



Development of a Shoulder Contracture Model in Rats

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Hypothesis: The capsular tissue is responsible for the pathogenesis of the shoulder contracture.

Materials and methods: The glenohumeral joint of Sprague-Dawley rats was immobilized using internal fixation (immobilized group). The control group underwent a sham operation (sham group). To assess the range of motion, the glenohumeral joint angle was measured repeatedly under same torque in 6 conditions: after removal from the trunk, after removal of the outer muscles other than rotator cuff after removal of the rotator cuff muscles, and after 3 types of partial capsulotomy. The abduction angle and total rotation angles were measured. The length of the synovial intima was measured with hematoxylin-eosin-stained specimens. Immunohistochemical study for type III collagen was also performed.

Results: No significant differences were found in the range of motion until all the muscles were removed. The abduction angle increased significantly after serial capsulotomy in the immobilized group. Even after the capsulotomy, however, this angle remained significantly less than that in the sham group. There was a similar trend for the total rotation angle. There was morphological change in the synovium of the immobilized group; the significant decrease of synovial length and strong staining of type III collagen.

Discussion: Our results show that capsule might play important role for contracture formation. Decrease of the synovial length might reflect synovial adhesion. Strong expression in Type III collagen might be related to joint stiffness.

Conclusion: A contracture model was successfully established. Changes in the capsule and synovium might play an important role in occurrence of contracture.

Level of evidence: Basic Science Study, Animal Study.

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Keywords: Animal model; shoulder; contracture; capsule; synovium

Shoulder contracture is one of the most common causes of dysfunction of the shoulder joint. Although the clinical features of shoulder contracture have been well documented, little is known concerning its pathogenesis or pathophysiology. Previous magnetic resonance imaging studies revealed that there was a thickening of the joint capsule in patients with frozen shoulder syndrome.^{12,14-16} Histologically, Ozaki et al¹⁹ and Bunker et al³ reported

that fibrosis of the capsule was main lesion, but neither inflammation nor synovitis was observed. Wiley et al³¹ and Uitvlugt et al²⁹ reported that synovitis was the most predominant intra-articular pathologic finding in patients with frozen shoulder. Other authors reported that inflammation in the synovium and fibrosis occurred together in the capsule.^{9,20} Type III collagen deposition in the capsule and synovium was thought to be an important pathogenetic factors in human frozen shoulder.^{3,20,28} Unfortunately, we could not conclude which tissue is principally responsible for the pathogenesis of frozen shoulder syndrome in humans.

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To investigate these issues, it is necessary to develop an animal model simulating *primary* frozen shoulder syndrome. However, because no such models are available, one should use a *secondary* contracture model with joint immobilization. A number of studies have used immobilization to investigate joint contracture, but most have focused on the knee joint.^{1,2,5,7,8} To date, Scholmeier et al^{22,23} have reported the only shoulder contracture model. They immobilized the canine shoulder using a cast and successfully developed shoulder contracture by decreasing range of motion and increasing intra-articular pressure. The presence of an adhesion in subacromial bursa was histologically confirmed concomitant with increasing numbers of synoviocytes and vessels. They also reported that these changes recovered after the cessation of immobilization and concluded that these findings were consistent with reports about human frozen shoulder.^{10,11,31}

One of the major drawbacks of their study, however, was that the position of the limb was varied between each animal or changed with time because they used a cast for immobilization. To control the limb position consistently, it seemed to be better to apply internal fixation than to apply a cast. On the basis of these facts, we attempted to establish a new shoulder contracture model using extra-articular rigid internal fixation.

Furthermore, we hoped to clarify which tissue was the most responsible for the development of shoulder contracture after immobilization using the current immobilization model. We hypothesized that the capsular tissue including lining synovium plays the most important role in the pathogenesis of the joint contracture. To test this hypothesis, histologic changes including synovial adhesion and type III collagen an were also assessed in the current study.

Materials and methods

The current study was conducted under an experimental protocol approved by the Institutional Animal Experimentation Committee of the Tohoku University (19-6, 20HpA-2), Japan.

Development of the animal model

The study used 24 male Sprague-Dawley rats (12 weeks old), which were divided into 2 groups: the immobilized (12 rats) and sham groups (12 rats). The animals were anesthetized with an intraperitoneal injection of pentobarbital sodium (40 mg/kg).

A longitudinal skin incision was made parallel to the humeral shaft. The deltoid and biceps muscles were retracted to expose the lateral aspect of the humeral shaft. The insertion of the deltoid muscle was minimally detached from the humeral shaft. Another skin incision was made perpendicular to the scapular spine on the scapular side, from the upper to lower edge of the scapula. The origins of both infraspinatus and subscapularis muscles were minimally elevated from the scapular body only for their middle

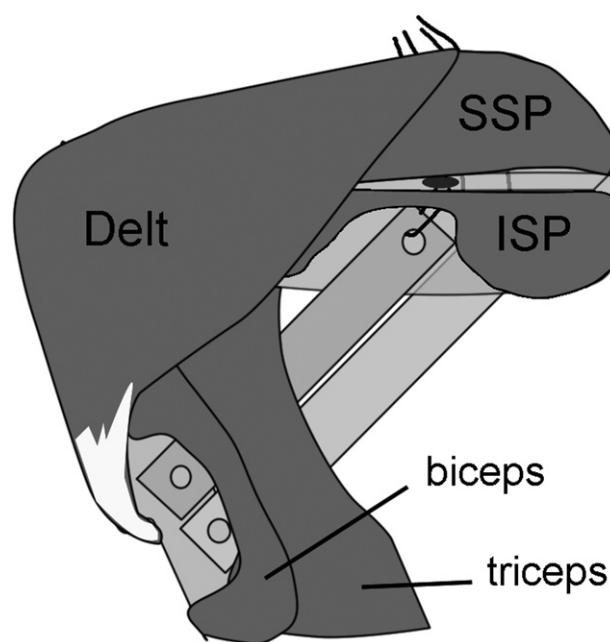


Figure 1 A schematic diagram drawn with muscles shows the immobilized shoulder. In the humeral side, the deltoid (*Delt*) insertion is excised minimally and the biceps is retracted. Two plates, which are fixed with humeral shaft with screws, penetrate the triceps. In the scapular site, the infraspinatus (*ISP*) and subscapularis are both elevated at the middle of scapula. The scapular body is pinched by 2 plates with prethreaded, soft flexible wires. A bony hole is made on the scapular spine to pass through two flexible wires, which are then twisted and tied securely on the scapular spine. *SSP*, Supraspinatus.

third parts (Fig. 1). Care was taken not to damage the glenohumeral joint capsule during this step. The humeral insertions of these muscles were not detached in the current study.

Two plastic plates (Senko Medical, Tokyo, Japan), screws (J. I. Morris, Southbridge, MA), and flexible wires were used to immobilize the shoulder (Fig. 2). The scapular body was pinched by these 2 plates with prethreaded soft flexible wires. For the ventral side, 1 plate with flexible wire was inserted from the upper side of the scapular body. For the dorsal side, another plate with flexible wire was also placed directly on the bony surface of the scapula. A bony hole was made using an 18-gauge needle on the scapular spine to pass through 2 flexible wires, which were then twisted and tied securely to fix the scapular body with the plates. These implants were covered by the muscle bellies of subscapularis and infraspinatus. Rigid immobilization was achieved by fixing these 2 plates on the lateral aspect of the humeral shaft using the screws.

In the current model, the shoulder joint was immobilized at 60° abducted position. The abduction angle of the shoulder joint was defined as the angle between the humeral shaft and the scapular spine. In the rat shoulder, 60° abduction was the maximum adducted position because of the presence of the muscles. Because we hoped to simulate 0° abduction of the human shoulder, we chose this angle for the immobilization in the current study. To precisely measure the abduction angle, 2 lines were first drawn from the edge of the scapula to the humeral head and from the

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