Anterior Shoulder Dislocations

Jack Yu, MD
Glendale, California

The glenohumeral joint of the shoulder is the most commonly dislocated joint in the body. When dislocation occurs, it is usually anterior. The primary care physician usually sees the patient after reduction has been performed. Thus, treatment is directed at recovery of function and preventing recurrences. The prognosis and likelihood of recurrence in a first-time anterior dislocation depends on the mechanism of injury, treatment, rehabilitation, sex, age, and complications. Nonoperative treatment after reduction usually involves immobilization and rehabilitation. Studies to date seem to indicate that age and activity level are more important factors for recurrence than length of immobilization. Traditionally, if there are recurrences after conservative treatment, then surgery is contemplated.

Key words. Shoulder dislocation; recurrence; immobilization; clinical protocols.
J Fam Pract 1992; 35:567-576.

Shoulder dislocations and their treatment have been written about and discussed since at least the 5th century BC, when Hippocrates described a number of methods of reduction.[1] During the Greek age, as now, shoulder dislocation was a very disabling injury causing many to "abandon gymnastic exercise, and . . . become inept in war time practices." Of the difficulty of treating this malady, Hippocrates wrote, "I have never known any physician to treat the case properly." He then described a "burning" of the shoulder to form eschars to prevent recurrent dislocations.[1] This is not unlike some modern procedures that prevent recurrences by limiting range of motion.

In the modern age of medicine, physicians have tried various postreduction treatments in an attempt to prevent recurrences. The length and type of immobilization has been studied, as well as the use of rehabilitation therapy; however, no uniform conclusions have been reached.

Normal Anatomy

The shoulder is one of the most flexible and mobile joints in the body. This is made possible by the minimal area of contact between the humeral head and the osseous portion of the joint, ie, the glenoid fossa of the scapula, as well as the shallowness of the glenoid fossa. As a consequence, it is also the most unstable and most often dislocated joint in the body. Dislocations usually occur anteriorly because of the lack of skeletal restraints, as well as the relatively weaker anterior capsule. Posterior dislocations are fairly uncommon (2.8% in one study) and usually require consultation with an orthopedist.[2]

The stability of the shoulder is in large part due to the shoulder capsule, which includes the synovial membrane, the fibrous capsule consisting of the coracohumeral ligament, and the superior, middle, and inferior glenohumeral ligaments. Other parts of the shoulder capsule include the fibrous glenoid labrum, which attaches to the anterior portion of the glenoid fossa, and the rotator cuff muscles, ie, the supraspinatus, infraspinatus, teres minor, and subscapularis, and their tendons.[3] These muscles maintain the humeral head in the glenoid fossa regardless of position.[4] The deltoid, triceps, and biceps muscles are also stabilizing structures[5] (Figure 1).

Prognosis

The prognosis and likelihood of recurrence of first-time anterior dislocations of the shoulder is related to (1) mechanism of injury, (2) postreduction treatment, (3) rehabilitation therapy, (4) patient age, (5) sex, and (6) complications.

Mechanism

In Rowe's study,[5] dislocations caused by twisting or forceful abduction had a higher recurrence rate (56%) than that of the group where dislocations were caused by a direct blow to the shoulder, anterior or posterior

Submitted April 20, 1992.

From the Family Practice Residency, Glendale Adventist Medical Center, Department of Family Medicine, Loma Linda University, and the Department of Family Medicine, University of Southern California School of Medicine, Los Angeles. Requests for reprints should be addressed to Jack Yu, MD, Family Practice Residency Program, 801 S Chevy Chase Dr, Suite 201, Glendale, CA 91205.

 ISSN 0094-3509

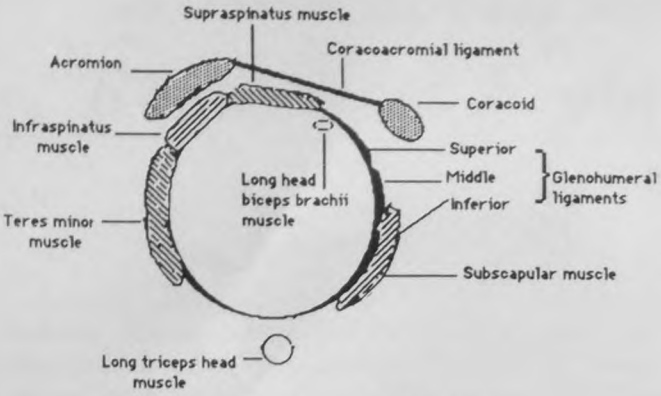


Figure 1. Lateral view of shoulder.

(19%). This increased rate may be due to the detachment of the glenoid labrum, known as the Bankart lesion, which was shown by Rowe to be associated with recurrent dislocations. Kiviluoto and co-workers[6] also found a greater number of recurrences in cases in which torsion had occurred (11/35, 31%) compared with dislocations caused by a fall in the same plane (9/92, 10%) or a fall from a height (0/15).

Traumatic dislocations are more often associated with a fracture or other complication that requires a long period of immobilization resulting in additional scar formation. This may ultimately contribute to greater stability and fewer recurrences.[7] It is thought that traumatic anterior dislocations usually occur with rupture or avulsion of anterior supports (subscapularis, glenohumeral ligaments, and capsule). Surgical studies of shoulders with recurrent dislocations have confirmed that there are anatomical abnormalities, eg, relaxed tendons or shoulder capsules, that allow the humeral head to dislocate, or detachment of the glenoid labrum, which allows the humeral head to dislocate anteriorly.[8–12]

Atraumatic dislocations are rare (4.4% in Rowe's study of 1956[5]) and are probably related to inherent instability. These dislocations may respond to resistive strengthening exercises, thus avoiding surgery in at least 50%.[5,13,14] Inherent instability may be due to an abnormal glenohumeral head or congenital hyperelasticity of the connective tissue throughout the body. Some adolescent girls with psychological problems but normal anatomy may be able to voluntarily dislocate their shoulders by manipulating their muscles.[5]

In terms of recovery of function, Pasila and coworkers[15] found that greater time was required to regain normal mobility after a fall from a height (median 60 days), as compared with falling (30 days) or torsion (24 days); thus, the greater the severity of the dislocating trauma, the longer the recovery period may be.

Simonet and Cofield[16] suggested that athletes with their greater exposure to trauma, irrespective of age, were at higher risk for reinjury. Of 32 patients less than 30 years old, athletes had an 82% recurrence rate as compared with 30% in nonathletes.

Epileptic patients represent a special subset of traumatic dislocations. The trauma from the force of their convulsion is evidenced in the increased incidence of humeral head defects. Rowe[13] reported a recurrence rate of 94% in primary dislocations that occur in patients with epilepsy. In his 10 cases, there was one shoulder with posterior dislocation, two had bilateral dislocations, and 10 of 11 shoulders had recurrences. This can be explained by the great forces that act with each successive convulsion. Because of this great force, the patient's prognosis is poor unless seizures are controlled.

Postreduction Treatment

Once a shoulder dislocation has been diagnosed, reduction can be performed by a number of methods. Postreduction treatment has been studied but no uniform conclusions have been reached.

Bankart[17] challenged the theory that recurrent shoulder dislocations were due to abnormal capsule laxity from too early or vigorous motion of the humeral head postreduction, thus stretching the capsule or not allowing it to heal properly. He proposed that the primary cause was the detachment of the glenoid labrum from the anterior glenoid fossa. This association was confirmed by Rowe.[5] The former theory persisted for many years and is the reason for the traditional immobilization postreduction of 3 to 6 weeks.

The efficacy of immobilization in preventing dislocation recurrences has been the object of a number of studies. Immobilization consists of either sling and swathe, or sling with a restriction on shoulder mobilization. Most studies seem to show that 1 to 3 weeks of immobilization is beneficial (except in the young) and that no additional benefit is seen with immobilization for more than 3 weeks. When stratified by age of patient, differences in rate of recurrence suggested that age may be the most important variable. Young patients are at much higher risk of recurrences. Studies by Hovelius[19] and Henry and Genung[21] involved only young athletic patients, and there were no significant differences in rate of recurrence with length of immobilization. Table 1 is a summary of the impact of both age and treatment in 10 studies.

In general, based on these studies, there appears to be an advantage in a 3-week immobilization period for a primary anterior shoulder dislocation. For the young, however, it seems to be more important to avoid stressing the joint, consciously avoiding external rotation and abduction for 3 to 6 weeks, and possibly even up to 3

DESCRIPTION

LEVO-T contains synthetic crystalline L-3,3',5,5'-tetraiodothyronine sodium salt [levothyroxine (T_4) sodium]. Synthetic T_4 is similar to that produced in the human thyroid gland. T_4 contains four iodine atoms and is formed by the coupling of two molecules of diiodotyrosine (DIT).

Levothyroxine (T_4) sodium has an empirical formula of $C_{15}H_{10}I_4NNaO_4xH_2O$ and a molecular weight of 798.86 (anhydrous).

LEVO-T tablets contain the following inactive ingredients: Microcrystalline Cellulose NF, Pregelatinized Starch NF, Lactose NF (Anhydrous), and Magnesium Stearate NF. The following are the color additives per tablet strength:

Strength (mcg)	Color Additive(s)
25	FD&C Yellow No. 6 Aluminum Lake
50	None
75	FD&C Red No. 40 Aluminum Lake FD&C Blue No. 2 Aluminum Lake
100	D&C Yellow No. 10 Aluminum Lake D&C Red Lake Blend (D&C Red No. 27 Lake and D&C Red No. 30 Lake)
125	FD&C Yellow No. 6 Aluminum Lake FD&C Red No. 40 Aluminum Lake FD&C Blue No. 1 Aluminum Lake
150	FD&C Blue No. 2 Aluminum Lake
200	D&C Yellow No. 10 Aluminum Lake D&C Red No. 27 Aluminum Lake
300	D&C Yellow No. 10 Aluminum Lake FD&C Yellow No. 6 Aluminum Lake FD&C Blue No. 1 Aluminum Lake

CLINICAL PHARMACOLOGY

The steps in the synthesis of thyroid hormones are controlled by thyrotropin (Thyroid Stimulating Hormone, TSH) secreted by the anterior pituitary. This hormone's secretion is in turn controlled by a feedback mechanism effected by the thyroid hormones themselves and by thyrotropin releasing hormone (TRH), a tripeptide of hypothalamic origin. Endogenous thyroid hormone secretion is suppressed when exogenous thyroid hormones are administered to euthyroid individuals in excess of the normal gland's secretion.

The mechanisms by which thyroid hormones exert their physiologic action are not well understood. These hormones enhance oxygen consumption by most tissues of the body and increase the basal metabolic rate and the metabolism of carbohydrates, lipids, and proteins. Thus they exert a profound influence on every organ system in the body and are of particular importance in the development of the central nervous system.

The normal thyroid gland contains approximately 200 mcg of levothyroxine (T_4) per gram of gland, and 15 mcg of triiodothyronine (T_3) per gram. The ratio of these two hormones in the circulation does not represent the ratio in the thyroid gland, since about 80% of peripheral triiodothyronine comes from monodeiodination of levothyroxine at the 5 position (outer ring). Peripheral monodeiodination of levothyroxine at the 5 position (inner ring) results in the formation of reverse triiodothyronine (rT_3), which is calorigenically inactive. These facts would seem to advocate levothyroxine as the treatment of choice for the hypothyroid patient and to militate against the administration of hormone combinations which, while normalizing thyroxine levels may produce triiodothyronine levels in the thyrotoxic range.

Triiodothyronine (T_3) level is low in the fetus and newborn, in old age, in chronic caloric deprivation, hepatic cirrhosis, renal failure, surgical stress, and chronic illnesses representing what has been called the "low triiodothyronine syndrome."

Pharmacokinetics: Animal studies have shown that T_4 is only partially absorbed from the gastrointestinal tract. The degree of absorption is dependent on the vehicle used for its administration and by the character of the intestinal contents, the intestinal flora, including plasma protein, soluble dietary factors, all of which bind thyroid and thereby make it unavailable for diffusion.

Depending on other factors, absorption has varied from 48% to 79% of the administered dose. Fasting increases absorption. Malabsorption syndromes, as well as dietary factors (children's soybean formula, concomitant use of anionic exchange resins such as cholestyramine), cause excessive fecal loss.

More than 99% of circulating hormones are bound to serum proteins, including thyroxine-binding globulin (TBG), thyroxine-binding prealbumin (TBPA), and albumin (TBa), whose capacities and affinities vary for the hormones. The higher affinity of levothyroxine (T_4) for both TBG and TBPA as compared to triiodothyronine (T_3) partially explains the higher serum levels and longer half-life of the former hormone. Both protein-bound hormones exist in equilibrium with minute amounts of free hormone, the latter accounting for the metabolic activity.

Deiodination of levothyroxine (T_4) occurs at a number of sites, including liver, kidney, and other tissues. The conjugated hormone, in the form of glucuronide or sulfate, is found in the bile and gut where it may complete an enterohepatic circulation. Eighty-five percent of levothyroxine (T_4) metabolized daily is deiodinated.

INDICATIONS AND USAGE

LEVO-T is indicated:

1. As replacement or supplemental therapy in patients with hypothyroidism of any etiology, except transient hypothyroidism during the recovery phase of subacute thyroiditis. This category includes cretinism, myxedema, and ordinary hypothyroidism in patients of any age (children, adults, the elderly), or state (including pregnancy); primary hypothyroidism resulting from functional deficiency, primary atrophy, partial or total absence of thyroid gland, or the effects of surgery, radiation, or drugs, with or without the presence of goiter; and secondary (pituitary), or tertiary (hypothalamic) hypothyroidism (see **CONTRAINDICATIONS** and **PRECAUTIONS**).

2. As a pituitary TSH suppressant, in the treatment or prevention of various types of euthyroid goiters, including thyroid nodules, subacute or chronic lymphocytic thyroiditis (Hashimoto's), multinodular goiter, and in the management of thyroid cancer.

3. As a diagnostic agent in suppression tests to aid in the diagnosis of suspected mild hyperthyroidism or thyroid gland autonomy.

CONTRAINDICATIONS

Thyroid hormone preparations are generally contraindicated in patients with diagnosed but as yet uncorrected adrenal cortical insufficiency, untreated thyrotoxicosis, and apparent

hypersensitivity to any of their active or extraneous constituents. There is no well documented evidence from the literature, however, of true allergic or idiosyncratic reactions to thyroid hormone.

WARNINGS

> Drugs with thyroid hormone activity, alone or together with other therapeutic agents, have been used for the treatment of obesity. In euthyroid patients, doses within the range of daily hormonal requirements are ineffective for weight reduction. Larger doses may produce serious or even life threatening manifestations of toxicity, particularly when given in association with sympathomimetic amines such as those used for their anorectic effects.

The use of thyroid hormones in the therapy of obesity, alone or combined with other drugs, is unjustified and has been shown to be ineffective. Neither is their use justified for the treatment of male or female infertility unless this condition is accompanied by hypothyroidism.

PRECAUTIONS

General: Thyroid hormones should be used with great caution in a number of circumstances where the integrity of the cardiovascular system, particularly the coronary arteries, is suspected. These include patients with angina pectoris or the elderly, who have a greater likelihood of occult cardiac disease. In these patients, therapy should be initiated with low doses, ie, 25 to 50 mcg levothyroxine (T_4). When, in such patients, a euthyroid state can only be reached at the expense of an aggravation of the cardiovascular disease, thyroid hormone dosage should be reduced.

Thyroid hormone therapy in patients with concomitant diabetes mellitus or insipidus or adrenal cortical insufficiency aggravates the intensity of their symptoms. Appropriate adjustments of the various therapeutic measures directed at these concomitant endocrine diseases are required. The therapy of myxedema coma may require simultaneous administration of glucocorticoids (see **DOSAGE AND ADMINISTRATION**).

Hypothyroidism decreases and hyperthyroidism increases the sensitivity to oral anticoagulants. Prothrombin time should be closely monitored in thyroid treated patients on oral anticoagulants and dosage of the latter agents adjusted on the basis of frequent prothrombin time determinations. In infants, excessive doses of thyroid hormone preparations may produce craniosynostosis.

Information for the Patient: Patients on thyroid hormone preparations and parents of children on thyroid therapy should be informed that:

1. Replacement therapy is to be taken essentially for life, with the exception of cases of transient hypothyroidism, usually associated with thyroiditis, and in those patients receiving a therapeutic trial of the drug.

2. They should immediately report during the course of therapy any signs or symptoms of thyroid hormone toxicity, eg, chest pain, increased pulse rate, palpitations, excessive sweating, heat intolerance, nervousness, or any other unusual event.

3. In case of concomitant diabetes mellitus, the daily dosage of antidiabetic medication may need readjustment as thyroid hormone replacement is achieved. If thyroid medication is stopped, a downward readjustment of the dosage of insulin or oral hypoglycemic agent may be necessary to avoid hypoglycemia. At all times, close monitoring of blood or urinary glucose levels is mandatory in such patients.

4. In case of concomitant oral anticoagulant therapy, the prothrombin time should be measured frequently to determine if the dosage of oral anticoagulants is to be readjusted.

5. Partial loss of hair may be experienced by children in the first few months of thyroid therapy, but this is usually a transient phenomenon and later recovery is usually the rule.

Laboratory Tests: Treatment of patients with thyroid hormones requires the periodic assessment of thyroid status by means of appropriate laboratory tests, by full clinical evaluation, or both. The TSH suppression test can be used to test the effectiveness of any thyroid preparation bearing in mind the relative insensitivity of the infant pituitary to the negative feedback effect of thyroid hormones. Serum T_4 levels can be used to test the effectiveness of levothyroxine sodium. When the total serum T_4 is low but TSH is normal, a test specific to assess unbound (free) T_4 levels is warranted. Specific measurements of T_4 and T_3 by competitive protein binding or radioimmunoassay are not influenced by blood level of organic or inorganic iodine and have essentially replaced older tests of thyroid hormone measurements, ie, PBI, BEI, and T_4 by column.

Drug Interactions: *Oral Anticoagulants:* Thyroid hormones appear to increase catabolism of vitamin K-dependent clotting factors. If oral anticoagulants are also being given, compensatory increases in clotting factor synthesis are impaired. Patients stabilized on oral anticoagulants who are found to require thyroid replacement therapy should be watched very closely when thyroid is started. If a patient is truly hypothyroid, it is likely that a reduction in anticoagulant dosage will be required. No special precautions appear to be necessary when oral anticoagulant therapy is begun in a patient already stabilized on maintenance thyroid replacement therapy.

Insulin or Oral Hypoglycemics: Initiating thyroid replacement therapy may cause increases in insulin or oral hypoglycemic requirements. The effects seen are poorly understood and depend upon a variety of factors such as dose and type of thyroid preparations and endocrine status of the patient. Patients receiving insulin or oral hypoglycemics should be closely watched during initiation of thyroid replacement therapy.

Cholestyramine: Cholestyramine binds both T_4 and T_3 in the intestine, thus impairing absorption of these thyroid hormones. *In vitro* studies indicate that the binding is not easily reversed. Therefore, 4 to 5 hours should elapse between administration of cholestyramine and thyroid hormones.

Estrogen, Oral Contraceptives: Estrogens tend to increase serum thyroxine-binding globulin (TBG). In a patient with a nonfunctioning thyroid gland who is receiving thyroid replacement therapy, free levothyroxine may be decreased when estrogens are started thus increasing thyroid requirements. However, if the patient's thyroid gland has sufficient function, the decreased free thyroxine will result in a compensatory increase in thyroxine output by the thyroid. Therefore, patients without a functioning thyroid gland who are on thyroid replacement therapy may need to increase their thyroid dose if estrogens or estrogen-containing oral contraceptives are given.

Drug/Laboratory Test Interactions: The following drugs or moieties are known to interfere with some laboratory tests performed in patients on thyroid hormone therapy: Androgens, corticosteroids, estrogens, oral contraceptives

containing estrogens, iodine-containing preparations, and the numerous preparations containing salicylates.

1. Changes in TBG concentration should be taken into consideration in the interpretation of T_4 and T_3 values. Pregnancy, estrogens, and estrogen-containing oral contraceptives increase TBG concentrations. TBG may also be increased during infectious hepatitis. Decreases in TBG concentrations are observed in nephrosis, acromegaly, and after androgen or corticosteroid therapy. Familial hyper- or hypothyroxine-binding-globulinemias have been described. The incidence of TBG deficiency approximates 1 in 9,000. The binding of thyroxine by TBPA is inhibited by salicylates. In such cases, the unbound (free) hormone should be measured. Alternatively, an indirect measure of free thyroxine, such as the Free Thyroxine Index (FTI) may be used.

2. Medicinal or dietary iodine interferes with all *in vitro* tests of radioiodine uptake, producing low uptakes which may not indicate a true decrease in hormone synthesis.

3. The persistence of clinical and laboratory evidence of hypothyroidism in spite of adequate dosage replacement indicates either poor patient compliance, poor absorption, or inactivity of the preparation. Intracellular resistance to thyroid hormone is quite rare, and is suggested by clinical signs and symptoms of hypothyroidism in the presence of high serum T_4 levels.

Carcinogenesis, Mutagenesis, and Impairment of Fertility: A reported association between prolonged thyroid therapy and breast cancer has not been confirmed and patients on thyroid for established indications should not discontinue therapy. No confirmatory long-term studies in animals have been performed to evaluate carcinogenic potential, mutagenicity, or impairment of fertility in either males or females.

Pregnancy—Category A: Thyroid hormones do not readily cross the placental barrier. The clinical experience to date does not indicate any adverse effect on fetuses when thyroid hormones are administered to pregnant women. On the basis of current knowledge, thyroid replacement therapy to hypothyroid women should not be discontinued during pregnancy.

Nursing Mothers: Minimal amounts of thyroid hormones are excreted in human milk. Thyroid is not associated with serious adverse reactions and does not have known tumorigenic potential. While caution should be exercised when thyroid is administered to a nursing woman, adequate replacement doses of levothyroxine are generally needed to maintain normal lactation.

Pediatric Use: Pregnant mothers provide little or no thyroid hormone to the fetus. The incidence of congenital hypothyroidism is relatively high (1 in 4,000) and the hypothyroid fetus would not derive any benefit from the small amounts of hormone crossing the placental barrier. Routine determinations of serum T_4 and/or TSH is strongly advised in neonates in view of the deleterious effects of thyroid deficiency on growth and development.

Treatment should be initiated immediately upon diagnosis, and maintained for life, unless transient hypothyroidism is suspected; in which case, therapy may be interrupted for 2 to 8 weeks after the age of 3 years to reassess the condition. Cessation of therapy is justified in patients who have maintained a normal TSH during those 2 to 8 weeks.

ADVERSE REACTIONS

Adverse reactions other than those indicative of hyperthyroidism because of therapeutic overdosage, either initially or during the maintenance periods, are rare (see **OVERDOSAGE**).

OVERDOSAGE

Signs and Symptoms: Excessive doses of thyroid result in hypermetabolic state resembling in every respect the condition of endogenous origin. The condition may be self-induced.

Treatment of Overdosage: Dosage should be reduced or therapy temporarily discontinued if signs and symptoms of overdosage appear. Treatment may be reinstated at a lower dosage. In normal individuals, normal hypothalamic-pituitary-thyroid axis function is restored in 6 to 8 weeks after thyroid suppression.

Treatment of acute massive thyroid hormone overdosage is aimed at reducing gastrointestinal absorption of the drugs and counteracting central and peripheral effects, mainly those of increased sympathetic activity. Vomiting may be induced initially if further gastrointestinal absorption can reasonably be prevented and barring contraindications such as coma, convulsions, or loss of the gagging reflex. Treatment is symptomatic and supportive. Oxygen may be administered and ventilation maintained. Cardiac glycosides may be indicated if congestive heart failure develops. Measures to control fever, hypoglycemia, or fluid loss should be instituted if needed. Antiadrenergic agents, particularly propranolol, have been used advantageously in the treatment of increased sympathetic activity. Propranolol may be administered intravenously at a dosage of 1 to 3 mg over a 10-minute period or orally, 80 to 160 mg/day, especially when no contraindications exist for its use. Other adjunctive measures may include administration of cholestyramine to interfere with thyroxine absorption, and glucocorticoids to inhibit conversion of T_4 to T_3.

DOSAGE AND ADMINISTRATION

The dosage and rate of administration of LEVO-T are determined by the indication and must in every case be individualized according to patient response and laboratory findings.

Hypothyroidism: LEVO-T is usually instituted using low doses, with increments which depend on the cardiovascular status of the patient. The usual starting dose is 50 mcg, with increments of 25 mcg every 2 to 3 weeks. A lower starting dosage, 25 mcg/day or less, is recommended in patients with longstanding hypothyroidism, particularly if cardiovascular impairment is suspected, in which case extreme caution is recommended. The appearance of angina is an indication for a reduction in dosage. Most patients require not more than 200 mcg/day. Failure to respond to doses of 300 mcg suggests lack of compliance or malabsorption. Adequate therapy usually results in normal TSH and T_4 levels after 2 to 3 weeks of the maintenance dose.

Readjustment of LEVO-T tablets dosage should be made within the first 4 weeks of therapy, after proper clinical and laboratory evaluations.

TSH Suppression in Thyroid Cancer, Nodules, Euthyroid Goiters: Exogenous thyroid hormone may produce regression of metastases from follicular and papillary carcinoma of the thyroid and is used as ancillary therapy of these conditions following surgery or radioactive iodine. Medullary carcinoma of the thyroid is usually unresponsive to this therapy. TSH should be suppressed to low or undetectable levels. Therefore, larger amounts of thyroid hormone than those used for replacement therapy are frequently required. This therapy is also used in treating nontoxic solitary nodules and multinodular goiters, and to prevent thyroid enlargement in chronic (Hashimoto's) thyroiditis.

Thyroid Suppression Therapy: Administration of thyroid hormone in doses higher than those produced physiologically by the gland results in suppression of the production of endogenous hormone. This is the basis for the thyroid suppression test and is used as an aid in the diagnosis of patients with signs of mild hyperthyroidism in whom base line laboratory tests appear normal, or to demonstrate thyroid gland autonomy in patients with Grave's ophthalmopathy. ^{131}I uptake is determined before and after the administration of the exogenous hormone. A 50% or greater suppression of uptake indicates a normal thyroid-pituitary axis and thus rules out hyroid gland autonomy.

For adults, the average suppressive dose of levothyroxine (T_4) is 2.6 mcg/kg of body weight per day given for 7 to 10 days. These doses usually yield normal serum T_4 and T_3 levels and lack of response to TSH.

Levothyroxine sodium should be administered cautiously to patients in whom there is a strong suspicion of hyroid gland autonomy, in view of the fact that the exogenous hormone effects will be additive to the endogenous source.

Pediatric Dosage: Pediatric dosage should follow the recommendations summarized in Table I. In infants with congenital hypothyroidism, therapy with full doses should be instituted as soon as the diagnosis has been made. Levothyroxine sodium tablets may be given to infants and children who cannot swallow intact tablets by crushing the proper dose tablet and suspending the freshly crushed tablet in a small amount of water or formula. The suspension can be given by spoon or dropper. DO NOT STORE THE SUSPENSION FOR ANY PERIOD OF TIME. The crushed tablet may also be sprinkled over a small amount of food, such as cooked cereal or apple sauce.

Table I
RECOMMENDED PEDIATRIC DOSAGE FOR CONGENITAL HYPOTHYROIDISM*
LEVO-T (Levothyroxine Sodium Tablets, USP)

Age	Doses per day	Daily dose per kg of body weight
0-6 months	25-50 mcg	8-10 mcg
6-12 months	50-75 mcg	6-8 mcg
1-5 years	75-100 mcg	5-6 mcg
6-12 years	100-150 mcg	4-5 mcg

*To be adjusted on the basis of clinical response and laboratory tests (see **Laboratory Tests**).

HOW SUPPLIED

Levothyroxine Sodium Tablets, USP are supplied as scored, color-coded, potency-marked tablets as follows:

25 mcg, orange, marked 25 and LL on one side and LEVO-T on the other side:
NDC 0205-3610-43 Bottle of 100
NDC 0205-3610-34 Bottle of 1000

50 mcg, white, marked 50 and LL on one side and LEVO-T on the other side:
NDC 0205-3611-43 Bottle of 100
NDC 0205-3611-34 Bottle of 1000

75 mcg, violet, marked 75 and LL on one side and LEVO-T on the other side:
NDC 0205-3612-43 Bottle of 100
NDC 0205-3612-34 Bottle of 1000

100 mcg, yellow, marked 100 and LL on one side and LEVO-T on the other side:
NDC 0205-3613-43 Bottle of 100
NDC 0205-3613-34 Bottle of 1000

125 mcg, brown, marked 125 and LL on one side and LEVO-T on the other side:
NDC 0205-3614-43 Bottle of 100
NDC 0205-3614-34 Bottle of 1000

150 mcg, blue, marked 150 and LL on one side and LEVO-T on the other side:
NDC 0205-3615-43 Bottle of 100
NDC 0205-3615-34 Bottle of 1000

200 mcg, pink, marked 200 and LL on one side and LEVO-T on the other side:
NDC 0205-3616-43 Bottle of 100
NDC 0205-3616-34 Bottle of 1000

300 mcg, green, marked 300 and LL on one side and LEVO-T on the other side:
NDC 0205-3617-43 Bottle of 100
NDC 0205-3617-34 Bottle of 1000

Store at controlled room temperature 15°-30°C (59°-86°F).

CAUTION:
Federal law prohibits dispensing without a prescription.

Manufactured by
MOVA PHARMACEUTICAL CORPORATION
Caguas, Puerto Rico 00725

For
LEDERLE PARENTERALS INC.
Carolina, Puerto Rico 00630

Distributed by
LEDERLE LABORATORIES DIVISION
American Cyanamid Company
Pearl River, New York 10965

Rev. 5/91
MV114258-91

STANDARD PRODUCTS

MOVA

*Synthroid® is a registered trademark of Boots Pharmaceuticals, Inc.

References:
1. Data on file, Lederle Laboratories, Pearl River, NY.
2. Medi-Span's **MASTER DRUG DATA BASE-SELECT(MDDB-SELECT™)**. Indianapolis, IN: Medi-Span, Inc; August 1992.

Lederle Laboratories
A Division of American Cyanamid Company
Wayne, New Jersey 07470

Table 1. Studies Showing the Impact of Age and Treatment on the Rate of Recurrence of Shoulder Dislocation

Study	Total Patients	Impact of Age on the Recurrence of Dislocation		Impact of Treatment on the Recurrence of Dislocation	
		Age (y)	% Recurrence	Treatment	% Recurrence
MacLaughlin and Cavallaro[9]	101	<20	90	Sling and swathe 3+ wk	0
		20–40	60	Sling and swathe 2–3 wk	1
		>40	10	Sling and swathe 1–2 wk	6
				Sling and swathe 0–1 wk	14
Rowe and Sakellarides[13]	324	<20	94	Sling 3–6 wk	37
		20–40	74	Sling 1–3 wk	46
		>40	14	No treatment	70
				Sling and swathe 3–6 wk	33
				Sling and swathe 1–3 wk	26
Kazar and Relovszky[18]	566	<20	46	Immobilized 15 d	4.5
		21–30	31	Immobilized 8–14 d	6
		31–40	6	Immobilized 0–7 d	16
		>41	<6		
Hovelius[19]	63	<20	90	Immobilized >20 d	75
		20–25	65	Immobilized 10–20 d	87
		>25	50	Immobilized <10 d	68
Kiviluoto et al[6]	53	16–20	56	Immobilized 3 wk	22
		21–30	26	Immobilized 1 wk	56
		>30	<8		
Yoneda et al[20]	104	21.5	17.3	Sling and swathe 5 wk, then limited exercise 6 wk	17.3
Henry and Genung[21]	121	19 (average)	—	Immobilized	90
				Not immobilized	85
Simonet and Cofield[16]	116	<20	66	Immobilized, then	44
		20–40	40	activity restricted 6 wk	
		>40	0	No activity restricted	85
Hovelius[22]	254	12–22	55	Age <22 y	
		23–29	37	Immobilized 3–4 wk	64
		30–40	12	Mobilized early	58
				Mixed treatment	17
				Age 23–29 y	
				Immobilized 3–4 wk	58
				Mobilized early	47
				Mixed treatment	17
				Age 30–40 y	
				Immobilized 3–4 wk	63
				Mobilized early	33
				Mixed treatment	20
Wheeler et al[23]	38	—	—	Immobilized 3 wk, then 3 mo physical therapy and activity restriction	92

months in athletes.[16,23,24] It takes that long for tendons to heal. The patient's age is also important for prognosis of contractures; in the older population, prolonged immobilization may lead to a "frozen" shoulder. Pasila and colleagues[15] showed that a longer period of immobilization (from 1 to 3 weeks) was associated with a longer period needed to recover normal mobility (from 3 to 5 weeks). Kazar and Relovszky[18] found 37% of patients over 50 years of age had contractures after suffering a shoulder dislocation, compared with 14% in the 31 to 50 years age group and 2% in the group under 30 years of age. They recommended a shorter period of immobilization for those over 50 years of age.

Rehabilitation

Since the subscapularis is one of the major components in preventing anterior laxity of the shoulder, strengthening it through rehabilitation therapy should decrease the recurrence rate. (The Appendix describes an 8-step pro-

gram for rehabilitation of the muscles around the shoulder.) Rowe believed that resistive exercises did not help patients with established recurrences and normal muscle mass, but did help those with poor shoulder musculature and those who complained of subluxations.[13] The program recommended by Rowe and Zarins[11] will decrease pain and disability to the extent that surgery is not required. In older patients, however, these exercises may irritate or worsen a rotator cuff tear.

Simonet and Cofield[16] found that physical therapy made no difference; however, their therapy consisted of only a single teaching session followed by a patient-initiated home program. Aronen and Regan[24] in their small study of 20 Naval Academy midshipmen found that a restrengthening program of the muscles of internal rotation and abduction, plus rigid restriction of activities until the program was completed, resulted in a recurrence rate (including two subluxations) of 25% over an average follow-up period of 35.8 months. This uncontrolled study of a highly athletic group showed a significant reduction in risk. Another small uncontrolled study of 38 army cadets found recurrent dislocations or subluxations in 92% following 3 weeks of immobilization, completion of a supervised physical therapy program, and 3 months of restriction from contact, throwing, and overhead sports.[23] The study conducted at the Naval Academy describes in detail their rehabilitation program, whereas there is very little description of the therapies used in the other two studies. Thus, it is difficult to assess how rehabilitation therapy might improve the prognosis, since it is so individualized, and may vary from institution to institution. Nevertheless, rehabilitation therapy may still be useful in strengthening and helping regain mobility of the shoulder after a period of rest and immobilization during which muscles tend to atrophy (Table 2). It is also a noninvasive, conservative means of therapy to be used before resorting to surgery. The cost of therapy three times a week for 6 weeks must be considered as well as possible reinjury if overly aggressive strengthening and range-of-motion exercises are prescribed.

Table 2. Shoulder Rehabilitation Following Anterior Dislocation

Range of motion
Passive
Done by therapist
Home program using a wand*
Active
Pendulum (Codman) exercises
Wall climbing—lateral, anterior
Strengthening
Isometric
Resistive exercise using surgical tubing, bungee cords, Theraband, free or pulley weights
Abduction
Adduction
Forward flexion
Backward extension
Internal rotation in abduction and extension (elbow on hip)
External rotation in abduction and extension
Specific functional strengthening, eg, throwing motion against resistance

** Gripping the wand with both hands, the good arm moves the injured side through its range of motion.*

Age

In Rowe's original study in 1956[5] and in the follow-up study by Rowe and Sakellarides in 1961,[13] age was found to be the biggest determinant in the prognosis of recurrences in primary anterior dislocations. Of 324 patients, those less than 20 years of age had a 94% recurrence rate, those 20 to 40 years of age had a 74% recurrence rate, and only 14% of those over 40 years of age had a recurrence.

This was also observed in the study by MacLaughlin and Cavallaro in 1950.[9] In their series of 101 patients, there were 7 recurrences (90%) in those less than 20 years of age, 10 (60%) in the group 20 to 40 years of age, and 4 (10%) among those older than 40 years of age. This has been confirmed by Kazar and Relovszky,[18] Hovelius,[19,22] Kiviluoto and colleagues,[6] and Simonet and Cofield,[16] who found recurrence rates of between 46% and 90% for those under 20 years of age. The reason is not clear but may be the greater physical activity of the younger age group or possibly greater noncompliance with therapy, home exercises, or activity restrictions (Table 1). Two studies had lower recurrence rates: in the study by Yoneda et al[20] 104 athletes with an average age of 21.5 years had a 17.3% recurrence rate; in Aronen and Regan's study[24] 20 naval cadets with an average age of 19.2 years had a 25% recurrence rate. Both studies included a supervised rehabilitation program.

Sex

As one might expect, it has been reported that the incidence of shoulder dislocations is generally higher in men than in women at a ratio of 2:1. In adolescence this difference may not be as great and may actually be reversed after the age of 60 years. In the largest study (Rowe[5]) involving 323 men and 165 women, there were approximately twice as many recurrent injuries in men as in women. In other smaller studies involving 254 patients (Hovelius[22]) and 116 patients (Simonet and Cofield[16]), little difference between the sexes was found.

Complications

Complications that occur with anterior shoulder dislocations principally involve osseous elements, nerves, or the rotator cuff muscles. Vascular injuries do occur, but are

rare and usually occur in the elderly who may have abnormal blood vessels.[3]

Osseous complications include fractures of the shoulder girdle, ie, the surgical neck, clavicle, scapula, acromion, the greater tuberosity, the anterior glenoid rim, and the humeral head.

In his study of 500 patients, Rowe[5] found 122 fracture-dislocations; 75 involved the greater tuberosity, 10 the shoulder girdle, and 27 the anterior glenoid rim. In 125 primary dislocations, 38% had humeral head defects compared with 57% found in recurrent dislocations. This Hill-Sachs lesion or posterior-lateral notch in the humeral head is from a compression fracture of the softest portion of the humeral head occurring when it slips over the rim of the glenoid. When this is found, it is associated with an increased incidence of recurrences.[5,22] Moseley[3] believed that this defect, while not predisposing to dislocation, tended to facilitate the development of the recurrent state.

Fractures of the anterior glenoid rim also predispose patients to recurrences. Pushing the humeral head anteriorly and comparing it with the opposite shoulder may aid in the detection of fractures. Unfortunately, often these labrum defects can only be detected through invasive procedures such as arthroscopy or open surgery.

Patients with fracture dislocations including greater tuberosity fractures had a low incidence of recurrence (5.6%).[5] This may be because these patients were older and less active, or had greater trauma causing the dislocation, which may lead to increased scarring and immobilization. Hovelius[22] theorized that either the younger skeleton was stronger or the capsule more lax so that the humeral head passed the glenoid rim without causing a fracture.

Rotator cuff tears can also occur with shoulder dislocations. Pasila and colleagues[15] found 26 rotator cuff tears in 226 primary dislocations. In these shoulders with tears the median time for normal mobility to be regained was 1 year, compared with 51 days in the 31 shoulders with nerve lesions and 27 days in the uncomplicated dislocations. Regaining full mobility was slowed by advanced age, long immobilization, long periods before the dislocations were reduced, rotator cuff rupture, and severe primary dislocation.

Nerve injuries are relatively uncommon (5% to 30%) and are often related to severe dislocations. Fortunately, these lesions are transient, and the prognosis for recovery is good, especially if only one nerve is involved. Injuries involving a combination of nerves are more likely to be permanent. The brachial plexus, the median cord, the ulnar nerves, and the radial and axillary nerves are the nerves usually damaged, in decreasing frequency. If nerve damage is permanent, weakening of the shoulder muscles can predispose the patient to an increased recurrence rate.[4–7,15,25]

In summary, complications are usually associated with traumatic dislocations; the more severe the trauma, the worse the complications. Most fracture dislocations not involving the glenoid labrum or humeral head defects have a lower recurrence rate. Injuries with multiple nerve defects have a poorer prognosis of recovery and sometimes have an increased recurrence rate. Rotator cuff tears are also not common, but when they occur, significant loss of mobility results; however, the redislocation rate is low.

Surgery

The role of surgery in anterior shoulder dislocations is principally to prevent recurrences, or, rarely, to reduce an otherwise nonreducible dislocation. If recurrences are frequent, ie, more than several times a year, or if there is symptomatic anterior instability with activities of daily living or overhead activities, the patient would benefit from surgery. Before surgery is considered, rehabilitation and strengthening of the rotator cuff muscles may be attempted, especially in infrequent recurrent dislocations or recurrent subluxations. If the patient's shoulder is constantly exposed to violent forces, eg, epileptic convulsions, surgery may not help. In young, athletic patients exposed to trauma, nonsurgical treatment is unlikely to succeed and surgery should be given higher consideration.

In a first-time anterior shoulder dislocation, identifying the prognosis for recurrence can assist one in predicting those patients who will eventually need surgery. If surgery is desired, the patient must understand the long rehabilitation period postoperatively. Without surgery it is difficult to identify pathologic conditions that predispose to recurrences. One of many procedures could be used, depending on the expertise and skill of the surgeon, the type of lesion(s) thought to be present, and the patient's particular circumstances, eg, professional athlete. Rowe[14] believed that 6 of the 300 or more procedures had stood the test of time. His preferences in order are: the Bankart, Putti-Platt, Hybbinette-Eden, Magnuson-Stack, Gallie-LeMesurier, and the modified Bristow procedure.

Almost all procedures fall into one of three groups: (1) shortening of the subscapularis tendon (Magnuson-Stack, Putti-Platt), (2) alteration of inherent bone stability (Bristow, Eden-Hybbinette), and (3) anterior capsulorraphy (Bankart). Unlike the others, the Bankart procedure is an anatomically normal repair. It reattaches the anterior capsule to the anterior glenoid fossa in a layer-by-layer fashion, thus repairing the Bankart lesion. The Putti-Platt shortens the subscapularis, thus protecting the anterior rim by reinforcing the muscles and limiting external rotation. The Hybbinette-Eden grafts an osseous barrier along the ante-

continued on page 575

continued from page 571

rior glenoid rim, thus preventing a humeral head defect. The Magnuson-Stack, the Gallie-LeMesurier, and the modified Bristow transplant muscles or fascia, thus constructing a sling to restrain the humeral head and prevent instability. Maximizing the stability of the shoulder results in a loss of mobility.

Recently, early arthroscopy with anterior glenoid abrasion or capsulorraphy or both was evaluated in 47 army cadets in a prospective nonrandomized study where the cadets were given the surgical option. In this population, where those treated nonsurgically (35 of 38) had a 92% rate of recurrence (either subluxation or dislocation), the rate of redislocation in those treated arthroscopically was 22% (2 of 9). Both groups received the same rehabilitation.[23] Arthroscopy may be a useful intermediate step before an open operative procedure is considered in a very high risk and elite athletic group, but more study needs to occur before this is generally recommended as an alternative to conservative rehabilitation. If conservative therapy can obviate the need for surgery, it would be more cost effective, though it may be more time-consuming and inconvenient. These different procedures have different failure rates and require different amounts of exploration; therefore, surgery for recurrent shoulder dislocations should be carefully considered.

Summary

Primary anterior shoulder dislocations are the most common of dislocation injuries. The shoulder's stability is largely dependent on its soft tissue components. Dislocation usually occurs with a traumatic event. This trauma may cause increased complications and result in a long period of immobilization and disability. Traumatic dislocations with injury to the shoulder ligaments or humeral head defects or glenoid labrum defects may predispose the patient to increased recurrences. Atraumatic dislocations usually reflect an anatomically unstable shoulder or, occasionally, psychological factors. Recovery after reduction usually involves a period of immobilization. In the young, decreased stress and range of motion from 6 weeks to 3 months may be beneficial. This should be followed by rehabilitation consisting of shoulder muscle strengthening and range-of-motion exercises. The patient older than 50 years, would best benefit from a short immobilization period followed by range of motion to prevent loss of mobility. Young age and increased level of activity may be the most important variables in predicting prognosis after a primary anterior dislocation. Persistent recurrences and disability following nonoperative treatment are often an indication for surgical treatment. The main complications of shoulder dislocations are fractures, nerve lesions, and rotator cuff tears, all of which usually occur as a result of traumatic injuries.

Acknowledgments

I am grateful for the help of James Puffer, MD, in initially supervising and reviewing this manuscript. I also thank Rebecca Short, MD, and Janet Cunningham, MD, for their critical review of the revised manuscript, and Chau Thang for her assistance in preparing the text.

References

1. Hippocrates. Theory and practice of medicine. New York: The Citadel Press, 1964.
2. Kroner K, Lind T, Jensen J. The epidemiology of shoulder dislocations. Arch Orthop Trauma Surg 1989; 10:258–99.
3. Moseley HF. The basic lesions of recurrent anterior dislocation. Surg Clin North Am 1963; 43:1631–4.
4. Depalma AF, Flannery GF. Acute anterior dislocation of the shoulder. J Sports Med 1973; 1:6–15.
5. Rowe CR. Prognosis in dislocations of the shoulder. J Bone Joint Surg 1956; 38A:957–77.
6. Kiviluoto O, Pasila M, Jaroma H, Sundholm A. Immobilization after primary dislocation of the shoulder. Acta Orthop Scand 1980; 51:915–9.
7. McLaughlin HL, MacLellan DI. Recurrent anterior dislocation of the shoulder, a comparative study. J Trauma 1967; 7:191–201.
8. Turkel SJ, Panio MW, Marshall JL, Girgis FS. Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. J Bone Joint Surg 1981; 63A:208–17.
9. McLaughlin HL, Cavallaro WU. Primary anterior dislocation of the shoulder. Am J Surg 1950; 80:615–21.
10. Depalma AF. The role of the subscapularis in recurrent anterior dislocations of the shoulder. Clin Orthop 1967; 54:35–49.
11. Rowe CR, Zarins BZ. Recurrent transient subluxation of the shoulder. J Bone Joint Surg 1981; 63A:863–71.
12. McLaughlin HL. Recurrent anterior dislocation of the shoulder, morbid anatomy. Am J Surg 1960; 99:628–32.
13. Rowe CR, Sakellarides HT. Factors related to recurrences of anterior dislocations of the shoulder. Clin Orthop 1961; 20:40–7.
14. Rowe CR. Acute and recurrent anterior dislocations of the shoulder. Orthop Clin North Am 1980; 11:253–70.
15. Pasila M, Kiviluoto O, Jaroma H, Sundholm A. Recovery from primary shoulder dislocation and its complications. Acta Orthop Scand 1980; 51:257–62.
16. Simonet WT, Cofield RH. Prognosis in anterior shoulder dislocation. Am J Sports Med 1984; 12:19–24.
17. Bankart B. The pathology and treatment of recurrent dislocations of the shoulder joint. Br J Surg 1938; 26:23–9.
18. Kazar B, Relovszky E. Prognosis of primary dislocation of the shoulder. Acta Orthop Scand 1969; 40:216–24.
19. Hovelius L. Shoulder dislocation in Swedish ice hockey players. Am J Sports Med 1978; 6:373–7.
20. Yoneda B, Welsh RP, MacIntosh DL. Conservative treatment of shoulder dislocation in young males. J Bone Joint Surg 1982; 64B:254–5.
21. Henry JH, Genung JA. Natural history of glenohumeral dislocation—revisited. Am J Sports Med 1982; 10:135–7.
22. Hovelius L. Anterior dislocation of the shoulder in teen-agers and young adults. J Bone Joint Surg 1987; 69A:393–9.
23. Wheeler JH, Ryan JB, Arciero RA, Molinari RN. Arthroscopic versus non-operative treatment of acute shoulder dislocation in young athletes. Arthroscopy 1989; 5:213–7.
24. Aronen JG, Regan K. Decreasing the incidence of recurrence of first time anterior shoulder dislocations with rehabilitation. Am J Sports Med 1984; 12:283–91.
25. Pasila M, Jaroma H, Kiviluoto O, Sundholm A. Early complications of primary shoulder dislocations. Acta Orthop Scand 1978; 49:260–3.

Appendix

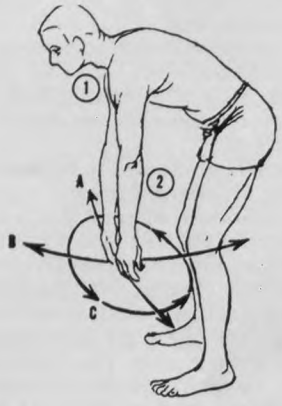
EXERCISES FOR MUSCLES AROUND THE SHOULDER

GRAVITY EXERCISES

Exercise 1
Bend far forward holding shoulders and trunk still (Step 1).

Exercise 2
Let arms hang loosely and swing like a pendulum *(A)* across the front of the body, *(B)* backward and forward, and *(C)* in a circle (Step 2).

Note. The arc should be small at first, and then gradually increased.



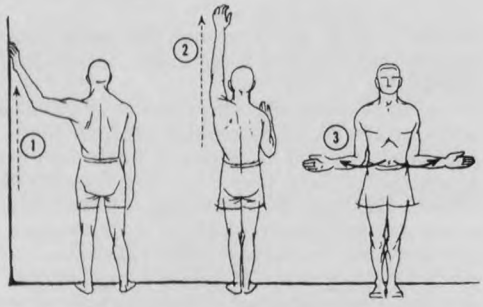
ANTIGRAVITY EXERCISES

These exercises will be added after you have mastered gravity exercises.

Exercise 1
Arm is to the side. Raise and lower with fingers "climbing" the wall (Step 1). Repeat 10 times.

Exercise 2
Arm is to the front. Raise and lower with fingers "climbing" the wall (Step 2). Repeat 10 times.

Exercise 3
Back is to the wall. Move your arms through the arc shown in Step 3. Repeat 10 times.



Exercise 4
Lie flat on your back and move your arms through the arc shown in Step 4. Repeat 10 times.
Exercises 3 and 4 are intended to increase external rotation.

Exercise 5
Lie flat on back with hands under head and touch the elbows to the table or floor (Step 5).

Exercise 6
Pulley exercises (Step 6). Perform as illustrated for 5 minutes, four times daily.

Note. These exercises must be executed on a regulated program, for a certain period and a specific number of times each day.

From H. W. Griffith. *Instructions for Patients.* 4th ed. Philadelphia: W. B. Saunders, 1989: 139. Reprinted with permission.