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SYMPOSIUM: TRIBUTE TO DR. ANTHONY F. DEPALMA, FIRST EDITOR-IN-CHIEF OF CLINICAL ORTHOPAEDICS AND RELATED RESEARCH

The Classic

Recurrent Dislocation of the Shoulder Joint*

Anthony F. DePalma MD

Dr. Anthony F. DePalma is shown. Photograph provided with kind permission of the Art Committee of Thomas Jefferson University, Philadelphia, PA.

Dr. DePalma was the first editor of Clinical Orthopaedics and Related Research, established by the recently formed Association of Bone and Joint Surgeons. The idea of forming the Association of Bone and Joint surgeons had been conceived by Dr. Earl McBride of Oklahoma City in 1947, and organized by a group of twelve individuals (Drs. Earl McBride, Garrett Pipkin, Duncan McKeever, Judson Wilson, Fritz Teal, Louis Breck, Henry Louis Green, Howard Shorbe, Theodore Vinke, Paul Williams, Eugene Secord, and Frank Hand) [9]. The first organizational meeting was held in conjunction with the 1949 Annual Meeting of the AAOS [9] and the first annual meeting held April 1–2, 1949 in Oklahoma City. Drs. McBride and McKeever invited Dr. DePalma to attend that meeting and join the society. According to DePalma, "Even at this small gathering, there were whisperings of the need of another journal to provide an outlet for the many worthy papers written on clinical and basic science subjects" [7]. The decision to form a new journal was finalized in 1951, and Drs. DePalma and McBride signed a contract with J.B. Lippincott Company. Dr. DePalma was designated Editor-in-Chief, and the journal became a reality in 1953 with the publication of the first volume. From the outset he established the "symposium" as a unique feature, in which part of the articles were devoted to a particular topic. Dr. DePalma served as Editor for 13 years until 1966, when he resigned the position and recommended the appointment of Dr. Marshall R. Urist. At his retirement, Clinical Orthopaedics and Related Research was well established as a major journal.

Dr. Anthony F. DePalma was born in Philadelphia in 1904, the son of immigrants from Alberona in central Foggia, Italy [1]. He attended the University of Maryland for his premedical education, then Jefferson Medical College, from which he graduated in 1929. He then served a two-year internship (common at the time) at Philadelphia General Hospital. Jobs were scarce owing to the Depression, and he felt fortunate to obtain in 1931 a position as assistant surgeon at the Coaldale State Hospital, in Coaldale, Pennsylvania, a mining town. However, he became attracted to orthopaedics and looked for a preceptorship (postgraduate training in specialties was not well developed at this time before the establishments of Boards). In the fall of 1932, he was appointed as a preceptor at the New Jersey Orthopaedic Hospital, an extension of the New York Orthopaedic Hospital. In 1939 he acquired Board certification (the first board examination was offered in 1935 for a fee of $25.00 [2]) and was appointed to the NJOH staff [1].

Dr. DePalma volunteered for military service in 1942, and served first at the Parris Island Naval Hospital in South Carolina, then on the Rixey, a hospital ship. In addition to

serving to evacuate casualties to New Zealand, his ship was involved in several of the Pacific island assaults (Guam, Leyte, Okinawa). In 1945, he was assigned to the Naval Hospital in Philadelphia [1].

On his return to Philadelphia, he contacted staff members at Jefferson Medical College, including the Chair, Dr. James Martin, and became good friends with Dr. Bruce Gill (a professor of Orthopaedics at the University of Pennsylvania, and one of the earliest Presidents of the AAOS). After he was discharged from the service, he joined the staff of the Department of Orthopaedic Surgery at Jefferson, where he remained the rest of his career. He succeeded Dr. Martin as Chair in 1950, a position he held until 1970 when he reached the mandatory retirement age of 65. He closed his practice and moved briefly to Pompano Beach, Florida, but the lure of academia proved too powerful, and in January, 1971, he accepted the offer to develop a Division of Orthopaedics at the New Jersey College of Medicine and became their Chair. He committed to a five-year period, and then again moved to Pompano Beach, only to take the Florida State Boards and open a private practice in 1977. His practice grew, and he continued that practice until 1983 at the age of nearly 79. Even then he continued to travel and lecture [1].

We reproduce here four of his many contributions on the shoulder. The first comes from his classic monograph, "Surgery of the Shoulder," published by J. B. Lippincott in 1950 [2]. In this article he describes the evolutionary development of the shoulder, focusing on the distinction between various primates, and relates the anatomic changes to upright posture and prehensile requirements. The remaining three are journal articles related to frozen shoulder [1], recurrent dislocation [3], and surgical anatomy of the rotator cuff [6], three of the most common shoulder problems then and now. He documented the histologic inflammation and degeneration in various tissues including the coracohumeral ligaments, supraspinatus tendon, bursal wall, subscapularis musculotendinous junction, and biceps tendon. Thus, the problem was rather more global than localized. He emphasized, "Manipulation of frozen shoulders is a dangerous and futile procedure." For recurrent dislocation he advocated the Magnuson procedure (transfer of the subscapularis tendon to the greater tuberosity) to create a musculotendinous sling. All but two of 23 patients he treated with this approach were satisfied with this relatively simple procedure. (Readers will note the absence of contemporary approaches to ascertain outcomes and satisfaction. The earliest outcome musculoskeletal measures were introduced in the 60s by Larson [11] and then by Harris [10], but these instruments were physician-generated and do not reflect the rather more rigorously validated patient-generated outcome measures we use today. Nonetheless, the approach used by Dr. DePalma reflected the best existing standards of reporting results.) Dr. DePalma's classic article, "Surgical Anatomy of the Rotator Cuff and the Natural History of Degenerative Periarthritis," [6] reflected his literature review and dissections of 96 shoulders from 50 individuals "unaware of any (shoulder) disability" and mostly over the age of 40. By the fifth decade, most specimens began to show signs of rotator cuff tearing and he found complete tears in nine specimens from "the late decades." He concluded,

> "Based on the…observations, one can reasonably construct the natural history of periarthritis of the shoulder. It is apparent that aging is an important etiological factor, and with aging certain changes take place in the connective tissue elements of the musculotendinous cuff…it is also apparent that in slowly developing lesions of this nature compensating adjustments in the mechanics of the joint take place so that severe alterations in the mechanics of the joint do not appear. However, one must admit that such a joint is very vulnerable and, if subjected to minor trauma, the existing degenerative lesion would be extended and aggravated."

Thus, he clearly defined the benign effects of rotator cuff tear in many aging individuals, but also the potential to create substantial pain and disability.

Dr. DePalma was a prolific researcher and writer. In addition to his "Surgery of the Shoulder," he wrote three other books, "Diseases of the Knee: Management in Medicine and Surgery" (published by J.B. Lippincott in 1954) [4], "The Management of Fractures and Dislocations" (a large and comprehensive two volume work published by W.B. Saunders in 1959, and going through 5 reprintings) [5], and "The Intervertebral Disc" (published by W.B. Saunders in 1970, and written with his colleague, Dr. Richard Rothman) [8]. PubMed lists 62 articles he published from 1948 until 1992.

We wish to pay tribute to Dr. DePalma for his vision in establishing Clinical Orthopaedics and Related Research as a unique journal and for his many contributions to orthopaedic surgery.

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A survey of the literature on the pathogenesis of recurrent dislocation of the shoulder reveals that there is total lack of agreement on the causative factors responsible for this lesion. It is interesting to note that most investigators, when considering the pathology of this disease, fail to evaluate the glenohumeral joint in its entirety but rather restrict themselves to some local irregularity observed in the labrum glenoidale, fibrous capsule or head of the humerus. Another observation that is outstanding by its very inconsistency is that these same workers believe and teach that stability of the glenohumeral joint is directly dependent upon the surrounding intricate muscular apparatus that motivates it; yet, when a state of instability exists, local defects in other components of the joint are credited as causative agents.

Bankart stoutly defends his belief that the pathology in all instances is either a detachment of the labrum from the anterior glenoid brim or a tearing away of the capsule from the labrum. He goes so far as to express the view that the recurrent lesion is a different entity from the ordinary acute traumatic dislocation. The recurrent lesion, according to his view, is invariably an anterior dislocation which tends to recur because the fibrocartilaginous labrum fails to re-attach itself to the glenoid margin. The acute traumatic lesion is an inferior dislocation, the head being forced through a rent in the fibrous capsule which heals readily to bone, thereby preventing recurrences. These features of the morbid anatomy of recurrent dislocation were recorded by Broca and Hartman as far back as 1890. These observers also noted a defect on the posterior aspect of the humeral head which they believed facilitated intracapsular subluxation of the head of the humerus.

Some observers are convinced that defects in the humeral head are capable of producing dislocation and believe that the frequency of the dislocation depends upon the size of the humeral defect. As the defect increases in size, the tendency to dislocation becomes greater.

The great discrepancy in the etiologic factors responsible for the lesion, and the lack of uniformity in the surgical principles employed to effect a cure, prompted this investigation. Observations noted herein lead one to conclude that the local pathologic abnormalities recorded above are not the true causative agents, and that many of the surgical procedures performed to cure this malady are based on erroneous interpretations of normal or variational anatomy. Moreover, this investigation provides an explanation for the numerous failures which occur in procedures utilizing the principle of suspension to effect a cure, and for the success of those procedures which shorten the structures on the anterior aspect of the glenohumeral joint. The conclusions arrived at in this investigation are based on: (1) a study made on 36 recurrent dislocating shoulders. These shoulder joints were thoroughly explored and all abnormalities of the inside of the glenohumeral joint recorded; (2) gross and microscopic observations noted in 88 shoulder joints explored postmortem of 44 individuals ranging in age from 18 to 79 years who, prior to their death, gave negative histories and showed no clinical evidence of dysfunction of the shoulders; (3) determination of the range of external rotation of the arms in 800 normal individuals, 100 for each decade, from the first to the eighth inclusive; and (4) a postoperative end-result survey of 23 recurrent dislocating shoulders treated by the Magnuson (modified) procedure, the shortest interval since operation being 17 months, the longest three and a half years.

Anatomy of the Inside of the Glenohumeral Joint

At this point a review of the variational anatomy of the inside of the glenohumeral joint is imperative. In a study

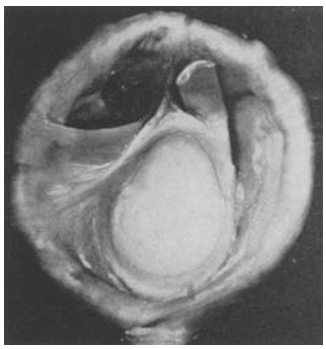

Fig. 1 Specimen exhibits one large subscapularis recess immediately above the middle glenohumeral ligament. Superior, middle and inferior glenohumeral ligaments are well defined. Note the intimate relationship of the biceps tendon and the glenohumeral ligament as they blend with the labrum glenoidale. There is early recession of the labrum from the brim of the upper half of the glenoid cavity.

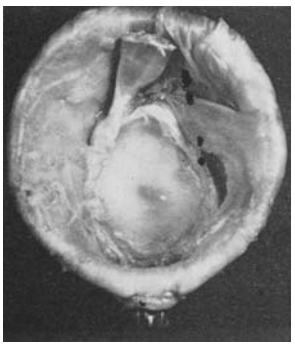

Fig. 2 Two subscapularis recesses exist; one above and one below the glenohumeral ligament. Note the severe degenerative changes that are apparent in the entire labrum glenoidale; these changes comprise shredding, thinning and villous formation on all surfaces of the fibrocartilaginous structure. Figures 1 and 2 comprise 88.6 per cent of all specimens studied.

conducted by Dr. G. Bennett, Dr. G. Callery and the author[1] on the variational anatomy and degenerative lesions of the shoulder joint, it was noted that the fibrous capsule in the anterior portion of the joint is not, as so many believe, continuous with the capsular surface of the labrum glenoidale. Instead it is projected mesially as far as the subcoracoid region and then reflected onto the anterior surface of the neck of the scapula until it reaches the periphery of the labrum. This out-pouching of the capsule forms the subscapularis recesses, one or two, depending on the variable middle glenohumeral ligament. In joints possessing one bursal recess it is usually located below the middle glenohumeral ligament; those possessing two disclose one recess above and one below the ligament. These two patterns comprised 88.6 per cent of all the specimens studied (96 shoulder joints). In the remaining 11.4 per cent there were no recesses demonstrable; the capsule in these cases was continuous with labrum (Figs. 1, 2 and 3). Although there is pronounced variation in the size of the subscapularis recesses, in general they are rather spacious. It is conceivable that one not familiar with this variational anatomy might readily misinterpret these recesses as tears or rents in the capsule. The subscapularis muscle and tendon are in intimate relationship to the anterior surface of the bursal recesses.

It becomes apparent that it is impossible for the head to pierce the capsule in the anterior portion of the joint unless it is forced through a rent below the subscapularis muscle. The head, therefore, in recurrent dislocations lies within the subscapularis recess or recesses, which are stretched to accommodate it. Both the middle and inferior glenohumeral ligaments may be the only feeble barriers to dislocation.

[1] All figures in this article dealing with the variational and gross degenerative changes of the inside of the glenohumeral joint were taken from this study which appeared in the American Academy of Orthopedic Surgeons Instructional Course Lectures,Vol. VI, 1949.

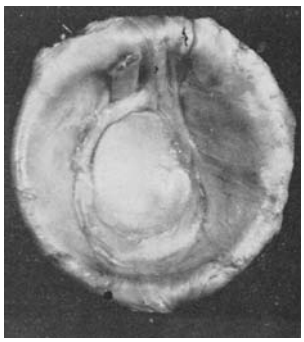

Fig. 3 No subscapularis recesses are demonstrable in this specimen. This pattern of the inside of the glenohumeral joint comprises 11.4 per cent of the specimens studied. Note the definite separation of the labrum from the upper half of the brim of the glenoid cavity.

Pathologic Observations Noted at Operation on 36 Recurrent Dislocating Shoulders

Thirty-six individuals provided the 36 recurrent dislocating shoulders studied; there were 35 males and one female; the age ranged from 18 to 36 years.

Musculotendinous Cuff

An observation of considerable significance was the pronounced laxity of the musculotendinous cuff in all cases. It appeared as if the short rotators were unduly stretched and lacked normal tonicity. Mild traction in all instances, with the cuff intact, readily separated the articulating surfaces of the humeral head and glenoid cavity. Such laxity in the musculotendinous cuff is not demonstrable in normal shoulders even under deep anesthesia.

In two instances small tears not over one centimeter wide were demonstrable in the supraspinatus region of the cuff (Fig. 4). The margins of the defects were smooth, indicating that the lesions were not recent. Three shoulders

Fig. 4 The type of complete tear noted in two of the 36 recurrent dislocating shoulders explored.

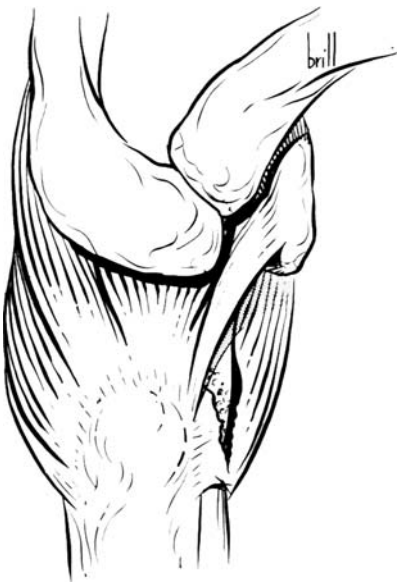

Fig. 5 The type of tear noted in the subscapularis region of the musculotendinous cuff in three of the 36 shoulders explored.

disclosed partial detachment of the inferior portion of the subscapularis tendon at its insertion into the lesser tuberosity (Fig. 5). Upon division of the subscapularis tendon at its insertion into the anterior lip of the bicipital groove, the humeral head could be readily dislocated anteriorly by external rotation of the extremity.

Labrum Glenoidale and Fibrous Capsule

Some degree of labral detachment was discernible in 29 cases (80.5 per cent). The detachment was invariably

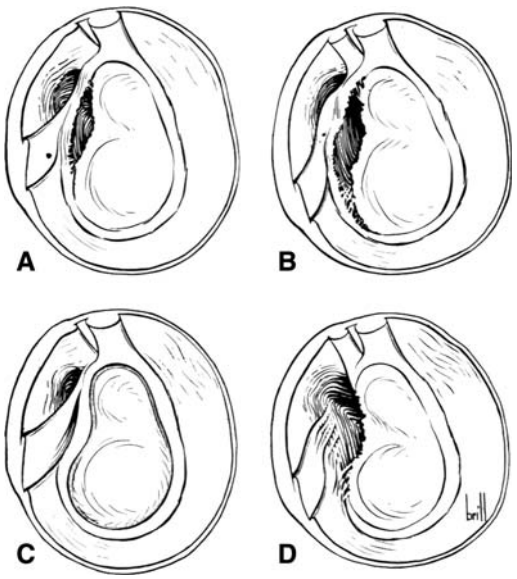

Fig. 6 (a) Only moderate detachment of the labrum. (b) Severe detachment of the labrum from the entire anterior surface of the glenoid cavity. (c) Slight elevation from its bony attachment of glenoid border of the fibrocartilaginous structure, resembling in a large measure the menisci of the knee joint. This type of separation was found in 7 cases or 19.4 per cent of shoulders explored. (d) Detachment and complete degeneration of the anterior portion of the labrum and also fraying and some shredding of the middle glenohumeral ligament.

from the anterior or antero-inferior portion of the glenoid rim. All degrees of detachment were noted, varying from one or two centimeters in length to complete detachment of the entire anterior one-half of the fibrocartilaginous ring (Fig. 6). In most instances the capsule and periosteum together with the labrum were stripped for varying distances from the anterior surface of the neck of the scapula. This last feature was more pronounced in cases with extensive labral detachments. Such a defect comprises the classic "Bankartian Lesion." Seven cases (19.4 per cent) disclosed the labrum to be firmly anchored to the glenoid margin by its capsular border, its glenoid border, however, being free like a meniscus (Fig. 6c). Many of the detached labra disclosed advanced fraying, shredding, and thinning.

Glenohumeral Ligaments and Subscapularis Recesses

In many instances the middle ligament could not be identified. In a few, small shreds of tissue indicated the remains of the ligament (Fig. 7). In most shoulders, the inferior ligament was discernible but it was greatly attenuated and stretched. All shoulders revealed marked stretching of the subscapularis recesses, and one could pass a probe mesially on the anterior surface of the neck of the scapula as far back as the coracoid process.

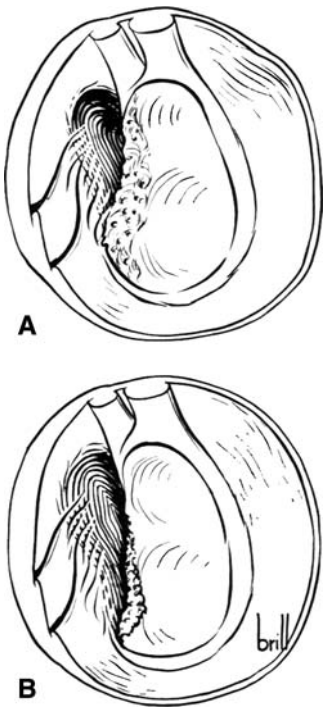

Fig. 7 (a) Complete degeneration of the labrum is noted anteriorly with some involvement of the middle glenohumeral ligament and also degenerative bone changes along the anterior margin of the glenoid cavity. (b) Note the defect on the anteroinferior aspect of the glenoid cavity resulting from actual loss of bone substance, as if a piece of bone had been sheared away from this region.

Bone Changes

Varying degrees of erosion and eburnation were found on the anterior lip of the glenoid fossa in cases in which there were labral detachments. In one instance a large irregular defect was demonstrable, leading to the conclusion that a fragment of bone had been sheared away from this area (Fig. 7b). Some osteophytes were observed on the anterior surface of the neck in three cases with extensive labral detachment.

Pathologic Observations Noted in the Musculotendinous Cuff of 44 Normal Shoulder Joints

It was interesting to note that the alterations noted above in the labrum glenoidale, fibrous capsule, and glenoid cavity did not essentially differ from the degenerative lesions observed in normal shoulder joints. Also, the lesions found in the normal shoulders were identically the same, in the respective decades, as those discernible in the aforementioned investigation, Variational Anatomy and Degenerative Lesions of the Shoulder Joint, a study conducted on cadavers on which no medical histories or physical examination prior to death were available.

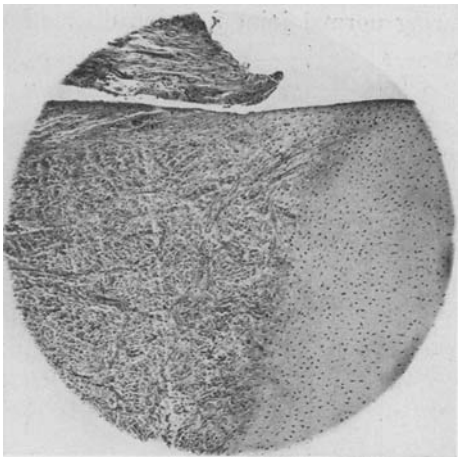

Fig. 8 Microscopic section through the junction of the labrum and the glenoid cavity of a child one year of age. Note the intimate blending of the fibrocartilage on the left with the hyaline cartilage of the glenoid cavity on the right. No definite line of demarcation exists. Both the fibrocartilage and the hyaline cartilage appear to fuse into one structure.

In infant shoulders the labrum blends with the hyaline cartilage of the humeral head; so complete is the fusion of these two structures at this period of life that, microscopically, they appear as one structure (Fig. 8). As early as the second decade, evidence of tearing away of the triangular fibrocartilage is manifested (Fig. 9a and b). This gradual separation is noted grossly in the second decade and increases in frequency and severity in each subsequent decennium; after the sixth decade it is demonstrable to some degree in approximately 100 per cent of the cases (Fig. 10a and b). In the light of this information it becomes apparent that labral detachment is associated with advancing age. The cause for labral detachment is found in the topographical relation of the biceps tendon and glenohumeral ligaments to the labrum.

Both the biceps tendon and the glenohumeral ligaments blend with the fibers of the labrum; any traction on these structures will tend to tear away the labrum from its bony anchorage on the glenoid (Fig. 1). Comprehension of this mechanism is facilitated by the knowledge that the glenohumeral ligaments act as check reins to external rotation of the limb. It becomes apparent that during normal joint function distracting forces are acting constantly on the labrum through the medium of the biceps tendon and glenohumeral ligaments; these forces achieve, gradually, separation of this triangular fibrocartilage from its bony attachment.

Grossly, another observation of great significance was the gradual but progressive increase in degenerative changes in each subsequent decade after the third in the synovialis, fibrous capsule, glenohumeral ligaments, musculotendinous cuff, and biceps tendon. The lesions comprised tearing, shredding, fraying, and villous formation of the synovialis;

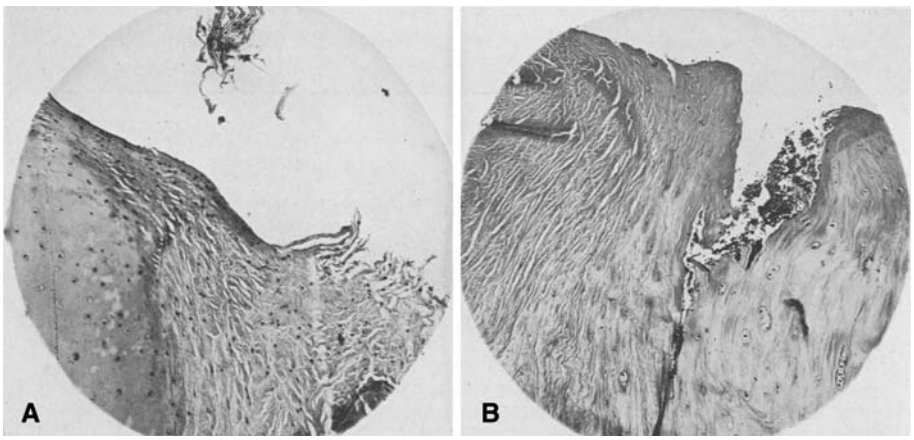

Fig. 9 (a) Specimen through the junction of the cartilage and the labrum from an individual in the second decade of life. Note the gradual tearing away of the labrum from its attachment to the hyaline cartilage. (b) Specimen in the third decade of life. Note the actual splitting away of the fibrocartilage on the left from the glenoid cavity on the right.

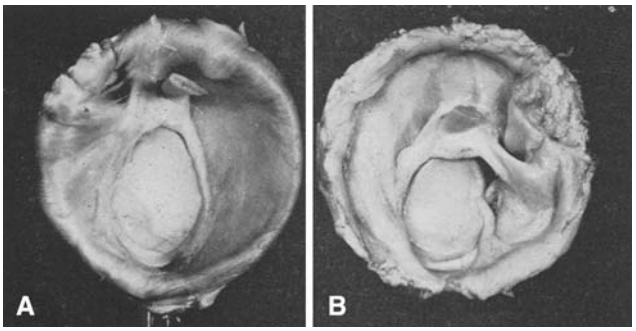

Fig. 10 (a) A specimen from the second decade in life. Note the separation of the labrum from the glenoid cavity along its upper borders. (b) Note the complete separation of the labrum from the entire brim of the glenoid cavity. The only ligament that is firmly attached to the glenoid cavity is the inferior glenohumeral ligament.

tearing and shredding of the cuff, particularly in the supraspinatus and infraspinatus areas; and a diffuse thickening of all the above components of the joint (Fig. 11a, b and c). Microscopic study disclosed a generalized increase in fibrous tissue in all the aforementioned structures. In some instances the fibrosis, especially in the later decades, was so pronounced that it partially obliterated the bursal recesses (Fig. 12a, b and c). It was also interesting to note that in the late decades of life (especially after the sixth decade) the hyperplastic process was exceedingly pronounced; it appeared as if nature were attempting to re-attach detached labra to the glenoid brim. In many instances the attempt was in part successful. It is logical to assume that replacement of normal elastic tissue by fibrous tissue must result in varying restriction of motion of the joint depending upon the severity of the process of fibrosis. This destruction led to the next step in the investigation.

Determination of the Range of External Rotation in 800 Normal Individuals

Eight hundred individuals, 100 in each decade from the first to the eighth inclusive, were studied. All possessed

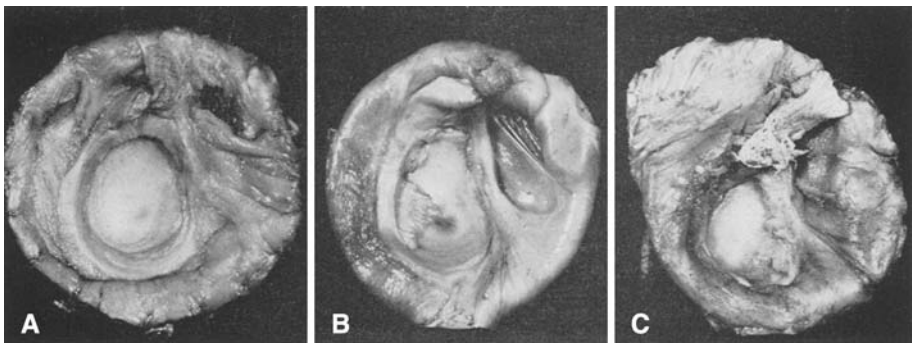

Fig. 11 (a) There is marked thickening of all the soft tissue structures comprising the inside of the glenohumeral joint. Note the marked thickening of the labrum, middle glenohumeral ligament, the inferior and superior glenohumeral ligament, and all the synovial tissues lining the joint. It appears as if the hyperplastic process is gradually obliterating the subscapularis recess in the upper right hand corner. (b) Again there is marked thickening of all the structures of the inside of the joint. There is detachment of the labrum from the upper half of the glenoid cavity, also an attempt is made by nature to re-attach the labrum to its original bony site by the formation of villi and tabs between the labrum and the glenoid cavity. This is particularly noted in the posterior aspect of the joint. (c) There is profound thickening of the synovial and sub-synovial tissues, the fibrous capsule, the labrum glenoidale, and all the glenohumeral ligaments. The process is so marked that it is hardly possible to distinguish the different glenohumeral ligaments. Also note marked degenerative changes in the form of villi, shredding and thickening of the biceps tendon.

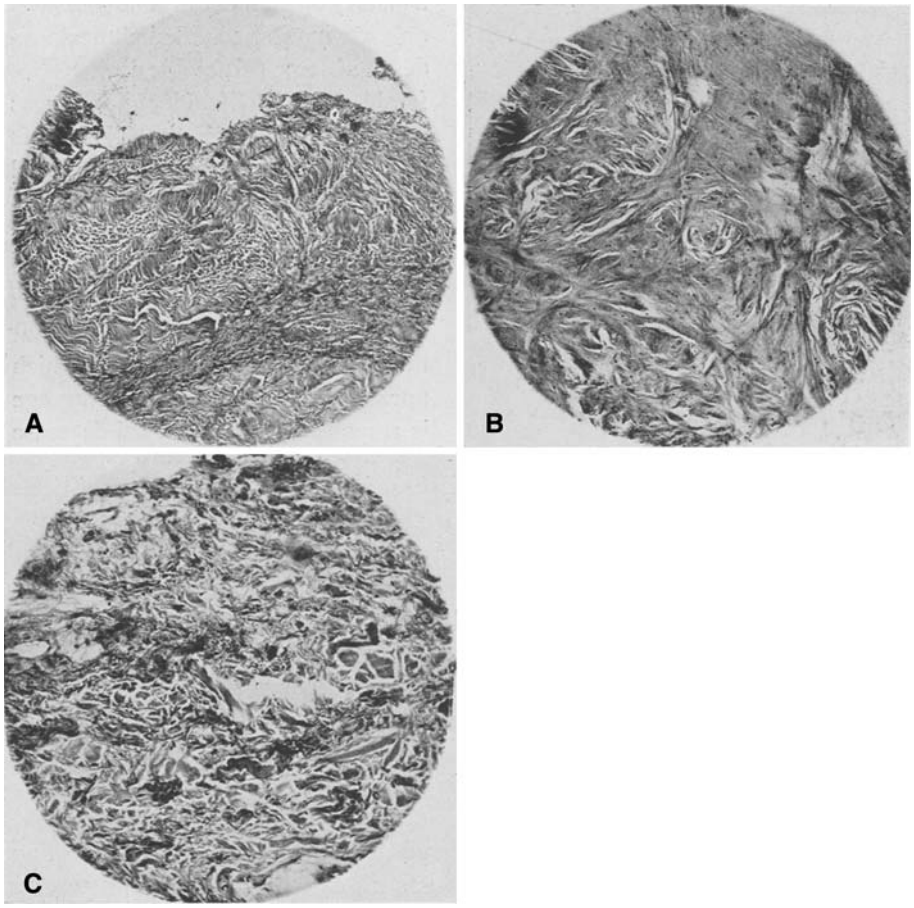

Fig. 12 (**A**) Microscopic section through one of the glenohumeral ligaments. Note the thickening of the entire synovial tissues which has been entirely replaced by fibrous tissue. (**B**) This represents a section through the synovial membrane of an individual in the fifth decade of life. Note the marked fibrosis present and hyalinization of the fibrous tissue. (**C**) Microscopic section through the musculotendinous cup in the region of the supraspinatus tendon. Note the advanced degenerative process characterized by fragmentation, shredding, tearing, of all the tendon fibers and replacement of the degenerated tissue by fibrous tissue.

normal shoulders. No individual with a history of shoulder disability or who on examination revealed clinical evidence of some pathologic disorder referable to the shoulder joints was included in this survey. There was found an average range of external rotation in the second decade of 105 degrees, in the third of 85 degrees, in the fifth of 78 degrees, and in the seventh and eighth of 68 degrees (Fig. 13). It becomes obvious that the above increase in fibrosis which normally occurs with advancing age is responsible for a gradual decrease in the range of external rotation in each successive decade.

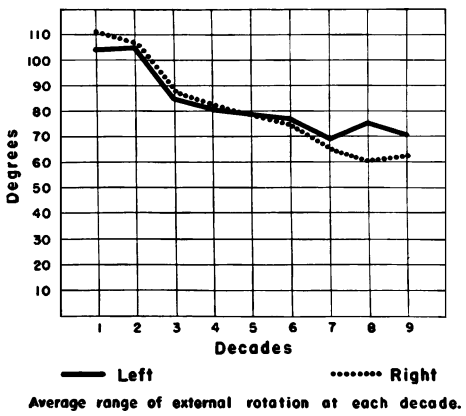

Fig. 13 Graph showing the gradual restriction of external rotation from the first to the eighth decades of life.

Correlation of Observations and Clinical Facts

Progressive fibrosis of all capsular tissues, followed by restriction of external rotation, provides an explanation for the following clinical facts, namely, that recurrent dislocation of the shoulder is seldom encountered after the fourth decade and that the malady is a self-limiting disease. Many instances are known of individuals who had in early life numerous recurrences which, with out treatment, steadily decreased in frequency as the individual approached middle life; finally no more occurred. In other words, nature's method of curing the disease is by producing scarring and fibrosis of the soft tissues sufficient to limit markedly external rotation.

Another observation of significance is that in those decades in which labral detachments are most frequent and most severe (after the fourth decade) recurrent dislocations are rarely encountered. In the light of these observations one is forced to conclude that local lesions such as labral

defects and tears in the capsule and defects in the humeral head are not the causative agents of the disease, that a true Bankartian lesion does not exist, and that some other disorder is the responsible agent.

Concept of Pathogenesis of Recurrent Dislocation of the Shoulder

Capsular, labral lesions and humeral head defects are not the prime causative agents of recurrent dislocation; they are changes associated with ageing but may be produced or aggravated by trauma. Neuromuscular imbalance, chiefly of the short rotator muscles, is the most single important causative factor. Such a neuromuscular state follows severe stretching of and direct injury to the involved muscles, particularly the subscapularis muscle, at the time of injury. Pronounced capsular stretching and enlargement of the bursal recesses to accommodate the head are secondary adaptive changes. Repeated dislocations increase the neuromuscular imbalance, hence increasing the tendency to recurrences until nature overcomes the laxity of all tissues by a progressive process of fibrosis which limits external rotation and stabilizes the glenohumeral joint.

If the aforementioned concept of the pathogenesis of recurrent dislocation is accepted, it becomes apparent that such a complication may follow any initial mechanism of anterior dislocation, provided the short rotators, particularly the subscapularis muscle, are severely stretched and traumatized, and if the primary dislocation has not been adequately treated. I am in total disagreement with Bankart's postulate that recurrent dislocation is a different lesion than ordinary acute traumatic dislocation and that it can occur only in the presence of capsular or labral detachment.

Capsular and labral lesions, as well as defects in the humeral head, have been given an undeserved place of importance in the pathology of recurrent dislocation, because it has been clearly revealed in this study that they are normal degenerative changes associated with wear and tear and senescence—although it must be admitted that trauma plays a part in their formation.

Analysis of Operative Procedures

A survey of the many operative procedures devised for re-dislocation reveals that the essential feature which affects a cure is restriction of external rotation. Regardless at what region of the glenohumeral joint, or at what components of the joint the operative attacks are aimed, limitation of external rotation will effect a cure in the great majority of cases. This is also nature's method of eliminating the disability. Therefore, the simplest procedure which will bring about restriction of the arc of external rotation will eventually be uniformly adopted.

Magnuson points out that by the transference of the subscapularis tendon to the greater tuberosity a musculotendinous sling or cup is formed around the humeral head in both external and internal rotation which counter-balances the powerful pull of the adductor muscles (pectoralis major, latissimus dorsi, and teres major) which tend to force the head downward and forward. We are of the opinion that the Magnuson procedure properly performed will supplant all other operations.

The Magnuson procedure was modified in order to increase the efficiency of the musculotendinous sling. This has been accomplished by transferring the subscapularis tendon across the bicipital groove and anchoring it at a lower level than its original insertion onto the humeral shaft.

Magnuson Operation (Modified)

(Fig. 14) An "S"-shaped skin incision, beginning at the inferior margin of the acromioclavicular joint, is made on the anterior aspect of the shoulder. The interval between the deltoid and the pectoralis major is developed, taking care not to injure the cephalic vein which is retracted medially with the pectoralis major muscle. By external rotation of the shaft of the humerus, the subscapularis tendon, as it inserts into the lesser tuberosity, comes into view. A blunt dissector is passed under the subscapularis tendon in order to determine more clearly its upper and lower borders. An incision is made in the interval between the supraspinatus and subscapularis muscles, beginning proximal to the blending of the subscapularis tendon with the fibrous capsule; the incision is continued to the anterior lip of the bicipital sulcus. A second incision, the same length as the first, is made along the lower border of the scapularis muscle. The tendon between the two incisions is then freed from the anterior lip of the bicipital groove by sharp dissection. Retraction medially of the tendon and capsule affords a clear view of the humeral head, anterior glenoid margin, and the anterior portion of the synovial capsule with its glenohumeral ligaments and bursal recesses.

The greater tuberosity is visualized by internal rotation of the arm. The subscapularis is then pulled, by means of a suture through its substance, across the bicipital groove to a point below the greater tuberosity and its site of reattachment is determined. The tendon should be anchored to the humeral shaft below the level of its original insertion, under moderate but not severe tension. With a thin osteotome a slot one-quarter inch wide and as long as the width of the subscapularis tendon is made parallel to the posterior lip of the bicipital groove below the greater tuberosity. Four drill holes are then made in the posterior lip of the

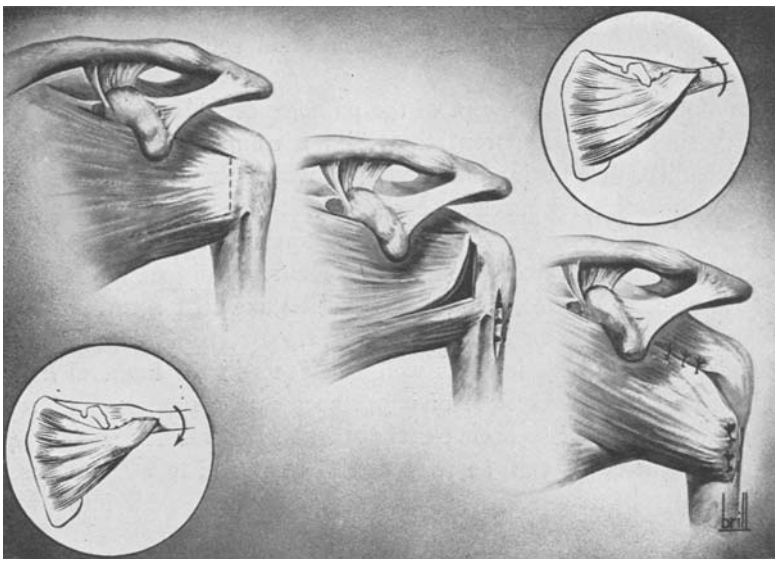

Fig. 14 Note that in the modification of the above Magnuson operation the subscapularis tendon is anchored in a bony trough below the level of its original site on the other side of the bicipital sulcus.

newly formed slot and the end of the tendon is buried in the bony trough with silk mattress sutures. The upper border of the subscapularis muscle is approximated to the supraspinatus muscle by side-to-side sutures, while its lower border is sutured to the capsular tissues under the head of the humerus.

Considerable restriction of external rotation is demonstrable at the completion of the procedure. The subscapularis muscle and tendon fibers can also be seen to form a sling under the head, on abduction of the arm in internal and external rotation. The procedure is completed by wound closure in layers with interrupted sutures.

Postoperative Treatment

For the first two weeks, the arm is fixed to the side with the forearm across the chest by a Velpeau dressing. For the next two weeks the arm is kept in a sling. Motion is then begun but not permitted above the horizontal plane for two more weeks. Forceful external rotation during this period is prohibited, but full resumption of all motions should be attained by the eighth week. As a rule, abduction is restricted a few degrees and external rotation may be restricted as much as 50 per cent. This, however, produces no functional disability and is assurance against re-dislocation. The amount of external rotation lost is but a small price to pay for the cure of such a disabling malady by so simple a surgical procedure.

Analysis by many observers of the different types of operations performed discloses that the Bankart and Putti-Platt procedures give a high percentage of cures while the Nicola and Henderson operations result in a high percentage of failures. Adams recorded the following data in III cases under observation not less than two years from the time of operation. Re-dislocation occurred in 21 (36 per cent) of 59 cases, in which the Nicola operation was employed; in two of 37 cases in which the Putti-Platt operation was done and in one of 18 cases in which the Bankart procedure was performed. Leeds reported recurrences of dislocation in nine out of 13 cases treated by the Henderson operation. Re-dislocation occurred in all cases in which the sling was constructed of fascia lata and in four of seven cases in which the tendon of the peroneus longus muscle was used as a sling.

In this investigation 23 recurrent dislocating shoulders were treated by the modified Magnuson Operation; there were recurrences of dislocation in two out of 23 cases (8.7 per cent) (Table 1).

Success of the modified Magnuson and Bankart procedures can be directly attributed to the known restriction of external rotation which is achieved by these operations. Failure of the Henderson operation can be explained on the basis that it fails to limit external rotation while the high percentage of failures occurring in the Nicola procedure results from failure to restrict external rotation and pulling away of the labrum from the glenoid rim when the weight of the extremity acts upon it through the medium of the proximal end of the biceps tendon which now functions as a suspensory ligament.

Conclusion

1. Local lesions of the labrum glenoidale, fibrous capsule, glenoid cavity, and humeral head are not the causative agents responsible for recurrent dislocation of the shoulder.
2. These lesions are observed in normal shoulders and are manifestations of wear, tear, and senescence.

Table 1. Analysis of 23 Magnuson procedures (modified)

Name	Sex	Age	Date operation	Number recurrences	Pain	Satisfied
C.F.	M	28	5/26/48	1	Yes	No
R.M.	M	23	11/22/47	0	No	Yes
R.C	M	30	5/30/48	0	No	Yes
G.H.	M	24	10/3/47	0	Occasionally	Yes
W.M.	M	24	6/28/48	0	No	Yes
A.A.	M	23	2/30/48	0	No	Yes
M.K.	M	21	3/21/48	0	No	Yes
E.H.	M	22	4/29/48	0	No	Yes
J.C.	M	29	6/25/48	0	No	Yes
J.F.	M	26	7/30/48	0	No	Yes
W.V.	M	20	7/19/48	0	Yes	Yes
A.V.	M	25	8/24/48	6	After dislocation	No
W.S.	M	21	3/3/48	0	No	Yes
J.N.	M	18	11/15/47	0	No	Yes
T.J.	M	36	8/4/48	0	No	Yes
D.L.	M	17	5/21/45	0	No	Yes
W.B.	M	30	1/21/48	0	No	Yes
C.M.	F	29	3/26/48	0	Slight	Yes
J.C.	M	19	8/24/47	0	No	Yes
L.B.	M	33	6/12/47	0	No	Yes
T.N.	M	22	8/18/46	0	No	Yes
D.O.	M	29	10/15/47	0	No	Yes
J.N.	M	22	2/23/48	0	No	Yes

Analysis of cases:

Total number of cases	23
Age range	17 to 33 years
Males	22
Females	1
Recurrences of dislocation	2 cases (8.7 per cent)
Pain present in	2 cases
Not satisfied with operation	2
Satisfied with operation	21
Period of observation	17 months to 3½ years

3. Trauma does play a role in their formation.
4. They occur most frequently and exhibit the greatest severity in decades in which recurrent dislocations are seldom encountered.
5. Recurrent dislocation of the shoulder is a self-limiting disease; nature affects a cure by a progressive process of fibrosis which restricts external rotation.
6. Operative procedures which mimic this mechanism are favored by a high percentage of cures; those that do not, result in a high percentage of failures.
7. The modified Magnuson operation is offered as a simple and effective method to achieve the desired restriction of external rotation.

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