence of good faith and of a desire to avoid future similar errors. Registration should be a medico-legal protection rather than the reverse.

The Registry plans to send the collection about like a circulating library in boxes containing twelve of these envelopes, in rotation, to all pathologic laboratories interested in helping in this research. Remember that the data in these envelopes, though often obviously incomplete, is the best we can get. Don't find fault with it or laugh at it until you have registered the material from your own clinic! Every hospital makes errors in these cases, and yours probably does too. The time to register is when you have the material — not after you have lost it, as many hospitals of wide reputation do. It is not necessary, but advisable, to give the patient's name to avoid duplication. The Committee wishes to study the Collection with all other laboratories and clinics. We feel that the best way to agree on the same names for the same entities is to pass this collection about as it accumulates, and as our mutual knowledge about Bone Sarcoma and its treatment crystallises. The material of the Registry is at the service of any one taking part in the research.

LIST OF DESCRIPTIVE ADJECTIVES AND PREFIXES used in the Literature for Bone Tumors. These terms should not be used as clinical entities, but to describe characteristics of tumors. When correctly used they are not objectionable, but they are often used incorrectly and lead to confusion, leaving the impression that a large number of different clinical entities exists.

CELL RESEMBLANCES
- Round { small / large
- Spindle { small / large
- Oat cell
- Giant cell { foreign body / true tumor
- Mixed cell

TISSUE RESEMBLANCES
- Fibro-
- Myxo-
- Chondro-
- Osteo-
- Osteoid
- Osteogenic
- Osteogenetic
- Osteoblastic
- Ossifying
- Xantho-
- Sclerosing
- Cellular

RESEMBLANCES TO VASCULAR RELATIONS
- Endothelio-
- Hem-
- Lymph-
- Angio-
- Cavernous
- Capillary
- Telangiectatic
- Perithelial

ANATOMIC RELATIONS
- Multiple
- Diffuse
- Periosteal
- Extraperiosteal
- Paraosteal
- Subperiosteal
- Central
- Invasive
- Solid
- Dural
- Alveolar
- Medullary
- Myeloid
- Myelo-

SUBDIVIDING MYELOMA
- Lymphocytic
- Myelocytic
- Erythroblastic
- Plasma cell

LIST OF CLINICAL ENTITIES recommended by the joint Committee of the Registry of Bone Sarcoma and Clinical Pathologic Association, for general use by Clinicians, Pathologists, and Rontgenologists.

1. METASTATIC TUMORS
2. PERIOSTEAL FIBROSARCOMA
3. OSTEOGENIC TUMORS
   - Benign
     - a. Exostosis
     - b. Osteoma
     - c. Chondroma
     - d. Fibroma
   - Malignant (OSTEOGENIC SARCOMA)
     - a. Anatomic types
       - Medullary and subperiosteal
       - Periosteal
       - Sclerosing
       - Telangiectatic
     - b. Undifferentiated sarcoma
4. INFLAMMATORY CONDITIONS THAT MAY SIMULATE BONE TUMORS
   - Myositis ossificans
   - e. g. Osteoperiostitis
     - a. Traumatic
     - b. Syphilitic
     - c. Infectious
   - Ostetitis fibrosa
5. BENIGN GIANT CELL TUMOR
6. ANGIOMA
   - Benign
   - Malignant (angio-sarcoma)
7. EWING'S TUMOR
8. MYELOMA.

It is believed that this list covers all bone tumors which are known to have a natural history distinct enough to justify prognosis or to indicate special treatment. If you believe there are others, please register illustrative cases.

THIS INDIVIDUAL CASE is classified by the Registrar as:

Osteogenic Sarcoma.

If you agree, sign your name below. If you disagree, write "disagree," and give the term you prefer and sign your name. Write on the "Classification Sheet," within the envelope, your reason for disagreeing. That is also the place to write anything that you think may be helpful to others who study the cases.

Date *Name of Pathologist*

For varied opinions of the following:

Jan 2-21. J. C. Bloodgood
DEC 23-20 } J. Ewing
May 15-21 }
July 12-22. F. B. Mallory
July 1922. J. H. Wright
Aug 1-22. S. B. Wolbach.
Nov 30-22. V. H. Kuehler.
" " J. L. Taylor
DEC 1922 A. G. White.
" " H. Fox
" " J. Speese.
Apr 18-23. Sir Harold Stiles.
July 11-22. J. Warren White.

Any case alive on or treated since January 1st, 1921, is eligible for registration. Any case of unusual interest which died before that date is also eligible.

tially, these two types are the same and have the same prognosis, and are clearly not different clinical entities. It is, therefore, not logical to say that a tumor arose in either situation because the bulk of it is extra- or intra-cortical.

In my opinion, we should also have left the terms sclerosing and telangiectatic in the left-hand column. They are descriptive adjectives, useful for individual instances, but do not define real clinical entities. It was then held that "sclerosing" cases had a little better prognosis and telangiectatic a little worse than the average. Certainly among the shoulder bones this has not been confirmed, for all the sclerosing cases are dead and one very bad telangiectatic case is a five-year cure (Registry No. 156).

The second subdivision of osteogenic sarcoma, "undifferentiated sarcoma," has practically become an unused pigeon-hole, because the cases which we intended to put in that division are now carried under Ewing's sarcoma. Ewing's sarcoma is characterized by absolutely undifferentiated cells which have no intercellular substance. There may be a few undifferentiated sarcomas which are not Ewing's sarcomas, and which we cannot pigeon-hole in any other place, but as a matter of fact, such tumors are extremely rare, if they exist at all. It is an almost useless division. I prefer to carry the few cases under "Atypical Sarcomas."

The subdivisions of inflammatory conditions were not intended so much as actual subdivisions as examples. Of course, other inflammatory conditions of bone might have been included. I think we might have saved ourselves many explanations by stating that callus and cysts are considered as "inflammatory" in the sense of a tissue reaction to trauma. Normal repair following trauma without infection may be considered inflammation. Inflammation does not necessarily mean infection.

Benign giant cell tumor has proved a fairly distinct class, but there are a few cases in the Registry series, the outcome of which suggests the advisability of dropping the adjective *benign*. Experience shows that we have had to acknowledge in a very small number of cases that our original diagnoses were incorrect, or else to admit that a malignant tumor became superimposed on a benign condition, or to admit that benign giant cell tumors were not always benign. This subject has been discussed by Geschickter and by Simmons recently, and will not be discussed further by me here, especially as no such instances have occurred in the shoulder bones. Later in the chapter reasons will be given for the belief that benign giant cell

tumors of the upper end of the humerus have many characteristics which differ from most of the giant cell tumors in other bones.

Our class of angioma was necessary, and the subdivisions were necessary, for we have had a few unquestioned cases of benign cavernous angioma of bone which have a distinct X-ray appearance and a benign course, in spite of the presence of large tumors in two of the cases. On the other hand, the malignant division of angioma is still of doubtful value. I could not pick out a case from the Registry series which would be a typical angio-sarcoma. The type of tumor called Endothelioma by Kolodny would be the nearest that I know of; yet, since this type is so very rare and still doubted by so many pathologists, and since the few cases we have are regarded by some pathologists as perhaps instances of metastasis from cancer, the term angio-sarcoma is of doubtful value, but it cannot be discarded.

The seventh division (Ewing's tumor) should certainly be modified to Ewing's sarcoma, because this type of tumor is malignant, and since we have used the term tumor in benign giant cell tumor instead of sarcoma, to denote that the giant cell type is benign, we certainly should use the term sarcoma with Ewing's name, because his tumors are surely malignant. For the eighth division (Myeloma), I believe we should use the term Multiple Myeloma, because the English and Canadians, following the British custom, have for years used the term myeloma to signify what we know as giant cell tumor. So far as the experience of the Registry goes, myeloma is always multiple, although we have had a few cases where a single tumor appeared to be present for some time before others developed. It is important to emphasize this multiple character of myeloma on account of the differential diagnosis of Ewing's sarcoma. Ewing's sarcoma is certainly sometimes multiple, but the bulk of evidence goes to show that the tumor is usually primary in one bone and that the involvement of other bones is of a metastatic nature. An increasing number of amputation cures proves this. Myeloma, however, seems to arise in different foci in many parts of the body at once. Ewing prefers the term endothelioma for his tumor, but this is confusing, and most pathologists do not admit that it is endothelial. It certainly is quite different from multiple myeloma, although the two are the only round-celled primary bone tumors. We should simply modify these two terms by saying: (7) Ewing's sarcoma. (8) Multiple myeloma. This would indicate that while Ewing has convinced the American pathologists that his sarcoma is a clinical entity, he has not yet convinced them that it is entitled to the name of endothelial

myeloma or endothelioma. The above modifications of the Registry Classification are used in the table.

Under Atypical Sarcomas would fall cases which we cannot assign to any one group, yet we have evidence enough to feel that they are primary sarcomas and not benign conditions or metastases from cancer. So the term is not the same as Miscellaneous, for it implies "surely sarcoma." Under "atypical" might be put such cases as the sarcomas arising in giant cell tumors or in old osteomyelitis, or any malignant tumor with peculiar histology which is not considered to be a metastasis. The few cases of questionable periosteal fibro-sarcoma might be put in this class also, if it seemed advisable.

The group "discarded" means that the case has been so poorly registered and has such poor data that conclusions drawn from it would be of little or no value for the present purpose. Such cases are not valueless entirely; I have several times learned something from cases reported with very incomplete data. In fact, few of our cases do have absolutely complete data, and it must not be understood that the cases in the table are all positively and permanently diagnosed.

Notice that those tumors above the heavy line are primary, malignant tumors of bone—the kinds which we asked surgeons to register. Those below the line we did not and still do not wish to have registered. We asked for registration of cases of sarcoma; the cases below the line were registered because some one thought they were sarcoma. The inference from this is, that the relative frequency of the tumors below the line with those above the line cannot be estimated from this chart. Actually, the occurrence of the kinds listed below the line is probably greater, for many more of the types below the line might have been registered if we had asked for them. On the other hand, I see no reason why we should not estimate the relative frequency of the occurrence of the tumors above the line from this table, or a similar one drawn from all the Registry cases, including the other bones. Possibly some giant cell tumors have not been registered because some surgeons have thought that we did not wish them, since they were not sarcomas. Time has proved the desirability of following up the giant cell tumors, since we have now a number which have proved to be malignant. None of these were in the shoulder bones, however. Perhaps other surgeons may have refrained from registering myelomas because they were not sarcomas. At any rate, I can see no reason for doubting that the

proportion of osteogenic sarcomas and Ewing's sarcomas can be obtained from this chart, except that the diagnosis of Ewing's sarcoma can seldom be made from the X-ray alone, while those of osteogenic sarcoma may be. A significant point is brought out by this chart—that Ewing's sarcoma is quite common in all three shoulder bones.

The chart also shows the probable relative proportion of errors in practical diagnosis, since those cases above the line stand to those below the line as 102 to 32. This is an improvement on Greenough's, Simmons', and Harmer's figures in 1921 in reference to the cases at the Massachusetts General and the Huntington Memorial Hospitals. These authors say:

"Perhaps the most surprising fact of the whole study is that out of 148 cases sent in as possible bone sarcoma, only sixty-six could be considered in fact to be cases of malignant new-growth of bony origin; the remaining eighty-two cases proving on more detailed study to be metastatic tumors of bone (twenty-nine cases), sarcoma primary in the soft parts (twenty-eight cases), inflammatory conditions (eleven cases), or tumors of a non-sarcomatous type (fourteen cases)."

I feel very confident that the degree of accuracy of diagnosis in bone tumors throughout America has greatly improved since the Registry was established in 1920. In the last thirty-eight shoulder tumors registered there were only five to be placed below the line.

Among the first 1,058 registered cases, ninety-six involved the humerus, twenty-three the clavicle, and twenty-five the scapula. So we may say that about every tenth bone tumor will involve the humerus, or out of every ten bone tumors, about one or two will involve one of the shoulder bones. When we confine our study to the portions of these bones which lie near the bursa, we find

	Up to No. 1,059	*No. 1,059 to No. 1,335*
Upper end of Humerus	55	20
Outer Clavicle	11	2
Acromion	6	0
	72	22

The writer (Bone Sarcoma: Prevalence in Massachusetts. *Bos. Med. & Surg. Jour.*, 1922, Vol. 187, pp. 208-211 and pp. 543-545.) after a postal card canvass of the physicians of Massachusetts, estimated that only one out of every 100,000 of the population at any one time was afflicted with bone sarcoma. This estimate would be decreased to less than one in 1,000,000 if we specify a sarcoma in the

neighborhood of the subacromial bursa. In other words, most physicians will see perhaps one such case in a lifetime. Why occupy a whole chapter of this book with discussion of such a rare shoulder condition?

A glance at the chart gives one good reason, for the cases whose deaths have been reported are in **heavy type**. Obviously some of these tumors are very fatal and others are not, and while at any one date there may be only one fatal case progressing among every million persons, that means at least a hundred fatal cases in the United States. Delay in diagnosis is the most important obstacle to the prevention of this mortality, and the cause of deep regrets for time and money wasted by treatment for erroneous diagnoses of "rheumatism," etc. Bone sarcoma is practically the only fatal lesion which arises in the shoulder. We must learn to recognize it and to cure it. It should be our first thought in the case of a young patient complaining of a dull aching pain; if any thickening of the bone were palpable or the pain persisted more than a week, we should seek help from the X-ray.

The details of this chapter need not be remembered by the average physician if he will only bear in mind his obligation to see that every patient complaining of pain or swelling about the shoulder is promptly referred to a röntgenologist. It is to the röntgenologist even more than to the surgeon that this chapter is addressed. The present-day röntgenologist is the diagnostician of the community, not only in bone tumors, but in almost every department of medicine and surgery. He must know all the specialties so far as diagnosis is concerned, and some of them so far as treatment is concerned. His danger at present is in accepting cases referred for treatment by practitioners not qualified to decide for the patient as to whether or not X-ray therapy should take the place of surgery.

Relation of Pathologic Conditions to X-ray Appearances.

As a first step in this study of the Registry cases all the X-rays of the 144 cases were taken out of the envelopes and arranged on tables, classifying them according to this chart from X-ray diagnosis alone. In very few cases was there any difficulty in putting them in their proper places. I also had the same experience with an independent examination of the slides. Then the clinical histories and the written opinions of the pathologists who had examined the slides and the Registry classification of each case were compared. In the main, from whatever angle I approached, I found I could make the diagnosis and prognosis. Either from an excellent X-ray or an excellent

slide, and in most cases from an excellent clinical history alone, the diagnosis could be made. Most of the difficulties came from incomplete or poor technique. Very few cases were registered with excellent data in all three respects.

A few cases were doubtful even with excellent technique. Another fact was obvious. If a good X-ray was puzzling, the slide of that case and the clinical history were, too. Such cases have been classed as "Atypical Sarcoma," if I felt tolerably sure that they were cases of primary *malignant* disease of the bone, although not confident of the variety. Some of the atypical sarcomas are unique and waiting for other similar ones to form a new group. Let us follow the table and first consider the Röntgenological appearances of tumors of the upper end of the humerus.

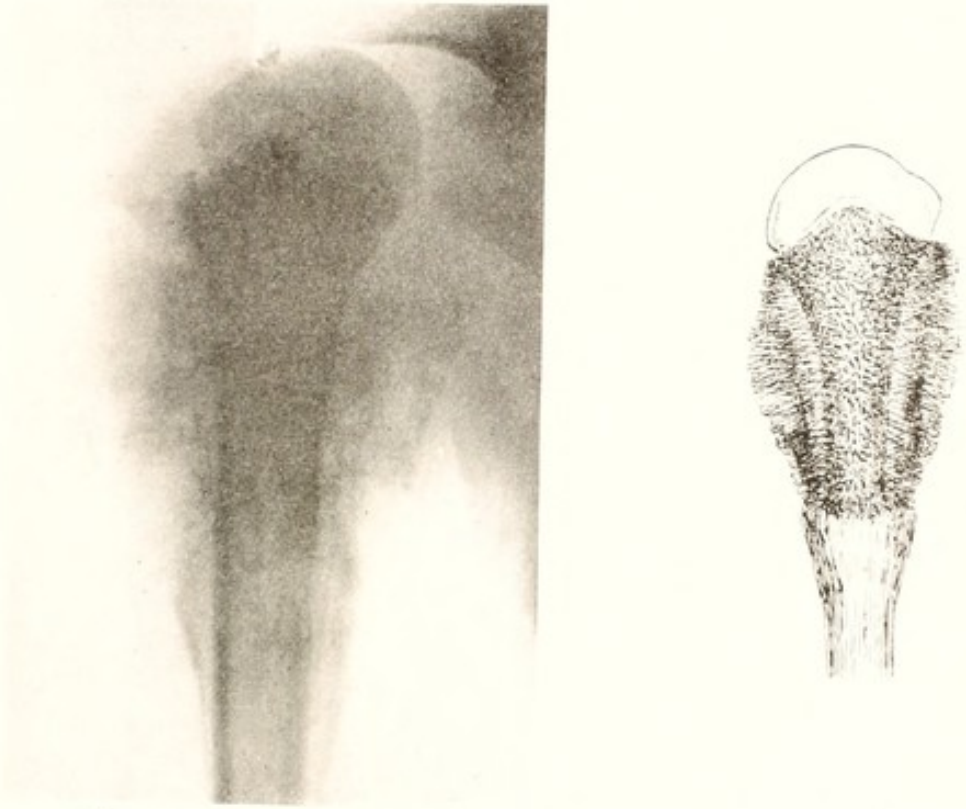


FIGURE 68. INTERPRETATION OF X-RAY OF OSTEOGENIC SARCOMA

Osteogenic Sarcoma. X-ray and schematic drawing of osteogenic sarcoma. The outline of the shaft still shows within the tumor, which ceases at the epiphyseal line and does not invade the head of the bone. The triangle at the lower margin of the tumor is, in reality, a cuff like a candlestick, made of defensive new bone, which will be destroyed as the tumor progresses downward. The radiating spicules shown in the X-ray are probably formed by the action of tumor cells which are essentially osteoblasts, although they have received the malignant impulse which makes them cease to respect the normal laws of growth. In the dissected specimen of such tumors the outer surface is much more smooth than is suggested by the X-ray, and does not have the prickly roughness which seems to be indicated in this diagram. The reader must not expect to find radiating spicules in all cases of osteogenic sarcoma. Sometimes there is no bone production and very often, as in the next figure, one cannot recognize any radiating structure.

Osteogenic Sarcoma. There were twenty-four which showed fairly typical X-ray appearances of osteogenic sarcoma; *i.e.*, the old shafts were visible in the tumors, which were evidently both intracortical and extracortical; there were "reactive triangles" and "sunray spicules." These tumors all occupied the metaphysis up to the articular head, the contour of which was not destroyed. These tumors as a rule are enlarged outward beneath the deltoid, but some are almost globular in shape. In two cases in which photographs of the patient were given, the external swelling presented a fusiform appearance, with the greatest diameter distinctly below the region of the tuberosities. In all cases in which we had slides, the histologic type was very clearly osteogenic sarcoma. The diagnosis was further confirmed by the report of death in all the cases but No. 49, traced three years only, and No. 812, traced five years. All but five showed pathologic fractures, and of these five, four were of the "sclerosing" type. Dr. Bradley F. Coley was, I believe, the first surgeon to call attention to the interesting fact that pathologic fracture is more common in the humerus than in the femur, which is so distinctly a weight-bearing bone. (Bradley F. Coley and George S. Sharp. *Amer. Jour. Surg.*, Vol. ix, August, 1930, pp. 251-263.)

FIGURE 69. SCLEROSING OSTEOGENIC SARCOMA

This case illustrates the fact that osteogenic sarcoma in this region tends to grow outward under the contour of the deltoid, and does not tend to invade the articular head of the bone and the joint.

Five cases presented so much new tumor bone that one might be justified in calling them instances of the "sclerosing" type. Only one of these had shown definite evidence of fracture, and even in this

case, the fracture was of slight extent. Most of the new bone deposited was to the outer side, so that the contour corresponded with that of a large deltoid. Histologically, the soft parts of these tumors were characteristic of osteogenic sarcoma. The evidence of their belonging to the "sclerosing" variety was chiefly from the X-ray. Death had been reported in all of these cases.

Five cases might be classed as belonging to the osteolytic type of osteogenic sarcoma, with much disintegration of the bone and no bone formation. All of these tumors were very large and very far advanced. In only one was a section obtained, so the diagnosis was not certain in the other four. In their earlier stages they may have shown typical X-ray appearances with radiating spicules, etc., which later were again dissolved by the tumor. In X-ray interpretation, röntgenologists must bear in mind that many tumors cause bone proliferation and then devour the new bone. The reactive triangles at the bases of osteogenic sarcomas are normal new bone with which nature endeavors to check the progress of the disease. In the macerated specimen, it is seen that this triangle, since it is present at the periphery from all angles, really forms a trumpet-shaped affair which holds the old shaft as a candlestick holds a candle. This collar of bone is constantly being absorbed and renewed at a lower level.

So far as this collection is concerned, I do not place much significance on the five cases of osteolytic type, especially since four of the five were not confirmed by slides or autopsy, and all were in advanced stages. Two, at least (Nos. 484 and 643), were in cases of Paget's disease, and such cases are always atypical, and very apt to be of the osteolytic type, both from the X-ray and microscopic point of view. Microscopically, osteolytic tumors are usually very cellular and very actively growing, but occasionally a purely cartilaginous tumor is very osteolytic and advances through the bone as if melting it away. The non-radiable lime salts are completely absorbed and replaced by radiable cartilage, and yet the line between is sharp under the microscope. No. 1,000 is such a case.

Dr. Phemister (*Surg., Gynec. & Obstet.*, January, 1930, p. 214) recently brought up the question of whether chondro-sarcoma should not be separated from the other forms of osteogenic sarcoma as a clinical entity. He feels that if an osteogenic sarcoma is largely composed of cartilage, the natural history of the case is longer and the prognosis of operation better. I do not agree with his point at present, but other members of the Registry Committee do. If we go back to the old nomenclature of chondro-sarcoma, we shall also have

to go back to osteo- and fibro- and then to their mixtures. We should wait for a demonstration of some distinct difference in the clinical course of the chondromatous type in its way of responding to treatment before we accept Phemister's suggestion. We have only a handful of cures of all forms of osteogenic sarcoma in all parts of the body, and the chondro-type forms no large proportion of these. It may be that any fully differentiated form, whether osteo- or chondro- or fibro- has a better prognosis than the usual case, which generally contains a mixture of all three intercellular substances. However, this theory is not borne out by the tumors of the shoulder bones, for only one is still living as a five-year cure and this is Phemister's Case No. 812. His other case, No. 1,031, I do not consider a sarcoma, but as a giant cell tumor, and will discuss it later in this chapter. Other than this frail evidence, there is none to encourage his suggestion, so far as the shoulder bones are concerned, unless the group of epiphyseal giant cell tumors is included as showing instances of chondro-sarcoma. In Geschickter's opinion this should be done.

Ewing's Sarcoma. Osteogenic sarcomas of the upper end of the humerus are pretty uniformly confined to the metaphysis as opposed to Ewing's sarcoma, which involves the shaft (diaphysis) from epiphysis to epiphysis. Two cases of osteogenic sarcoma, No. 852 and No. 1,002, extend well down the shaft, but the radiating spicules are much longer than in any cases of Ewing's sarcoma, in which the spicules, if present at all, are only about one-fourth inch in length. One may pretty safely say that most sarcomas involving the shaft of the bone will be Ewing's, and that Ewing's sarcomas do not occur in the upper end of the bone without showing involvement of the major portion of the shaft.

The usual X-ray criteria for Ewing's tumor, broadening of the shaft by separation of the lamellæ and the formation of onion-like layers of periosteal new bone as the tumor advances beyond the cortex, are beautifully shown in the cases in the humerus. I am inclined to think that Ewing's sarcoma in the shaft of the humerus very frequently shows fine, short radiating spicules outside the onion-like layers. But the new bone is merely reactive bone.

Ewing's sarcoma outnumbers all the other tumors of the shaft of the humerus. The only other condition with which one familiar with its typical appearance could confuse it, would be osteomyelitis, especially because both of these lesions cause febrile symptoms. The diagnosis is confusing on clinical grounds because in Ewing's sarcoma

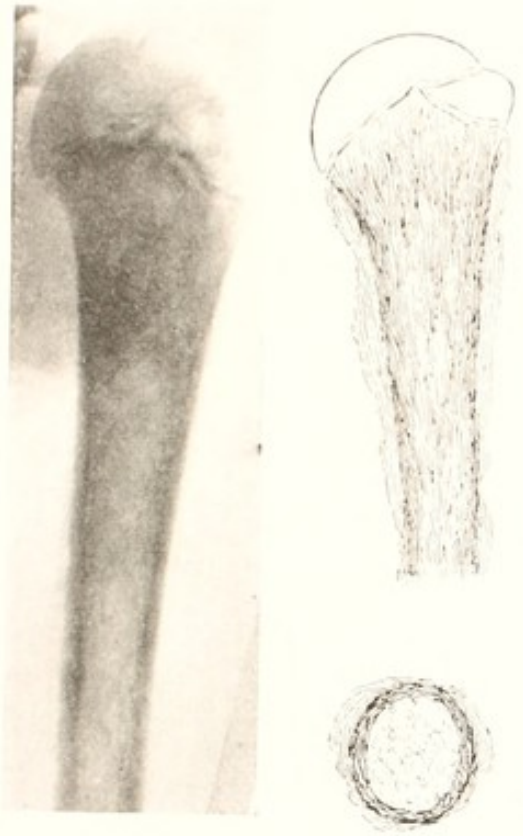
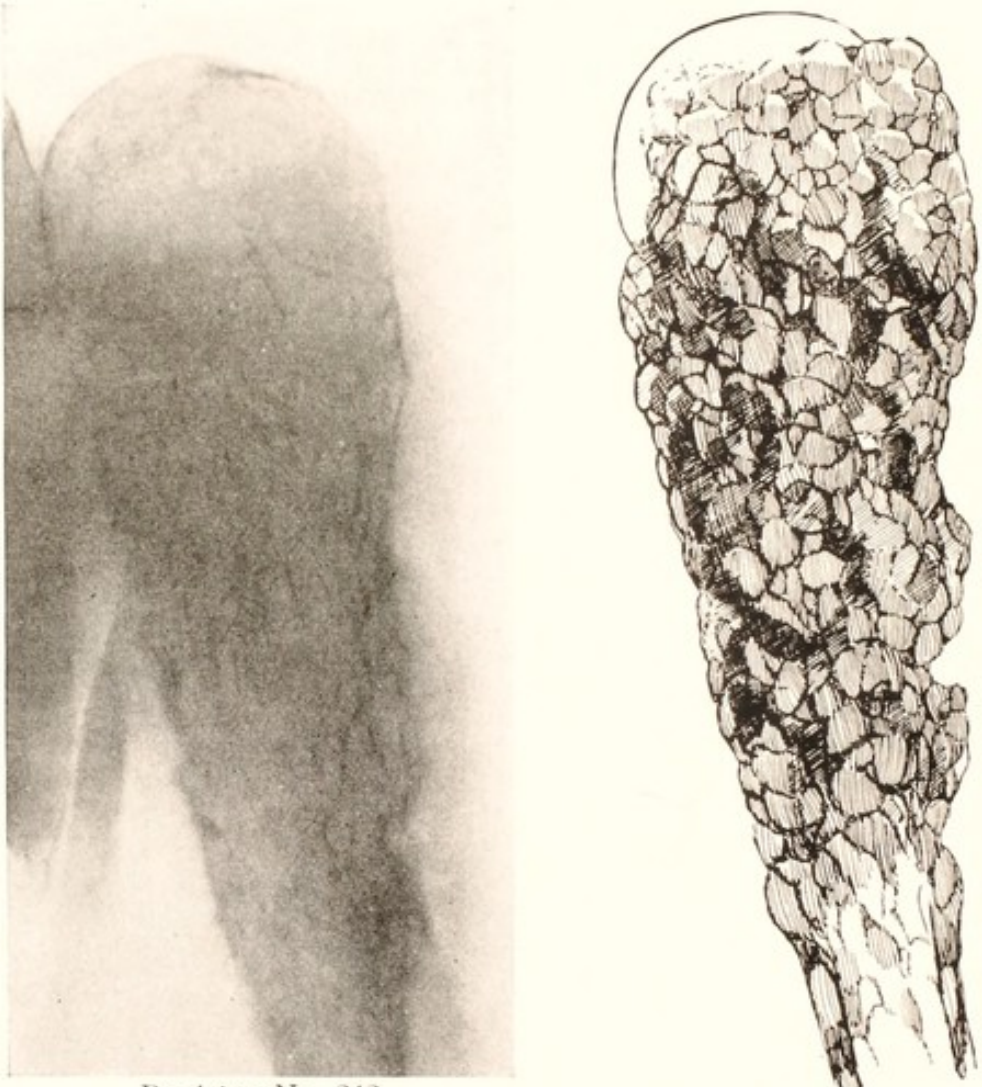


FIGURE 70. INTERPRETATION OF X-RAY OF EWING'S SARCOMA

Film and schematic drawing of method of invasion of Ewing's sarcoma. The tumor involves the shaft of the bone, invades between the lamellæ and destroys the trabeculæ even in the cortex. It stimulates defensive periosteal new bone, which it may in part destroy, although new layers form again in defense; hence the "onion-like layers." A cross section of such a bone would show that this process is centrifugal, so that the whole shaft appears somewhat widened.

pain is usually intermittent at first. It usually appears in healthy, rapidly growing children, while osteomyelitis as a rule has a definite history of infection in some other part of the body. Nevertheless, any one who operates for osteomyelitis in any bone should exclude Ewing's sarcoma or be prepared to recognize it. The X-ray appearances of the two are similar, but not alike. When the pain in cases of Ewing's sarcoma is severe enough to make the patient consult a doctor, the X-ray will show well-advanced changes, but in acute osteomyelitis the pain precedes the changes visible in the X-ray by some weeks.

Multiple Myeloma. We may readily dispose of this lesion in a differential diagnosis of pain in the shoulder. The X-ray appearance is characteristic and it does not involve the head alone, but extends down the medulla or forms lacunæ in the cortex. It is among the few really endosteal tumors. It is truly inter- and intra-cortical, and advances in the spongiosa and medulla more definitely than is characteristic of any other tumor, even of a cancerous metastasis, the bone tumor which most resembles it in the X-ray. Yet one case of early myeloma in the femur, I recall diagnosing as a cyst! Once having seen a good X-ray of a myeloma and having realized the essential elements of its pathology, one should readily separate it from other tumors of the bone. It is essentially a hyperplasia of



Registry No. 242

FIGURE 71. INTERPRETATION OF X-RAY OF MYELOMA

X-ray and diagrammatic drawing of a myeloma of the upper end of the humerus. Even little marrow spaces in the cortex have developed foci; the whole upper end of the bone is blown up as if by a mass of bubbles. The method of invasion of this tumor is essentially one of expansion of each little marrow space. In this specimen there were more foci than usual.

Myeloma of the clavicle, which shows the tendency of the tumor to expand the bone as if blowing it up by small bubbles.

marrow cells, not a tumor with intercellular substance. It is multiple in origin and can only arise where there are marrow cells and then dilate the spaces in which these cells normally lie. We know such cells are normal in the medulla and in the spongiosa chiefly, but they may occur in small spaces in the cortical bone. As the hyperplasia in each space—large or small—increases, the spaces are enlarged and break into one another and the intervening trabeculæ are absorbed. The result is an appearance as if the bone were blown up from inside, as by a series of bubbles, large and small. The periosteum is somewhat stretched and as the process advances, lays down a film of new bone so that a pencilled outline is given to the whole. The bubbles may be single or multiple. The process is quite different from that which takes place in Ewing's sarcoma, where it is essentially invasive and peculiarly apt to be cortical, since the tumor cells penetrate every part of the bone following the lacunæ and Haversian canals and separating the lamellæ around the circumference, dissolving the bone as they go.

The process of dilatation in giant cell tumors is also different, for instead of blowing the bone up with large and small bubbles, as does myeloma in most cases, there is one large bubble which expands by pulsation, destroying all the spongiosa on its periphery until it reaches the resilient medulla on one end and the resilient cartilage on the other. It does not, like myeloma, extend in the marrow, but stops abruptly. I think the reason is that the medulla also pulsates and stops the advance of the tumor. The apparent trabeculæ in giant cell tumors are merely ridges in the cortex, not evidence of loculi in the tumor. It is my favorite theory that the origin of all giant cell tumors is a ruptured branch of a nutrient artery which does not clot. Expansion takes place from this single point. Giant cell tumor always arises in spongy bone, myeloma in the marrow of the shaft and also in the spongiosa or cortex.

Giant Cell Tumors. In the head of the humerus giant cell tumor does not behave in a typical way, and my study in 1930 of this group of registered tumors of the shoulder bones resulted in the following paper, which is here reprinted with the permission of the editor of *Surgery, Gynec. & Obst.* No other tumors of this type were registered in the last 277 cases up to April 7, 1931. (1,060 to 1,335.)

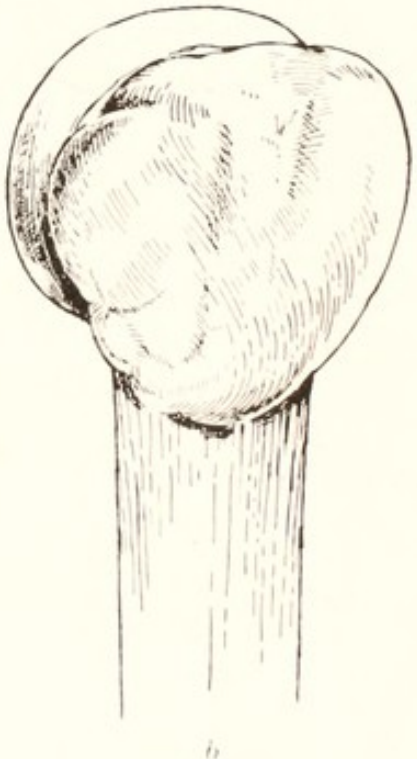
Reprint from
SURGERY, GYNECOLOGY AND OBSTETRICS
February 15 (No. 2A), 1931, Vol. LII, 543-548

EPIPHYSEAL CHONDROMATOUS GIANT CELL TUMORS OF THE UPPER END OF THE HUMERUS

Giant cell tumors of the upper end of the humerus are either very rare or else they should include the nine tumors in the cases I am about to discuss, which in our series have in some instances been called giant cell tumors and in others chondromata, or chondrosarcomata. Evidently a giant cell tumor affects the upper end of the humerus in a different way from that in which it affects the other long

REGISTRY NO. 117

a



b

FIGURE 72. INTERPRETATION OF X-RAY OF GIANT CELL TUMOR ARISING IN AN ADULT

Interpretation of Giant Cell Tumor arising in an adult. Diagrammatic drawing representing the probable condition which would be shown by the dissected bone in such a case. Note the biceps groove in both drawing and X-ray. The apparent trabeculations in these tumors are usually only local ridges or thickenings in the outer walls; few, if any, interior compartments exist in them.

bones. Case 117 registered by Bloodgood is the only one which seems to resemble a typical giant cell tumor, and even in this case very little expansion of the bone is shown.

There are two other cases, 4 and 556, which are perhaps pure giant cell tumors, but owing to the poor data we have received concerning them, they cannot be seriously considered. I do not know of a single case of typical giant cell tumor in which the growth has expanded the whole upper end of the humerus to the very cartilage of the joint, and which shows the typical trabeculæ which are observed in such tumors in other bones. Phemister tells me there is such a specimen at the Presbyterian Hospital in Chicago. It is my belief that the peculiar structure of the head of the humerus makes the picture of giant cell tumor so modified that typical cases do not often occur.

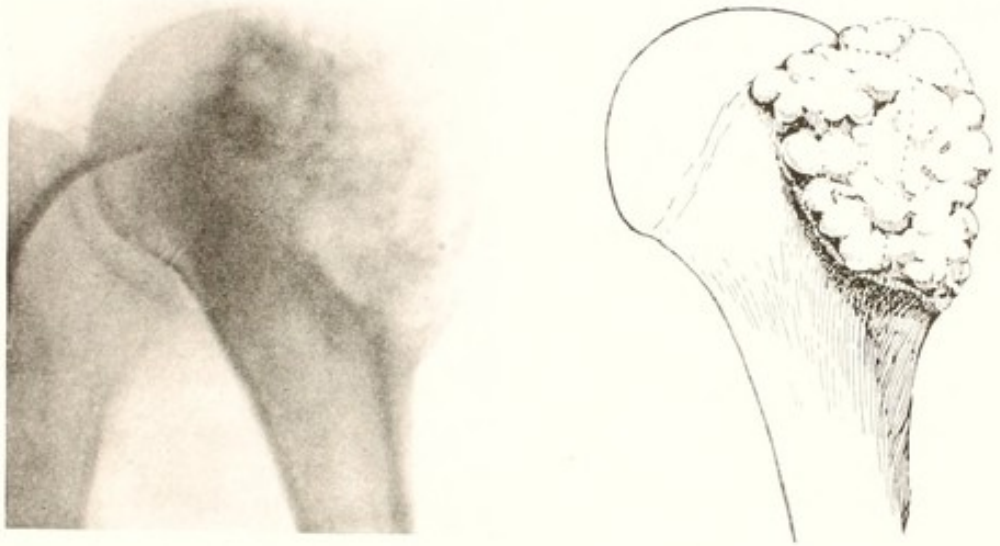


FIGURE 73. INTERPRETATION OF X-RAY OF GIANT CELL TUMOR ARISING IN A YOUTH

These tumors are modified in their structure because the epiphyseal lines are involved in them, and therefore contain irregular proliferating cartilaginous areas, and in these some bone production.

The type of case I allude to in the title is illustrated in the Registry Series by nine tumors of the upper end of the humerus, involving only the portion of the bone in the region of the tuberosities. I wish to call attention to the fact that all the cases in this class are very atypical for either giant cell tumor or osteogenic sarcoma, and that they have a characteristic appearance both by X-ray and in the microscopic slide. It is of interest that the first case (Croxton, Registry 5) has recently been reported by Geschickter and Copeland as a sarcoma.[1] On the other hand, Kolodny has reported the same

[1] Arch. Surg., 1930, May, pp. 731-733.

case as a giant cell tumor and illustrates his book (Figs. 88, 89, and 98) with photomicrographs and X-rays from it. The last case in the group, 1,031, has been recently reported by Phemister[2] as one of chondro-sarcoma. Three of the other cases were registered by Coley. Since these men have given serious study to the subject, I cannot lightly take the position that their conclusions have been erroneous, and that in reality, their cases were not malignant but were benign giant cell tumors of this chondromatous type. However, the intermediate seven similar cases in the Registry present such uniformity in the long history prior to operation, the ages of the patients, the good results following conservative treatment, and the unanimity of X-ray and microscopic appearance, that I am personally convinced that all the cases, theirs included, had essentially benign tumors. I believe that this group of tumors is a clinical entity which the surgeon should bear in mind when confronted with tumors of the upper end of the humerus.

In regard to one of these cases (391), of which we have no X-ray, registered by Coley, but which had been operated on in January,

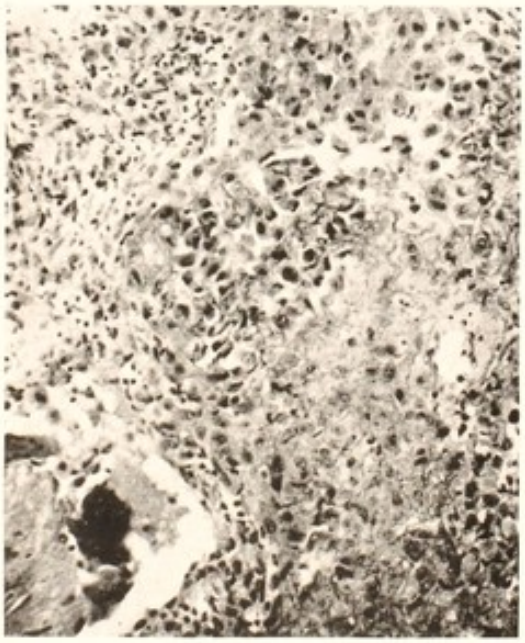


FIGURE 74*a*

Registry 391. Registered by Coley, but had been operated on at the University of Michigan, in January, 1915. The patient, a boy of 15 years, had a tumor of the right shoulder. He was unable to raise his arm. Pain had been present for eight months. He gave no history of injury. He was treated afterward with toxins. The slides show the characteristic appearance of these tumors. No X-ray. This is the case on which we quoted Ewing's pathological report. Well in October, 1923.

1915, at the University of Michigan, Ewing wrote on the Registry Classification sheet as follows:

"Dec. 18, 1923. This appears to be one of the cases of giant cell tumor associated with the absorption of cartilage. *Such tumors are generally found at the head of the humerus.* The giant cells are rather numerous, typical epulis type, and most numerous about blood spaces. The accompanying cells are peculiar, and are rather large polyhedral

[2] Surg., Gynec. & Obst., 1930, l. 216.

granular cells occurring in sheets and clumps. In some areas the giant cells are missing and the polyhedral cells appear exclusively. There are several small foci in which there is dark, staining fibrillar or partly hyaline material, which appears to be degenerating cartilage. There may be some new formation of this imperfect cartilage. There is no sign of bone formation. It does not resemble any of the well-known forms of osteogenic sarcoma. Tumor is relatively benign, but probably more active than most giant cell tumors."

The line in italics in which Ewing says, "Such tumors are generally found at the head of the humerus," shows his recognition of this type of tumor. He has also alluded to them in his paper at the 1929 London Congress under the caption of "Calcifying Giant Cell Tumors," as follows: "I also feel that these tumors are essentially giant cell tumors, and that one must not be deceived by the appearance of chondro-sarcoma which they give."

The much-discussed Croxton case, Registry 5, was that of a boy of 16, registered by F. G. Bunts, Cleveland, one of the first cases in the Registry Series. There has been much argument about this case

FIGURE 74*b*

SHOWING TYPICAL APPEARANCE OF ONE OF THESE TUMORS AFTER CURETTING

Registry 5. Bunts first saw the case May, 1919, five months after the boy had hurt his shoulder while wrestling. During these months, there was a history of the shoulder having slipped out of place and readily slipping back again into the socket. In the last two months there had been much pain, especially at night. There was also scapulo-humeral spasm. Bunts first treated the shoulder conservatively with a rubber bandage, but in October, 1920, he had X-ray pictures taken which showed the tumor. He then consulted Bloodgood, and operated on October 11, 1920. The patient was reported to be well ten years later.

among those interested in the Registry. Bunts consulted Bloodgood and the case appears in Bloodgood's writings as P. N. 26792. Bloodgood favored a pre-operative diagnosis of benign tumor, but changed this diagnosis to central sarcoma after seeing the specimen. Bunts, however, using his clinical judgment at the operation, decided to treat the case conservatively. He did a

very thorough curetting of the tumor and packed with gauze; eight days later he cauterized with zinc chloride, implanted radium, used postoperative X-ray and Coley's toxins. The patient has remained well. Geschickter and Copeland, in their recent article of 1930, still consider the case a sarcoma. In the early days of the Registry, it

FIGURE 75*a*

Registry 86. Registered by C. C. Simmons, of Boston. A boy of 13 years had had for one year, a hard, smooth tumor, the size of an egg. Curettage was done August 6, 1918. The X-ray picture which was typical of this kind of tumor was poor and was probably taken after curetting. The slide is a good example of this kind of tumor. It presents more giant cell tissue than some of the others. The giant cell tissue verges on the true giant cell tumor type. Patient was well in November, 1922. No further note.

was considered sarcoma by Ewing, Mallory, Wolbach, Wright, White, Fox, Taylor, and Phemister. Later, Ewing and Bloodgood changed their opinions and agreed with Graham, Stout, Connor, Morton, and the writer, who had considered it a giant cell tumor. It seems to me that in consideration of the other eight cases here presented and the outcome of this case that the diagnosis of giant cell tumor is the correct one. The nine cases may be summarized as follows:

SUMMARY OF NINE CASES

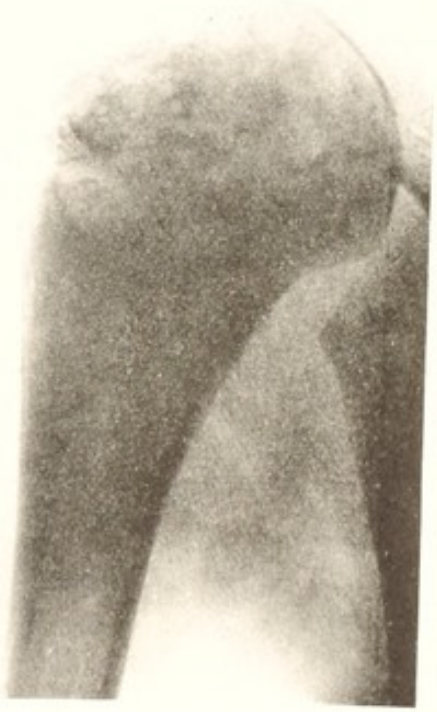
X-ray characteristics. These giant cell tumors or chondromata(?) of the upper end of the humerus lie in the angle between the upper and lower epiphyseal lines, *i.e.*, in the greater tuberosity, and are checked at the epiphyseal line of the articular head. Fractures of normal bones in this region are very apt to separate the portions of the bone at the lines of cleavage of the old epiphyses. It appears that in normal bone there is a line of cleavage separating the portion of the bone which is covered with cartilage from the rest of the head, and that this line of cleavage persists long after the epiphyses have united. It may be this line of resistant bone which keeps these tumors from reaching the articular head. At any rate,

these tumors do not, as do giant cell tumors in the neighborhood of other joints, actually destroy the bone to the very cartilage. Thus these tumors affect the bone after the manner of osteogenic sarcoma and one always sees in the X-ray picture a small portion of normal bone near the articular surface. This appearance in the lower end of the femur is very characteristic of an osteogenic sarcoma, but it should be discounted when it is found near the head of the humerus. In other words, these peculiar tumors break the rule of giant cell tumors in other bones of progressing through to the very cartilage of the joint, and in the head of the humerus do not extend much beyond the epiphyseal line.

They do not present the characteristic trabeculations which are seen in the X-ray picture of giant cell tumors in other bones. Instead they have a fluffy, "cotton-wool" look. After curettement, they present a fairly characteristic appearance, as if the tuberosities had been gnawed away, thus leaving the articular head and a very definite neck of bone on the inner side. Before curettement, the X-ray examination shows that there is a rather sharp line of demarcation separating the irregular, flocculent mass occupying the region

FIGURE 75*b*

Registry 603. Registered by Coley. Male, aged 15 years, was admitted to hospital February 18, 1924. Six months ago he had pain in the right shoulder, followed by stiffness three months later, with some swelling. He had had electrical treatments with no relief. He was treated with X-ray by Herendeen, and was reported well in July, 1931. No slides were made. X-ray films in this case indicate a certain amount of separation of the greater tuberosity, but are fairly characteristic of the group. No history of injury was elicited.



of the greater and lesser tuberosities from the articular head, the inner portion of the neck of the humerus, and the rest of the shaft. A very characteristic feature in all the X-ray films is a buttress of apparently thick, normal, cortical bone on the outer portion of the

shaft just below the tumor. This appearance suggests that the tumor has been of quite long duration and nature has supplied reactive bone to strengthen the cortex below it. There is also a thick endosteal layer of bone especially evident in Phemister's case 1031, which had been curetted twice before the resection. The outer buttress is not in appearance like the so-called "reactive triangle," in which there is a triangle between the raised periosteum and the old cortex. It is much more dense and extends much farther down the bone. The buttress is solid and is merely an increase of normal cortical bone, with no invasion of tumor tissue between the old cortex and the periosteum as we see in osteogenic sarcoma. All nine of these tumors are comparatively small, none being twice as large as the normal head of the humerus.

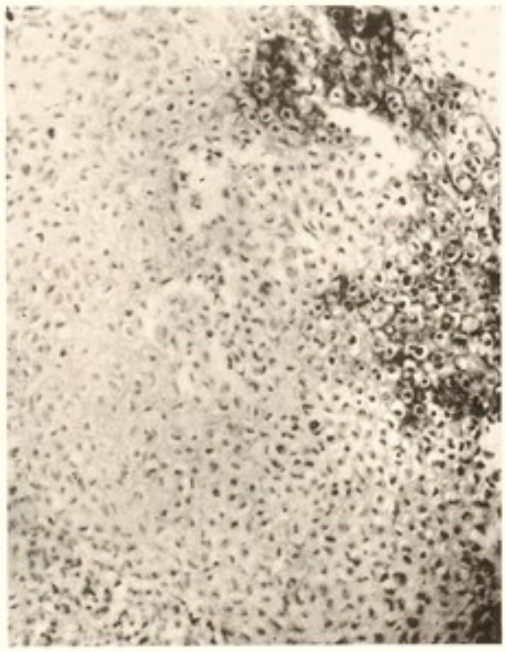


FIGURE 76

Registry 1031. Registered by D. B. Phemister, in August, 1919. Male, aged 22 years. Three and a half years ago he had slight pain, followed by slight limitation. Operation was done two years ago, by another surgeon; the area of reduced density in the tuberosity being curetted. Symptoms soon recurred, a second curetting was done but no sections were made. Limitation of movement and pain became worse. Phemister resected the upper six inches of the humerus, and applied a bone graft in August, 1919. The outcome was successful, and the patient was well eleven years later. The X-ray is fairly typical of this condition and the slide is very typical. The relative proportion of chondromatous material to giant cells is greater than in the other cases, but this case was operated upon twice before, and had in the meantime passed the age of epiphyseal union. The X-ray was made from the specimen after removal.

The microscopical appearances are chiefly distinguished by the presence of peculiar epithelioid cells which merge into a low-grade type of cartilage cell on the one hand, and into the cells of the tumor on the other. Sometimes the newly formed cartilage forms the bulk of the tumor, and sometimes a tissue indistinguishable from giant cell tumor occupies most of the space. The gross curettings are composed partly of red, friable tissue and partly of whitish, sago-like bits of material or frank cartilage. Sometimes the bulk of one of these tumors is almost wholly cartilage. Perhaps these instances are in slightly older patients, near or just beyond the age of epiphyseal union, *e.g.*, Phemister's case 1031. Sometimes there is a deposit of calcium in the cartilage, and new bone formation. It is very likely that some cases may be complicated by infraction and callus.

We frequently find newly formed cartilage and bone toward the periphery of a giant cell tumor, and we sometimes see in giant cell tumors in other parts of the body small areas which correspond to the peculiar tissue seen in these tumors of the humerus. However, we seldom see, in tumors of the other parts, the bulk of the tumor made up by this low-grade cartilage and epithelioid cells.

Since we may find similar tissue in true osteogenic sarcomata which involve the epiphyseal lines in this region, it cannot be too strongly stated that the presence of this tissue does not prove that the tumor is benign. I merely wish to make the point that this peculiar tissue probably indicates a benign process and together with the charac-

FIGURE 77

Registry 174. Registered by Coley. No operation was done and hence no slides were made. Patient was a pregnant female, colored, aged 24. She was admitted to Memorial Hospital, February 1, 1922. She had had pain, with increasing severity, for six months, and a tumor soon followed, July, 1921. The method of treatment was not stated. In August, 1930, Ewing reported that she was well. The notes were very brief. The X-ray is fairly characteristic of this type of tumor.

teristic X-rays and clinical history would justify conservative treatment. In fact, I believe that characteristic X-rays alone are sufficient evidence to justify the institution of radiotherapy without incision. It seems to me that there is reason to believe that if one of these patients were treated with the X-rays alone, the ultimate result would be better. Unfortunately, there are no detailed notes on the end-result of Case 174, which is the only one in which no incision was made.

Clinical points. The age may prove of importance in diagnosis, when we get a larger series. It is highly probable, as suggested by J. H. Wright, that this cartilage-like tissue is derived from the epiphyseal line. It will be observed that the ages in the cases here presented were in the neighborhood of the time when the epiphyseal lines were still present (given as twelve to twenty-four, but the tumor must have begun earlier in each case). One must bear in mind, too,

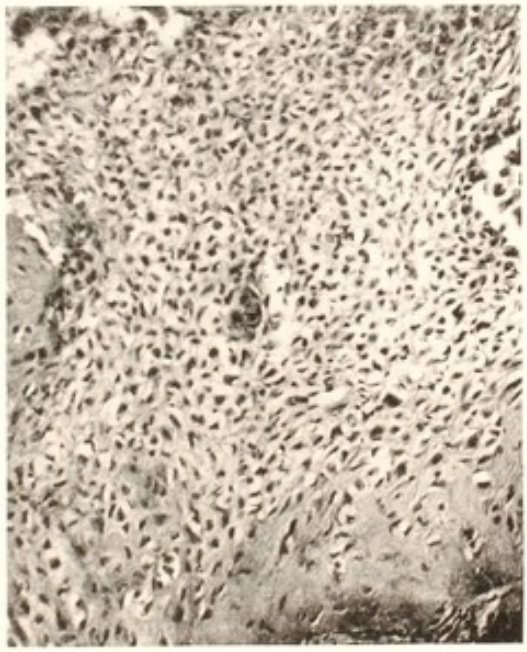


FIGURE 78

Registry 816. Registered by E. T. Dickinson, Greenville, North Carolina. A girl of 17 years, about a year before entering the hospital, noticed a dull pain in the right shoulder. The pain had increased until she could barely use the arm at all. Abduction to more than twenty degrees was impossible. Operation was done October 5, 1926. The tumor was curetted and swabbed with zinc chloride. The supraspinatus and subscapularis were removed and sutured back to the rims of the head. After operation Coley toxins and X-ray treatment were given. She was well, with some limitation of arm movement, in August, 1930. X-ray pictures before and after operation were typical of this kind of tumor. The slides were very typical. There was more bone formation than usual. There were areas of giant cells mixed with cartilage and bone.

that the epiphyseal lines in the region of the head of the humerus occupy a relatively larger part of the bone than do such lines in other situations, because there are three separate centers of ossifications: one for each tuberosity, and one for the head. The upper surface of the diaphysis, adjacent to the epiphyseal line, is cone-shaped.

FIGURE 79

Registry 815. Registered by W. A. Clark of Pasadena, California. A boy aged 20 years, colored, had pain in his arm which began in November, 1926. The arm was stiff. He was operated on March 10, 1927, at which time the entire upper end of the humerus was resected. He was working October 13, 1931. The X-ray was very typical, as was the microscopic slide.

Another clinical point is the relatively long history of symptoms prior to any operation, or the formation of a large tumor, *i.e.*, at the least, six months and an average of over a year. It is of interest to us also that the early symptoms are very similar to those of subacromial bursitis, *i.e.*, pain and restriction of motion. As I have never taken care of one of these patients myself, I cannot speak positively of the early symptoms, and the notes in our cases are very meager, but all mention pain and restriction. However, since these tumors lie just beneath the bursa, it is highly probable that they do produce a bursitis by crowding the enlarged tuberosities under the acromion. There should be a question of this diagnosis in cases with symptoms of subacromial bursitis in persons under twenty, for sub-

acromial bursitis is rare at this time of life, and these peculiar tumors occur only at this time of life.

Since all twenty-four patients with true osteogenic sarcoma of the head of the humerus are dead or very recently registered, *a most important clinical point in the nine cases reported here is the history of recovery without amputation of the arm.* In three of our cases curettements were done, excision was done in one case, resection including the head of the bone was done in three cases. The two others had no operations. Coley's toxins were also used in Registry cases 5, 174, 391, and 816.

I have described these cases in some detail for two reasons. First, because I believe they represent a subordinate clinical entity which should be recognized in the literature; and second, because they are among the few bone tumors which lie in direct contact with a part of the subacromial bursa and give bursal symptoms. In fact, an operator could not thoroughly curette or excise one of them without opening the bursa. If the growth were malignant, one could hardly fail to leave some cells in the bursa. That seven were thus curetted

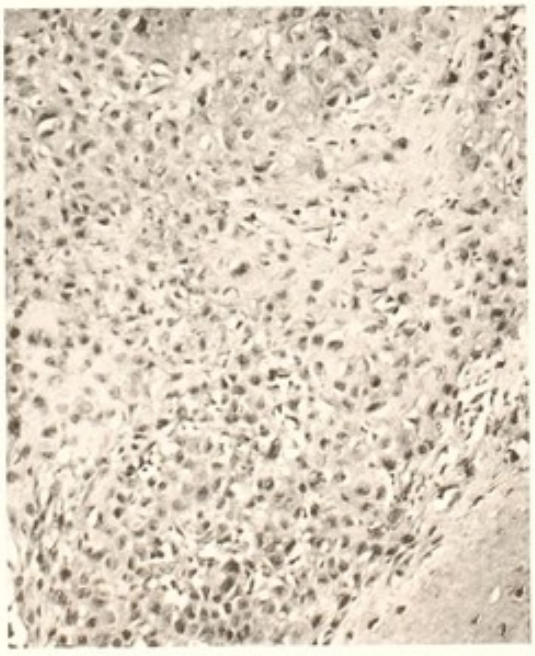


FIGURE 80

Registry 902. Registered by H. B. Thomas, Chicago Research Hospital. A girl of 14 years had had pain and difficulty in putting on her coat for a year. She gave no history of trauma. Some swelling was present. She had had some chiropractic treatments. She was operated upon by Hedblom, March 10, 1927, at which time the upper end of the humerus was excised. June 27, 1927, the upper end of the left fibula was transplanted. The wound healed with primary intention. She was well, July 27, 1931, with no sign of recurrence. The slide and the X-ray picture were very typical.

or excised with the bursæ opened (and probably also the joint), and extension of the disease did not result, seems strong evidence that the tumors were essentially benign.

Another point is that in our nine cases the clinical judgment of the surgeons in charge was evidently in favor of conservative treatment, for no amputations were done in spite of the pathology.

Treatment. Since the cases are few in number and the treatment has been outlined in each case, the reader may take his choice of methods. I think I should incline toward mild X-ray therapy, but if I decided to operate, the procedure followed by Dickinson (Registry 816) appeals to me as the best, namely, to excise the tumor and to suture the short rotators to the defect in the head of the humerus. Unfortunately, we have no details about the function of the arm in these nine cases. All are said to have useful limbs, but the degree of usefulness with and without bone grafts cannot be compared without examination of the individual cases.

It is highly probable that similar giant cell tumors may arise in patients between ten and twenty years, in the region of the other skeletal epiphyses, especially in the homologous region of the great trochanter of the femur. I believe that Case 277 is such an instance, although in the Registry it is carried as a case of osteogenic sarcoma.

COMPARATIVE DATA OF THE NINE CASES

Registry No.	Age	Date of onset	Duration before treatment (Mos.)	X-ray	Slide	Nature of operation	Date of operation	Yrs. since onset	Date Last Note
5	16	5-19	18	+	+	Curettage	10-19-20	10	9-29
86	12	8-17	12	+	+	Curettage	8-8-18	5	11-22
174	24	7-21	6	+	0	X-ray ?	(?)	9	8-30
391	15	5-14	8	0	+	Curettage	1-29-15	8½	10-23
603	15	8-23	6	+	0	X-ray	2-12-24	8	7-31
815	20	11-26	5	+	+	Resection	3-10-27	5	10-31
816	17	9-25	12	+	+	Excision	10-5-26	5	8-30
902	14	2-26	12	+	+	Resection and transplantation	3-10-27	5	7-31
1031	19	8-16	36	+	+	Resection and transplantation	8-19	14	10-30

NOTE.—The years since onset have been brought up to date so far as may be from data recorded with the Registry on April 7, 1931.

With regard to the name by which to designate this type of tumor. I have used the adjective epiphyseal because I believe that these tumors are characteristic of the period of life when the epiphyses are uniting and that their peculiar cells arise from the epiphyseal cartilage. I have used the adjective chondromatous as descriptive of their histology. I believe that they are essentially benign giant cell tumors, and thus that part of their name is justified. Yet I am not satisfied with such a long, cumbersome name. Ewing speaks of them as calcifying giant cell tumors, but that does not seem to me descriptive enough. We must find a name which will not tie the tongue, but it should associate adolescence with this puzzling type of lesion.

(*End of reprint.*)

Metastatic Cancer. The advance of a malignant metastasis may mimic the methods of invasion of any of the primary bone tumors. It usually begins in the neighborhood of the nutrient artery or in one of its branches as an embolus. The cells multiply and have an osteolytic and invasive power. The bone melts before them or, if they are slow growing, piles up new bone to check their advance. Rapid vascular metastases may dilate the bone by pulsation as do giant cell tumors. If the osteolytic quality of the cells is predominant, a local defect is produced in the bone and transverse fracture occurs. If there are multiple emboli, the bone may be riddled by many foci which all advance together and melt into one another as do the lesions of myeloma, but the X-ray appearance is not quite like the latter.

The very diffuse form of cancer involving the whole bone without changing its shape, is probably due to lymphatic extension. Although many writers have denied the existence of intraosseous lymphatics, Kolodny believes he has proved their presence in bone. (Kolodny, Anatole. The Relation of the Bone Marrow to the Lymphatic System. *Arch. of Surg.*, 1925, Vol. II, pp. 690-707.)

On the whole we may say that cancer is so variable in the way it attacks the bone, that any unusual X-ray of a bone tumor should arouse the suspicion of metastasis. In an adult, a localized osteolytic tumor in the shaft of the humerus or involving the upper end of the bone from the mid-shaft upward, will usually prove to be cancer. In our table, it will be seen that all six cases of metastatic cancer occurred in the shaft of the humerus, and none in the ends. One case probably started in the shaft not far above the nutrient foramen and the whole upper third of the bone was destroyed. While this series is very small,

it accords with my general experience, which is, that a tumor of the upper end of the humerus, without involvement of the shaft, will rarely prove to be a metastasis. There are exceptions, however. In generalized carcinomatosis of the skeleton the humerus is, of course, involved. Myeloma may involve the shaft. Metastatic cancer seems to be the only lesion which invades the head of the bone to the very cartilage.

Of the five cases confined to the shaft, two present dilatation of the cortex, while the other three show only localized bone destruction, without dilatation. The dilatation probably depends upon the vascularity of the tumor, for the blood pressure working from within the bone destroys the firm cortex after the manner of an aneurysm,

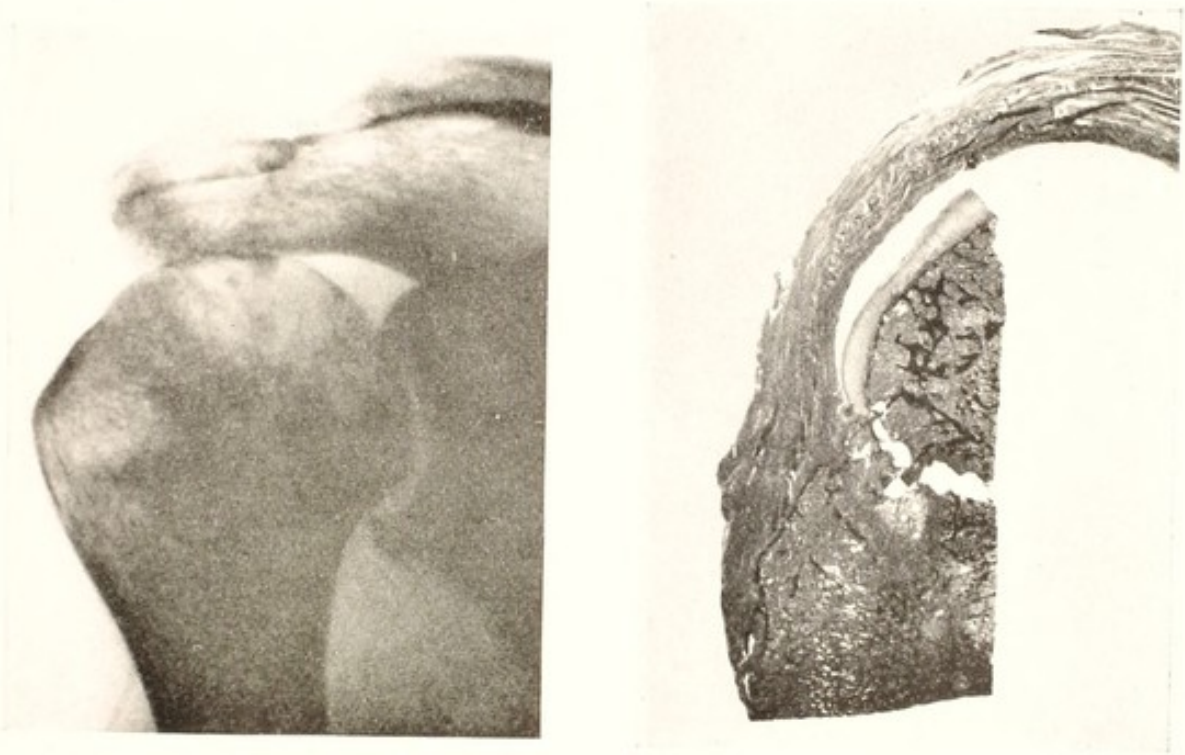


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FIGURE 81. METASTATIC CANCER INVADES THE MARROW SPACES OF THE ARTICULAR HEAD WITHOUT CHANGING THE CONTOUR

a. Metastases of cancer of the breast. Reduced one-half. Both the head of the humerus and the acromion are involved. The opposite shoulder was also similarly affected, as were many of the other bones, but the outlines as shown by X-ray were not dilated or otherwise changed. Notice that the involvement, as shown in radiability, extends into the articular head of the humerus.

b. Photomicrograph of section through the attachment of the supraspinatus tendon in a case of cancer of the prostate with diffuse skeletal metastases. Enlarged two diameters. The section was broken when being cut. The marrow spaces are filled with cancer cells.

In films which show disorganization of the interior of the articular head but without change of outline, the presumption should be that the disease is cancer, for not only does cancer generally involve this region, but sarcoma very rarely passes the old epiphyseal line.

or of a giant cell tumor. Hypernephroma metastases frequently have this expansile quality. As in giant cell tumors, the periosteum may keep endeavoring to surround the growth with new bone, and unless the advance of the tumor is rapid, maintain a thin bone shell about it.

It is a fair working rule that a localized tumor of the mid-shaft will prove to be a metastasis, although occasionally a myeloma (No. 676) or an osteogenic sarcoma (No. 156) occupy this position.

Benign Osteogenic Tumors. This heading includes tumors which formerly appeared in the literature as osteomas, chondromas, exostoses, ecchondroses, enchondromas, and the combinations of these tumors with fibromas and myxomas, *e.g.*, fibro-myxo-chondro-osteoma, etc. These tumors are as a rule congenital, but may not be noticed until about the time of union of the epiphyses and seldom late in life. Usually they come to the notice of the parents at about the time when the child reaches the age of ossification of the various epiphyses. They are usually pedunculated in other bones, but are apt to be sessile about the head of the humerus. They may be single or multiple.

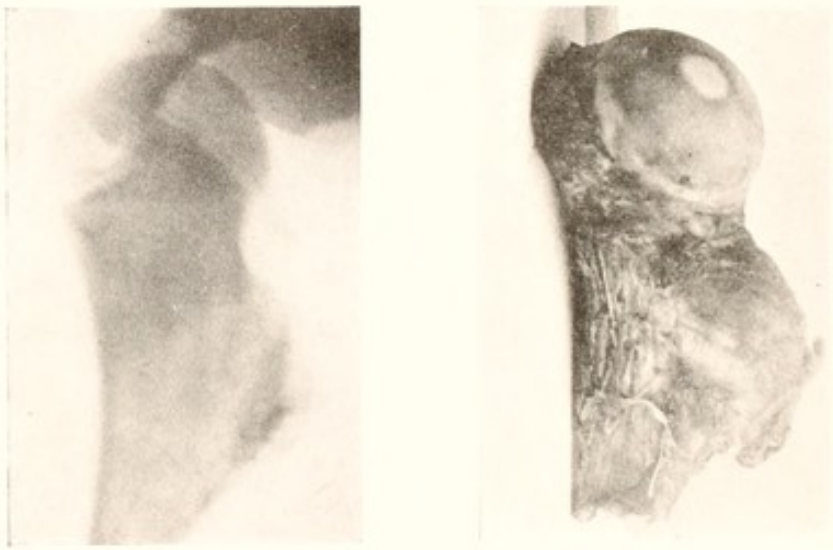

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FIGURE 82. BENIGN OSTEOGENIC TUMOR
Registry No. 31

Benign osteogenic tumor of the upper end of the humerus with the X-ray projection. Although poor, the X-ray is presented because it is taken from the bone shown in the photograph. This form of tumor is not uncommon in this situation. In the writer's opinion radical excision of the tumor, leaving only enough shaft to form a reasonably strong bone, is indicated, rather than total excision.

The tumors are not invasive, and while they push aside other tissues, they do not either destroy or penetrate them. The proportions of combinations of osteo-, fibro- and chondro- elements appear to have little significance, except that the softer the cartilage be-

comes, tending to be myxomatous in character, the more rapid the rate of growth is likely to be. These tumors are not uncommon near the head of the humerus, and it is rather remarkable that only one instance of a benign osteogenic tumor of the humerus has been registered. We do not ask for registration of these cases, but one would think that a considerable number would have been sent us, because they had been mistaken for malignant tumors.

The one instance which was registered, No. 31, although quite a small tumor, was excised by a surgeon under the impression that it was a sarcoma, and his diagnosis was confirmed by the hospital pathologist. In this particular case, the members of the committee have never seen the slides on which the diagnosis of sarcoma was made, for although the surgeon was quite willing, the hospital authorities did not wish the slide taken from the laboratory to be recorded with the case. We therefore cannot say positively that the tumor was benign. We can only judge from the X-ray which is here reproduced, and from the clinical fact that there was complete recovery after excision—the patient being reported well nine years later. The X-ray is very typical of the sessile tumors which occur in this region. Complete removal was perhaps wise, although the tumor might never have caused metastasis. Thorough excision of the base should be as effective.

The age of the patient is an important factor in deciding whether to operate in the case of such a tumor. My opinion is that operation should be postponed until after the epiphyses are united, for it is quite likely that the tumor will cease to grow at that time. Rapid rate of growth, or growth after the twentieth year, would speak for excision.

So far as benign osteogenic tumors are concerned, I shall have to draw on my own experience rather than on the recorded data of the registered cases. I vividly recall, before the days of the Registry, two exaggerated cases of such tumors of the upper end of the humerus which I had great difficulty in removing. It is one of the characteristics of this type of growth that it tends to extend along the fascial planes without invading the tissue, and if the tumors are large, one finds the muscles, nerves, and vessels buried in channels in the growths. I also have had experience with similar tumors of great size in other bones and I believe that it is of importance, if one operates on these tumors at all, to be very thorough in removing the base of the pedicle through the whole cortex. One should be very careful to remove from the tissues every particle of chipped material in

case one has to take the tumor out in pieces. When these tumors are pedunculated, their removal is not difficult, as they are not adherent to any tissue, and usually shell out easily, after the pedicle is divided. Often their surface is covered with cartilage and over this is an adventitious bursa on which the soft tissues and muscles slide.

The operative difficulties will come in those tumors which are sessile and are so large that they have branched between the normal fascial layers and surround the anatomic structures. In the two cases above alluded to, the deltoid was detached from its tubercle and turned upward over the shoulder. The humerus was then sawed directly across, and holding the lower end of the upper fragment, the nerves and vessels were dissected from their canals in the tumor, which was removed piecemeal with the whole upper end of the humerus. This very extensive excision was accomplished in both cases without dividing any of the important structures, but it necessitated removing portions of the tumor with a hammer and chisel, and much chipping of the tumor into small bits which were as far as possible removed. At the end of the operation, the deltoid was resutured to the upper end of the distal fragment of the humerus. Both patients obtained remarkably useful arms, having very nearly as much function as the classical cases of excision, where only the articular head of the humerus is removed.

One of these cases was that of an extremely stout woman with a very large tumor. In spite of my greatest care to remove chips of the tumor, bits of cartilaginous material remained entangled in the tissues, between the joint and the distal fragment. Many of these bits of tumor tissue afterward grew independently, and for some years I occasionally had to remove some of the bits which had grown to be the size of robins' eggs. They were always definitely encapsulated. These secondary tumors, on section, were almost pure hyaline cartilage. Eventually, so many of these bits recurred, and their consistency became so myxomatous, that I did a shoulder-girdle amputation. It happened that Dr. Bloodgood came to Boston within a few days after this, and I showed him the specimen before he left. Responding to his exemplary enthusiasm, I presented the limb to him. The picture remains with me of enthusiastic Joe Bloodgood with this stout shoulder and arm wrapped in brown paper, stepping aboard the night train for Baltimore.

Although an exaggerated case of its kind in regard to the size of the tumor, and the clear character of the cartilage in the implantations, it is only an example of a fairly common type of tumor

which is benign, for this patient is still living nine years since the last operation. I have seen other cases in which, owing to the fact that the operator had not thoroughly removed the pedicle of the tumor, recurrence at once took place. Cases are on record where these tumors at first are histologically of a distinctly benign character, and have become true osteogenic sarcomas after repeated ineffective surgical measures, or even where no surgery has been done.

Many of these pedunculated, osteo-cartilaginous, benign tumors are multiple. I have been able to follow one of these multiple cases over many years (Registry No. 343), and have seen the individual tumors take on rapid growth and become histologically malignant tumors. The poor fellow had, in all, three limbs amputated, but ultimately died of acute appendicitis with peritonitis. This case does not appear in the table because it is tabulated with the lower extremity. In one case (Registry No. 1016) one of the tumors appeared in the unusual position of the center of the shaft of the humerus. As a rule, operation should not be done in these multiple cases, until the tumor renders the limb inferior to an artificial one. Even when rapidly growing they are not very malignant so far as metastasis is concerned.

Personally, I am inclined to think that the Registry should recognize less sharp divisions between osteogenic sarcomas and benign osteogenic tumors. We should have a Borderline Class, which should include such cases as the two mentioned above. Some of these cases are certainly borderline, and I think it is the failure to recognize this borderline class that gives some surgeons and pathologists the excuse for using the word "chondro-sarcoma." The prognosis will depend on the surgical circumstances which attend the operation, as to whether the tumor can be excised entirely with the cortical bone from which it springs. The histologic criteria which help to form a prognosis in these borderline osteo-chondromatous tumors are not distinct, and depend on one's estimate of what degree of distortion normal cartilage cells may assume without becoming malignant. Mitotic figures will not help us, for we do not find them in cartilage cells. The clinical history, X-ray and gross pathology are of more importance than the histology. The pathologist should be able to see the extent of involvement shown in the X-ray before being asked to make a prognosis. If the X-ray shows intra- as well as extra-cortical involvement, malignancy is more likely. The size of the pedicle is important.

In the multiple cases some of the tumors may be intra- and some extra-cortical. If a tumor is extracortical and also intracortical,

suspicion of malignancy is aroused. Flocculent deposits of calcium near the periphery in these borderline tumors give a characteristic appearance in the X-ray.

Sarcomas Occurring in Paget's Disease. Only two cases of the twenty-four osteogenic sarcomas of the upper end of the humerus (No. 484 and No. 643) were over fifty years of age. Both of these patients had Paget's disease. This concurs with the finding which I have mentioned elsewhere, that most cases with osteogenic sarcoma, who are over fifty years of age, have Paget's disease of the skeleton. This conclusion was drawn about five years ago, after a review of the whole Registry series, including all bones. Since then, I have seen a few, but very few, exceptions. Of the seventeen cases of osteogenic sarcoma of the upper end of the humerus registered since 1932, there were three of about fifty. All three had signs of Paget's disease of the bones. Search for signs of Paget's disease in any case of bone tumor in patients about fifty, and search for sarcoma in every case of Paget's disease, for about fourteen per cent of all cases of Paget's disease develop it. (Sarcoma Complicating Paget's Disease of the Bone. Clarence E. Bird. *Arch. of Surg.*, June, 1927, Vol. 14, pp. 1187-1208.)

The histology of the sarcomas that develop in Paget's disease is varied and not typical. In one of our cases (Reg. No. 297), the slide resembles that of a benign giant cell tumor. This case is the only Paget's case in the Registry series in which the slide does resemble a giant cell tumor. Most of the other cases are quite atypical in structure, but their intercellular substance is distinctly osteoid, and by X-ray they are usually of the osteolytic type.

The facts that (1) osteogenic sarcoma is usually found in youth; (2) Paget's disease is seldom found before fifty; (3) less than one in 100,000 normal people have bone sarcoma anyway; (4) fourteen per cent of cases of Paget's may be expected to have it; (5) Paget's disease shows an instability of bone metabolism with enlargement accompanied by loss of lime salts; should give a medical Sherlock Holmes the key to the mystery of sarcoma.

Inflammation.

Under the heading "Inflammation" of the upper end of the humerus, we have six cases. All these six tumors were in our opinion cysts, although they were registered with the idea that they were sarcomas, or giant cell tumors. It may well be disputed whether cysts are to be classed as tumors or inflammation. All these cases, and practically all cysts that I have seen which were not registered,

have been in the same situation in the metaphysis and limited by the epiphyseal line, for they nearly always occur in young people, usually under ten years. In all six cases, a pathologic fracture without much displacement had occurred. Pathologic fracture appears to be the rule in these cysts, and repeated fractures may be sustained, but the amount of displacement is very little. A patient may even have a cyst in childhood which later leaves the bone weak, so that pathologic fracture occurs again after the age of epiphyseal union. X-rays in such cases are quite deceptive, for the walls may have become quite thick.

Figure 83. Cyst of the Upper End of the Humerus

An old bone cyst in which repeated infractions and healing had occurred.

Very frequently the cyst is not noted until a fracture occurs. The X-ray appearance is so characteristic in these bone cysts, and has now become so well known, that there is little danger of confusing the lesion with any other, except giant cell tumor. The amount of anxiety caused by one of these cases has changed very much in the last decade, for ten years ago it was not at all unusual for bone cysts to be misdiagnosed as sarcomas, and to cause a great deal of discussion and consultation. Dr. Bloodgood should be credited with much of this improvement, for he has for many years reiterated the diagnostic points which distinguish cysts from sarcomas. Even to-

day, it is always with a sense of responsibility that one decides against operation in one of these cases, because the penalty of mistaking an osteogenic sarcoma would be so great. It is a fact, however, that reliance can be placed on the X-ray. In the case of a child, a good, clear-cut picture of a central tumor, extending for several inches below the epiphyseal line, slightly dilating the bone, with an abrupt concavo-convex outline on the lower medullary end, almost certainly indicates a bone cyst. The bone cyst is unlike the epiphyseal, chondromatous giant cell tumors of the humerus in that the latter are between the epiphyseal lines and do not involve the inner contour of the neck, and occur in the teens rather than before ten. I do not know of any case of a giant cell tumor in the upper end of the humerus below the epiphyseal line, but in all the cases of cysts which I have known, that had any operative interference, there was a greater or lesser zone of tissue, resembling giant cell tumor lining the bone, so that many have been classed as giant cell tumors.

Geschickter (*Arch. of Surg.*, August, 1929, Vol. 19, pp. 169-271) has produced a very interesting theory, on a parallel of the normal method of cartilage absorption, for the common origin of cysts and giant cell tumors. I agree in the idea of their merging, but I am more inclined to feel that their origin is generally due to intraosseous hemorrhage from a fissure fracture, dividing a nutrient vessel in the spongiosa. I feel that the difference between cysts and giant cell tumors is probably connected with the age of the patient and the condition of the bone near the epiphyses. Apparently in cysts the artery is soon occluded, and the defect, instead of being filled with proliferative endothelium (which to my mind the cells of giant cell tumor are), through which the blood corpuscles circulate, becomes filled with serum, and has a lining membrane surrounding it, often partly composed of a tissue similar to giant cell tumor.

No tumors of the upper end of the humerus have been sent to the Registry which we thought were of an inflammatory nature, except the cysts. As a matter of fact, in my experience apart from the Registry, I have rarely seen in this region cases of inflammation, even including osteomyelitis and fracture, which simulated real neoplasms in the X-ray. Osteomyelitis in this region in adults must be a very rare condition.

There remain, in the first column of the table, the Atypical Sarcomas and "Cases Discarded" on account of poor data. It would be out of place to attempt to draw conclusions from either of these classes of cases, but we may at least find some encouragement, for

a few of the atypical cases of undoubted malignancy are well and will be considered under prognosis.

Shaft of the Humerus. (Second Column in Tables.) Following the policy of relating the contents of this book to lesions in the neighborhood of the subacromial bursa, I shall not discuss tumors of the middle and lower third of the humerus. The reader may readily see the comparative rate of occurrence of these by reference to the chart, and if his curiosity tempts him further, he may send to the Registry for the envelopes of the cases in any particular cross-section of the chart. This gives me an opportunity to say that it is a policy of the Registry to permit any qualified person to study the Registry series and to use the material for his surgical papers as I am using the present study for this book.

The chart confirms the current belief that tumors in the long bones are more common in the ends of the bones toward which the main nutrient arteries point. The tumors of the upper end of the humerus stand in relation to those of the lower end as fifty-five to eleven.

Tumors of the Clavicle.

Only tumors of the outer end of the clavicle concern us, and it appears that following the nutrient foramen rule, more than half of the tumors of the clavicle occur in the outer end. We have in the Registry only a small series—eleven in number. The tumors of the clavicle, as a whole, follow the general rules as laid down for similar

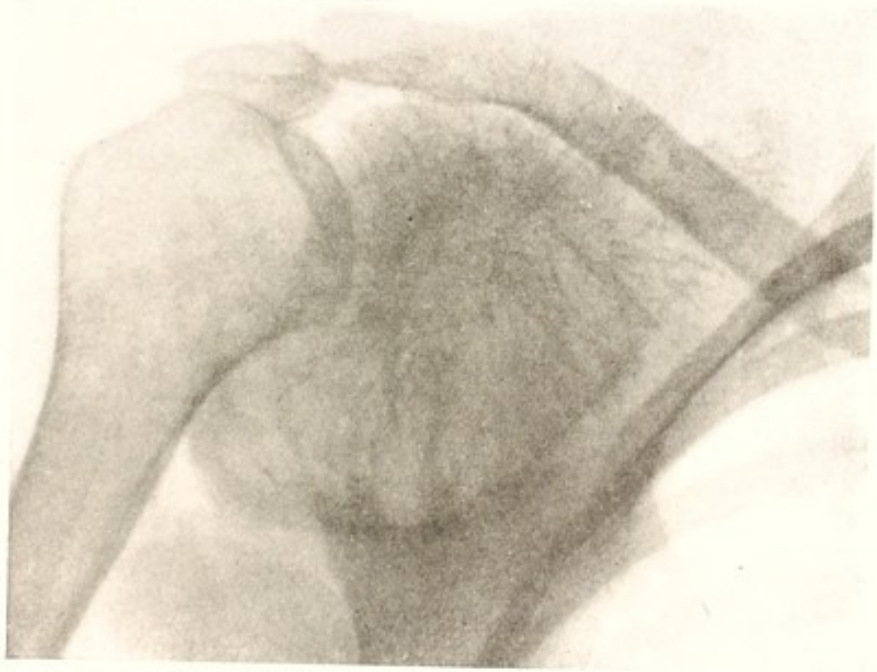


Figure 84. Angioma of the Scapula

The glenoid and coracoid process are involved. The peculiar radiating striation seems to be characteristic of the method in which cavernous angioma destroys bone.

tumors in the other bones. It is not difficult to diagnose from the X-ray the osteogenic sarcomas on the principle of the presence of the old shaft in the tumor and the other criteria spoken of in the humerus. The two Ewing's tumors are nearly exactly alike, and have the same characteristic form of invasion of the bone as do these tumors in other regions. The myelomas and giant cell tumors are also very typical in the way they affect the bone. In other respects, this small series of tumors of the clavicle yield nothing of importance for record.

Tumors of the Scapula.

Unless they were in the acromion or coracoid, tumors of the scapula would not be in relation with the bursa. We find that the rest of the bone is much more susceptible to tumors than is the acromion process. However, we have a very good example of each of the typical tumors in the acromion, and furthermore, the prognosis appears to be good, for none of the six cases recorded, whatever the variety of the tumor, have yet been reported to have died.

In one of the cases I personally had the clinical responsibility. A young woman (Registry No. 392) with a conspicuous tumor of the acromion was referred to me by the surgeon who had explored the tumor and found it of the giant cell type, although the pathologist considered it malignant. I wrote this surgeon that I thought I should have to do a shoulder-girdle amputation, but that Ewing and Herendeen had recently been using X-ray treatment with some success in this type of tumor, and I thought it was only fair to try the X-ray before removing the arm. Dr. L. B. Morrison kindly undertook the X-ray technique. For a few months I had much worry. The tumor swelled and softened until it seemed it must break the skin. I barely withheld myself from operating, but felt that it was now too late, and we must trust the X-ray and Ewing's statement that while these tumors appeared to grow worse following the use of the X-ray, they afterward shrank and the bone returned to approximately a normal condition. The result, six years later, is shown in Fig. 85. The patient is well. During this period of time this method of treatment has become accepted, but the course of my first case was followed with anxious attention.

Angioma.

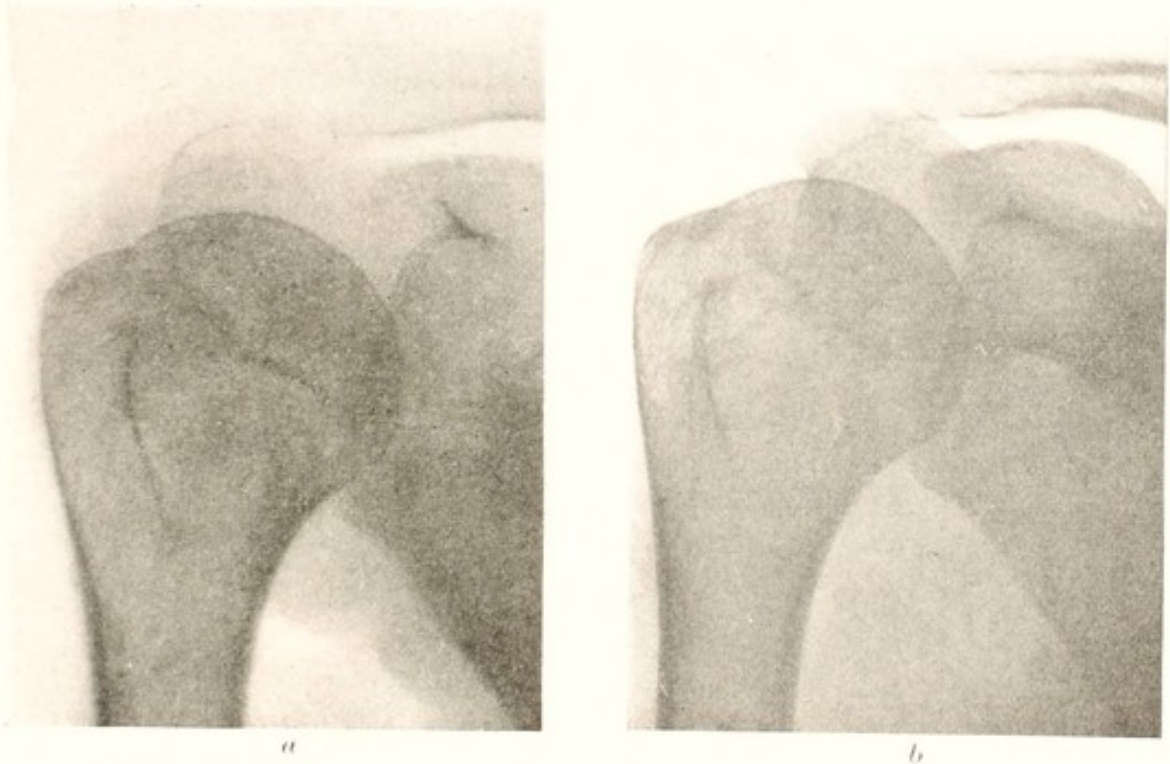
Cavernous angioma of bone is rare, but there is one instance in this series with a very beautiful X-ray which illustrates the manner in which this condition attacks the bone. The tumor has involved the whole glenoid region, invading down the acromion to within an

inch of its tip. A very complete account of what is known regarding cavernous angioma of the bone will be found in the *Journal of Radiology*. (Paul C. Bucy and Charles S. Capp in *Amer. Jour. of Röntgenology*, January, 1930, Vol. 23, pp. 1-33.)

Prognosis of Bone Tumors About the Shoulders.

The table speaks for itself, for those cases are underlined in which death has been reported. The cases not underlined will be alluded to below. It goes without saying, that cases of cancerous metastasis are always fatal. The prognosis of multiple myeloma is little better, except that it is more favorably influenced by radiation.

From the table we see that prognosis in osteogenic sarcoma of the upper end of the humerus is certainly very bad. Only two cases (No. 49 and No. 812) were living when last heard from. Case No. 49, registered by Dr. Emmet Rixford of San Francisco, had a large osteogenic sarcoma of the upper end of the humerus. A shoulder-girdle amputation was done on June 11, 1901. The patient was well on April 16, 1904, and has not been heard from since. Onset of



a *b*

FIGURE 85. GIANT CELL TUMOR OF THE ACROMION BEFORE AND AFTER X-RAY TREATMENT

Films of case referred to on page 454, taken before and after treatment. Before treatment the acromion process appears to be nearly destroyed and shows only as a faint outline. In the second film the structure of the acromion appears to be nearly normal. In such tumors, although they appear to be quite diaphanous, the periosteum is capable of regenerating new bone just as it is when a piece of bone is excised subperiosteally.

the disease in this patient was two years prior to the amputation, so that the patient may be considered to have been alive five years from the onset. Five years from the onset and three years from the date of operation are encouraging, because they are longer than the average duration of life for osteogenic sarcomas of the upper end of the humerus, namely, about two years. Even among the five cases each of the sclerosing and osteolytic types, there appears to be no better prognosis than in the average type. The death of every case of these types has been reported.

There may be a slightly better prognosis for osteogenic sarcomas of the chondromatous type, as alleged by Phemister in *Surg., Gynec. and Obst.*, January, 1930. One of the cases (No. 812) has been reported in full by Phemister. This was a large tumor of the chondromatous type of osteogenic sarcoma and is alleged to have survived shoulder-girdle amputation for five years (3-15-31). In considering the chondromatous type, we must be very sure that we are not dealing with the epiphyseal chondromatous type of giant cell tumor, or benign or borderline osteogenic tumors. If the nine cases of epiphyseal chondromatous type were included as sarcomas, the prognosis would be much better.

Osteogenic sarcomas of the shaft may possibly have a better prognosis, for case No. 156 had a huge sarcoma and is well eight years after amputation. This case had prolonged radium treatment at the Memorial Hospital of New York prior to amputation. It was a very large tumor of telangiectatic type. We also have full data of another case, No. 147, well nine years after amputation, registered by Dr. C. C. Simmons. Registry No. 1016 is a recent case.

Osteogenic sarcoma in the lower end of the humerus. Case No. 299 was one of sarcoma occurring in Paget's disease and could not be traced. The patient is undoubtedly dead. Case No. 357, registered by Memorial Hospital Clinic of New York, was reported to be well three years after amputation, and since then could not be traced. No X-ray was registered.

All four cases of *osteogenic sarcoma of the clavicle* are dead. Three out of ten cases of *osteogenic sarcoma of the scapula* are still living. Case No. 759 was well four years after operation and radiation. Case No. 867 was a remarkable one which has survived ten years since excision of the blade of the scapula, *followed by an attack of erysipelas.* It is not supported by an X-ray, but there is unanimity of opinion among the pathologists on the fairly satisfactory slide.

Ewing's sarcoma in the humerus, as in other parts of the skeleton, appears to be more favorable in prognosis than osteogenic sarcoma. Case No. 246, reported by Bloodgood, was said to be well nine years after exploration and radium treatment, but the diagnosis is doubtful in my opinion. Case No. 326, registered by Meyerding of the Mayo Clinic, had survived eight years after amputation and radiation. In this case, the tumor had extended well into the upper end of the bone. Case No. 576, which was a tumor of the upper half of the humerus, treated by Coley with radium and toxins without amputation, was said to have been well six years after treatment. It is not supported by a microscopic examination, but the X-ray is very suggestive of a malignant tumor. It was complicated by a fracture.

That *Ewing's sarcoma in the clavicle* is sometimes amenable to excision, followed by radiation, is shown by case No. 28. This was registered by Dr. Andrew Hosmer of Salt Lake City, and was well supported by X-ray and microscopic evidence. A tumor of the outer end of the clavicle was resected with the whole outer half of the clavicle on March 28, 1919. The patient was reported to be well nine years later. This case was complicated by a small local recurrence about a year after the first operation. This was removed and postoperative radiation given.

Cases of *Ewing's tumor of the scapula* are also encouraging. Bloodgood has registered a case, No. 137, in which one of these tumors involved the whole spine of the scapula. A portion of the scapula was excised with the outer end of the clavicle in July, 1920, and in September, 1921, the remaining portion was excised. The patient was reported to be well about nine years after the second operation. This case was reported to Bloodgood by Dr. Buist of Charleston, S. C. The X-ray and slides are characteristic of this tumor.

Case No. 515 was a similar one, and was registered by Dr. H. E. Isaacs of New York with satisfactory data. The lower portion of the scapula was resected on March 28, 1923, and the patient was reported to be well in September, 1929, but to have died of pneumonia March 5, 1931.

Case No. 919 has not been heard from since operation.

The *Atypical Sarcomas* are also encouraging. Case No. 409, a large tumor of the upper end of the humerus, resembling a giant cell tumor in the X-ray, was treated conservatively after a biopsy. The patient was given toxins and radium by Dr. Coley, and later con-

solidation and bone formation took place in the tumor. She was reported to be well eight years later. The microscopic slide is poor and the pathologists cannot make a diagnosis from it. While the case is obscure, it is encouraging, for the tumor was certainly large and one would have given a bad prognosis. Cases No. 421 and No. 998 have not been traced. Case No. 83, a very large malignant tumor of the whole shaft and upper end of the humerus, was registered by Dr. Simmons. The patient is reported well nine years after amputation, although from the slide and from the X-ray a very bad prognosis would have been given. Although the data are well registered, there is no unanimity of pathologic opinion. Connor has included it in his series of Ewing's tumors, but others consider it an angiosarcoma. A somewhat similar case has also been registered by Dr. Simmons, No. 564, involving the lower end of the humerus and also much of the shaft. The data are also good but confusing. The case was reported well six years after amputation, but a year later there was a recurrence in the axilla, which was excised. While both cases are atypical, they were very large tumors, very invasive and histologically very malignant.

The one atypical sarcoma of the clavicle, No. 1059, is living three and a half years after excision.

The two surviving cases of atypical sarcoma of the body of the scapula were probably as near to our former definition of periosteal fibro-sarcoma as any cases we have had. No. 447 was registered by the Presbyterian Hospital of New York and was reported well, except for tuberculosis of the lungs, eight years after partial excision of the scapula. Case No. 111, registered by Bloodgood, was reported as well nine years after excision of a similar tumor.

There are probably many surgeons who feel discouraged about our progress in the treatment of bone sarcoma. I hope they will feel somewhat more optimistic, for I do after making this study. Hope in prognosis may be emphasized again by calling attention to the following cases of five-year cures among primary malignant tumors of the shoulder bones.

Osteogenic sarcomas, total	47	Five-year Cures	4	No. 147 No. 156 No. 812 No. 867
Ewing's sarcomas, total	21	Five-year Cures	5	No. 28 No. 137 No. 246 No. 326 No. 576
(Possibly, if No. 515 be included, 6)				

Atypical sarcomas, total	16	Five-year Cures	5	No. 83 No. 111 No. 409 No. 447 No. 564
			15 or 14	

To find fourteen or fifteen living over five years out of eighty-four cases is certainly encouraging, and if we omit the twenty-four cases of osteogenic sarcoma of the upper end of the humerus, thirteen or fourteen out of sixty! Or, if we include the nine living cases of epiphyseal giant cell tumors, which some pathologists believe are sarcomas, we find ninety-three cases of sarcoma with twenty-three or twenty-four five-year cures. In my opinion this last should not be done.

THE RELATION OF TRAUMA TO BONE SARCOMA

The American College of Surgeons has widely advertised the Registry of Bone Sarcoma and has made repeated appeals to surgeons, röntgenologists and pathologists to register their records of instances of this disease. This propaganda has continued over ten years, and some $20,000 has been spent. Yet only 1,335 cases have been collected and studied. Only 655 of these are true osteogenic sarcomas. The Laboratory of Surgical Pathology at Johns Hopkins, together with Dr. Bloodgood's personal collection, had at their latest report only about 400 case histories of osteogenic sarcoma after more than thirty years. Coley, after forty years of the most arduous polemics in favor of his toxins and yearly papers before scientific societies as propaganda, has probably seen more cases than any other surgeon in the world. Yet, in a recent paper, he states that the combined records of the Hospital for Ruptured and Crippled and of the Memorial Hospital since 1890 show 855 cases. This included all forms diagnosed as bone sarcoma, and perhaps many were not sarcoma at all. Greenough, Simmons and Harmer found that in the Massachusetts General Hospital in a ten-year period, there were only twenty-seven cases of osteogenic sarcoma, although many other cases had been erroneously diagnosed as such. Since many of the cases of Bloodgood, Coley, Greenough, *et al.*, are among those registered, the total number is not great. In 1922 from a postal card canvass of the doctors in Massachusetts it was estimated that at any one time there were not more than one of these cases in 100,000 of the population alive in this state.

According to the best information at present available (March,

1933) at the Massachusetts State Board of Health, there are not more than four cases of bone sarcomas in every 100,000 persons now living in Massachusetts. This estimate is based on the fact that in 1932 there were 58 deaths said to be from this cause reported from a population of somewhat over 4,200,000. If the average duration of life after the development of a bone sarcoma be assumed to be three years, we may estimate that at any one time there are about $3 \times 58 = 174$ persons in the state who have this disease. This is likely to be a maximum estimate because we may be confident that some of these 58 cases were probably erroneously diagnosed bone metastases from cancer, so that the real figure is somewhere between that arrived at by my postal card canvass and this one from the Board of Health statistics; *i.e.*, between one and four per every 100,000 of population.

These figures show that either the profession is excessively remiss in consulting experts on this disease and in recording their cases, or that the disease is an extremely rare one, notwithstanding the immense literature which has grown up about it. Yet the courts continue to give credence to expert testimony that in particular cases particular traumas cause particular bone sarcomas. How can any expert be justified in such statements when the disease is so rare? Trauma is an everyday event to normal boys and girls, and to many adults. Consider the frequency of traumas compared to the frequency of bone sarcoma.

How severe must a trauma be to act as a potential cause for one of these tumors? Theoretically, as a minimum, only of sufficient degree to stimulate the process of repair, and from that point up to a complete fracture of the bone. Certainly it does not vary directly as to the degree of injury, because thousands and thousands of people fracture their bones and it is almost unknown to have a sarcoma develop at once at the site of fracture. Moreover, many cases are recorded where an X-ray taken immediately after the injury has shown signs of a sarcoma already at the site of fracture. The degree of trauma sufficient to make an ordinary black and blue spot, which necessarily causes discoloration of tissue and bleeding beneath the skin, would surely be sufficient to start the process of repair. We may, therefore, accept an ordinary bruise as a trauma of sufficient degree to initiate the repair process, which, either lacking a normal check or becoming unduly stimulated, runs wild and might become a sarcoma. What is the relative frequency of black and blue spots to sarcoma in 100,000 individuals? I think any mother or

father who has noticed these spots on their children, even if only on Saturday nights during the few years that the bath is given and not taken, would agree that 10,000 bruises in a lifetime would not be an excessive estimate for each individual.

There is an impression among some doctors that bone tumors are much more common than I have estimated. I believe it is a false impression, although I will admit that *many cases are never registered* for two main reasons. First, these patients are notoriously difficult to diagnose and to cure. They usually go to several doctors before the diagnosis is made, and several more in hope of a promise of successful treatment, before death occurs. They often go from hospital to hospital. Second, being rare and spectacular cases, they are usually demonstrated at the Clinics as "interesting cases," and most of the doctors at every hospital see every case. Since each case requires the service of the X-ray Department, of the Pathologic Department and of the Surgical Department, the members of each service have the case impressed on their memory. The pathetic character of these cases, which are usually in strong young people and yet rapidly fatal, also makes them memorable.

I think enough has been said to show that these cases are rare, and I feel that I am conservative in saying that one case in 100,000 persons is a maximum estimate. If every person has 10,000 bruises, 100,000 persons would produce one billion combinations of person and bruise, to only one bone sarcoma. This seems a difficult argument to combat for those who wish to show that a given trauma caused a given case of sarcoma. You must turn your mind away from mathematics and think from a human nature point of view to get the idea that a certain trauma caused a given case of sarcoma.

It was *your* boy who had the bump which was followed two months later by the tumor. You find that one of your friends has a friend whose boy had sarcoma preceded by a bump. You become more convinced that bumps cause sarcoma. One of your neighbors heard of a boy who had this trouble and he recovered damages because the bump was received while the boy was at work. Your lawyer confirms this and adds that many such cases have received compensation for such injuries. You become more convinced that the bump in your boy's case caused the sarcoma. You feel sure he had no sarcoma until he had the bump. Your lawyer tells you that famous medical experts have testified that a large percentage of all cases of bone sarcoma had bumps within a year prior to the time the sarcoma was noticed. These experts have testified that they believe

bumps cause sarcoma, even if they will not say that the particular bump in question caused the particular sarcoma. And some will even swear to that. By this time you are convinced that if your boy is not awarded compensation a great injustice will have been done. And furthermore, you will hear from your lawyer that insurance companies are generally ready to settle such cases out of Court, so great is the probability that Accident Boards and jurors will decide in favor of the poor boy with an amputated leg or arm.

At this point, the argument that 100,000 boys each bump themselves 10,000 times without having sarcoma, will go in one of your ears and out the other. You will say that 10,000 aviators might each fly 100,000 miles, yet one bump might kill any one of them, and think you have drawn a parallel. But the two propositions are not parallel and are widely different. It would be as logical to say that if one aviator in 100,000 had a bone sarcoma, aviation causes sarcoma. If all the aviators bumped in every mile the parallel would be more reasonable, for aviation would stop, and so would the human race if trauma caused sarcoma.

Suppose that your boy had a very slight prominence on his shin. You happened to notice this slight prominence. There is a beginning to everything. From your experience in counting the black and blue spots on his shins, how long will it take him to get one on that particular lump? Not long, you must admit. Then you and he will notice the lump and observe that unlike the other spots this lump will not go away altogether. In a month or two it may enlarge still more and become painful, and prove to be a sarcoma. Which black and blue spot caused the sarcoma? One of those previous bruises you have seen in the last five years, or the one that came after you had already noticed the lump? You will have to admit that if you have a boil or a bruise on some part of your person, some friend downtown is sure to hit it. How often has your other boy, who has no sarcoma, complained that when you grabbed him you hurt his sore arm or sore leg? Confess that you are too busy to count the lumps and black and blue spots on your boy every evening. Yet you say you are convinced that this particular bump caused this particular lump, and your lawyer thinks so, too. I admit that you may find the jury sympathetic and ready to be convinced. When the experts take the stand and swear they believe bumps are contributory causes of bone sarcoma, very likely you will get a verdict. Yet personally, I think the chance of any one boy getting a bump on a particular sarcoma is much greater than the billion to one chance of any particular bruise causing a sarcoma in 100,000 boys.

Let us see what grounds any experts have for such a statement as that sarcomas are caused by trauma. I will ask the questions and answer them as truthfully as I can, even though I favor the billion to one idea. I will now appear for the plaintiff.

Question. Do you believe that a blow or a bump may be a cause of a bone sarcoma? (Notice wording is not *may cause*.)

Answer. Yes. (*Sotto voce* explanation to my conscience.) Maybe a contributory cause.

Question. Why?

Answer. *First*, because a high percentage of these cases give a history of a local trauma preceding the discovery of the tumor by a number of months, and such a trauma may be assumed in the others. (This is entirely to impress the jury, because it does not impress me a bit, although I cannot be called a liar for saying it.) *Second*, because certain doctors, experimenting with malignant tumors in animals, found that when tumor cells were injected into the blood of these animals, tumors developed at places in the parts of the bodies of the animals which were purposely traumatized. (Also impressive but quite illogical.) *Third*, because the structure and the microscopic appearance of bone sarcomas vaguely resemble the process of repair which takes place when a bone is broken. (The only reason that I personally have any respect for.)

Question. Any other reasons?

Answer. Yes. (Followed by some learned "hot air," which is of no real importance.)

Question. Do you believe that the particular trauma caused this particular tumor?

Answer. It may have. If the patient had such a trauma as he claims it may have caused the tumor. (Drawing a red herring across the trail by focussing attention on whether or not he had the trauma.) (It may have—one chance in a billion!)

Now I will appear for the defence and answer the same questions.

Question. Do you believe that a blow or a bump may be a cause of a bone sarcoma?

Answer. Do I understand you to say cause or be a cause? I do not believe that sarcomas are caused by blows.

Question. Why?

Answer. Billion to one argument. Disease far too rare, etc.

Question. Do you believe that the particular trauma caused this particular tumor?

Answer. No—not a chance in a thousand.

I am not a very popular expert, for these are about the extreme limits to which I could force my conscience. Fortunately for these pathetic patients, there are experts whose minds work differently.

I have come to the conclusion that there are two kinds of minds, each of which may honestly answer the same question in diametrically opposite terms, although the same facts are known to each intellect. One kind of mind will say that because the ratio of occurrence of bone sarcoma to trauma is one in one billion, it is folly to attribute a given case of sarcoma to a given trauma. The other kind of mind will conclude that although the ratio of sarcoma to trauma may be only one in a billion, if, in a thousand cases of sarcoma there was history of trauma in every one, it would prove that sarcoma was caused by trauma. Now, the first kind of mind might admit on purely theoretical grounds that sarcoma may be caused by trauma, and yet deny that one could associate any particular sarcoma to any particular trauma. Number two mind might disregard all theory in the matter, and simply assert that because every one of the thousand cases which he had ever heard of followed trauma, sarcoma must be caused by trauma. As a matter of fact less than one-half of the cases give a history of trauma.

TABLE

	Humerus		*Scapula*		*Clavicle*		*All Other Bones*		*Total*
	Trauma	*None*	*Trauma*	*None*	*Trauma*	*None*	*Trauma*	*None*	
Osteogenic Sarcomas	32	34	4	8	5	2	265	266	616
Ewing's Sarcoma	7	7	0	5	0	5	58	58	140
Myeloma	2	1	0	0	2	3	14	22	44
Metastatic Malignant	1	6	0	0	1	1	11	15	35
Benign Giant Cell Tumors	6	9	2	5	1	0	117	115	255
Unclassified Sarcomas	1	3	0	0	2	1	11	23	41
Inflammatory	4	4	0	0	2	0	11	44	65
All Other	5	4	2	14	0	0	54	53	132
Total Registered	58	68	8	32	13	12	541	596	1,328

It seems to me we can make the subject a little plainer by comparing it with the condition known as hemophilia. Hemophilia is a hereditary condition, characterized by the absence of the ferment in the blood which normally causes clotting. These patients sometimes bleed to death from a trivial cut, from a nosebleed, or from the socket from which a tooth has been pulled. Hemophilia probably occurs in about the same frequency as bone sarcoma. A cut never causes hemophilia. The condition is there from the time of the birth of the individual. One particular minor cut may cause the patient's death in the ordinary, everyday sense of "cause." If the patient bleeds to death from that particular cut, was not the cut the cause? One may

say normal persons do not bleed to death from such a cut; it was this condition, hemophilia, which was the cause of death. Now hemophilia differs from bone sarcoma in at least one very important respect, although the two conditions are comparable in the way I have stated above. In the case of bone sarcoma, the *supposition is that there may be* a control lacking, comparable to the absence of the fibrin ferment, so that the patient repairs to death, just as the hemophilia patient bleeds to death, in consequence of the trauma. The difference is that in hemophilia, any particular cut may be definitely associated with the trauma, for the condition, hemophilia, is well understood, while the condition, bone sarcoma, is still unexplained. We merely guess that it is a result of repair which goes on unchecked. There is no definite proof that this disease is due to the lack of normal repair check. It is merely the most plausible theory yet offered. There are many other theories, some of which would be entirely incompatible with a trauma being the cause of the onset.

If it is just that damages should be paid for a patient with hemophilia who bleeds to death from a trivial cut, it would seem to me fair enough, although by no means so definite a matter, to compensate a patient with bone sarcoma, provided it could be shown that there was a reasonable relation of elapsed time between the injury and the first positive X-ray. Such a decision could not be given because there was as clear a relation as in the case of hemophilia, but because it is desired to give the benefit of the doubt to the unfortunate person who was the sufferer. Practically, I do not believe that one case in a thousand of bone sarcoma is caused by the particular trauma to which the patient attributes it. From experience with little injuries of my own, those of my family and of my personal friends, and of many patients, I know that any sore spot will soon be bumped and that many a sore spot is not noticed until it is bumped. It is far easier for me to believe that the average person would be ignorant of a lump on one of his bones until it was bumped and then notice it for the first time, than it would be for me to believe that any one of the thousands of bumps that a normal person receives in a lifetime could in itself cause a tumor. There is very much less evidence of a relationship between trauma and bone sarcoma than there is between hemophilia and trauma. Trauma may cause death in a patient with hemophilia by starting bleeding which may not stop. It is hypothetical to suppose that trauma may cause death in a patient who lacks the normal check to repair, and therefore repairs to death with a bone sarcoma.

Whichever kind of mind is right about trauma being an exciting cause, there is some reason to believe that a trauma may disseminate the disease and make it worse. It might, for instance, break the fibrous capsule limiting the tumor and allow the cells to get into the veins and hence to the lungs. But in nearly all bone sarcomas this happens within a few years, at any rate. It has never been shown that those cases with traumatic histories die sooner than other cases, but it seems probable that they may. Nearly all these cases die within two years from the time when the tumor is first noticed. In other words, every one is always receiving bumps. The Registry will have to collect many more cases before an intelligent opinion can be formed as to how much a blow decreases the chances of a patient with bone sarcoma. This question is intimately bound up with the question of how much chance there is of spreading the disease by an exploratory incision, or by spontaneous fracture.

The Registry Committee, after thirteen years of effort, has collected only sixty-three cases of five-year cures of osteogenic sarcoma (all bones included), in which the diagnosis could be considered as even moderately well substantiated. Of the sixty-three, at least twenty-three had had exploratory operations prior to amputation. An exploratory operation is certainly as likely to spread the disease as is any other trauma. Since most cases of bone sarcoma, with few exceptions, die within five years, and since a large percentage of these few exceptions are known to have had exploratory operations, pathologic fracture or other trauma, it may be seen that an expert opinion stating that a certain trauma spread the disease in any individual case and hastened the patient's death, would rest on frail evidence.

There are other reasons, which we may briefly mention, leading to the conclusion that it is unlikely that trauma is an important factor in the causation of bone sarcoma.

If trauma were a cause we might expect the bones most often traumatized to show the highest proportion of malignant conditions. This is not the case, for every one interested in these questions knows that the phalanges of the fingers and toes are rarely, if ever, affected by sarcoma, yet they are the most common seats of trauma. The middle of the shin bone, which is very often traumatized, is not a frequent seat of the disease, nor is the skull. Many of the parts commonly affected are in positions where the bone is shielded from trauma by large muscles.

On the other hand, it is well known that these lesions most com-

monly occur near the ends of the bones toward which the nutrient arteries point. The Registry statistics unequivocally support this statement, which cannot easily be reconciled with the traumatic theory.

Cases of metastatic lesions from cancer, which occur in the bones, give histories of preceding trauma almost as frequently as do those who have primary sarcoma, yet no one even pretends to believe that metastatic lesions are caused by trauma; located possibly, but never caused.

The table of registered cases shows the proportion giving traumatic histories under the different diagnoses. The number having a history of trauma is unduly large, because, in both the primary and secondary lesions, every possible history of trauma is included, even if, as in some cases, a pathologic fracture was the first symptom. Thus, in only a minority of these cases, even if we accepted the largest possible figure, could the trauma have a causative relation with the tumor, so that the proponents of trauma as a cause would have a very poor argument. To my mind the large percentage giving a history of no trauma is of more significance than the large percentage alleging possible trauma. In fact, if all recorded cases gave a history of trauma, I should still think that judgment was in favor of the billion to one argument, and that the burden of proof as to causative relation still remained with those who held that such a relation existed.

REFERENCES

The Registry of Bone Sarcoma of the American College of Surgeons at 40 East Erie St., Chicago, maintains an up-to-date bibliography of the subjects considered in this chapter.

It seems to me that Kolodny's book, which is in fact an official publication of the Registry and which may be obtained from the American College of Surgeons, 40 East Erie Street, Chicago, Ill., covers the subject of malignant bone tumors in general, better than any other publication which has come to my attention. I regard it as unfortunate for progress in knowledge of this subject, that Geschickter and Copeland in a more recent book (published by the American Society for the Control of Cancer, 1932) have taken the responsibility of making radical changes in the classification which has been offered by the Registry as a standard. If the authors had registered cases illustrative of their new nomenclature, or better still had re-classified the Registry cases with publication of the Registry numbers, their book would have been more instructive to the members of the committee and to future students. However, the book is evidence of a great deal of painstaking study, and I am sure that the other members of the committee feel as I do that it deserves praise and not blame, even though it seems to ignore the nomenclature at which we had unanimously arrived after days of discussion. We welcome any addition to the world's knowledge concerning malignant tumors of bone, but we would like to have illustrative cases of new entities registered as examples, or to have our attention directed to specific instances of our errors in classifying our old cases. We know that the future will show in many of our cases that our opinions, written before the final result in each case was known, will prove to be wrong. That is a part of the game we undertook to play. Changing the rules during the game, on mere authority without registered example, seems a little childish.

Chapter XV

RARE LESIONS OF THE SHOULDER

The use of the adjective "rare," in the title of this chapter, merely signifies that the author believes that the lesions to be considered are relatively uncommon when compared to those which have already been discussed. Yet some of the basic principles of science are implied when this word is used. In common parlance, an insect, a bird or a disease may either have an identity or be unidentified and yet be called rare, but in science it must be described, named, classified, and the members of its class shown to be in a numerical minority in relation to those of similar classes, before being thus designated. A flower, common in Borneo, may be rare in London, but from a scientific point of view it cannot be so classified. Thus the word should not be used as relative to the extent of knowledge of the observer or in relation to a locality, but to scientific knowledge the world over. It implies that science recognizes the identity of the unit, its class and the relative numerical occurrence of the individuals of that class, compared to that of individuals of similar classes. An unidentified bird is not considered rare until its species has been described with sufficient accuracy to show that it is essentially different from other known species. There is a large group of obscure shoulder lesions, which probably contains many, as yet, undescribed entities. I have attempted by description and naming to subtract from this group, what I believe to be certain new but common species, but there are, I am sure, several still left to be described by other explorers. My descriptions and names will persist only until some other investigator takes the trouble to write a better book. I hope there may never be a new specialist (the omologist ?), but that in the near future, there may be some surgeon in each hospital who is *interested* in shoulders and *could* write a better book, if he could spare the time. As yet there has not been even an attempt to classify shoulder lesions, although in the index of this book a tentative nomenclature will be offered.

One might contend that the degree of advance in any branch of science may be estimated by the accuracy with which it is possible to apply the word "rare," to any of the individual units with which the science deals. By this criterion there is a woeful lack of scientific knowledge concerning the lesions which affect the human shoulder, not so much as to the available descriptions of the individual units, as in their nomenclature and classification, and in the recorded facts about their numerical incidence of occurrence. We need the names of all the clinical entities, a list of these names and a knowledge of

their relative frequency, in order to teach future medical students how to recognize and to treat at least the more common ones. Moreover, there is evidently a considerable ratio of unidentified cases, which are not yet accurately described and named, and which can only be classified under a general term as "sprains" or "strains." The difficulties presented in any attempt to classify the diseases of a given part of the body are, however, far greater than those with which the ornithologist has to deal, but in spite of these we should at least make an effort to succeed although we may be pardoned for lack of success, or of clarity, in our attempt. The difficulties are many. For instance, species seldom mix, but several lesions of the shoulder are often combined in the same individual; lesions may arise from many different causes as congenital, inflammatory, traumatic, etc.; symptoms are often given names as if they were clinical entities, *e.g.*, arthritis might be due to tuberculosis or to syphilis; we deal with a variety of anatomic structures, several of which may be damaged by a single accident; some local conditions are due to generalized disease and others are secondary effects of lesions at distant anatomic locations.

I do not know where to find a nomenclature for lesions of the shoulder. If a thousand patients with such lesions consulted in turn two different hospitals, the diagnoses, under which they would be classified, would be hopelessly at variance. Unanimity could be obtained on only a minority of the cases when classified under very general headings such as those used by the Massachusetts Industrial Accident Board in the table on p. 160, and even then would be subject to the fallibilities discussed in the adjoining text.

At present my own records of most of the shoulder cases, which I have studied over and over again as a basis for this book, are filed for my convenience as follows:

I.	Complete Rupture of the Supraspinatus	115
II.	Partial Rupture of the Supraspinatus	119
III.	Tendinitis	110
IV.	Calcified Deposits	135
V.	Villi and Bands	57
VI.	Fractures and Dislocations	102
VII.	Nerve Injuries (including root pressure)	46
VIII.	Constitutional Arthritis	29
IX.	Ruptured Biceps	18
X.	Acromio-clavicular Arthritis or Dislocation	43
XI.	Hysteria, Malingering, etc.	36
XII.	Rare Lesions	42
O.	Trivial or no Diagnosis	118
Total		970
Tumors from Registry Collection		181
		1,151

1,151 is the actual number of cases; the number of diagnoses would be more than twice this, because several lesions often occur in one case.

No other surgeon or hospital would classify these cases as I have done. Were I to attempt to rearrange them today, I should very likely shift them about to some extent as I have done many times in the past. If I considered "bursitis" an entity, it would cover half these cases. The scheme on p. 124 represents cases picked from this same file to illustrate the four most common groups and even these would be to some extent interchangeable. Many of them were seen in consultation and had already puzzled other doctors, so that they are in a way picked cases. The list, therefore, does not represent the routine shoulder lesions which would be seen by the average practitioner or general clinic. From my (perhaps prejudiced) point of view, it seems that lesions of the supraspinatus are far the most common, not always isolated, but occurring as important complications of the common sprains, dislocations, fractures and paralyses of this part of the body. The "rare" cases, with which this chapter deals, were only forty-two in number, but there must be more among the 118 which were not diagnosed.

So little interest has been taken in sore shoulders that as far as I know, no one has even attempted to list the lesions of this joint, much less to place them in their order of frequency. It is possible, however, to find such a list in a roundabout way. Since X-ray films are demanded for nearly all cases of pain in the shoulder, it would seem that a fair idea of the relative frequency of the conventional diagnoses might be obtained by the study of a consecutive series of shoulder cases in the Röntgenologic Department of some large hospital of high standing. Fortunately such a list exists in an article by King and Holmes. Their study was made to determine the reasons why the X-ray is of so little help in shoulder conditions, but the table they compiled of the cases reviewed gives some idea of the relative frequency of the different diagnoses. This table probably illustrates very well the customary diagnoses used for shoulder lesions in the United States in 1927. It is significant chiefly in showing that in two-thirds of the shoulder cases referred to a large Röntgenologic clinic (The Massachusetts General Hospital), during a year and a half, not much help in diagnosis was obtained by the use of the X-ray. Definite diagnoses were made in only 150, or one-third of all the cases. Let us consider in a general way, some of the items on this list, which we may use as a guiding thread to follow through the mazes of this somewhat irregularly arranged chapter, although we must also introduce some other descriptive names. Little space can be given to each heading, but an effort will be made to refer the reader to a few outstanding articles on the different subjects.

KING AND HOLMES' LIST

	No. of Cases	*Percentage of Cases*
Arthritis	20	4.4
Tuberculous	5	1.1
Luetic	1	0.2
Hypertrophic	3	0.6
Infectious	3	0.6
Acrom.-Clav.	8	1.7
Fractures and dislocations	97	23.3
Frac. head other than greater tuberosity	9	1.9
Frac. acromion	1	0.2
Frac. glenoid	1	0.2
Frac. gr. tuberosity (uncomplicated)	52	11.4
Frac. gr. tuberosity + relaxed capsule	1	0.2
Frac. gr. tuberosity + dislocation	8	1.7
Dislocation, uncomplicated	22	4.8
Separation ac.-clav. joint	3	0.6
Relaxed capsule	1	0.2
Old fracture scap. with spot atrophy	1	0.2
Bursitis, subacromial	13	2.8
Periarthritis	6	1.3
Postop. deformity malignancy	1	0.2
Deformity, birth palsy	2	0.4
Foreign body	2	0.4
Old traumatic	4	0.8
Osteomyelitis	2	0.4
Myositis ossificans	1	0.2
Negative	300	66.6
	450	

Under the first main heading of Arthritis there were twenty cases, but only six of these were cases to which we ordinarily apply this term. This tends to confirm what I have said in Chapter IV to the effect that a true monarticular arthritis of the shoulder is rare, and that even when the disease is generalized, the shoulder tends to escape, or if affected, to return to normal function. These authors, in alluding to the six cases, say, "They were all cases of at least fifteen years' duration, and the shoulder involvement was part of a generalized ankylosing process involving most of the major joints. We believe that the Röntgen ray has missed, and will continue to miss, most of the cases of shoulder arthritis." In my opinion, the reason that they found so few cases is that arthritis in this joint is rare.

Tuberculous Arthritis. This is surely a rare disease in the shoulder, and yet there were five cases in a large hospital in a year and a half. There are only five cases in my own series, but they comprise

the largest single group among my forty-two rare lesions. This condition gives a very definite Röntgen picture, so that every case would be picked up by the X-ray. In no other disease of the shoulder is the sulcus deepened and widened at the expense of the articular head. That it is rare may be judged by the following statistics: Townsend reported twenty-one cases out of 3,244 patients with bone and joint tuberculosis. Whitman had thirty-eight cases out of 1,833 tuberculous joints, exclusive of spines. Young, of Philadelphia, seven cases out of 5,680 tuberculous joints at the University of Pittsburgh. Of 1,900 cases of joint disease at Billroth's Clinics, the shoulder was involved in fourteen, or less than one per cent. At the Children's Hospital, Boston, Sever reports seventeen cases out of 7,474 patients with joint disease.

There is a wide age incidence of the twenty-one cases reported by Townsend, but most of the cases occurred in youth. The youngest patient was three and one-half years, while the oldest was thirty-five; ten were less than ten; seven were between ten and twenty, while four were more than twenty. In five cases the disease was secondary. The average duration of symptoms was one and three-fourths years. The longest duration was ten years; the shortest, three months. One patient, age sixty, has been reported.

Pathology. Usually two types of the disease as it affects the shoulder are described. My own experience indicates that such a distinction is artificial and is made by the clinical course rather than by the X-ray or the pathology.

(1) Caries sicca is a chronic, slowly acting form which may destroy the head of the humerus and the shoulder joint without acute inflammation. There is very little tenderness and the symptoms are not marked. With the gradual destruction of bone and constriction of the capsule, the joint is diminished in size. There is loss of motion, atrophy is extreme and ankylosis eventually results. Destruction of the head of the bone proceeds from the anatomic neck, which it deepens and widens, so that the articular area becomes smaller and smaller.

(2) Caries carnosa is a more acute fulminating type. It may complicate the first type with abscess formation. There may be communication between the joint cavity and medulla of the bone, and the latter may be filled with granulation tissue studded with tubercles. In advanced cases the axilla may be indurated and perforated by sinuses.

The common findings are dull, aching pain, sensitiveness, spasm with restriction of motion, atrophy, change in contour, loss of function, glandular enlargement, shortening of arm, abscesses, ankylosis. Pain is usually the first symptom noted. This is frequently misdiagnosed as chronic rheumatism. It may be mild or severe in character. It is sometimes referred to the deltoid tubercle or to the elbow. The discomfort is usually increased by attempted motion, especially rotation, and motions going beyond the limit of mobility of the scapula are very painful. There is frequently associated tenderness over the anterior portion of the joint. The pain is a serious symptom and is usually worse at night. The patient is unable to lie on the affected side, but this symptom is common in most diseases of the shoulder.

Reflex spasm is the most important sign. The fixation of the scapulo-humeral joint is almost absolute. Clinically, the early stages of the dry type (caries sicca) are much like the cases described under tendinitis, but the fixation is more pronounced and the deltoid is more apt to be atrophied and therefore the whole shoulder seems small. The X-ray usually shows the characteristic deepened sulcus by the time the patient seeks medical advice. The onset must be insidious usually, for I have never seen a case so early that a diagnosis could not be made by an X-ray.

If abscess forms, local heat, infiltration and swelling will be present. The humerus is apt to be shorter than on the normal side, for the disease usually begins in adolescence. There may be some glandular enlargement in the axilla. If abscess forms, it may point near the coracoid or make its way down the shaft of the bone and appear on the surface anywhere in its upper third or half. The subacromial bursa may fill with pus. Sometimes perforation occurs posteriorly beneath the acromion. *Abscess about the shoulder is more apt to be due to this than to any other cause.* Mondan and Audry reported abscesses in twenty-seven out of thirty-three patients. Townsend reports abscesses in all his cases. Sinuses remain and drain for long periods. Loss of function is due to muscular spasm in early cases and to ankylosis later. Ankylosis, partial or complete, is the usual termination, and thus the range of motion which may be attained depends entirely on that of the scapula.

Prognosis has to be considered constitutionally and locally, for there is frequently an associated pulmonary involvement. The patient may die of miliary tuberculosis or pulmonary complications. In children the outcome is usually more favorable than in adults. In

adults, as far as the joint is concerned, there is little hope of recovery without ankylosis unless operation is done. The duration of the active stage is from two to five years.

Treatment. This is general and local. The former is of great importance, as in all forms of tuberculous disease. It is usual to immobilize the joint by the use of a plaster cast. The arm should be abducted and brought forward slightly and somewhat externally rotated. Since the joint tends to become ankylosed in internal rotation and adduction, and as the scapula is very evasive in a plaster cast, over-correction of the desired position should be aimed at. When abscesses form they may have to be drained. Necrotic bone may require removal, but even such a minor operation is accompanied with the danger of causing miliary tuberculosis. If a localized area is found by X-ray, it may be removed, but, in my opinion, if any operation is done in adult cases, radical excision of the articular head with preservation of the short rotators is indicated. When excision is done and the tissues are well carbolized the disease tends to heal, even if it is not all removed. Other surgeons advocate ankylosis. We greatly need a large collection of postoperative statistics to determine the relative values of these two operations. At present ankylosis is more popular than excision. In children, interference with the humeral epiphysis may mean cessation of growth of the arm, and conservative treatment should be given a long and fair trial. In the acute stage traction sometimes has to be applied to relieve pain.

Syphilis in the shoulder joint must be very rare. The following remarks are made entirely from a perusal of the literature, for I have never recognized a case of this condition in the shoulder. The statements apply to the joints in general rather than to the shoulder in particular. That the joints are affected in this disease was recognized by Peter Martyr as early as 1498, but it was first accurately described by Ricket in 1853. The manifestations have been classified into arthralgia, acute synovitis, chronic synovitis, and gummatous affections. There may also be periostitis, osteitis, osteomyelitis and bursitis. The most outstanding symptom is pain, which is usually nocturnal. It may be dull, aching or sharp. It is often relieved by exercise.

Arthralgia often occurs in the primary stage. The onset may occur as early as six days after appearance of the primary sore, but usually comes much later than this. There is no demonstrable pathology. *Acute synovitis* occurs within the first or second year. The

joint is swollen and hot, but limited motion may be painless. The condition lasts one to two weeks or may become chronic. In *chronic synovitis* there is marked swelling with effusion and fluctuation, but there is little pain. Ankylosis may result. Late in the disease gummatous affections are found. In *periostitis* there is thickening of the periosteum as shown by X-ray. There may be a localized tender area. In *syphilitic osteitis* there is rarefaction of the bone. In *osteomyclitis* the symptoms depend upon the severity of the involvement. The X-ray appearances are fairly characteristic. The last two conditions are usually seen between the second and third years of the disease. *Bursitis* may be primary or secondary. This condition has been discussed by Churchman. He does not mention the subacromial bursa. I have never recognized a case of syphilitic bursitis in the shoulder, but I have seen on the superior surface of the acromion two cases of syphilitic ulcer, which presumably involved the subcutaneous bursa. It is very unusual to find any manifestation of this disease in the shoulder joint or in the structures near it. In a series of sixty-three cases of syphilitic arthritis reported by Baetz, and in eighty-three cases reported by Whitney and Baldwin, the shoulder is not mentioned. My assistant could find only four cases of osteitis mentioned in the literature. One was reported by Brickner in a man of thirty-two, who had pain and disability of his left shoulder. The X-ray showed fine lines indicating periostitis about the surgical neck and also beginning rarefaction, indicating necrosis of the greater tubercle. Another was reported by Wile and Senear, who state that the shoulders were definitely swollen. Meriel saw a juxta-epiphyseal fracture of the upper end of the humerus with gumma formation. Fred H. Albee reported a case of gumma of the shoulder which showed an area of increased radiability with disintegration at its center and bone sclerosis below this area. There was a periostitis about the surgical neck. A good illustration is given in the article.

Diagnosis is made on the basis of history, Wassermann, other signs of syphilis, and by X-ray. Luetic lesions occur in the epiphyseal end of the diaphysis and this distinguishes them from tuberculosis which arises in the epiphysis. Swelling of the soft parts is more common in tuberculosis, while in lues there is thickening of the bone. In tuberculosis there is a tendency to sinus formation. The outlines of the bone entering into the articulation are distinct in lues in contrast to the deep sulcus in tuberculosis.

Treatment is antiluetic and prognosis is good with proper treatment.

In my opinion, the chief danger of error in diagnosis in such cases is in crediting a pain in the shoulder to the disease, when in reality the fact may be that a syphilitic patient has a sore shoulder from some other cause. The lesions connected with the supraspinatus tendon described in this book are so common that patients with all sorts of general diseases may suffer from them. Since lues and its treatment tend to weaken the collagenous parts of the body, we might expect weak tendons in such patients.

Closely allied to syphilitic lesions of the joints are the neuropathic conditions, and while the majority of these are secondarily due to lues in cases of tabes dorsalis, there are other spinal lesions which produce similar arthritic changes. Although no neuropathic lesions are mentioned in King and Holmes' list, we may give them brief consideration. There were only two in my series, one due to tabes and the other to syringomyelia.

Charcot's Joint

In his original article in 1868, Charcot writes: "The disorder generally shows itself at a determinate epoch of the ataxia, its appearance coincides in many cases with the setting in of the motor incoördination without any appreciable cause. We may see, between one day and the next, the development of a general and often enormous tumefaction of the member, most commonly without any pain whatever or any febrile reaction; . . . a more or less considerable swelling of the joint remains owing to the formation of hydrarthus; and sometimes to the accumulation of liquid in the periarticular serous bursæ also. . . . One or two weeks after invasion, sometimes much sooner, the existence of more or less creaking sounds may be noted betraying the alteration of the articular surfaces, which at this period is already profound. . . . Besides the wearing down of the articular surfaces you notice here the presence of foreign bodies, of bony stalactites. . . . The articular affection in question is itself, also, the expression of trophic disorders directly dependent on the lesion of the spinal nerve center."

Little more can be said now, but contrary to this early description by Charcot, onset is often gradual. Since the joint is insensitive the first symptom is usually merely swelling, without the concomitant signs of inflammation. This is followed by an abnormal range of motion, so that deformity and instability result. There is practically no local pain or sensitiveness, but pain may result from distention. Subluxation is common. As the weakness and instability progress nature makes feeble attempts at repair with irregular for-

mation of fibrous tissue, cartilage, and even bone in the capsule. Thus the joint as a whole becomes enlarged and yet there is marked laxity of the ligaments and painless, abnormal mobility.

Involvement of joints is seen in or as a precursor of two to ten per cent of cases of tabes dorsalis. It may take the form of a simple chronic synovitis or a destructive osteoarthritis. The latter form, or Charcot's joint, is seen more often in the lower extremity and may be multiple. It rarely affects the shoulder joint. In 155 cases reported by Flatlow, quoted by Whitman, twenty-seven involved the shoulder. In 217 cases of Chipault this joint was not involved. In eighty-eight cases Wile and Butler found four in the shoulder. In 167 cases reported by Sutherland from the Mayo Clinic the shoulder was involved twice.

This type of joint is not limited to tabes, for it or a similar condition is seen in syringomyelia, and very rarely in psoriasis, leprosy, spina bifida, and in cases of direct injury to or after cutting of dorsal roots. The spinal lesion is probably the basic cause, while trauma may precipitate the appearance of the local lesion.

Carman states that in most cases the X-ray alone is sufficient for diagnosis. He makes the following points: (1) Atrophy of articular cartilages. (2) Irregular destruction of bone, often associated with (3) irregular hyperplasia in the same joint, (4) detached bone masses and detritus, (5) translucent areas.

The presence of painless lateral mobility in any joint should suggest this diagnosis. Reliance should not be placed on the Wassermann reaction, which is usually negative both in the blood and cerebrospinal fluid.

Prognosis. This is not good, but if the tabetic processes can be halted, the change in the joint may also stop. The bone may regenerate somewhat and there may be some shrinking of the capsule. Fluid may be absorbed.

Treatment. The treatment is general and local. An effort is made to control the tabes. A support to prevent progressive distortion should be used. An airplane splint or elastic shoulder cap may be employed. Tapping of the joint for fluid may be done.

Syringomyelia is usually considered a separate disease entity characterized by formation of cavities in the spinal cord, but a similar condition may result from degeneration of a central glioma. It was first described by Charles Estienne Stephanas in 1546. Schlesinger collected 260 cases for his book, *Die Syringomelie*, published in 1902.

The disease is progressive. The characteristic symptoms depend upon the extent and area of the cord involved. Dissociated anæsthesia, loss of pain and temperature sense and retention of tactile sense are most important. Trophic disturbances, with involvement of skin and joints, are common. Joint affections are a very common complication. Schlesinger collected 105 cases with joint involvement. He estimates that this occurs in ten per cent of the cases. Unlike Charcot's disease, in which the knee is most often involved, in syringomyelia the shoulder is most commonly affected. In a series of ninety-seven, the order given by Schlesinger was: Shoulder twenty-nine, elbow twenty-four, wrist eighteen, hip four, knee seven, foot seven, other joints eight. The lesion resulting in the joint is very similar to that in Charcot's. Progressive muscular atrophy with paralysis is usually present. Deep X-ray treatment of the spinal lesion is often successful in checking the progress of this disease.

Hygroma of the Subacromial Bursa. The surgical literature of the last century occasionally mentions the term Hygroma, meaning a collection of fluid in an enlarged bursa or joint. This condition was more often described in connection with other regions, but occasionally hygromas of the shoulder were mentioned. The subject can best be discussed in connection with the following case:

Mrs. H. Massachusetts General Hospital No. 150673 E. S., Dec. 15, 1906. The patient was a stout woman of 51, who gave the history of having always been well. She was referred from the Dispensary with a diagnosis of a fracture of the head of the humerus, but the X-ray showed that there was no fracture. She stated that six years before she had strained her arm in lifting, and that since then it had been somewhat stiff and sore and painful on use. Eighteen months before entrance after carrying a heavy bag she noticed that the arm had become much swollen. After a time the swelling disappeared. Ten days before entrance she fell forward down two steps, striking on her head and shoulders. The arm again became swollen and painful, particularly around the shoulder joint. Since that time the swelling had increased and at the time of entrance it was very conspicuous, suggesting a tumor of the head of the humerus, but it was fluctuant. The record states: "Movements of the arm free and not painful; soft crepitus near head of bone; clavicle palpable over top of swelling and apparently loose at its outer end." The operation was performed by me and the notes were by the house officer. An incision was made over the subacromial bursa and it was found to be "enormously dilated to the size of a large grapefruit. Inside were numerous cartilaginous bodies in size from (word illegible) to a pea, attached loosely to the walls. The sac contained yellowish mucous fluid, semi-viscid. The head of the bone was much roughened and covered with bits of cartilaginous bodies similar to those found on the walls of the sac. The normal topography of the bones about the joint was destroyed by the pressure of the

fluid in the sac. The clavicle was lifted two inches from the acromial-clavicular attachment. The glenoid cavity was obliterated." I excised the head of the humerus and the redundant bursal sac. There was a mild staphylococcus infection. The patient was discharged from the hospital in six weeks with a small sinus. Her pain was relieved and she was intensely grateful, but I doubt if she ever had a very satisfactory arm. Unfortunately I was unable to follow up the case. Pathologic report No. 612-54. "The head of the humerus almost devoid of cartilage, to which several irregular pieces of capsule were adherent; also two pieces from the joint 1.5 cm. in length. Diagnosis, chronic synovitis."

This was undoubtedly a case which the older writers would have called a hygroma; even now I cannot state positively what the condition was, although I fully believe it was the late result of such a case as that described on page 389. The short rotators had been torn from the head of the humerus at the time of the accident six years before, leaving the head of the bone free under the deltoid; chronic synovitis, effusion and slow distention of the bursa and joint followed. The woman had continued to do her housework for six years in spite of the disability. The case mentioned on p. 389 very likely will eventually have a similar condition. In both cases movement of the joint was not painful because the short rotators had retracted to a fixed position on the glenoid.

Since I operated on the above case I have seen several others of a similar character in which there was a smaller quantity of fluid. Any patient with ruptured supraspinatus, who is able to continue to use his arm, may develop a large amount of fluid. It is my personal belief that most of the cases formerly diagnosed as hygroma were these extreme cases of accumulation of fluid due to the use of a shoulder joint in which the bursal floor was in communication with the true joint. Others may have been Charcot's joints.

My interpretation of this case may be incorrect. The trouble may have been a Charcot's joint. The record gives no reference whatever to the question of syphilis, but there was nothing in the physical examination to indicate it. The case was at the time diagnosed a Charcot's joint, and this shows that the question of syphilis was entered into, but that there was no positive finding in the history or physical examination. In those days we did not use the Wassermann test.

I feel very confident that most similar cases are primarily due to ruptures of the tendon. The case described in the chapter on Arthritis showed similar changes in both shoulders, but of less extent. Several of the cases Dr. Akerson has found at autopsy showed similar lesions.

Treatment. In the past, incision of the bursæ and evacuation of the fluid and necrotic villi was recommended. Withdrawal of the fluid, followed by the injection of irritants as in hydrops of the joints, was also done. The treatment which would be recommended now would be ankylosis of the joint.

In 1922, Aldo Avoni was able to find only thirty cases of hygroma in the literature. Churchman ably discusses the subject in relation to syphilis. H. T. Jones reviews the literature on Cystic Bursal Hygromas.

Progressive muscular atrophy, as the name implies, is characterized by progressive wasting of the muscles. It rarely begins before the twentieth year. Its onset is very slow and months or years may elapse before symptoms are present. It is dependent upon a chronic degeneration of the cells of the anterior horns of the spinal cord. The small muscles of the hands are usually involved first, although the disease may begin in the muscles of the shoulder girdle. It is usually bilateral.

The objective signs are: atrophy, fibrillary twitching, paralysis, reaction of degeneration, decreased or absent reflexes, deformity and changed attitude. Subjective symptoms are weakness, impaired function and clumsiness. There is no pain, and it is never accompanied by loss of sensation. In diagnosis we must consider:

(1) In syringomyelia there is sensory dissociation, trophic changes and unilateral distribution.

(2) In anterior poliomyelitis there is rapid onset, loss of power before atrophy.

(3) Amyotrophic lateral sclerosis shows spasticity with exaggerated reflexes.

(4) In lead palsy the lead line, colic and extensor paralysis occur.

(5) In caries of the vertebræ there are sensory symptoms, pain, positive X-ray findings.

It almost seems superfluous to consider this condition in a book on the shoulder, but practically I have several times seen cases in which the atrophy of the shoulder muscles from this cause has led to the conclusion that there was a local lesion in the shoulder joint. It is not uncommon to find some fibrillary twitching in the muscles about a sore shoulder, but this sign should always suggest the diagnosis of progressive muscular atrophy. We may make this diagnosis if the twitching and atrophy are not accompanied by pain or restriction of motion, or loss of sensation, especially if both shoulders are affected.

We may return now to the table of King and Holmes. We have considered the different forms of arthritis there listed as well as the forms of arthritis due to changes in the cord, and have already discussed in other parts of the book acromio-clavicular arthritis. It can be seen from the list, that King and Holmes found that the X-ray was chiefly useful in fractures and dislocations, for nearly two-thirds of the 150 cases in which the X-ray was useful were of this nature. This portion of the subject has been referred to in Chapters IX and X. Attention is particularly called to their figures on uncomplicated fractures of the tuberosity and on uncomplicated dislocation, which seem so contradictory to my remarks on the frequency with which these lesions are found in combination. The röntgenologist sees these cases after the reduction has taken place either spontaneously or as a first aid measure. We have also considered the diagnoses of subacromial bursitis and periarthritis. Of the last ten diagnoses on their list there are three which it seems worth while to discuss.

Osteomyelitis may be disposed of very briefly. While it may occur about the shoulder, its occurrence must be very rare, for in spite of my interest in the shoulder for many years, I have never seen a case of osteomyelitis of the upper end of the humerus in an adult. I have seen the condition in children who had osteomyelitis also in other bones. I do not refer, of course, in this connection to bone infection following surgical operations. I refer to inflammation of bone caused by blood-borne germs which produce suppuration. My experience with bone sarcoma has been, of course, unusual, so that it is perhaps not fair for me to draw comparisons between this disease and osteomyelitis, but I cannot help saying that when a surgeon sees a bony lesion of the upper end of the humerus I believe he should think of bone sarcoma first and osteomyelitis second, for while sarcoma is rare, I do not believe it is as rare as osteomyelitis in this region of the body.

Myositis Ossificans is to be thought of in the diagnosis of shoulder conditions, although it is very unusual for it to involve the shoulder muscles. In the traumatic variety the shoulder is rarely involved. The writer has never seen a case. In 127 cases reported by Strauss the shoulder is not mentioned. The most common sites are, in order of frequency: (1) Brachialis, (2) quadriceps femoris, (3) adductor longus, and (4) biceps brachii.

In 233 cases from the German military service, the brachialis and quadriceps were the only muscles involved in all except three cases. In eighty-six cases analyzed by Binnie, the "shoulder" was mentioned once, deltoid once and pectoralis major twice.

The progressive type (the ossified man of the circus) has been studied thoroughly by Rosenstirn. He reviews the literature and presents 115 cases extending back to 1692. Ossification of the supraspinatus was mentioned four times.

Relaxed Capsule. The term relaxed capsule is perhaps inappropriate as a diagnosis, because the causes of the condition may be of several different kinds. The term simply means a condition in which the head of the humerus can be pulled downward from the glenoid to an abnormal degree. Since the capsule of the joint is essentially made up by the short rotators, paralysis of these muscles permits a sagging of the humerus. We may expect, therefore, that the X-ray will show a large gap between the humerus and the glenoid if traction is exerted on the arm when these muscles are paralyzed or stretched. If the X-ray is taken while traction is being made, the gap may be one or two inches in extent. A similar condition occurs when the supraspinatus, together with other short rotators, has been torn and fluid has accumulated in the joint and bursa. Perhaps the condition may also appear in old cases of habitual dislocation. As a rule, however, the X-ray appearance of relaxed capsule means paralysis of the short rotators or deltoid. Investigation of this sign is much needed. It is a troublesome thing to take films of a shoulder joint and then repeat the same films with traction on the arm. In a normal person the joint surfaces may be separated to a certain extent by traction, and the degree of separation must vary with the amount of fluid present which may fill the gap as suction is made. We must remember too that the joint cartilages do not show in the picture. To obtain a correct estimate the direction of the rays must be parallel with the glenoid surface.

Cotton and Hammond have drawn attention to this condition. Cotton believes that the persistent dragging of the swollen arm, or the arm plus heavy splinting, gradually stretches the deltoid and brings the head of the humerus lower and lower in relation to the glenoid. Hammond shares the same views. Cotton makes an observation which is quite at variance with my views and those of Stevens, described in Chapter XI. He says, "Deltoid paralysis assumed to be from circumflex damage I do not see; all the cases seem to be either brachial plexus lesions or the sort of thing here described." I cannot agree with this statement, for I believe that circumflex damage is very common as a complication of dislocation. However, I am sure Cotton and I would agree that in all cases of fracture of the humerus with paralysis of the deltoid, it is wise to support the arm

so that the weight will not sag on the deltoid or on the short rotators. I should interpret Case 3 in Hammond's paper, and also one of Cotton's cases (Fig. 6) to be clearly cases of comminuted fracture of the upper end of the humerus. In all fractures of this kind, and in many of the minor ones, there is swelling and edema of all the tissues in the neighborhood, and this edema or joint fluid tends to permit the glenoid and head of the humerus to separate.

In some of the cases of circumflex paralysis described previously, after the arm has been put up in elevation, it may take twenty-four hours or more before the edema is forced out of the subacromial space to make room for the head of the humerus. The relief which so constantly occurs in these cases under this treatment is partly due to the displacement of this edema as well as to relieving the stretched condition of the deltoid.

In making the diagnosis of a relaxed capsule, one must always bear in mind that in order to determine the size of a gap between the head of the humerus and the acromion, we must be sure that the path of the X-ray is parallel with the plane of the acromion, so that the latter will be shown in profile. If the path of the X-ray is not in this line, the shadow of the acromion appears to fill up the gap. Films taken of the shoulder when the plate is anterior are much more likely to show the acromion in profile than are antero-posterior views. There is quite a large gap in normal cases. "Dropping shoulder" is a better term than "relaxed capsule," for the relaxation is in the muscles.

Referred Pain. Among their three hundred cases which were negative, King and Holmes suggest that perhaps there were many in which referred pain caused the symptoms. They list as possible causes the following:

1. Cardiovascular lesions, angina, aortitis, aneurysm, coronary sclerosis.
2. Pleural, pulmonary and mediastinal. Pleurisy, pneumonia, intrathoracic new growth, pneumothorax.
3. Abdominal lesions, gastritis, gastric ulcer or carcinoma, gall-bladder disease, hepatic or subphrenic abscess, pneumoperitoneum, ascites, rupture of ectopic pregnancy.
4. Neurological lesions, hemiplegia, new growth, cervical Pott's disease, cervical arthritis, cervical ribs, herpes.

They comment:

"Let it be remembered that these lesions can not only cause pain referred to the shoulder, but when severe will cause limitation in motion and localized tenderness, followed by atrophy of the muscles about the shoulder, moderate capsular relaxation and continued or increasing disability."

I am unable to agree with these authors as to the statements in the last paragraph. I have never observed cases of referred pain in their first three groups, which showed limitation of motion or true localized tenderness in the scapulo-humeral joint. Hyperalgesia to pressure on the trapezius or to pinching of the skin over the shoulder there may be, but not deep, local tenderness over the joint. Atrophy of the muscles and bones may perhaps be present after long-continued pain and disuse on account of pain, but only in a few of the above lesions, especially those associated with lesions in paragraph 4, such as cervical rib and cervical arthritis, which cause pressure on the nerve roots.

Shoulder pain, as has long been known, may appear in some cases of gallstones and of other lesions of the liver, and students for generations have been taught that this pain is a typical example of "Referred Pain." Cope lists the following conditions in which he has found shoulder pain to be of diagnostic value. (1) Liver abscess, (2) perforated gastric or duodenal ulcer, (3) subphrenic abscess, (4) cholecystitis with adjacent peritonitis, (5) perforation of the gall bladder, (6) splenic infarct, (7) spontaneous rupture of spleen, (8) acute pancreatitis, (9) appendicitis, (10) ruptured ectopic gestation, (11) dilated stomach, (12) actinomycosis of thoraco-diaphragmatic junction, (13) diaphragmatic pleurisy, (14) basal pulmonary infarct, (15) pericarditis.

It has been well established by surgeons since the use of local anæsthesia was introduced, that the viscera are as a whole incapable of sending sensory impulses to the brain in response to such injuries as cutting and bruising, although distention of their cavities may register on the brain local distress and even agony. Injuries to the parietal layer of the peritoneum or pleura, on the other hand, can usually be localized almost as accurately by the patient as if they were on the skin. On evolutionary grounds it seems probable that all the external parts of the body developed accurate protective sensitivity, while the vital organs did not, because injury to them from the claws of other animals meant death. If a beaten animal, even without a whole skin or abdominal wall, escaped, he might live to breed another day, but if his heart, intestines, lungs or liver were injured, he could leave no protective reflexes to his posterity. Therefore, nature has not developed useless sensory nerves in these organs to give us needless pain from local injuries. Yet these very organs are provided with the mechanism to give us pain when they are functionally abused. Distention from gluttony or from poisonous foods

may give us protective abdominal warnings. So may over-exertion cause a warning distress in the heart or lungs. Yet the heart and lungs, when torn or scratched, give us no distress. It is almost a rule that the better protected a vital organ is, the less will be its sensitiveness to touch stimuli. Capps has shown that the heart cannot convey to the brain any painful or localizing sensation when touched with a wire, nor can the inside of the pericardium which surrounds it, but the parietal pleura is sensitive and the patient can localize the point at which it has been touched.

Capps' work has extended over many years and has only recently (1932) been summarized in a little volume which will stand for a long time as a first-rate example of scientific clinical observation, experiment, logic and presentation. He has been able, by using the opportunities which have occurred in his practice in cases where it was necessary to tap the peritoneum, pericardium and pleuræ for therapeutic purposes, to ascertain quite definitely the sensitivity of most of the visceral organs, and not only to map out those areas capable of localizing painful stimuli, but also to determine certain areas, which, when touched, refer the sensation to other distant areas. His work has made the rather vague subject of referred pain, and particularly that of referred shoulder pain, far more succinct and intelligible than it was formerly, although many other investigators had prepared the way.

His essential contribution was made by the coöperation of his patients, who told him their sensations as he touched various spots on the heart or on the diaphragm with his probe when he was obliged to tap the peritoneum, pericardium or pleuræ to withdraw fluid. In this way it was found that when he touched from above or below the central area of the diaphragm or the outer lateral portions of the pericardium, shoulder pain was always experienced by the patient. He proved, as the anatomists already had suspected, that these areas were supplied by the phrenic nerve which arises in the fourth and fifth cervical segments, which also gives origin to the nerves which furnish the sensitivity to the skin of the upper posterior part of the shoulder (the trapezius ridge), to which area the pain sensation in these patients was invariably referred.

His experiments, therefore, confirmed the ideas previously worked out by others, which may be expressed in a general way thus: The afferent nerves from the viscera often terminate in the cord in close association with the afferent nerves from certain skin areas. When a powerful sensory stimulus is received from the viscera as a result of

disease, it may be projected to the peripheral distribution of those nerve fibers which terminate in the same segment of the spinal cord. Head suggests that this is an example of a general law; *i.e.*, when a painful stimulus is applied to a part of low sensibility in close connection in the cord with a part of much greater sensibility, the pain produced is felt in the part of higher sensibility.

Hypersensitivity, or hyperalgesia of the skin area, probably arises because repeated abnormal impulses from the diseased viscus irritate its particular region of the spinal cord, and this point becomes an "irritable focus." The intensity of impulses entering this area of the cord from areas not diseased becomes increased, and stimuli applied to the sensitive areas of the body surface which would normally arouse only slight discomfort, now become endowed with distinctly painful properties, *i.e.*, hyperalgesia is present in the corresponding segment of the body wall. Pain may or may not be felt over the organ involved.

Capps defines referred pain as follows:

1. It is often remote from the site of irritation. (This is especially well demonstrated in phrenic shoulder pain.)
2. It follows lines on the skin of spinal segmentation rather than the course of peripheral nerves.
3. It is usually associated with cutaneous hyperesthesia and tenderness to pressure (over the referred area).
4. Often the pain fails to involve a whole segmental area of skin, but finds expression in one or more points of maximal tenderness and spontaneous pain.

The phrenic shoulder pain varies in intensity and quality. It may shoot down over the outer aspect of the shoulder along the distribution of the 5th cervical nerve. Cope says, "Usually it has the quality of an ache and is regarded by the patient and often by the doctor as a rheumatic pain. Sometimes it is very sharp and stabbing or it may feel as if a nail were being driven into the spot. Some patients may say that they have no pain, but that there is a sensation of stiffness in the affected part." The pain is in the skin or superficial tissue and is always associated with hyperalgesia. It is said that there may be muscle spasm. I have never observed a case where this was present. In Capps' experiments the area affected was often small and very definite. He does not speak of spasm.

Practically it is very seldom that confusion arises in the diagnosis between actual lesions of the shoulder tissue and lesions causing shoulder pain by way of the phrenic. Usually much more pronounced

symptoms indicate the serious lesions which cause it. However, the possibility should be borne in mind in all cases of aching pain in or near the trapezius ridge accompanied by hyperalgesia. Such shoulder pain is apt to be enhanced if the patient takes a deep breath. It is also possible that a mistake might occur in the reverse way and a true shoulder lesion be ignored in a patient suspected of or found to have a visceral lesion, for shoulder lesions are very common and may affect a patient with gallstones as well as one without them.

Referred Pain from Pressure on the Roots of Cervical Nerves. Confusion in diagnosis is much more likely to arise in the fourth division of the causes listed by King and Holmes, than it is in the first three divisions. The reader who wishes details on the first three divisions is referred to Capps's book, for the cause of the pain in all the lesions mentioned is the phrenic pain we have just considered. The kinds of pain listed in the fourth division are quite different in character. The explanation usually accepted in such cases is that pressure on the nerve roots of the brachial plexus may give pain associated with the sensory areas supplied by the plexus. Any lesion which causes a swelling of the tissues in the spinal canal or in the bony gutters or in the part of the first rib which the nerves cross, may cause this kind of pain. The diagnosis, therefore, becomes complicated, for we must consider all the lesions which may affect the nerves themselves or the vertebræ between which they find their points of exit. For instance, in my opinion a very common cause of shoulder pain is hypertrophic arthritis of the cervical spine. I frequently make this diagnosis.

Bassoe says, "I have become more and more convinced that a great many cases of painful shoulders and arms in middle-aged people are caused by osteoarthritis of the neck and 'funiculitis' rather than by arthritis of the shoulder or toxic neuritis or neuralgia. I have repeatedly found röntgenologic evidence of such arthritis and seen relief from it by stretching the neck, which automatically relieves the pressure on the funiculi."

Cervical ribs and enlarged transverse processes on the 7th cervical vertebra are also, I am convinced, common causes of shoulder pain. Other rarer conditions, such as new growths or tuberculosis of the cervical vertebræ, produce the pain after the same manner, *i.e.*, by local pressure of the nerve roots. It seems to me that this is to be considered "referred pain" rather than pain from direct pressure. Certainly the sensory disturbance felt in the toe of an amputated leg, but caused by a neuroma in the stump, must be of a some-

what similar character, so far as the mechanism of cord and brain is concerned. In the same way a cervical rib or an hypertrophic arthritis may produce pain felt in the hand. I am very confident that most of these lesions which press on the nerve roots cause a vague pain of segmental type rather than pain of a type associated with peripheral nerve distribution. It therefore seems very unlikely that restriction or atrophy could accompany this type of pain, except secondarily from disuse on account of the pain.

We may speculate a little more in regard to the mechanism of shoulder pain caused by direct pressure on the nerve roots. Each of these roots contains efferent fibers (motor), afferent fibers (sensory) and sympathetic fibers. Besides these we may assume local nerve endings, because local pressure on a nerve trunk causes distinct local pain which the brain can accurately locate. I know of no positive evidence for or against such an assumption.

Granted that pressure on the afferent fibers would only give a false sense of pain in the shoulder, which could cause no changes such as spasm or atrophy or restriction, could pressure on the efferent motor fibers produce such changes? Two ways in which this might happen are obvious. *First*, the local pressure on the motor fibers might persistently stimulate the muscles to contract. Spasm would occur which would be followed by exhaustion and atrophy, and thus by restriction. Spasm itself is painful. *Second*, local inflammation within the fibrous nerve sheath of the trunk might be first caused by the pressure. A local lesion would be produced and the edema caused within the trunk gradually destroy or weaken the true motor nerve fibers, so that impulses could not travel normally to the muscles, which in consequence would atrophy. We know that when we cut the nerves which give the motor supply to a muscle, the muscle first becomes flaccid and then atrophic and later contracted. Perhaps tension confined within the fibrous nerve trunk may have the same effect.

Another way for pressure on the nerve roots to cause actual physical changes in the muscles and joints supplied by those nerves, would be by interference with the sympathetic fibers which lie in the nerve roots and are in control of expansion and contraction of, not only the main artery of the limb, but of each of its branches. Obviously, if pressure on these sympathetic fibers could cause undue interference either with the contraction or expansion of the arteries of the limb, physical changes might be produced. We know, for instance, that ischæmia and even gangrene occur sometimes in cases of cervical rib, but perhaps this is only when the artery is also involved.

We know also that removal of the sympathetic fibers about an artery causes hyperæmia below.

A fourth hypothesis might be to suppose that the pressure first stimulates the local afferent protective nerve endings in the root itself, and this centripetal impulse causes a command to return from the brain to produce a constant local contraction of the muscles thus leading to spasm, atrophy and restriction. In this case all the muscles supplied by that trunk would be affected.

We know from sad experience that pressure produced by tight bandages may lead to ischæmia in the muscles of the forearm (Volkmann's contracture), although we do not know whether this occurs through interference with the sympathetic or motor fibers. Could the much slighter and much slower pressure from cervical ribs act in the same way?

On the other hand, we know that bony tumors may grow to large size, obviously pressing large nerve trunks from their normal paths, without producing pain or paralysis in the muscles which these nerves supply. Sometimes such changes take place; sometimes they do not. I have read the detailed histories of many cases of bone sarcoma and do not recall such symptoms, except in a few cases of giant cell tumor of the upper end of the fibula, where mild symptoms of toe drop and tingling in the peroneal nerve area occurred. The pressure in these cases must have been excessive. I have seen chondromatous tumors, which have developed to enormous size, with no nerve symptoms, although nerve trunks were pushed far from their normal places and held in canals in the tumors.

It therefore seems to me that the nerve symptoms caused by pressure in the cases of cervical rib and hypertrophic cervical arthritis must have some additional explanation besides mere irritative pressure. Perhaps there is some strangulation of the nerve root as well. As a matter of fact, we do not understand the exact reasons for the shoulder and arm symptoms in these cases, although we call them "pressure neuritis." Many physicians are satisfied to treat for "neuritis" any painful arm without even taking the trouble to use the X-ray. Brachial neuritis is for them a satisfactory diagnosis, as if there were such a pathologic entity.

Cervical ribs, according to Keen, were mentioned by Galen, but our real knowledge of the subject dates back to Hunauld in 1742. The presence of a cervical rib is no longer considered a rarity. It may occur in one to two per cent of all subjects, but in probably ninety per cent of these it causes no symptoms. Eighty per cent or

more of cervical ribs are bilateral, but ninety-five per cent of the cases where pain results give unilateral symptoms. About seventy per cent of the clinical cases occur in females.

Cervical ribs can be divided into four classes: (1) a node that does not extend beyond the transverse process; (2) a blunt projection of bone four to five centimeters long; (3) ribs long enough to articulate with the first rib or be attached to the sternum with a ligament; (4) complete rib with vertebral origin and costosternal cartilage.

Symptoms may be divided into local, vascular, neurologic and muscular. A bony tumor can be felt in the supraclavicular region. Pressure on it causes pain. Bruit may be present where the artery crosses it. There may be disturbance of blood supply to the hand, usually causing only pallor, but sometimes gangrene of the fingers. It is disputed as to whether this is due to pressure on the brachial plexus or to pressure on the axillary artery. The neurologic symptoms depend somewhat upon the part of the plexus affected, but the pain is apt to be of segmental type. Pain is often severe and aggravated by certain movements, or it may be dull, continuous, or only a heavy feeling. The patient may have numbness, tingling, neuralgic pains, and weakness. A sensation of fatigue in the arm is very common. Muscular atrophy, loss of power or restriction are seen only in very severe prolonged cases. Sir Harold Stiles has pointed out that pressure of the normal first rib may also cause similar symptoms which may be relieved by removal of the rib. Sometimes a short cervical rib may have cartilaginous or tendinous prolongations that provoke symptoms. These may be attached to the first rib or to the dome of the pleura.

In my personal experience I cannot recall seeing any cases of shoulder pain caused by cervical ribs which were associated with a decided atrophy of the shoulder muscles or stiffness in the joint. I have seen a few very severe cases in which there was atrophy of the hand muscles. The pain was so severe as to demand excision of the rib. The type of case in which I personally make the diagnosis, and I have done this a number of times before receiving confirmation from the X-ray, is characterized by the vagueness and yet persistency of intermittent pain without definite disturbance along any particular peripheral nerve; without restriction or atrophy, and usually relieved by hunching the shoulder or holding the arm up so that the arch formed by the clavicle and scapula is raised. At the same time there will be found a slight enlargement in the region of

the cervical rib, and a slight amount of localized tenderness as compared with the other side. These signs are exactly similar to the signs which one finds in the next condition to be considered, cervical arthritis. I cannot differentiate between the two without the help of the X-ray, and even then sometimes am inclined to compromise.

Cervical Hypertrophic Arthritis. While one must rely on the X-ray for demonstration of this condition in most cases, we must always remember that the swelling of the soft parts adjacent to the hypertrophic ridges may be of considerable extent without showing in the film. Rigidity in the muscles of the neck must be present to some extent if this diagnosis is to be made. Lateral motion of the head and neck will not be symmetrical, and will cause local pain or increase the pain in the shoulders. As pointed out by Bassoe, there may be relief of the pain when the neck is passively stretched. The pain may be relieved by a Thomas collar, but is usually so mild and irregular in character that the patient soon wishes to abandon the collar!

I do not make a diagnosis of this condition or of cervical rib or of any other condition causing nerve root pressure, if I find any restriction of the scapulo-humeral joint. Even atrophy of the spinati without restriction would incline me to rule out this diagnosis, because this combination is the rule in cases of rupture of the supraspinatus. Pressure on the nerve roots makes a false pain, just as the pressure of a neuroma gives pain in the amputated limb. There is no more reason to expect physical changes in the scapulo-humeral joint from such pressure than there is to expect physical changes in the amputated toe. As in the case of the phrenic nerve pain above described, this pain is referred by the cord and brain and is not caused by a physical disease of the part where the pain is felt.

X-ray evidences of hypertrophic changes in the cervical spine are so common that we must be on our guard not to have them mislead us in cases where the real pathologic condition is in the tendons of the short rotators. The symptoms characteristic of a "frozen shoulder" cannot be explained by pressure on the nerve roots, but pain about the shoulder without limitation of motion may be due to this cause.

Brachial Neuritis is a very indefinite term meaning literally, inflammation of a nerve in the arm, but clinically, applied to pain in the arm from various causes. In my own opinion such cases are usually due to traumatic or inflammatory changes in the musculotendinous cuff or in the subacromial bursa, and are in no way con-

nected with true inflammation of the nerves themselves, although "brachial neuritis" symptoms are present. The proof is that most cases can be at once relieved by appropriate treatment for the bursitis; miraculously in a few days in the cases of calcified deposit, and surely, though less rapidly, in a few weeks in cases of tendinitis.

I have come to believe there is no such thing as a brachial neuritis *per se*. The nearest approach to such a condition is in the cases of pressure on the nerve roots we have just been considering. Possibly in these there is a "neuritis" at the point of local pressure, but it seems to me more likely that there is no inflammation even at that point, and that the symptoms are due to mechanical irritation of the nerves, either of the afferent or more probably of the sympathetic fibers. Perhaps the nearest thing to a real neuritis is what we know as Herpes Zoster.

Herpes Zoster sometimes causes pain in the shoulder, and if the characteristic lesions of the skin are not present might be confused with other pathology in this region. Mehlis, in 1818, was the first to suggest that the eruption follows the distribution of the nerves, but von Bärensprung was the first to prove the hypothesis. This condition has been presented thoroughly by Head. Very little light has been shed upon the subject since. Head collected twenty-one cases which had been examined at autopsy. The 3rd cervical nerve was involved twice and the 4th once. In 416 clinical cases he found the 3rd cervical involved fifteen times, the 4th cervical twenty-one times and the 5th cervical twice. The skin over the shoulder is supplied by these nerves, and by the posterior branches of the next four or five lower nerves. It is unnecessary to review here the whole subject of herpes, but it is perhaps well to remind the reader that long after the skin lesions have disappeared there may be subjective pain of neuralgic character associated with anæsthesia. This is due to destruction of the nerve endings by the herpetic lesion. In this condition there is some pathologic evidence of actual lesions in the nerve and in the spinal ganglia.

In the diagnosis of cases of painful shoulder this condition must be borne in mind. We are so accustomed to think that "shingles" occur on the sides of the chest that we are apt to forget that the peripheral nerves supplying the skin over the backs and tops of the shoulders are subject to the same painful malady. I recently saw a working woman who for several weeks was quite incapacitated by such a lesion. The characteristic rash extended over the dorsum of the shoulder just above and just below the spine of the scapula. At

first sight I was deceived, because acne over the shoulders is so common and so apt to follow the use of any plasters, hot applications, and other remedies. Once having thought of herpes the diagnosis became clear, for the rash was unilateral and characteristic in appearance and in distribution. There was no atrophy of the muscles or restriction of motion. The patient was obliged to continue her work and the atrophy of disuse did not set in. If this patient had been a woman of the leisure class and could have coddled the arm, she would perhaps have had atrophy from disuse, a frozen shoulder, and have been many months in recovering from these secondary symptoms. As it was, the patient suffered for three months and still some hypersensitivity of the skin remained.

Whether or not there is a neuritis *per se* which is not secondary to the common shoulder lesions or to pressure on the nerve roots, and which is not accompanied by an herpetic rash, there certainly are forms of toxic neuritis capable of giving shoulder pain. That caused by lead poisoning may be taken as a type. However, the symptoms in this condition and in alcoholic and other forms of toxic neuritis are quite different from the condition which doctors treat as "brachial neuritis," for the element of muscle paralysis is dominant instead of scapulo-humeral spasm. The symptoms are usually bilateral. To my mind the presence of scapulo-humeral spasm is not consistent with a true neuritis. In shoulder lesions the spasm itself produces the neuritic symptoms. The pain is from the nerve endings in the spasmodic muscles, not from disease in the nerve trunks, although the nerve trunks themselves become sensitive to the touch, even to the very roots of the plexus. If the nerves themselves were really inflamed, removal of a small calcified deposit or the breaking up of adhesions would not produce immediate relief. Their sensitiveness may be due to hyperæmia, but I know of no real explanation nor of experiments to determine whether a normal nerve trunk is capable of transmitting to the brain the exact location of touch stimuli.

Tumors of Spinal Cord and Nerves may cause shoulder pain, but they are rare. Among 35,000 autopsies Schlesinger found only 104 cases in which the spinal cord was involved. The cervical region is involved in about twenty-five per cent of the cases. In 264 cases he found it involved in seventy. In sixty-four cases reported by Ayer, twenty-six showed cervical lesions. In eight hundred cases referred to by Elsberg, twenty-four per cent were cervical. In his personal series of one hundred, C1-4 was involved six times; C5-8, twenty-six times.

The first symptom is usually root pain which is sharp, sudden and severe. It is aggravated by motion and patient holds his neck stiff. Tumors in the region of C4 or C5 give pain on the top of the shoulder which is distributed along the roots. Numbness, paresthesia, spasm and paralysis soon follow. Wasting of the muscles about the shoulder is often marked. There may be a dull "spinal pain." Lumbar puncture (Froin's syndrome) combined with cistern puncture and lipiodal injection help in diagnosis.

Tumors of peripheral nerves sometimes occur in the shoulder region and give rise to constant pain. Symmers has reported a case. Gatch and Ritchey reported two cases in which the brachial plexus was involved. Cushing reported a case in 1904 in which the pain radiated from the shoulders into the arm. He also reviewed the operated cases. If I have ever seen a case where shoulder pain due to this cause was the first or only symptom, I have failed to recognize it.

Cervical Potts Disease or tuberculosis of the cervical vertebræ may cause shoulder pain but is very rare in adults. Whitman says that in a series of 1,355 cases of tuberculosis of the bones, the fourth cervical vertebra was involved twenty times and the fifth thirteen times. Pain is sometimes referred to the distribution of the cervical nerves. A report based on eighty-seven cases has been given by Volkmann.

Lead Palsy may take a number of forms. Its onset may be gradual or sudden. Frequently there are prodromal symptoms such as general lassitude, muscle cramps or tremor, numbness, dull ache or formication in the affected area. The palsy may manifest itself as muscular weakness or motion may be lost. The common type is the antibrachial with wrist drop. This may gradually increase until all the muscles supplied by the radial nerve are involved, except the brachio-radialis, anconeus and usually the abductor of the thumb. This is an important point in a difficult diagnosis. There is marked atrophy. A second or brachial type involves the deltoid, biceps, brachialis anticus and brachio-radialis, thus simulating an Erb-Duchenne palsy. The pectorals may be involved and sometimes a scapulo-humeral palsy results. "It is very rare for this to occur without wrist drop; but even when associated with palsy of forearm and hand, this type of paralysis is not a common manifestation of lead poisoning." (Aub.)

A third form is characterized by progressive atrophy of the thenar and hypothenar eminences and small muscles of the hand.

In diagnosis poliomyelitis, progressive muscular atrophy, amyotrophic lateral sclerosis, cervical rib, arsenic or alcoholic neuritis have to be ruled out. Colic, lead line, stippling and lead in the urine are very important. Prognosis is good.

Diabetic Neuritis. While neuritis in diabetes usually manifests itself by aching or gnawing pains in the legs and feet, with hyperesthesia and burning sensation, it is said that it may also involve the brachial plexus in rare cases and give rise to similar symptoms in the arm and shoulder.

A good review of the subject with bibliography is given by Woltman and Wilder (*Arch. Int. Med.*, 1929, 44: 576). Joslin says, "Neuritis is most uncommon among my patients. . . . Indeed, primary 'neuritis' in diabetes is usually a misnomer. It is secondary to some form of circulatory obstruction or some form of arthritis."

Tendinitis and consequent subacromial bursitis are not uncommon in diabetic patients, and it is probable that most cases considered as "neuritis" in diabetics, just as in non-diabetics, are due to this cause. The similarity of degenerative changes in the arterial walls and in the tendons has been alluded to under Pathology, and it is noteworthy that these conditions both appear with unusual frequency in diabetics.

King and Holmes also give a list of what they call "The more common shoulder lesions causing pain and disability," which are *not* demonstrable by Röntgen rays. We may briefly consider these diagnoses.

Postural
Subcoracoid bursitis
Tenosynovitis biceps
Myositis—traumatic and infectious
Attrition—slight to moderate
Relaxed capsule—slight to moderate
Tight axillary capsule

Postural. There can be little doubt that a poor posture may cause shoulder symptoms just as certainly as that poor posture tends to cramp all the viscera and lead to a general condition of lowered vitality. Goldthwait, who has always been interested in this matter, is insistent on its effect on shoulder conditions. The type of patient in whom we might expect shoulder pain from poor posture, would be an adult past middle life with head held somewhat forward, exaggerated dorso-lumbar curves, protuberant abdomen and sloping shoulders. Such cases not infrequently have in addition hypertrophic changes in the cervical spine. I recently saw such a pa-

tient with vague shoulder pain, without restriction of motion or atrophy of the muscles. I was uncertain whether to attribute the pain to the hypertrophic arthritis or to the posture. The two conditions are bad companions, and it is not improbable that they interact on one another. The forward position of the shoulders tends to drag the nerve roots down across the first rib, and to exaggerate any irritation which the hypertrophic changes about the intervertebral foramina are already producing. Such patients often have sedentary occupations which compel them to sit for hours in a slumping position. In my experience, shoulder pain from this cause is never unendurable or of a very severe nature. Obviously the treatment should be general, and no time and energy should be wasted on local physiotherapy, for the patient will need all his courage to change his habits. The subject of posture as related to the education of children is undoubtedly of more importance than has been realized, and improvement in the condition of shoulders will be but one of the minor results of its general appreciation.

Subcoracoid Bursitis. In my early papers I alluded to this condition and sometimes made the diagnosis as if it were a distinctly separate condition from subacromial bursitis. As I have explained on page 27, I now feel that it should be considered one and the same thing, although doubtless the subcoracoid portion of the bursa might be inflamed without involving the subacromial bursa. Certainly there are some cases in which a calcified deposit forms in the subscapularis tendon and therefore may involve the subcoracoid portion of the bursa. Goldthwait, in 1908, wrote two articles on the shoulder in which he made more of the subcoracoid bursa than I had in my original article. He insisted on its cause being related to poor posture. I believe that his articles are misleading, particularly as to his idea that the lesser tuberosity impinges on the coracoid process. As a matter of fact, the lesser tuberosity is almost wholly covered by the muscle and tendon of the subscapularis, so that such impingement is next to impossible. In the normal working of the joint not only the lesser tuberosity but the bicipital groove may pass beneath the coracoid process in internal rotation.

Tenosynovitis of the Biceps. When I was in the Medical School, I used to hear this diagnosis made. At that time there was a general impression that if you moved the forearm up and down you moved the tendon of the biceps in its groove. This, as explained previously, is erroneous. There may be such a lesion as tenosynovitis of the long head of the biceps, but I have never demonstrated it either clinically,

at operation or at autopsy, except in conjunction with rupture of the supraspinatus. I cannot say that it does not occur, but practically I do not make this diagnosis, and have no temptation to do so. I think that tenderness over the bicipital groove is more apt to be due to inflammation at the insertion of the pectoralis tendon or that of the teres major or of the bursæ which accompany them, than it is to inflammation in the groove itself. It is not uncommon for persons after unusual muscular effort to have tenderness and lameness for a few days in this region.

Tuberculosis in the shoulder joint may involve the groove and the tendon, and, if it is accompanied by suppuration, the pus may run down the course of the sheath and make an abscess in the upper anterior part of the arm. Fracture of the tuberosity very commonly causes hemorrhage to run down parallel with but not in the groove, but I am quite confident that inflammation of the tendon is rare. I have occasionally thought that I recognized a mild tenosynovitis in young people who have used their arms to excess in playing games, particularly such games as Ring Toss and Quoits, which require antero-posterior motion of the humerus.

Myositis. Muscular rheumatism used to be a diagnosis for the cause of pain of an unexplained nature in the region of the muscles. It was a diagnosis commonly applied to what we now call subacromial bursitis. Doubtless it does occur as a complication of other lesions, such as fractures, but as a matter of fact all surgeons would agree with me that inflammation of a muscle, except as a secondary complication from infection of neighboring structures, is very uncommon. During the war I saw some instances of streptococcus infection of the muscles, but I have rarely seen such cases in civilian life. In my opinion "myositis" as a diagnosis, which explains any ordinary shoulder pain, has no pathologic or even intellectual foundation. We might as well be satisfied with having "caught cold" in the shoulder. The transient soreness in muscles after unusual or prolonged effort is the only evidence of even a temporary inflammation of which I know.

Attrition. Dr. A. W. Meyer, Professor of Anatomy at Leland Stanford University, has been the great exponent of this condition. His researches have been almost wholly from a purely anatomic standpoint, and they have not yet been accepted by clinicians. His articles are well worth reading, and there is some reason to believe that his contention, that the tendons and other portions of the joints may actually wear from continued use, has much to support it.

Dislocation of the Long Head of the Biceps.

According to Dr. Meyer, dislocation of the biceps tendon is a lesion which has not been sufficiently recognized clinically, for he found four instances in 286 cadavers. He was able to find only two cases in the literature. One of these was proved at operation. He believes the condition results from destruction of the superior part of the capsule from attrition. He also believes that actual wear plays a part in rupture of the biceps tendon.

I have seen one case in which dislocation of the tendon was probably the cause of symptoms which were much like those of a complete rupture of the supraspinatus. There was a loud, painful "snapping" as the arm was elevated. I opened the bursa, but not the joint, and as no rupture was found I closed the wound. The snapping continued. Later I found that I could prevent the snapping by making the patient rotate the humerus internally with the elbow flexed; then put the humerus in dorsal flexion; then extend the forearm and rotate externally. When this was done he could elevate the arm in the coronal plane without pain and without the snapping. This maneuver would have placed the biceps tendon in the groove in internal rotation; held it snug in the groove by extension of the forearm; permitted external rotation while the tendon was taut and therefore could not slip out of the groove. Once in external rotation the arm could be elevated in the coronal plane without releasing the tendon.

From Meyer's findings in the dissecting room, the reason why the tendon may slip out, is denudation of the capsule at the inner edge of the intertubercular notch, so that in external rotation the tendon slips over the upper part of the lesser tuberosity. The symptoms in my case and their relief by the maneuver described are compatible with Meyer's findings. However, in the case in question, the X-ray showed a small fragment or "mouse" in the neighborhood of the tuberosity which may have caused the symptoms, so that I do not feel sure of the diagnosis. The patient refused further exploration. In a few other cases I have been inclined to make this diagnosis for similar reasons, but the symptoms were not severe enough to justify operation.

Meyer's careful observations deserve more attention than they have received from clinical surgeons. My own opinion is that the cases he describes are usually the result of ruptures of that portion of the musculo-tendinous cuff which is inserted into the inner side of the intertubercular notch. This portion is made up by the joined edges of the supraspinatus and of the subscapularis. I do not believe

that bare tuberosities are the results of attrition; I think they are due to evulsion of the tendon fibers. This is shown by the fact that in most cases the evulsion of fibers begins on the cartilage side of the tendon.

Whatever the cause, it is undoubtedly true that in the cadaver one occasionally finds that the long head of the biceps can slip out of the upper end of the groove on to the lesser tuberosity. It may be that the maneuver I have described may some day help to make this diagnosis clinically. In such a case it should not be difficult to promote adhesions over the point where the tendon slips and thus hold it in place. There are several ways in which the surgeon could accomplish this.

The remaining two items on King and Holmes' list, relating to the condition of the capsule, have already been considered. It is my belief that most of the three hundred cases in which they state that the X-ray films had been negative were cases of lesions of the tendons of the short rotators, such as those described in Chapters III to V. These lesions do not give pronounced X-ray findings and need special technique for their demonstration. Each facet should be taken in profile separately, and even then the lesions which I have described as excrescences, caverns, and bursal osteitis, are small and inconspicuous. When the attention of the profession has been aroused to the fact that these small but painful lesions exist, Röntgenologists will certainly find the technique to demonstrate them. The average surgeon is not interested in them, but they are of great importance to the patient and to industrial insurance companies. In all such cases the Röntgenologist should take comparative plates of both shoulders to determine the relative amount of bone atrophy. Most of the conditions to which this book is chiefly devoted show bone atrophy at least as early as the fourth week.

It does not seem worth while to enter into the diagnosis of still rarer lesions than those mentioned in this chapter. My list of forty-two consists mainly of one or two instances of each of these unusual conditions, and others could be collected from the literature. We might, for instance, find the report of a case of echinococcus cyst, of a monstrosity or of involvement of the shoulder bones in certain rare general diseases which affect all the bones, such as leprosy, lymphoma, osteomalacia, etc. Our real troubles in diagnosis come, not from the rare diseases, but from the common conditions. A list of such rare cases might be interesting but would not be of great importance. However, there are a few more subjects, if not diseases, which should

be mentioned in this connection. These subjects are: bone atrophy, ruptures of muscular insertions, congenital anomalies, the normal epiphyses and variations in their time of union; lastly, zero cases.

Bone Atrophy. Acute bone atrophy was established as a clinical entity by Sudeck. He attributed the condition to low-grade inflammation, but later modified his view and asserted that it was essentially a trophoneurosis. Atrophy occurs more rapidly than it does in the chronic form resulting from disuse and may occur when there is no disability.

The condition is most often studied in the bones of the hands because it is easily recognized in small bones. There is usually a history of trauma with or without fracture, although the condition may arise spontaneously. Onset of symptoms may occur almost as soon as the initial shock has worn off or may be delayed several days. Pain is usually a prominent feature. The overlying tissue may become swollen and glazed. Motion is limited. The pain is more marked with motion. There may be hyperesthesia over the part.

The X-ray shows rarefied areas in the spongiosa. The Röntgenologists speak of it as "spot-atrophy." The trabeculæ fade into one another and produce a poorly defined shadow. The rarefied areas disappear and the individual longitudinal striations are thicker and stronger. The bones are represented by mere outlines in extreme cases.

Acute bone atrophy in the shoulder, particularly in the humerus, has not received any attention. In fact, acute bone atrophy, as a distinct lesion apart from the atrophy of disuse, is still *sub judice.* In my dispensary work, years ago, I thought that I had many occasions to verify the entity described by Sudeck. I had read his original article in 1900, and many times thought that I had made the diagnosis, particularly in the bones of the hand, in cases of low-grade inflammation not accompanied by suppuration or perhaps by a focus distinct from the atrophied bones, for instance, in cases of felons of the finger. Sometimes in a few weeks following such a lesion the bones of the hand would show extreme atrophy in the X-ray. I soon came to recognize that atrophy occurred very early, and to a great extent, in some of the cases of frozen shoulder. I now know that within about three weeks, if not before, after the onset of restriction of motion, the humerus, and sometimes also the scapula, show marked signs of atrophy. This atrophy is much more marked in some cases than in others. Of late years I have come to think of atrophy of the bone in all cases as being pretty well proportionate to the atrophy of

the short rotators. It appears in all these cases to some degree, and involves the whole humerus and especially the spongy bone in the upper end. Very often it is closely associated with the bursa, so that the X-ray shows atrophy which corresponds quite exactly with the part of the bone which is covered by the base of the bursa. Normally, there is less density in the spongy bone in this region, but where atrophy sets in, not only is the density less, but the peripheral outline of the area of absorption is irregular. It seems to me that it is best explained by supposing that an active or passive congestion of the medullary spaces in the spongy bone produces absorption of the intervening trabeculæ.

Since the interesting experiments of Allison and Brooks conducted on dogs to study the atrophy of disuse, my convictions have been more or less shaken. Their experiments seemed clearly to indicate that fixation *only* was responsible. They could detect X-ray changes in the lower extremities as early as seven days after fixation. They concluded that the changes observed were the same whether non-use resulted from nerve paralysis, injury to joints or simple fixation. The degree of atrophy was proportioned to the degree of non-use. My own theory that the trabecular absorption is from congestion seems also to be confuted by the experiments of Grey and Carr, from which they concluded that alterations of the blood supply had no effect. A good review of the literature has been given by Noble and Hauser in *Arch. of Surg.*, Vol. 12, p. 75, 1926.

I still cling to the feeling that there is an atrophy of bone which is produced independently of fixation, and is, as claimed by Sudeck, a trophoneurosis. It is hard for me to believe that a few weeks of fixation could produce the extreme atrophy of the humerus which we find in some cases of tendinitis. I do not find bone atrophy in the shoulders of malingerers or of hysterical cases. When I am in doubt of the preponderance of the mental element in a given case, atrophy demonstrated by the X-ray strongly influences me to believe that there is a physical lesion as a basis for the symptoms.

At any rate, I feel confident that many cases of atrophy would have been demonstrated in the three hundred negative cases listed by King and Holmes if an effort had been made to find it, by taking comparative films of both shoulders.

Rupture of Muscles and Tendons. While I may be prejudiced enough to think that rupture of the supraspinatus tendon is more common than that of any other muscular insertion in the shoulder, or even in the whole body, my mind has not become so distorted in

writing this book that I ignore other tendons and muscles either about the shoulder or elsewhere. In fact, the reverse is the case, for now I am inclined to the opinion that minor ruptures of tendons about various other joints may initiate the phenomena which we call arthritis. The same degeneration of collagen which I find in the shoulder probably occurs in other tendons and ligaments; slight, unusual efforts tear a few fibers and the same vicious circle which arises in the shoulder conditions, spasm, adhesion, non-use from pain, further degeneration, more ruptured fibers, etc., produces the picture of arthritis.

In the shoulder we have already seen that the long head of the biceps and the tendons of the short rotators may individually, or to a greater or less extent together, become evulsed. I have seen two cases where almost the whole of the great pectoral insertion tore out. Several times I have seen partial rupture of this tendon. No doubt the insertions of the rhomboids, of the latissimus and of other tendons at times tear out and produce soreness and pain and muscular spasm. Yet the literature concerning these lesions is scanty. I am apt to make provisional diagnoses of these lesions when I find sharply localized tenderness at the insertion and spasm in the belly of a muscle, especially if there is also spasm in the antagonistic muscles.

According to Loos, Petit was the first to report a case of ruptured muscle, in 1722. The muscles most commonly injured, according to Gottlieb, in a review of five hundred cases, are: (1) back, (2) calf, (3) quadriceps, (4) adductors, and (5) biceps.

The muscles about the shoulder, other than the biceps and supraspinatus, are seldom injured, if we may judge by statistics. In the library of the Surgeon General, one case of rupture of trapezius, three cases of deltoid, two cases of pectoralis major, and four of triceps are listed. Rupture usually results from indirect violence. Sudden sharp pain is experienced. There may be a loud snap. A break in continuity can be made out and loss of function results. Contraction of the muscle may cause a swelling, but atrophy results later. Suture of the muscle or tendon, if it can be done soon, will hasten recovery.

Since writing the above I was consulted by a colleague (age about 60), who had a fluctuant tumor, the size of a hen's egg, situated in the anterior lower portion of the right deltoid. He stated that on the evening before he had noticed some pain in the region, and when undressing at night had found the tumor. It was far too large and conspicuous to have escaped his notice if it had been there previously. He recollected that in the morning he had been stooping over and had made quite a violent effort to open a

drawer, but did not recall any special pain in doing this. The tumor is firm when the muscle is contracted, and soft when it is relaxed. The X-ray shows an oval cyst-like shadow in the muscle beneath the fascia. There has been no ecchymosis. The tumor caused slight pain for a few days, but since then it has neither caused pain nor interfered with the use of the arm. Two months have elapsed without any apparent change in the size or in other characteristics of the tumor. No operation has been done. My diagnosis of this case is that some of the deltoid fibers in one of the compartments of the muscle were ruptured, and the hematoma being confined between the intermuscular septa has dilated the space, which would normally be shaped somewhat like a section of a melon, until it has assumed the contour of an egg. The case is unique in my experience, and in that of the röntgenologists who have seen the films.

Congenital Anomalies are rare about the shoulder. Absence of the whole or of a portion of the clavicle occasionally occurs. Congenital elevation of the scapula ("Spengel's Deformity") is well known, though rare. The scapula is small and broad. In addition to elevation, internal rotation is also present. A fibrous band may attach the scapula to the vertebral column, and this interferes with motions of the shoulder. I have had no personal experience in treating these cases. Horwitz reviewed 136 cases in *Am. Jour. Orth. Surg.*, 1909, Vol. VI, No. 2.

The only anomaly which the writer has frequently noticed is a protuberance on the clavicle adjacent to the coracoid. In one case which I observed, this protuberance touched the coracoid and there formed, according to the X-ray, a small joint. I do not know whether this protuberance is to be explained by any hypothesis founded on embryology. I do not know whether it has any practical significance. In the case I observed there had been a trauma, the patient complained of pain in the region, the X-ray showed an abnormality and compensation was awarded. I have not seen this condition described elsewhere.

"Snapping Shoulder" is a term used to describe certain cases where there is a definite audible snap in the shoulder when the arm is abducted and externally rotated. The condition may or may not cause pain. I do not understand it, but it may be due to the lesser tuberosity catching as it passes under the short head of the biceps.

As is obvious to every one, there is a wide variation in the shapes of the shoulders of different individuals, and when one studies a large series of macerated specimens, many details in each of the three bones which compose the shoulder are found to vary, as do their mutual relations, and their positions as a group in regard to the rest of the skeleton. The variations of the humerus and of the clavicle have not

received as much attention as have those of the scapula, which have been intensively studied by Dr. W. W. Graves (Director, Department of Neuro-Psychiatry, St. Louis University School of Medicine), not as a contribution to anatomy or to explain local clinical manifestations, but for the broader object "of laying the foundation for investigations on the vital and enduring problems of individual, family and racial fitness." After a series of careful studies, he has concluded that the outline of the greater portion of the vertebral border of the scapula below the base of the spinous process is the most practical, convenient and reliable, single, skeletal landmark yet known to science, for use as a criterion to determine the fitness of individuals, of groups or even of peoples. The valuable anatomic data that he has accumulated are merely by-products of his examinations of large series of scapulæ, including anatomic and anthropologic museum specimens from monkeys, apes, primitive man, racial types, and even from collections of fœtuses. Data have been derived from healthy and sick groups for comparative study. In a book of this kind I can only afford space for a brief abstract of his evidence, argument, and conclusions.

1. All scapulæ may be classified into several distinct types and each type has a characteristic contour of the vertebral border.

2. The general contour of the vertebral border remains the same from the latter part of fœtal life until death.

3. Omitting subordinate or incomplete specimens there are two main types of scapulæ, and these are found in both ancient and modern men, the scaphoid and the convex.

4. The outline, once formed, is not changed by age, nutrition, occupation, disease, or by any other factor.

5. He has found no inherited characters, common to human beings, to be more constantly transmitted from parents to progeny, than scapular types: the *scaphoid* types showing Mendelian dominance.

6. Statistics of age incidence show that decade by decade the proportion of the *convex* type increases, so that the incidence of occurrence from birth to the age of sixty-five is almost reversed; *i.e.*, two scaphoid instances to one convex become one scaphoid to two convex.

7. Hence, many individuals possessing the scaphoid type cannot be as resistant to the exigencies of life as are the possessors of the convex types of scapula.

8. Statistics from hospitals, insane asylums, pauper institutions, etc., show that the scaphoid type is represented in greater than normal proportion; therefore, this type indicates a tendency to lack of adaptability as well as to diminished longevity.

Dr. Graves does not assert that the type of scapula is of major importance in a person's psycho-physical make-up; he simply argues that the vertebral border furnishes a conditional index to determine probable longevity, health, and adaptation in general. As a practical example one might say that if one had to pick a baseball team, an army or a board of trustees from groups of otherwise unknown applicants, one would be wise to select those whose scapulæ were of the convex type, for the psycho-physical mechanisms of these individuals would probably be of a higher order than those of the remainders of the groups.

Among the by-products of these years of research, Dr. Graves has called attention to an anatomic finding which he calls "the acromial plaque," a condition to which I have alluded in Chapter III, p. 68, as "hypertrophic changes at the acromial edge." Dr. Graves in his paper of 1922 has described these plaques very carefully and has noted their incidence of occurrence. I am inclined to interpret them as results of irritation of the region in response to friction in the bursa, due to irregularity in its base from ruptures of the tendons or from hypertrophic changes in the tuberosities. Their age incidence and percentage of occurrence are confirmatory of this supposition. Dr. Graves states:

"(4) that neither in man nor in other primates has it thus far been found in young and relatively young, but invariably in old and relatively old bones; and (5) that it is indicated as early as the thirtieth year in human scapulæ, increasing in size and frequency of occurrence in succeeding age periods, although it may be either rudimentary or absent even in senile bones. The precise structure or structures involved in the genesis of the plaque; what relation, if any, its presence may bear to pathological processes in general, aside from those incident to age, are problems for further investigation."

Dr. Graves' intensive studies of the age changes in this one bone would make his opinion on anomalous conditions of great value. It is always difficult to say whether such hypertrophic changes and "lippings" on the edges of other bones where ligaments, tendons or muscles have their origins and insertions, are due to anomalies, age changes, excessive use of the structures or to toxic conditions in the blood. In these instances at the acromial edge it may be that continued irrita-

tion of the tuberosity by the acromial plaque produces the changes in the insertion of the tendon, but my present view is that the reverse is the case.

A very important result of Dr. Graves' studies is, that he has found and used a principle applicable to the evaluation of other criteria on which life expectation may be estimated; *i.e.*, the age incidence of any given inherited morphologic criterion which, after type differentiation, remains throughout life, may be thus used. He merely claims that the shape of the vertebral border of the scapula is a most practical unit. He definitely shows that many of those who possess what is called scaphoid types are less fitted to meet the world, not on account of their scapulæ, but because the shapes of the vertebral borders are indicative of other mental and physical characteristics. Dr. Graves' work is a fine example of how the scientific method carefully applied year after year, on some apparently trivial circumstance, may lead to conclusions of great importance to future generations.

Epiphyses in the Shoulder Bones. Since the centers of ossification may be mistaken for fractures, the following table (Cunningham) may be of use in the diagnoses of shoulder lesions occurring in youth.

Center	*Appears at*	*Fuses*
Scapula		
1. For coracoid	1st year	Puberty
2. Subcoracoid for lateral part of root of coracoid and upper third of glenoid cavity	10th year	Puberty
3. Margin of glenoid cavity	Puberty	20-25
4. Inferior angle	Puberty	20-25
5. Vertebral border	Puberty	20-25
6. Two for acromion	Puberty	20-25
Humerus		
1. Shaft	8th week fetal life	
2. Head	1st year	
3. Greater tubercle	3rd year	20-25
4. Lesser tubercle	5th year	

Occasionally the epiphyses of the acromion fail to fuse with the rest of the bone, and the greater part of the acromion is throughout life a separate bone, united to the rest of the process by periosteum and a strip of cartilage, or by a joint which appears like a line of fracture in the X-ray. I have known one such case to be mistaken for a fracture. It is possible that the coracoid epiphyses might cause a similar error, but I have never observed such a case. The coracoid would appear to be broken off at its base. (See Fig. 56.)

In preparation for this book I took my records of shoulder cases and endeavored to file them under definite headings, as in the table on page 469. The task was a difficult one, so far as about one-fifth of the cases were concerned, because for one reason or another the diagnosis was complicated by other considerations than mere symptoms and signs. In some cases I suspected exaggeration, if not actual malingering; others were complicated by coincident injuries; some had trivial or mild symptoms; some had no organic signs to confirm the existence of alleged pain; others had unusual symptoms. Finally, I concluded to classify under the main headings only cases in which the diagnosis was reasonably sure. I marked 0 on the records of the doubtful or trivial cases; thus I have come to think of this group as zero cases.

Zero Cases. It is easy enough to detect typical cases of extreme degrees of rupture of the tendons or of calcification in them, but the cases which are really troublesome in diagnosis are those where the symptoms are mild and transient, where varying degrees of pain and restriction exist or are complicated by the mental attitude of the patient. This is particularly true in industrial cases where the element of compensation enters the problem.

Many of these mild cases, without clear-cut signs, can only be classed as strains; the shoulder remains sore for a few days or weeks, and recovers under palliative remedies. We can no more absolutely define strain in the shoulder than we can strains and sprains in the other joints or muscle insertions. My belief is that most of these cases are due to what I call "rim rents," *i.e.*, the evulsion of a few of the fibers of the short rotators on the joint side. In such cases, with almost negligible physical signs and a mere complaint of pain, I cannot be positive as to the exact nature of the lesion. The more my experience has increased, however, the more sure I am able to feel of this conviction. Oftentimes I can palpate very distinctly the tender spot on the tuberosity which is, I believe, the location of the actual lesion, although the patient refers the pain to the spasmodic muscles. Patient, repeated examinations, which show consistent accuracy in the location of tenderness, may strengthen one's conviction that there is a real injury, even though it is pretty obvious that the employee is making the most of his symptoms in order to get compensation. "Strain" is the word which, by long custom, we apply to indefinite lesions of the muscles and tendons, and we do not yet know of any technique by which these lesions can be actually demonstrated. However, the cases of complete rupture of the supraspinatus described in this book must be promptly diagnosed and not passed off

as strains. At present the only way to be sure in a suspicious case is to make a small exploratory incision in the bursa. Improved Röntgen technique aided by injection of air or of opaque fluids will make this Zero Class smaller.

Of course, many of these cases I saw only once, and any doubtful case needs repeated study. However, even with more study there would have been a large class in which I was in doubt of the real nature of the lesion, which caused the patient to claim disability. Usually this doubt was more of a mental than of a physical nature. How much more satisfactory the world would be if we gave up lying! How inefficient it is, particularly in regard to medical science and its results! What with patients lying to doctors, and doctors lying to patients and our lying to each other at scientific meetings, "we tend to become confused," as the patient, polite, dignified man of science would put it.

Of course, this group of zero cases interests me just because I do not understand them. I think I see some promising leads which will throw light on their nature, but I must admit that I do not know enough about one-fifth of the shoulder cases I see to be *confident* of the detailed diagnoses. I could write a most uninstructive book about this group, for each case is a puzzling and unsolved complex of phases of human nature, vague physical signs and inaccurate statements of the clinical history. I alternately wonder at the good will in human society which has produced the workmen's compensation ideals, and at the viciousness in human nature which constantly interferes with their practical application.

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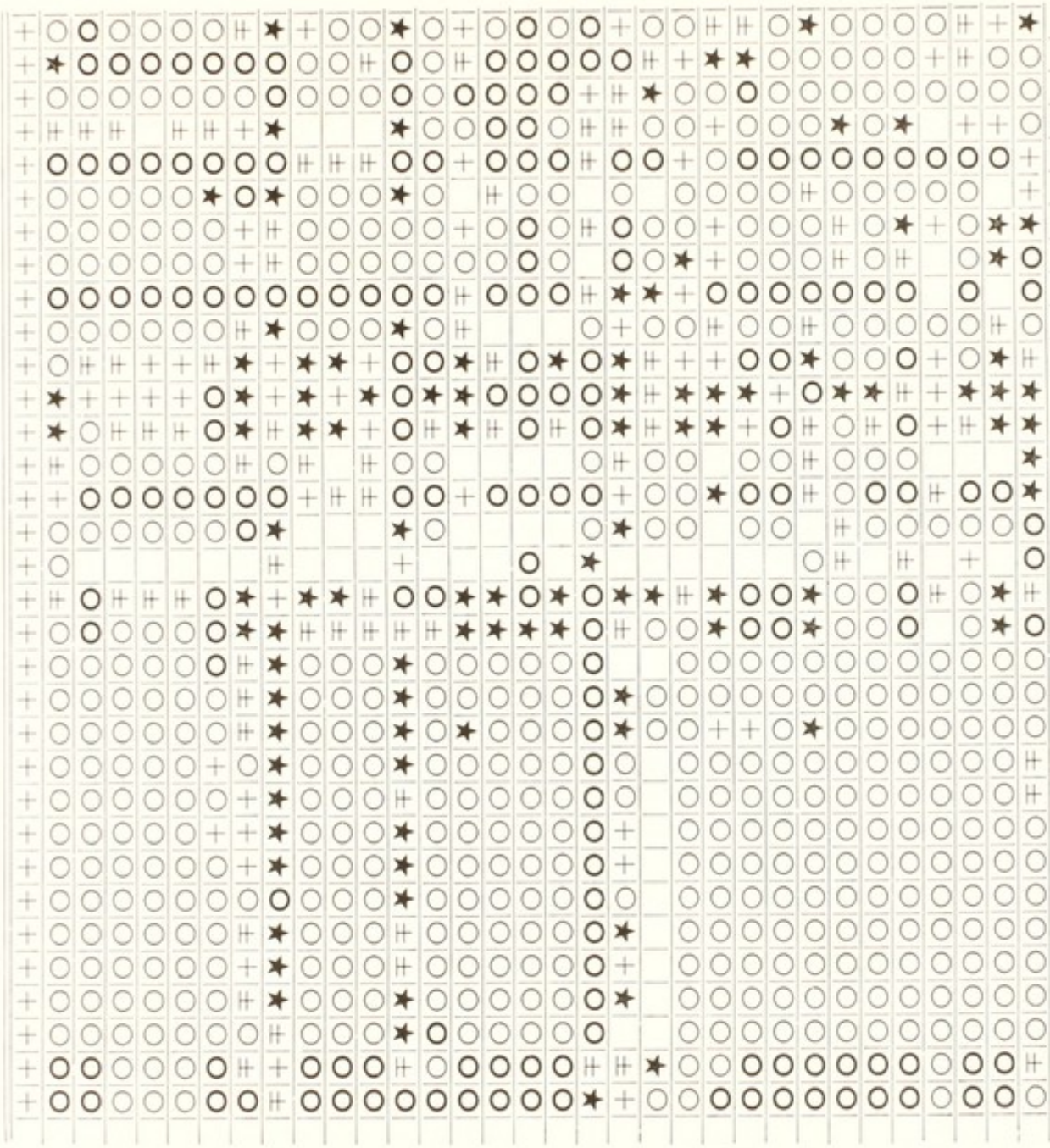
INDEX

A diagnostic chart combined with indices of shoulder symptoms and of conditions which may cause them.

DIAGNOSTIC POINTS	described on→ ↓	CLINICAL ENTITIES	Arthritis, acromio-clavicular	Arthritis, scapulo-humeral	Bursitis, subacromial	Calcified deposits	Cervical rib	Charcot's joint	Congenital conditions	Cord lesions	Dislocation, acromio-clavicular	Dislocation of long head of biceps	Dislocation, scapulo-humeral, anterior	Dislocation, scapulo-humeral, habitual	Dislocation, scapulo-humeral, infantile	Dislocation, scapulo-humeral, old	Dislocation, scapulo-humeral, posterior	Fracture, clavicle	Fracture, humerus, head of	Fracture, humerus, tuberosities of
		described on→	307–309	108–118	119–122	178–215	489–490	476–478	503–506	480	307–309	498	262–293	293–301	301–307	291–292	291, 303	314	315–331	281–291
Accident, type of		140–144, 262–279	±	○	±	±	○	○	○	○	★		★	★	±	★	★	★	★	★
Atrophy of spinati		152, 195	○	★	±	+	○	+	+	+	○	±	±	○	★	★	±	±	★	★
Crepitus		151	±	±	+	○	○	★	○	○	★	+	±	○	○	±	○	+	+	±
"Down-it-will-go"		196	○	○	±	+	±	+	○		○	○	○	○	○	○	○	○	○	○
"Dropping shoulder"		482	○	○	○	○	○	★	○	○	○	○	○	○	○	○	○	+	±	○
Eminence on tuberosity		150	○	±	○	★	○	○	○	○	○	○	○	○	○	○	○	○	±	★
Fluid sign		94, 115, 155	○	○	○	○	○	★	○	○	○	○	○	○	○	○	○	○	○	○
"Frozen shoulder" see Restriction		216	○	★	+	+	○	○	±	○	±	○	±	○	★	★	★	+	★	★
Hypertrophy of deltoid		154	○	○	○	○		○	○	○	○	○	○	○	○	○	○	○	○	○
Jog in motion		150, 503	○	○	+	+	○	○	±	○	○	★	○	○	○	○	○	○	○	±
Lack of scap.-hum. rhythm		147, 188	±	+	★	+	○	+	±		±	★	±	○	+	+	+	±	±	±
Occupation		135–136	+	+	+	±	○	○	○	○	○	○	○	○	○	○	○	○	○	○
Pain, neuritic		190	○	○	±	+	★	○	○		○	○	○	○	○	○	○	○	○	○
Pain, nocturnal		191	○	★	+	★	±	○	○	○	±	○	±	○	○	±	±	±	±	+
Sensory changes		396–399	○	○	○	○	±	★	○	★	○	○	○	○	○	○	○	○	○	○
Restriction of scapulo-humeral motion	adhesions	72, 147	○	★	±	+	○	○	±	○	○	○	○	○	+	+	○	○	+	+
	ankylosis	58–59	○	±	○	○	○	○	±	○	○	○	○	○	○	±	○	○	±	○
	contracture	189, 196	○	★	±	±	○	○	±	○	○	○	○	○	+	+	+	○	±	±
	spasm	149	○	★	+	+	○	○	○	○	±	±	±	○	○	+	+	+	★	★
Sulcus on tuberosity		149–150	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○
Swelling or enlargement		411–467	★	±	○	±	○	★			★	○	★	○	○	★	★	★	★	★
Tenderness, over bursa		148–149	○	+	+	★	○	○	○	○	○	±	○	○	○	○	○	○	+	★
Tenderness, over lower deltoid		191	○	+	★	+	○	○	○	○	○	○	○	○	○	○	○	○	±	+
Tenderness, over teres major		195	○	★	★	+	○	○		○	±	±	±	○	○	±	±	○	★	★
X-Ray signs			★	+	+	★	★	★	★	○	★	○	★	±	★	★	★	★	★	★
bone atrophy		93, 500	○	+	+	★	○	★	+		○	○	±	○	+	+	±	±	+	+
calcification		68, 75–84	○	○	±	★	○	+	○	○	○	○	○	○	○	○	○	○	+	+
caverns		91, 103	○		○	±	○	○	○	○	○	○	○	○	○	○	○	○	○	○
eburnation		92	○		○	○	○	○	○	○	○	○	○	○	○	○	○	○	○	○
excrescences		91	+	±	±	○	○	+	±	○	○	○	○	○	○	○	○	○	+	±
recession		92	○	○	○	○	○	±	○	○	○	○	○	○	○	○	○	○	○	+
Wince		150, 189	+	+	±	★	○	○	○	○	+	★	○	○	○	○	○	○	○	±
Other symptoms			+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+



313	Fracture, scapula
71–72	Fringes, villi and bands
492	Herpes Zoster
478–479	Hygroma
400–410	Hysteria and allied conditions
481	Myositis ossificans
332–379	Paralyses from nerve injuries
494	Paralyses from other causes
483, 491	Neuritis, brachial (for toxic see paral.)
481	Osteomyelitis
118–119	Periarthritis
123–155	Rupture of, supraspinatus complete
155–158	Rupture of, supraspinatus incomplete
501–503	Rupture of other tendons
507–508	Sprains or strains
474–476	Syphilis
477–478	Syringomyelia
216–224	Tendinitis (non-calcareous)
471–474	Tuberculosis
421	Tumors, angioma
425	Tumors, atypical sarcoma
432–444	Tumors, benign giant cell
446–450	Tumors, benign osteogenic
450–452	Tumors, bone cyst
427	Tumors, chondrosarcoma
428	Tumors, Ewing's sarcoma
411	Tumors, lipoma
444–445	Tumors, metastatic cancer
429	Tumors, multiple myeloma
425	Tumors, osteogenic sarcoma
418	Tumors, periosteal fibrosarcoma
488, 491	Vertebral lesions (referred pain)
483–488	Visceral lesions (referred pain)
495	Zero cases or other lesions

THE USE OF THE INDEX CHART

The numbers below the diagnoses refer to the pages where the lesions are described; those in the vertical column, next to the list of diagnostic points, refer to the pages on which these points are explained. Thus the student, when using the chart, may readily obtain more information about dubious points.

To use the chart to summarize the symptom-complex of any clinical entity, make separate lists of diagnostic points for each form or symbol in the vertical column under that entity, then write after each list as follows:

★ (list) "are of positive importance in this diagnosis."
+ (list) "are of positive importance, but not in all stages or in all cases."
± (list) "are sometimes present but are often absent."
O (list) "the presence of these would be contradictory to this diagnosis, so that their absence is important."
○ (list) "are usually not present, but their presence is not important."
□ (list) "the author has no opinion."

For instance, in complete rupture of the supraspinatus:

★ The type of accident, atrophy of spinati, crepitus, eminence on tuberosity, fluid sign, jog in motion, lack of scapulo-humeral rhythm, occupation, nocturnal pain, restriction from spasm, sulcus on tuberosity, tenderness over bursa and over teres major, bone atrophy, wince—are of positive importance in this diagnosis.

+ "Down-it-will-go," hypertrophy of deltoid, tenderness over lower deltoid, the X-ray signs of caverns, eburnation, excrescences, recession and other symptoms—are of positive importance, but not in all stages or in all cases.

± Dropping shoulder and neuritic pain—are sometimes present but are often absent.

O Frozen shoulder, sensory changes, restriction from adhesions, ankylosis or contracture; swelling or enlargement, calcification—the presence of these would be contradictory to this diagnosis, so that their absence is important.

To use the chart to find the clinical entities which any one symptom may indicate, follow the transverse column, making separate lists of the diagnoses above each kind of symbol. Combine these lists as before with the same sentences, using the singular instead of the plural, and *vice versa*, thus:

★ A jog in motion—is of positive importance in these diagnoses—dislocation of the long head of the biceps, fringes, villi and bands, complete rupture of the supraspinatus tendon.

\+ A jog in motion—is of positive importance, but not in all stages or in all cases of—subacromial bursitis, calcified deposits, incomplete rupture of the supraspinatus, benign giant cell tumor.

± A jog in motion—is sometimes present but is often absent in—congenital conditions, fracture of the tuberosity, fracture of the scapula.

O A jog in motion—the presence of this would be contradictory to these diagnoses, so that its absence is important—cervical rib, herpes zoster, hysteria and allied conditions, neuritis, periarthritis, sprains or strains, tendinitis, tuberculosis, vertebral lesions, visceral lesions.

○ A jog in motion—is usually not present but its presence is not important in—acromio-clavicular arthritis, scapulo-humeral arthritis, Charcot's joint, cord lesions, dislocations, hygroma, myositis ossificans, paralysis, osteomyelitis, etc.

The chart is capable of presenting a whole chapter on differential diagnosis, as the reader may prove, by writing out after this fashion the information contained in all the vertical and transverse columns.

The last transverse column shows none but + marks, indicating that there are other symptoms not typical of shoulder lesions but which help to clinch the diagnosis, such as the lead line in "paralysis, other forms," dissociated anæsthesia in syringomyelia, ecchymosis in the various fractures, the blood conditions in syphilis, tuberculosis, osteomyelitis, etc.

The last vertical column leaves room for the addition of new diseases, or of some of the rare ones briefly mentioned in the text.

There are of course many imperfections. For instance, the list of entities should not contain synonyms or terms partly synonymous, such as "tendinitis" and "periarthritis." Nor, in my opinion, should it contain "brachial neuritis." I have dared to leave out "rheumatism" and "neuralgia," but the believers in an idiopathic "neuritis" are too numerous to ignore. Its only symptom or sign is pain along the nerve trunks, and this symptom is a very common one in other conditions which can readily be relieved by treatment on the bases of other diagnoses!

Unsatisfactory as this list may be, I challenge any student or professor to produce a better one, for our science, as yet, is too imperfect. Who can make a list of the diseases of this or of any other part of the body without having the nightmare of symptoms appearing as diseases called by shifting, unstable synonyms? At any rate, it offers a form of mental exercise for any one who may be interested in lesions in and about the subacromial bursa.

AN EPILOGUE

THE ETHICS OF ADVERTISING BY THE MEDICAL PROFESSION

Advertise.

(1) To give notice, advice, or intelligence to; to inform or apprise; —followed by *of* before the object of information, as, to advertise a man of his loss.

(2) To give public notice of, or to describe with a view to sale or recovery and the like; as, to advertise goods; to advertise a runaway.

Synonyms: To apprise; inform; make known; announce; proclaim; promulgate; publish.

Webster's Dictionary.

THIS EPILOGUE is addressed particularly to those members of the American College of Surgeons who originally subscribed for this book. It is, therefore, like the preface, somewhat intimate, although quite as scientific as the meat layer of this literary sandwich, for it is also an effort to trace truth as it dodges about among appearances or hides in plain sight behind respectable customs.

Our profession is being more or less justly criticized in our own journals and in the lay press because we have provided no satisfactory method whereby the layman may be promptly and economically attended by the particular specialist best qualified to treat each of his ills. One who practices any specialty will readily admit the truth of this charge against our methods, for he daily sees patients who have suffered greatly, both physically and financially, because they did not have appropriate treatment as soon as the diagnosis could have been made, perhaps months or even years before. The lesion which is the subject of this book offers a striking example. Every specialist feels as I do, that his cases should have been recognized sooner, and yet every one of us will admit that while he is treating his special organ, he may be overlooking some other ailment of greater importance in some other organ. Knowing this, he insists on a general examination by the patient's own doctor or he calls in a diagnostician to make one. Few true specialists will receive, for more than a short time, the responsibility of the whole patient. They insist that the general practitioner should be his major adviser and friend. As a matter of fact, this is not as altruistic as it sounds, for many of us would have to admit that we do not even know how to make a good modern general examination. And what would we do in case some dangerous or disagreeable condition appeared, such as a contagious disease, delirium tremens, insanity, an incurable condition or the

complication of financial irresponsibility? The specialist for these things is apt to be the family doctor. Constantly under fire from the specialists for not having a consultation sooner, the practitioner is, at the same time, censured by the patients for his tendency to call in a specialist. The specialist charges more than he does, but the practitioner backs him in so doing for the following reasons among others. He knows that the specialist, to achieve his position, has had to spend most of his time and energy in unpaid hospital work and perhaps in expensive travel to observe the work at other clinics, and very likely, also, he may have written a book! The doctor also wants the specialist to charge significantly more than he does, so that his own patients will not all run away from him. Not infrequently he knows what the patient ought to have done, and that the specialist's glamour can make the patient have it done, although he cannot, himself, make the patient submit to it. This problem has become an economic one, and the modern "pay-clinic" is one very reasonable attempt at its solution. The question which I want to discuss is whether any amount or any manner of advertising could help to better our present methods. Far be it from me to suggest *more* advertising, for my waste basket daily receives material, which, if converted into cash at cost price, would easily support one person.

First let us consider what ways we have at present by which to apprise, inform or otherwise advertise a patient with a rare disease so that he may receive appropriate treatment, and *vice versa*, how may a doctor, who has given intensive study to some one condition, obtain the patients who need his help?

Our present *Tel-U-Where System* is made up of units scattered over our country and designated by Dr. or M.D. on conventional doorplates. Possibly his special field is also indicated on the doctor's sign, but this is not in general considered to be in good taste. We urge every person to attach himself or herself to one of these units as a permanent patient, so long as mutual trust is maintained. Every one should have a yearly physical examination by his physician, and if found to be sound, be instructed as to how to keep sound, and warned of the consequences of any bad habits or physical weaknesses which his physician may discover. Should the physician, at any time, find a minor condition requiring treatment which he can give effectively, he may treat the patient and charge a fee, limited in a general way by the local fee table of the County or State Medical Society. If a specialist's service be required, the physician should either refer the patient directly to a specialist competent to treat the condition, or call the consultant to make or confirm the diagnosis, and to instruct

him, the physician, as to how to give the appropriate treatment. The consultant's fee is not so much limited by the local fee table as by general custom, and especially by the statement of the physician to the consulting specialist of the patient's circumstances, while to the patient is explained the reputed standing of the consultant among his confreres and in the public eye.

Thus, we, the profession, appoint the physician an arbiter between patient and consultant, for he should have, on the one hand, an intimate knowledge of his patient's financial condition, and of the consultant's standing and attainments on the other. Consultants are expected never to attempt collection of fees of which the practitioner has expressed his disapproval. Since a consultant often responds to the call of a practitioner for a patient who can pay little or nothing, the practitioner usually urges his well-to-do patients to pay a handsome fee to the same consultant. Some consultants habitually refuse to accept any fee until the practitioner has been paid his moderate charge.

The patient, having once chosen his practitioner, is supposed to remain the patient of that practitioner and to consult no other physicians or specialists without his consent, as indicated in a letter to the new doctor. This point of "etiquette" has grown up to safeguard the patient, for there may be facts about him such as previous diseases, nervous or mental history, or social complications—financial responsibility, etc.—which the new physician should know. Moreover, it is merely ordinary courtesy for the patient to be frank with the doctor he trusts, although it is human nature to try to avoid hurting his friend's feelings by showing a lack of confidence in his professional knowledge. The patient may, at any time, leave one physician and go to another, but he should notify the first physician before so doing. The second physician necessarily receives him in a different spirit, with less feeling of responsibility for the trust imposed, if he does not do this.

Now this system has grown up more by custom than by the plans of the leaders of the profession. The fact that it exists is because our ethics in general are simply the dictates of the Golden Rule. The system is a good one, if not an entirely practicable one. It works, in fact, almost in proportion to the tendency of mankind, laymen and physicians, to abide by the Golden Rule in their daily lives. It probably works more nearly perfectly than other forms of etiquette or ethics in other classes of people for two main reasons. First, because the physician usually chooses his calling from high-minded motives and secondly, because a patient's moral resolutions are usually highest

when he is sick. The devotion of doctors and nurses to a sick man or woman is seldom unappreciated at the time it is given, though it may soon be forgotten, especially if the bill has not been paid. "When the Devil was sick, the Devil a Monk would be."

However, the burden of these customs lies on the conscience of the general practitioner. In addition to the obligation to know and recognize the early symptoms of hundreds of different diseases and injuries, which may need a specialist's attention, he is expected to know the reputations of the individual specialists themselves, whose more or less superficially concealed advertisements he has little time to read in our journals. It is perhaps even more difficult for him to choose a specialist than it is for the layman to choose his doctor. In both choices, reputation for honesty and for training are of more importance than for personal attainments. The practitioner, in making his choice, has much help from knowledge of the hospital positions held by the consultants, as well as from the general standing of the hospitals themselves in the community at large. Hence, all of us wish to stand well on the staff of a renowned hospital. Just as a layman in choosing a doctor may pick his medical school, and then select the nearest graduate of that school, so a doctor may pick his hospital before choosing his consultant.

Theoretically this system is a good one, and thinking men among physicians, statesmen, educators and philanthropists are constantly and patiently endeavoring to strengthen it rather than to plan a new one. Naturally the unit of the system where the sign M.D. hangs, is the chief point to strengthen. This may be done in several ways. The efforts of many of the best minds in the profession are given to teaching medical students the impossible task of caring for these unit stations. They still (I think futilely) hope that men can hold such superhuman jobs. Some of us have aimed to raise the standards of the hospitals, so that incapable or dishonest consultants will not be given places on their staffs, hoping that in time the public will not be satisfied with consultants who do not have, and take, full advantage of hospital opportunity. A much smaller, but still an active group of minds, aim by legislation to raise the standards of education required for a license to become a unit of the system.

The great majority of those of us who are consultants or specialists, occupy ourselves with post-graduate education in the form of advertisements addressed to the existing units, in order to keep them informed of the advances in our specialties and of our own hospital positions and professional standing. These, sometimes altruistic, advertisements are delivered in person at great national meetings or

at small local clubs; in print in countless medical journals and as reprints of such articles. Occasionally our time hangs so heavily that we write a book. In all this flood of literature directed to these units of our system, there is but a small modicum which the recipient doctor can assimilate. Too much of it is to tell him how much we know. Moreover, much of this material, especially when it is as exact as we can make it, proves to be very ineffective as an advertisement of ourselves. Judging by my own experience my papers represent time and energy wasted, so far as bringing patients to my door is concerned, because most of my papers have been on unsolved problems. However, these meetings and articles do help us to educate ourselves and to stimulate our colleagues, even if only a hazy amount reaches the units of our advertising system. Quantity probably counts with them as much as quality, for few busy practitioners can have time for much more than a glance at the titles of our papers.

Of late, there is a tendency to advertise our clinics as a whole, rather than our individual attainments. This is probably an improvement in effectiveness and perhaps in ethics. In our community the Medical School of Harvard University gives a series of Sunday afternoon public lectures at which the university presents its good and faithful medical servants directly to the public and to the newspapers. I certainly approve of this. The Massachusetts General Hospital has repeatedly told the public of the advantages of the Baker Memorial, even mentioning the prices of the professional service. I approve, although this seems to be a challenge to the individual local surgeon to put under his sign "Clearance Sale; prices less than those of the Baker Memorial." He certainly cannot hope to compete with either of these organizations in newspaper publicity. Nevertheless, notwithstanding the prestige of these renowned institutions, the majority of patients still go to the ordinary Tel-U-Where System described above.

Both the public and the profession are so in the habit of reliance on the conscience and common sense of the family doctor, that the advertising of great institutions, or even the purely advisory one of Dr. Evans, cannot change the custom. I, for one, believe that the conscience of the medical profession as a whole is a little better than that of the average man, but we are human and must earn our livings. In matters of life and death our system works tolerably well, because procrastination is apt to recoil on the practitioner; but when it comes to the case of a patient with some small matter like a sore shoulder, each unit feels that he owes it to his family to try his hand at treatment, for he knows that most sore shoulders get well after a time of

their own accord, and he needs the patient's money. Moreover, such patients do not take readily to the advice to see a specialist; it costs too much.

To illustrate what I believe happens with our Tel-U-Where System in cases of rupture of the supraspinatus, let us imagine the progress of a patient with this lesion who seeks relief. If the patient came to a doctor who was equipped with an electric baker, the baker would be used. If the doctor had an Alpine lamp, that would be used, and so on, whether the equipment was an infra-red or an ultra-violet lamp, a diathermy set or other form of electric apparatus. If the doctor were an osteopath, cheiropractor or employed as a masseur, some form of manipulative treatment would be given, or, if he had cultivated a reputation for intravenous therapy, colon irrigations, organotherapy, hydrotherapy, heliotherapy, and so on, these methods would be used. If the doctor had faith in drugs, an alarming list of soporifics would be at his command to palliate the pain. Any or all of these methods might help the patient to bear his suffering, but none of them would be at all curative. If the patient went directly to a busy surgeon he would probably be referred to some orthopedist, or to one of the above "specialists," who usually sent his abdominal cases to that surgeon. If he went directly to a busy practitioner, he might be told "There! there! don't worry," given aspirin and forgotten until the next visit, when the drug would be changed. Even if he went to a very painstaking, conscientious practitioner, the latter probably would not recognize the condition, and would not know what specialist to call. Within a few weeks of consulting any of these doctors he would perhaps be referred for an X-ray, and as this would be practically negative, still no diagnosis would be made.

If the patient happened to be an employee, he would lose in these ways the golden opportunity which is present immediately after the accident, and as week after week goes by, gets progressively more unfavorable. In Massachusetts our Compensation laws allow the patient to choose his own physician, and oblige the insurer to pay that physician for at least two weeks—the golden weeks for diagnosis and treatment of most injuries. The insurance examiner seldom sees the patient for some time after this, owing to certain forms of red tape, so that, as a matter of fact, no really early diagnosis can be made by even the examining physicians of insurance companies, to whom the early detection of this injury is most important from the dollar point of view. Those who will have the best chance to detect these injuries and treat them successfully will be the men on duty in great industrial plants and in the accident wards of great hospitals,

but these men are often inexperienced. At present the orthopedic surgeon is the most likely practitioner to know that this lesion does occur, and to recognize it, but he seldom sees any kind of case until it has become chronic. Then, too, he usually has a physio-therapy equipment with a large overhead expense, and this might tempt him to procrastinate.

If the doctor who originally sees the patient is puzzled and does recommend a consultant, the chances are that such consultant will be one of his own ilk, or if not, some surgeon who is not interested in shoulders, but who has operated on a member of the practitioner's family, gratis, and for whom, therefore, the latter would like to do a favor. When in doubt, any doctor would prefer a consultant from his own medical school or from the hospital where he himself has been an interne, for his teacher is sure to say a good word to the patient's family in regard to his own abilities and standing. For most patients, a distant consultant is out of the question on account of the expense, and if the disease is rare or new, there is usually no one near-by who has given any particular study to it, so that the doctor calls in a friend, quite likely one of his own religion. I have said nothing about fee-splitting, for I wish to speak only of fairly conscientious practitioners, the units of our Tel-U-Where System, to whom, I insist, that at least the first of my claims should be advertised.

My Claims in Regard to Complete Rupture of the Supraspinatus Tendon

1. The lesion exists, is not uncommon, causes prolonged disability, has a clear symptom complex, and may be relieved by a minor surgical operation, if it is promptly done.
2. Since it occurs at a time of life when general mental and physical degeneration readily ensues from enforced idleness, most patients never do heavy labor again, even after their compensation ceases. Thus the economic loss is great.
3. In Massachusetts the cost in compensation for this disability in an individual case is as great as from any major injury. To the man incapacitated it *is* a major injury. One hundred such neglected cases cost us more than the entire gross income of the average doctor during his lifetime.
4. Since the lesion is important to the employee and to his family, to the physician, to the hospital, to his employer, to the insurer, to the industry and to the consumer, the above facts should be advertised to all, because the relief of the patients, as well as a great saving, largely depend on its prompt recognition.

5. Hitherto, for twenty-three years the burden of advertising it by the usual professional methods has been assumed chiefly by me, at an expense greater than all my earnings from treating such cases.
6. My advertising has been ineffective, for I have not yet had a patient referred to me immediately after his injury. Moreover, the operation which I recommend is as yet rarely done in any hospital in the world; in fact, the lesion, frequent as it is, is still unknown, much less recognized in many of them.

Evidently there is a dense wall between the employee thus disabled and the writer, who thinks he knows how to relieve him. The patient consults other doctors who have not studied the shoulder; while I earn my living by caring for other conditions to which I have given no more, and perhaps less study, than have other surgeons. Could this wall be penetrated by any form of advertisement consistent with medical ethics? I believe that these patients and their insurers need me; I know that I need their money. How may I advertise to get such cases? All concerned, from the patient to the consumer, have at least some reason for having the symptoms of the lesion, if not the discoverer, proclaimed. Should the medical profession improve our advertising system or wait for business to do it badly through politics? These are my problems and yours.

Business has now tried the experiment of Workman's Compensation for some years, and is only just beginning to see that a large fraction of the expense is due to carelessness, ignorance or simply lack of being up-to-date, on the part of those doctors who give treatment during the first few weeks after all injuries. The fact that only one hundred neglected or unrecognized cases of any curable lesion may cost as much as the average doctor's earnings in a lifetime may engage attention, and lead business to conclude that our system for the advertisement of our units must be improved. This may be their point of view, although the same facts indicate that doctors should be paid more and expected to accomplish more.

Is it my fault that my advertisements have been ineffective? Look at the list on the chart in the preface. I have usually advertised debatable questions in their early stages, and most of the fields I helped to plow have flourished, although I have reaped little of the harvest. There has been delay on this matter of early operation for shoulder lesions, because the field was rocky and difficult, and in an out-of-the-way region. If I am right, surely I deserve more help, and if I am wrong I should, by this time, have been proved to be so. There should

be some method of "put up or shut up," in such cases. I am quite ready to go to any great clinic for a practical examination. Collect for me fifty patients disabled for six months or more with injured shoulders, X-rays of which are negative, and I will pick you out several instances of complete rupture of the supraspinatus, demonstrate the lesions through tiny incisions, and if the patients wish, make an attempt to cure them, although the operation would be long overdue. And here comes the real difficulty with personal advertising. I cannot guarantee cure; on this fact rests our principle, which is misnamed an ethical one, that the doctor should not advertise in the public press. We would find an excuse quickly enough, if we could deliver our goods with a high degree of certainty. We would even consider such advertisement a duty.

There should be some method by which claims can be tested before they are advertised, even to the general practitioner. Obviously the first checking should be at the hospital where the work is done, the detailed records are filed, and where the patients have been examined also by colleagues. The staff of the hospital, when convinced of the value of the contribution to clinical science, should recommend it for confirmation by the staffs of all other hospitals. When these have sufficiently agreed to the essential claims, they should be transmitted to all those who practice medicine, by some authority constituted for that purpose. In England this might be done through the panel system, but in our case it would have to be done through some great professional organization. The idea is present in the Year Books, but the material in them is not checked or corroborated in any way; it is only abstracted at the discretion of a few busy editors. It would be very unreasonable to hold doctors responsible for not reading the Year Books, but if there were an annual number of a great medical journal, which listed all important innovations *accepted by the hospitals*, all doctors might be expected to inform themselves on the practical details of each certified innovation.

If some such plan had existed twenty years ago, when I published the paper quoted on pages 126-129, by this time there would be thousands of workmen who would have benefited by it, and a vast expenditure would have been saved. To be sure, my contributions would have played an insignificant part among the many advances which have come during this period, such as Graham's dye, the Bucky diaphragm, insulin, liver extract and a hundred other far-reaching innovations of a striking and generally applicable nature. However, a shoulder which is too weak and sore to permit him to work, is just as important to an individual employee and to his insurer, as is a disability from

any interesting and spectacular cause. The more trivial the condition and the more study required to understand it, the more need of organized effort to spread the news about it. There is still much delay in diffusing the benefits of even the great discoveries.

We may now consider the questions of who may be interested in having such advertising done and whether they are doing anything about it.

1. Obviously the patient's interest in any plan is the first consideration, yet his share in diffusing the details can be but a very small one.

2. The practitioner can do somewhat more by telling his fellow practitioners about an individual case or two, but unless the condition is common and readily recognized, his chance of meeting another doctor who has such a patient is small at best. Like the patient, he may readily be deceived or may deceive himself.

3. A hospital does not concern itself with advertising its ability to treat new conditions, until the staff begins to see some profit in so doing. Yet a hospital, once it established an End Result System of organization, would be in the best position to answer any patient's question, "What doctor obtains the best results in conditions like mine?"

4. The employer does not advertise the new lesion; he gets another man and regrets the expense and trouble.

5. The insurer shuts his eyes to most medical aspects and does not even estimate the cost of errors of ignorance, carelessness and of lack of being up-to-date, on the part of the physician giving the first treatment. He concerns himself chiefly with distributing the cost of all errors, whether avoidable or not, between the industry and the consumer. When he once realizes that many disabilities are preventable, he may see his way to helping our profession to advertise new methods which may prevent them.

6. Industry is already writhing with the excessive cost of compensation; it vaguely thinks the doctors are making too much money; it does not realize that the cost of one hundred neglected cases would engage experts to treat a thousand, for it only thinks of the amount of its premiums.

7. Next to the individual patient, the consumer has the greatest interest, although he is unconscious of it. The patient feels the acute suffering caused by one neglected lesion, but the consumer bears his share of the total loss from avoidable errors in the treatment of *all* forms of accident. He could well afford to edit a volume to be distributed free to every physician to advertise approved steps in treat-

ment. As an example, the cost saved by the prompt treatment of two cases of rupture of the supraspinatus might distribute 1,200 copies of this book.

Actually, our present advertising is not done by any of the above parties for whose interest it would seem important, but by two others —the organized medical profession and the discoverer of the lesion— and our present methods are expensive and ineffective for both.

The medical profession, even against its apparent interest, has assumed a certain duty to present such information to its units, and, as we have previously mentioned, does this in a confused way, through its journals, its societies and its schools, for it is an enormous body, loosely organized at best. It has only recently recognized the duty of advertising to the public at large. The magazine *Hygeia*, the press articles of Dr. Evans and others, are highly commendable efforts to perform this function. Yet the knowledge which they endeavor to advertise often could not stand the acid test which I recommend, of subjecting claims to corroboration by hospitals, before they are put authoritatively before the units in a way which would compel the attention of every doctor who is in active practice.

To the layman the wranglings of the medical profession are so confusing that legislation can barely be secured to maintain proper boards of health, good standards of medical education and of licensure. Money can barely be obtained to permit health boards to advertise the public concerning serious epidemic and contagious diseases. However, I am confident that if the American Medical Association did publish a single annual number of its journal, with a *résumé* of tested and *accepted* advances in medicine and surgery, some one would see to it that such a publication was made available without cost to every doctor not a member of the association, and expect from him a certain degree of responsibility in return.

The Federal Government recognizes a duty to advertise the farmer in regard to the diseases which affect his crops and his cattle. It has laboratories for the investigation of these diseases. It is becoming more attentive to the diseases and injuries of man, but it waits for the medical profession to give some tested, authoritative list of *accepted* improvements in every field of medicine. Slowly it will see the economic importance of injuries and diseases which cause disability, even if they are apparently as trivial as is the one which is the subject of this monograph.

With one exception, we have briefly considered those who are chiefly interested in having new methods of diagnosis and treatment

diffused; that exception is the discoverer himself. It is human nature which is to be relied upon in his case, for his Ego usually impels him to seek money or fame. The pathfinders usually thrust the duty of advertising discoveries on themselves, for sometimes their own interests are involved. Even incredulity and opposition whet the ambition of some natures, and the self-imposed duty becomes an obstinate form of egotistic assertion. We enjoy the struggle to be believed. Such may be my own case.

If I could frankly and impersonally analyze my own feelings and ambitions in regard to bringing the lesion which is the main subject of this book to the knowledge of every general practitioner, I might throw a helpful light on the problem stated on the first page of this epilogue.

As a preliminary statement, I assert that I certainly should not have spent five years on this book merely for the philanthropic purpose of instructing the medical profession. It would not have been worth while, had I not felt impelled to use the subject to illustrate the End Result Idea, in order to point to the fact that any hospital which will follow up its cases of shoulder injury, will find instances of this lesion and be able to recognize and to relieve them, and that this would only be one instance among many. As a product of such analyses both the discovery and the discoverer would be automatically and effectively advertised.

I might have written this book twenty years ago, in a more active stage of life. I admit that while engaged with the far more interesting and varied experience of earning a living as a general surgeon, I feared that if I wrote a book on this subject, my friends (competitors) would specialize me. It is far more interesting when you get up in the morning to realize that today you may remove a gall bladder, a stomach or a colon, or do a circumcision in a millionaire's family, than it is to know that you will painstakingly do a fussy little shoulder operation just like one which you had done the day before, and the day before that, and the day before that. It is worse still to realize that meantime your reputation as a general surgeon is diminishing and the major surgical cases of absorbing interest are falling to your less studious colleagues.

To some extent my friends have thus specialized me, but I have postponed my decline by shirking writing a book on this subject. This I admit, but not with pride, for I regard my behavior as narrow-minded and due to silly human weakness. There is always a mental wrestle between the general and the particular. No ambitious man

cares to achieve in only one subject. We all want to be broad rather than narrow, and since few have the innate mental capacity to gain success in many or even in several paths, we become "jacks of all trades." However, it seems to me that a man is more likely to be happy if he follows the path he is fitted for, rather than to allow himself to dabble with the things that excite and interest him for the time. We have today thousands of surgeons, each doing hundreds of different operations on the same list, without having time to give thorough study to the anatomy and pathology of all the various regions of the body on which they are called to operate. For instance, most surgeons have paid little attention to the shoulder.

Each dreads that he may be specialized, especially in any minor, non-lucrative field, such as in that of this lesion, which does not occur among the well-to-do. To devote himself to such a thing would mean not only more study, but less pay. It is better to be known as a skillful operator, who may be assumed to be clever at everything, for the public knows little of the relative value of knowledge of anatomy and pathology as compared with dexterity, in determining the success of an operation. Thus it was perhaps worth while for me to write a few articles to draw attention to my ability as an observing young surgeon, but writing a book on the shoulder would have been very poor business, for, so far as my major surgery was concerned, my friends would have said, "He does only shoulder surgery."

Later (1928), when I laid out my plan to use this lesion as an illustration of the End Result Idea, it was at a time of plenty as shown by the chart in the preface. At that time my mind projected my ambitions in somewhat the following order:

1. To hasten better medical service to the public through improved hospital organization.
2. To illustrate to all hospitals some of the advantages of the End Result Idea.
3. To make the life of a doctor count more to himself and to his patients.
4. To enable great medical societies to be of more service.
5. To render our medical journalism more effective.
6. To make our medical education more logical.
7. To contribute to the advance of medical science.
8. To influence the H. M. S. to seize the E. R. I.
9. To help people with sore shoulders.
10. To obtain more such cases to treat.
11. To lay up money for my heirs.
12. To get some just to spend.
13. To enjoy my life.
14. Ego.

Now, 1933, turn to the chart again and behold the curves of income and their trends! I have not changed my investments for I have

neither bought nor sold, although I have lent and borrowed, and still owe more than I can pay. The rest of my book is actually in page proof, but I am confronted with a bill of five thousand dollars, for I had planned to have this peculiar book printed for that sum. Neither the printer nor I, both coming from generations of "respectable" people, who have prided themselves on paying their bills, dreamed that a condition would arise such that I might not be able to pay nor he to collect. Yet such is the fact, unless the book "sells." Thus I am compelled by circumstances, which have been largely out of my control, to invert this pyramid of purposes, although still, the satisfaction of the Ego forms the apex.

1. Ego.
2. To enjoy my life.
3. To get money just to spend.
4. To lay up some for my heirs.
5. To obtain more such cases to treat.
6. To help people with sore shoulders.
7. To influence the H. M. S. to seize the E. R. I.
8. To contribute to the advance of medical science.
9. To make our medical education more logical.
10. To render our medical journalism more effective.
11. To enable great medical societies to be of more service.
12. To make the life of a doctor count more to himself and to his patients.
13. To illustrate to all hospitals some of the advantages of the End Result Idea.
14. To hasten better medical service to the public through improved hospital organization.

Then, 1928, the satisfaction of the Ego was to be obtained by the broader purposes for which my effort was made, for I had money to spend and something laid up for my heirs. I was earning a living, and I was slowly teaching others what I could about shoulders. In like manner, my other ambitions were tending to become fruitful. Now, 1933, my Ego sees in large letters chiefly the narrow portion; to enjoy life, I need money, and I have none to spend. My fixed expenses so nearly equal my income that my heirs would receive nothing, should I die. I have even borrowed to the full extent on my War Risk Insurance. Beyond my immediate necessities, the other broader plans are rather hazy. Thus in these five years the subject of the ethics of professional advertising has become a very personal one to me. I must in some way earn my living or be dependent on others. It would seem reasonable that I should do so by treating patients with sore shoulders.

Now, then, my colleagues, I ask you how can I obtain such cases? You must admit that I have studied disabilities of the shoulder, more than any of you have done. Will you send me your patients? You have not done so in the past, for most of my cases have come through

a few personal friends. Or will you buy my book? I will admit that I rarely buy your books.

I feel that your answers will be negative, if not still stronger. You will say to me, "Leave out your outrageous egotistic preface, your insulting epilogue and your commonplace cartoons, and put your book in the hands of a publisher. He will call it to the attention of every doctor in the country, advertise you as 'the well-known authority,' and in every possible way, however flagrant, his agents will try to get their commissions. Leave it to him; don't soil your hands with advertising. Get your friends to write complimentary reviews for the medical journals, so that your publishers may quote the most florid phrases. Even if nobody buys your book, the salesmen will tell the practitioners throughout the land about you, for they can make no sales without praising your knowledge. Thus you will diffuse your ideas, and operations for your pet lesion will be done everywhere. You will soon have plenty of patients—many of them the failures of other surgeons, who have tried to follow your instructions. Make appointments so that there will always be some one in your waiting room, especially your most successful cases. Have your failures come by themselves, if they can pay; otherwise, don't see them at all. Stick to our regular methods and you will soon be an acknowledged expert, and your patients will expect to pay good fees. No layman appreciates a doctor's advice unless his waiting room is full and his fee is large. Make a success of your own practice, if you want young men to follow you. Efficiency! Nonsense! Don't be so foolish as to say you have spent five years in writing a book recommending immediate suture when you have never done it! Other people will mention your faults and failures enough. Don't write about them! Remember that in our business, as in many others, we must sell what the public demands. You cannot educate it to be End Result-minded in your time. People still believe in gods and fairies, in the serpent of the Garden of Eden, in the patent medicine advertisements, and in a hundred mystic cults. You will get no satisfaction when you are dead, for your memory would never be as cherished as that of Mary Baker G. Eddy or as those of the great spiritual leaders, whose psychic energy would make your helpless patients elevate their arms (unless compensation was being paid) and swear they had no pain! Be reasonable; treat the patient, not the lesion. Lie to him, and to his family if necessary. They expect it from you, just as they do from a political orator. Your own moderate success shows that you can be a humbug, when it is necessary for your personal comfort. Be moderate in your old age. Your friends and relatives

will be glad to have you retire, and go fishing, and so will your fellow members, if you will stop writing. The shoulder part of your book is all right; why not leave out this End Result Stuff?"

My answer is that to leave it out now would be to me like working for years on preparing a balloon for a long journey of adventure, and at the last moment having the builder insist on having my scientific apparatus removed for fear it would be too heavy, and prevent my return and the payment of the bill. The portion of the book on the shoulder is the balloon, but the really important part of the expedition is in the basket below, containing the preface and epilogue. I designed the balloon to advertise the End Result Idea, and I was planning to pay for the trip as a luxury, provided the whole affair was built on my specifications. It was my balloon, although five hundred members of the American College of Surgeons had promised to be backers to the extent of each agreeing to purchase a report of the trip for five dollars. I did not submit the manuscript to a publisher, for I felt there was little chance of one accepting it with the "sales value" destroyed by two cartoons, a preface and an epilogue, which ridiculed our most sacred medical institutions. This would have been his point of view, even though the central portion of the book might be an excellent and enduring monograph. He would point to that ugly truth displayed in the basket, that few hospitals have any individuals, committees or departments, whose duty it is to persistently investigate the results of medical and surgical treatment, in order to prevent waste products in the forms of unnecessary delay, suffering and failures in achieving relief or cure in each individual case.

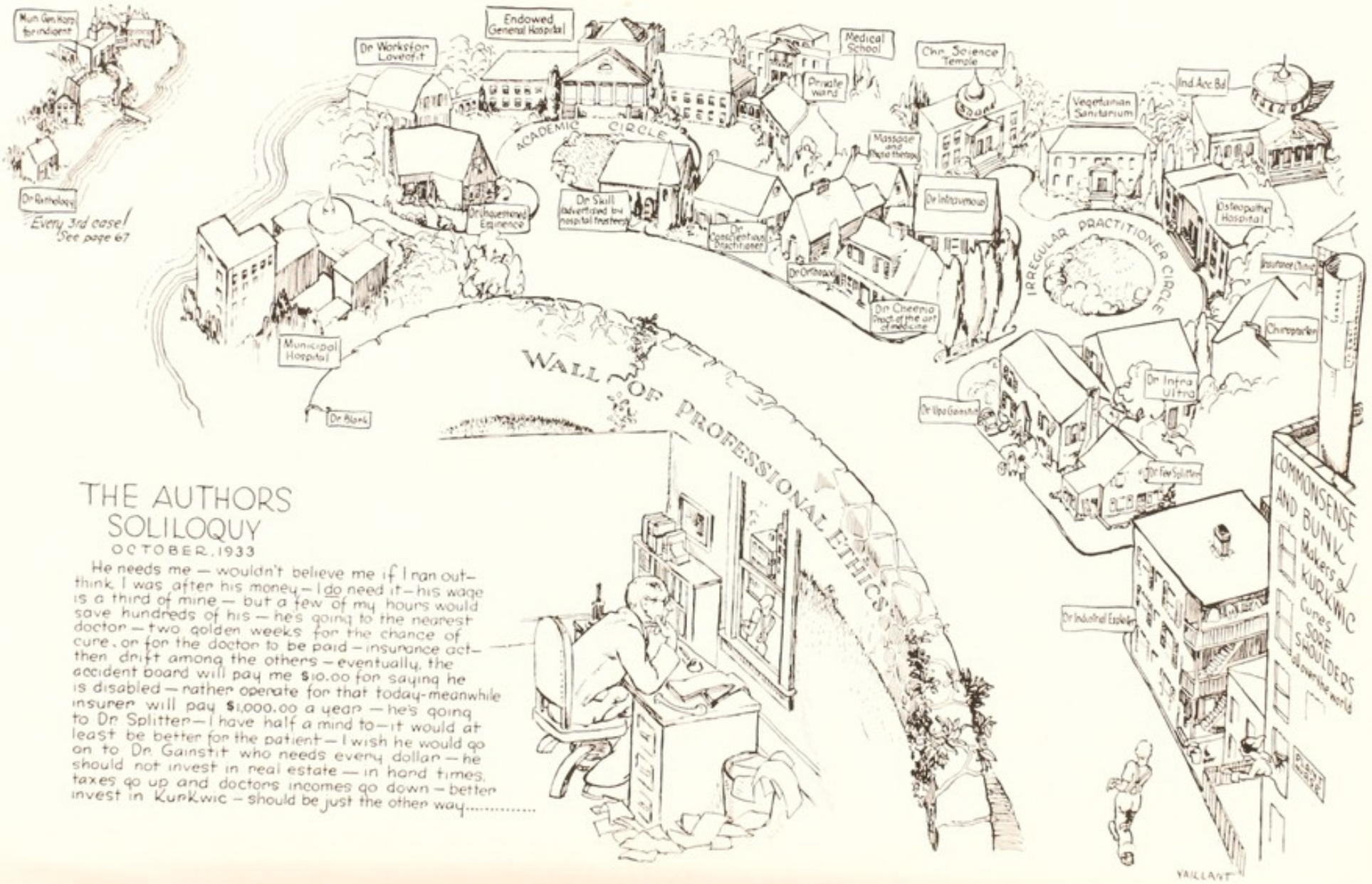
There has been a very earnest attempt to make the central portion of this book so good that it would be worth at least five dollars to each of the subscribers, and afterward go about the world for a decade or more as a standard work on the shoulder. I feel sure that it will be at least ten years before any other surgeon would, if he could, take the time and trouble to write a better one. The question is whether the buoyancy of the "sales value" of the shoulder part can carry, what seems to me, its far more important, though unpopular and heavy basket, which flaunts a banner at which presidents of endowed universities cannot afford to look, and even those of state universities would need their smoked political glasses, for the first shock. Even if the balloon floats across the sky, there will be but a small number of surgeons who will notice S H O U L D E R in large letters and care to study it carefully. However, it is not wholly through surgeons that I hope to plant my ideas in suitable soil where they may flourish. I must somehow reach those who think about hospitals in terms of dollars; those who arrange investments and engage auditors, execu-

tive officers who manage and make up budgets, and especially, the directors of insurance companies which pay the bills for our errors in diagnosis, in judgment, in skill, in care and in procrastination. Industry, through the insurance directors, must be made to realize that the cost of avoidable errors is greater than the cost of good medical care.

I must harp on the fact that only one hundred neglected cases of any lesion may cost them more than the gross income of the average doctor in a lifetime. When they realize this, the captains of industry, the bankers, the statesmen, the philanthropists, the politicians and the educators, will see to it that the hospitals take inventories of their products. My old slogan of the hospital standardization campaign: "Hospitals which do not take inventories of their product do not audit their accounts," will have meaning to these business men, once they overcome their mystic awe of our profession. They will ask questions of our trustees, superintendents and chiefs of staffs, and, after a time, will not be misled into thinking that new wards or new operating rooms are needed more than follow-up systems and efficiency analyses, as the successful physicians and surgeons would have them believe. These men, and they are good men, became successful under old ideas; of course they would advise spending a legacy on a new ward or operating room, rather than on a system to search out their personal errors. They want their new operating rooms and wards for more individual experimentation and publicity, and as training grounds to turn out more students "to take their responsibility as the physicians of the future." In other words, as soon as they are given their degrees, to begin to aim for the goal of increasing their reputations among the well-to-do classes, by the success with which they can evade responsibility for their errors, and give social publicity to their achievements. These are ugly things to put in the basket of my balloon for the enemies' guns to bring down amid the applause of the multitude, who have justly worshiped the old-fashioned, and now no longer possible, family physician.

Suppose that I could persuade some rich old lady to leave her millions to a certain hospital to be devoted to a constant efficiency analysis of the results of treatment—would the trustees accept the gift? Would she not be dissuaded by her lawyer and influenced instead to endow a new ward in her own memory?

Shame on me for thinking that doctors are as human as business men, bankers, insurance directors and politicians, and need periodic investigation! Shoot away at the balloon, down with the author, down with the End Result Idea; if the stuff he has written on the shoulder disappears in the sea, it is no great loss!



THE AUTHORS SOLILOQUY

OCTOBER, 1933

He needs me — wouldn't believe me if I ran out-think, I was after his money — I do need it — his wage is a third of mine — but a few of my hours would save hundreds of his — he's going to the nearest doctor — two golden weeks for the chance of cure, or for the doctor to be paid — insurance act — then drift among the others — eventually, the accident board will pay me $10.00 for saying he is disabled — rather operate for that today-meanwhile insurer will pay $1,000.00 a year — he's going to Dr Splitter — I have half a mind to — it would at least be better for the patient — I wish he would go on to Dr Gainstit who needs every dollar — he should not invest in real estate — in hard times, taxes go up and doctors incomes go down — better invest in KurKwic — should be just the other way.....

—Old Commonsense, who makes KurKwic is probably a hospital trustee—his business ability is respected—perhaps insurance director too—understands people and only takes Bunk as a necessary partner—would tell me thats just what doctors do—he's a sensible man on the whole—would tell me that his liniment would at least do no harm and my surgery might—he's right—under present hospital conditions—knows surgeons try everything new—pays us well if we attend his own family—knows we've learned by experience—(on others)—there goes that man into I.P. Circle—I wonder if old Bunk—or the factory nurse—advised—perhaps her brother-in-law's aunt swears by Dr Ultra—more likely Bunk himself—such canny old fish are often the best medical suckers—I wish that man would go to my conscientious classmate Dr C.P.—he would at least recognize whats the matter—to be sure he would not send the patient to me—knows I'm not in Academic Circle—yet he likes me I know—thinks I'm too cranky—poor chap he has to keep up with too many things—reads all the journals and refers all his patients to specialists—gets neurasthenics and moribunds in return—almost an angel himself—he had better stick by his academic friends—on the whole they are the best—although they won't come to a showdown—if that patient strays into their free clinic—it won't be Dr Skill or Dr Eminence who will see him—some hurried young assistant—who won't profit by the experience—until the trustees insist on a careful follow up system—our Academic Circle and I.P.Circle are close competitors—compare their temples—they will be—until we compare results—probably a draw at present—they may save as many by not interfering with nature as we do by butting in—I know a lot about the shoulder but a comparison of results might be in favor of any cult—their check books, yes—but could they write an intelligent book—here I sit and write one on that man's injury—in debt for it—my competitors would not study it as a gift—no show window, as when I had a charitable hospital racket—charity!—why not sell hospital positions—I'd pay for one to treat shoulders—consulting surgeon means "skill living" and little else—it seems hopeless at my age—yet—even if I cannot get patients I may advertise my lesion—if I can only get the A.C. of S. to appoint a man in each hospital to study it—my claims will surely be confirmed—might sell my book,—the insurance companies saved thousands—think of the pain and worry the patients would be spared—satisfy my instinctive desire to be of service—and not to be dependent—yet—if I advertise boldly the college must take some action—if they ignore this book, it will still stand as evidence—I know I'm right—only a few pages about some of our great taboos—to suggest their relation to analyses of results—advertise that I want a job—2000 hours to sell at $3.00 an hour—take less, if I liked the work—inventory of the results of some big general hospital—secretary of a committee to list recent advances in surgery—president of great national intelligence office for competent doctors—some insurance company might employ me—study costs of errors in diagnosis—especially those due to ignorance about new advances—I must earn in some way—almost any way is better than to sit here and see my pet lesions pass my door—23 years to collect 39 such operations!—only averaged about $25.00 for each—and tell the world about it!—one among the thousands of conditions a doctor should recognize—yet—as an example of many such picked up by the end result system—why write a book on such a little subject while in all the countries of the world the social structure has an earthquake balance—well—whatever the future—the E.R.I. must eventually be used—its commonsense as well as clinical science—what bondalog we are—better to leave our descendants instruction books about the care and repair of their inherited machines than to pray for their souls and let debt accumulate—to be sure, they won't bless me, but will damn the supraspinatus—when they break down on the road—if I had a patent it would pay to advertise—now I must pay to inform my competitors—however it will need many more experiments to perfect the best method—many millions of men and women will have to help to make the E.R.I. useful—yet—one rich man could hasten its coming by endowing one hospital with an E.R. fund—or one university president could be its sponsor—one hospital administrator could educate his trustees the American College of Surgeons will do much—after I have gone—how much easier it is to do something for future generations than to get money to spend in this!—yet—today—thats the first consideration—for me at least—this book (when paid for) and 2000 hours a year, for a brief and uncertain time are my chief assets—yet—many and many an old doctor after greater service to this generation is worse off than I am—and through no fault of his own—conducted my finances conservatively—(except for showing a cartoon at a medical meeting) but the depression and this book have put me in debt—in spite of my handicap of advantages and opportunities my earnings are only average—according to Committee on Cost of Medical Care—they tell us that 80% of our work can be satisfactorily done by the family doctor—how do they know—I say 80% could not be treated satisfactorily by all of us put together—they mean get away with 80% without arrest—they don't say enough about specialism—why a man who limits his practice to one field should charge so much more than a man who can treat 80%—perhaps because we cannot advertise—if I could get enough shoulder cases I could cut the price—as it is I cannot get enough to pay for this book—yet people insist that I'm a specialist—at any rate the Committee was nearly right about my income—it has been very nearly $6000 as they calculated—just a little below the average—in my own humbug—they did not say enough about the cost of wandering from one doctor to another—look at the number of doctors that poor fellow will consult in the next five years—the visit to each one counts among the 80% that each doctor can get away with—calculate the total cost of lack of advertisement!—if the Committee had studied in detail the patients who were treated in one large general hospital for one year their report would have—but they had to avoid hornets nests—you can't expect anybody to deliberately walk into hornets nests, if they know where they are—I, for one appreciate the work they did—I'm sure they agree that the cost of bad medical care is enormous—even if they fail to realize that you cannot know what is good care until you have an End Result System in the hospitals—two prompt operations for a ruptured supraspinatus tendon would save my salary for a year—the rest of the time I could wash instruments—I'm ready for technocracy—indeed the E.R.I. would fit into any scientific plan, although it seems unsuited to capitalism—doctocracy would be still better; for we are more needed than gold or technologists—yet—we still let the bankers lead us by the nose—we pay our taxes and also treat the poor, poorly—for nothing?—well—at any rate that man may turn up at the municipal hospital for the indigent—and—after death—prove that I am right—when will industry and pathology cooperate?—Akerson has saved his salary over and over again by what he has done—but only for future generations—for years I have received $10.00 for my opinion about any single patients shoulder—can I get $10.00 a copy for this book about all sore shoulders?—I wish I could give one to every practicing doctor—but must earn my living—no more philanthropy until out of debt—perhaps someone with greater optimism will buy copies—send them to university presidents, deans of medical schools, hospital trustees, insurance directors—I've done my bit—I'll watch my own tel-u-where station—treat anything from a cold in the head to major surgical cases—thats not disgraceful like advertising—compromise with humbug until this book is paid for—then for a farm in the country—by some lake—bass or trout—ducks—near the seashore—clams—lobster pots—an old Ford—a good dog—no telephone—but first—I must offer those resolutions before the Board of Governors—Oh! well—

As a matter of fact, I think I have a higher ideal of the character of the doctor than has the general public. If I did not believe that most doctors choose their profession in order to be of service, I would not appeal to the members of the American College of Surgeons. I believe we are much more interested in keeping our profession clean than is the public. The rich old lady wants to have her dear doctor at her bedside to inquire about the condition of her bowels, rather than to have him operating on a difficult emergency at the hospital, especially if the interne, who is taking his place, is her nephew, who is learning how to be a prominent surgeon. Under present conditions one cannot blame her and can only praise her nephew, if he seizes the opportunity.

It is because I do believe that there is a chance that the American College of Surgeons may adopt some of my ideas that I have continued to appeal to its members, and have, as a last effort, addressed them in this volume. The first printing of a thousand copies will be exclusively for them, but if my claims in regard to the supraspinatus are confirmed, I may eliminate the objectionable portions, and perhaps be able to sell the rest of the book to a publisher. However, at least for a time, a thousand copies of the balloon will float about our private sky to try the marksmanship of my friends. Let them think before they aim at either the balloon or the basket, for the balloon is, for a time, invulnerable. There is no use shooting at it with mere abuse. Ignore it, do not point to it, distract every one's attention from the banner it flaunts, but don't shoot at the basket—it might fall on you before you are ready to receive it. And besides shooting at it will only call attention to it. A few holes in the banner will do no harm, for the material is simple homespun truth. As for the balloon itself, the only missile which can bring it down will be a better book on the shoulder, and to write that will take some time. So long as it is the best book on the subject, it will carry its unpopular load, for doctors themselves sometimes have sore shoulders and will consult it in spite of the cartoons. Now and then some layman will find that although the shoulder part must be studied, the rest need only be read.

Who knows but that some day a copy may be dusted off in a library and shown to some lonely old hospital trustee, who has money which he may bequeath in order that an annual inventory may be taken of the products of his hospital. I am convinced that if a single great general hospital once did this thoroughly, the others would have to follow. Perhaps I might have made more use of my own life, if I had devoted my energies to exploiting some patent medicine or breakfast food, in order to leave a fortune for this purpose!

To make my suggestions more concrete, I submit the following plan to the American College of Surgeons:

(1) An annual letter to every approved hospital, asking for a list of the original contributions of the staff, which they consider should be known to every general practitioner.

(2) A Committee of the College to receive the answers to this letter, and to select from them a limited number of subjects for study and confirmation by other hospitals.

(3) A report from the Committee to all hospitals giving its selected list for confirmatory study, and also the entire list stating that confirmatory studies of the unselected innovations would also be welcome.

(4) On receiving these lists, each hospital would assign one or more members of its staff to give particular study to each of the selected subjects, and call for volunteers in the others. At the same time the staff would give authority to members who accepted these assignments to treat all such cases coming to the hospital, irrespective of what wards or on which services the patients might be admitted.

(5) At the end of a year or more these chosen members of the staffs would make critical reports to the Committee, confirmatory or otherwise of the originator's observations and claims.

(6) The Committee would then, in coöperation with the Committee on Scientific Meetings, arrange for discussion of the selected subjects at the annual meetings of the College, giving the originator in each case the opportunity to read the central paper, which would, of course, appear in *Surgery, Gynecology and Obstetrics*, together with the discussion it provoked.

(7) A single annual issue of *Surgery, Gynecology and Obstetrics* to be edited for the general practitioner, giving statements of those advances of which he should have knowledge, and the names of those in all hospitals who have confirmed these advances, and have thus fitted themselves to treat such cases.

(8) An announcement in this special Journal to the members of the profession not affiliated with hospitals, that original ideas which they individually may have, will be listed for investigation, if, in the opinion of the Committee, the ideas are important and the originator's claims are endorsed by other responsible individuals.

Among the advantages of such a plan would be the following:

(1) *The effect on individual hospitals.* In many hospitals at present there is little or no effort made to contribute to medical

science. Usually this inertia is due to modesty, but even in a most remote place, there may happen to be individuals who have unusual capacity to do original work, or, at any rate, a willingness to do their bit in helping to verify original work. Such men would be stimulated to make efforts which they do not now make at all. Their confirmatory work might be of great value, and would be sure to be of some value. Patients in their communities would benefit by their study, as would also their colleagues.

Hospitals which already have reputations would be eager to justify them. Such hospitals are usually manned by young men who desire to contribute to medical science; this plan would relieve these men of the often distasteful and expensive work of making their contributions known to the practitioner, who, in the end, will be the one to use them. Moreover, there is a self-depreciating type of man who must have his publicity done for him, and his hospital will want the credit for his work and make him present it properly. Another effect would, in my opinion, be still more important although less obvious. I think this plan would increase the spirit of coöperation in a hospital staff. The spirit of coöperation among the staff members of small hospitals is often high. If one of their number were willing to take part in such a research, either in presenting an original communication or in coöperating to verify one, I believe that others would give him all the help they could, not only at the hospital, but by asking him to see their private cases, for it is obvious that most men cannot devote much time to such studies without some thought of sooner or later profiting by them.

It is not unlikely that some of the best material would come from hospitals with no academic affiliations or time-honored traditions, for the cases treated at such hospitals are just as varied and interesting, and offer as much to the original mind, as do those at the most famous clinics. Some geniuses who are hidden might be thus revealed. The main objection would be more work, but this would be voluntary, and, in my opinion, would be welcomed by a few members of each staff. Of course there would be men who would shirk doing their bit, and this might create some hard feeling, but I do not believe that the total amount of hard feeling would be greater than at present, for this depends on the friction of characters rather than on facts.

(2) *The effect on the general practitioner.* One can hardly doubt that if next year the College asked every approved hospital to make an investigation of all shoulder injuries, that, at the end of the year most of the practitioners in the country would know at least that such a lesion as is the main subject of this book does occur, a fact

which certainly many do not know now, even in this community, where I have talked about it for years.

In the first place, in each hospital, the responsibility of studying the question would be assigned to one man, and he would soon teach the salient points to the other members of the staff. If he examined the bursæ at all autopsies, he would soon be able to demonstrate the lesion. When he had learned the local anatomy, it would not be long before a clinical case could be found; he would operate, and his colleagues would have a chance to see the lesion and observe his technique. At a meeting of the local medical society, he would show the case, explain the diagnosis and findings, and review the subject in general. He would accentuate the importance of prompt diagnosis and operation, and urge every one to be on the lookout for an early case, explaining that the College was asking for a general research, and he was doing his bit for a year. Practitioners generally take a certain pride in bringing to the hospital "interesting cases." Even this searching method would still not reach a considerable fraction of those who practice medicine. In most cities, and perhaps in many towns, our professional meetings, although open to the medical public, are not well attended. One reason is that the multiplicity of unauthoritative and often impracticable papers has worn down the enthusiasm of the local doctors, and what little desire they may have retained to keep up-to-date. I believe that sparks of interest would be fanned into flame, if they realized that we were making a national effort to study for a time a particular class of case. Then, too, they would know who to consult if a case turned up in their practices.

(3) *The effect on the patient.* The College was organized for service to ourselves and to the community at large, and it has had a remarkably successful record in so doing, particularly in the latter object. Service in advancing surgical science and in giving the community the benefit of that science efficiently, honorably and reasonably, has naturally been our chief field of endeavor. Service to the patient is the central idea, and insisting that hospitals ascertain the degree of service rendered, in order to constantly improve the value of service to be given future patients, should be almost as sacred a principle. The plan I have suggested is merely another phase in the campaign in which these two banners are carried. The present activities of the College have the same general purposes. The Committees on Fractures, Malignant Disease, Registry of Bone Sarcoma, Industrial Surgery, Scientific Meetings, Publications, etc., all aim to put at the service of the public, through the general practitioner, the best which surgical science can give. These efforts are expensive, our dues

are already a burden to many of us—why add another item to our budget in these hard times? Certainly there should be good reasons.

(4) *The effect on the College.* Apart from the satisfaction of performing a great human service, there would be certain points by which our present activities would be increased in value. For instance, the general meetings of the College at which the subjects of joint research would be eventually discussed, would be vastly more instructive and entertaining than under present methods, by which we listen to constituted authorities. To hear an authority on a subject of which one knows nothing, can never be so interesting as to hear him speak about something on which one has had some opportunity to form one's own opinion. If one's hospital has been doing its bit, each member of its staff will inevitably have some opinion, even if he himself has not done the work. Indeed, at the end of a year or two, when the subject is no longer an assigned one, every member of the staff would have to care for his own share of the cases in question.

The quality of the papers eventually presented in print would be better, because under the encouragement of their own hospitals, and in the limelight, the readers would take greater pains. Not only would the quality of the papers improve, but those of poor quality, read for purely personal advertising purposes, would tend to diminish in quantity, because discouraged by lack of recommendation from their own hospital staffs. Pointless papers read by local authorities would be less apt to be committed to print. Then, too, opportunity counts. For instance, I have no doubt that a surgeon for some great mining plant would see as many instances of rupture of the supraspinatus in one year as I do in ten, and could make a more informing study on that account.

Finally, the annual publication of one number of *Surgery, Gynecology and Obstetrics*, edited with the idea that it is to go to a vast number of general practitioners and contain only the salient points of new surgical discoveries, would be an interesting volume to all of us, as well as to the practitioner. It would chiefly concern itself with the diagnosis rather than with the treatment of the lesions in question. Results, supported by the reputations of the hospitals concerned, would be dwelt upon, for the practitioner needs to know what his patients may expect, rather than long descriptions of pathologic appearances, technical procedures and erudite theories such as most journals now contain. It would also give the practitioner a list of those who had taken part in confirming investigations, and this would serve as a directory to the nearest local consultant, who would be up-to-date in regard to that particular question. The authoritative and

relatively impersonal character of such a volume would intrigue, if not demand, their interest. It would contain also those portions of the reports of the special Committees of the College, which would be of interest to general practitioners. It would attract notice in the daily press.

Perhaps our example would be followed by the American Medical Association, which might issue a similar number regarding *accepted* medical advances. Such a volume might take some share of the attention which practitioners now give to the commercial druggists' advertisements, the volume of which proves that they are now read. In fact, some of these advertisements are perhaps more truthful and scientific than some of those which we publish as "papers." Our Tel-U-Where units need help. How can they function as advertising stations and individually select what is worth advertising? For instance, what does the general practitioner know of the early symptoms of poisoning due to the modern, complex chemicals which are of ever-increasing use in industry? The unexpurgated deluge of medical articles through medical journals and the bulletins of commercial druggists, to the abyss of proprietary advertisements, is constantly increasing. The most acute practitioner cannot distinguish the wheat from the chaff, and little organized effort is made to help him. He cannot even afford all the Year Books, or understand them if he reads them. In his confusion he finds it better to adhere to the old methods he was taught, years before, in the medical school. I need say nothing more of the evident importance to our national economics and to industrial insurance, which might result from some such plan as I have outlined.

Arguments have been presented to show the need and the possibility of a form of advertisement consistent with reason and with our ethics. What of the counter arguments? I have been unable to think of any of consequence, except the financial one and the difficulty which might be experienced in getting coöperation from hospital staffs. The former is clearly not a serious one, for a cost price could be charged for the single annual copy of the Journal, but I fear the latter may be. Surgeons are loath to make changes in their habits of work. We all like to try our hand at each new operation, and the idea of dividing up newly-discovered and interesting cases among our colleagues for purposes of intensive study, is not welcome to us. Those of us who have the gift of being successful, seldom have the time for intensive research and like "to try anything once" with a minimum of study and record of results, so that we may be somewhat prepared to use the experience for a private patient. To choose one

of our colleagues to study a series of cases for us and for the College, even though only for a year or two, would require a certain magnanimity. Yet surgeons have done this. The staff of the M. G. H. has now done this for many years, as I have explained in the preface. I can think of no other valid objections, except the question of an initial power sufficient to overcome the existing inertia in order to give momentum to this plan.

You may not approve of my suggestions. You may not even take the trouble to test the statements made, after our usual professional manner, in the central portion of the book, in which there is scarcely a chapter without one or more original observations which need confirmation by other students, before they should go to the practitioner. Yet, sooner or later, according to our usual customs, you will try by haphazard human experiments the operations which I recommend. You will do these experiments individually, without careful record or publicity of the failures, merely to satisfy yourselves that you each can make the diagnosis and accomplish the technical procedure. Why not try an experiment now, with this one relatively unknown form of injury to see whether by an organized effort you can test my claims and then diffuse those ideas which prove to be important? Would this not be more sensible than to permit me to write a book for a publisher to broadcast as authoritative, or to allow one of our colleagues to say in a respectable medical journal, that the intravenous injection of a certain drug is 100% effective in bursitis? Your present ethics encourage us to have such advertisements.

You may object to my personal form of presenting this problem and tell me to try the drug which my colleague recommends so highly, for my own analysis shows that my results are far less satisfactory than those which he describes, except in the milder forms of bursitis, which get well soon with no treatment but rest. Well, then, use his claims to test, and afterward to proclaim. Perhaps the makers of the drug will contribute the expenses for the experiment. Let them have your authority, as well as that of the prominent surgeon, whom they are now at liberty to quote in their publications to the practitioner, without any infringement of our ethics. If the fact is confirmed, it should have as wide a publicity as possible. Dr. Richards tells us that he made the discovery of this remarkable cure by accidental observation in a case where the drug had been given for other reasons, but promptly relieved a coincident bursitis, from which the patient was suffering. The surgeon repeated the experiment in seventy other cases with the same success; then he felt that he should

let others know of this simple procedure so that they might relieve their cases. He can no more be blamed if the makers of the drug advertise him to the ends of the earth, than I may be, if a publisher advertises me.

I might give you many other instances of innovations which, *if true*, should be broadcasted and which you may substitute for what I have to offer. It is safe to say there would be a hundred such in the journals which appear every month. You will say that you cannot investigate all these. I do not ask you to—only those on which the writers have the endorsements of the staffs of their own hospitals, and of these, only those selected by your committee as especially worthy of transmission to the general practitioner. You may rely on the combined staff of a hospital not to recommend the work of one of their number unless they are proud to do so. They are in the best position to know whether his work is sincere and accurate. One's colleagues are very critical; indeed, I think the danger would be that the annual reports from many hospitals would be negative as to new discoveries, for their reputations would be safer, if they kept the lid on boiling enthusiasm. The pressure of a real discovery, however, would soon be too strong for the combined efforts of jealous colleagues to resist. A young man could afford to take time to convince his fellow members, for after that was once done, he need not concern himself about convincing the rest of the world, piecemeal, as he must now. It is partly because I have had uphill work in fighting for my pet lesion that I desire to make a path for other more able and less obstinate young men, who are willing to work, but are too modest (or too poor) to battle for their discoveries. My circumstances have been such that I could afford to fight. If I had had children I could not have devoted so much of my life to such luxuries.

You may say, if you please, that I have written this book for my personal gain. I have. You may say that I want all hospitals to investigate shoulder lesions, so that I can sell a copy of this book to each hospital. I do. You may say that I want more consultations and operations on cases of shoulder disability. I do. You may say that I would be glad to have more surgery of any kind among well-to-do patients. I would. Does not your "shingle," like mine, tell the world much the same things? Such accusations I will not resent unless you allege that I have written this book *wholly* for my personal gain. I insist that you credit me with at least the ambitions listed in the order of my pyramid with the apex up, no matter how hazy the base may be in these hard times. Write down your own motives and compare them with mine; are they in a very different order, especially toward the apex?

Your signs of Dr. or M.D. tell the world that you are units of our advertising system, which should enable patients to reach appropriate doctors. Are they?

If you are a great statesman or scientist, the base of your pyramid may be so large and well-proportioned that it would form a sphere about your ego, while mine is not symmetrical, for it extends only in the portion limited to the surgical field. I feel no call, as must the president of a university, to study deeply into politics, law, finance, journalism, philanthropy, or into the many other worthy fields of human endeavor. My limited brain is fully employed in my relatively small field. You may be more conservative, and, avoiding all detailed study, feel that you do your bit by being a good-natured deadweight on all enthusiasts. You may feel that you are leading a more rounded life, but your ego will still be the center, and the circumference of your sphere perhaps be smaller than that of my unsightly pyramid, if I can extend it sufficiently in one section. On the other hand, your sphere may be large and still symmetrical, if you are always ready to give an ear to those who are devoted to special forms of service, and to champion them, if the evidence they have accumulated appeals to your intellect apart from your prejudices and emotions. If you are such a person, I crave your help, for it is mainly by convincing such as you, that such as I may succeed.

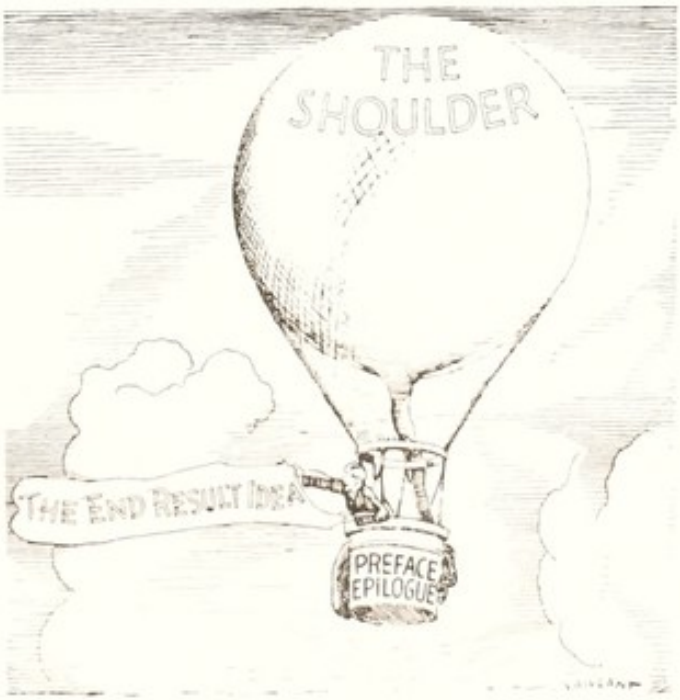
Thus for the present my ideas about the shoulder, as well as about advertising, are in the hands of those of my colleagues who have subscribed, or may subscribe, for this book. A thousand copies will not travel far without a publisher. You may prefer to let well enough alone, and continue to try your individual experiments for yourselves, to see whether you can make the diagnosis of complete rupture of the supraspinatus tendon and think out better methods for operating; my suggestion is merely that each hospital should appoint a young man to study the subject thoroughly so that he can do these experiments on a rational basis. If you each experiment to see whether you can do them, it will take much longer to establish routine and effective procedures. The subject is too complex, and there are not enough cases for you all to experiment upon, even if you each took the pains to read this book. Besides your errors are expensive for us all. Let the man who does this work make some reputation among your local practitioners and insurers, so that your communities may be saved an expense that will be greater than all the money he will earn in a lifetime. If he does this well, give him some other problem to solve, and if he does that well, credit it to him when there is a question of promotion on your staff. Remember that the years he devotes to

investigation of this subject for you, may set him backward in his ambition to be a general surgeon unless he makes some contribution to our knowledge of the subject. His capacity for original work and sincerity are on trial and he might fail.

What difference does it make to you if he buys my book? I believe I have something to sell him, although what I have for sale is too crude to sell to every practitioner. After all, I have never yet sutured a supraspinatus tendon immediately after the accident which ruptured it. I may not be right in spite of my evidence and my convictions.

My work may stimulate some one to write a better book, and that may be detrimental to the prospects of my heirs. However, if my work or my writings succeed in bringing about the establishment of an End Result System of Organization in our hospitals, even a few years earlier than it would otherwise have arrived, I shall have left to the children of my great nieces and nephews, more than a money value, although they will share it with all the other heirs of the world.

Most people desire to leave money to their heirs chiefly to protect them against sickness and injury. If our children's children have health, and are assured of the maximum benefits of medical science when sickness or injury does overtake them, they should enjoy looking out for themselves, and providing better conditions still, for their own third and fourth generations.



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