ulation of the nerve. This in turn evoked small, graded spikes not larger than about 25 mv.

Recordings of tension changes were made from innervated single muscle fibers by cutting the attachment to the scute at one end and clamping it in a small holder attached directly to the peg of an RCA 5734 transducer tube. There was no or very slight mechanical response to a single, small post-synaptic potential, but there was marked growth during trains at increasing frequency. Large post-synaptic potentials gave rise to brisk twitches. The responses to stimulation of opposite ends were similar.

A brief tetanus of only one end leads to tight closing of the valves. Stimulating the two nerves together more than doubles the force developed. Fatigue occurs after about 8 minutes when stimulation is continuously applied to one end. However, the dual nerve supply enables the animal to maintain contraction of the adductors indefinitely, without neuromuscular fatigue, by alternating activation between the two ends.

The depressor muscles each contain 30 to 40 muscle fibers in the range of 500 to 1,400 μ maximum width. These in turn contain densely-packed, coarse fibrils, but do not show the internal partitions or folding of the surface, commonly found in large crustacean fibers and also present in the adductors of *B. nubilus*. There is very little connective tissue between fibers, so they can easily be separated. Each has a fine tendon, 3 to 4 mm long, at its scutal-tergal end (Fig. 1). This greatly facilitates preparation for physiological work.

A single fiber may be partially isolated with its nerve supply intact. Force was measured by clamping the tendon in a device attached directly to a transducer tube, at the same time as membrane potentials were recorded with an intracellular electrode. The nerve, which enters from the scutal end, was stimulated electrically on the scute. It supplies two excitor axons to each muscle fiber and gives off multiterminal endings along the whole length of the fiber. The electrical responses are of two sizes: one, 15 to 20 mv, gives rise to a weak twitch, (about 0.3 g), and the other, 25 to 40 mv, gives rise to a much larger twitch, up to 6 g (Fig. 2). Tetanus tensions up to 50 g per fiber (5 kg/cm^2) were obtained.

The capacity of the muscle for shortening is remarkable. Repeated, fully reversible contractions of single fibers to one sixth of the resting length were obtained (Fig. 1) by bathing the fibers alternately in high potassium Ringer and normal barnacle Ringer solutions (*2*).

These extraordinary muscle fibers could be extremely valuable in fundamental muscle research (*3*).

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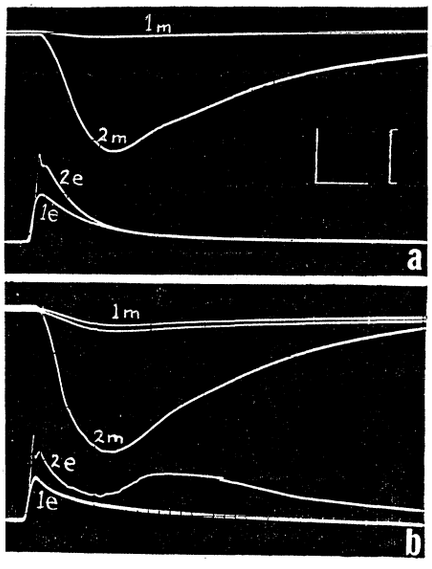

Fig. 2. Membrane potentials and force development in two (*a* and *b*) preparations of single fibers from m. depressor scutorum rostralis of *Balanus nubilus*. The muscle fibers were partially isolated, and their tendons were connected to a force transducer (force is registered on upper traces). An intracellular electrode was placed in the center of the fibers and used to record membrane potential changes (lower traces). (Note: Force [upper] traces start at level of zero membrane potential). Graded electrical stimulation, when applied to the nerve, produced two sizes of response, a pure end-plate potential (1*e*) giving rise to a small twitch (1*m*), and a larger end-plate potential (2*e*) evoking a small graded spike and giving rise to a much larger twitch (2*m*). Each muscle fiber was therefore dually innervated from "slow" and "fast" axons. In the fiber shown in *b* the small response was obtained twice in succession at a stimulus interval of 2 seconds. Note potentiation of mechanical response. Calibration: vertical bar, 20 mv; horizontal bar, 100 msec.; bracket, 1 g.

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2. Barnacle Ringer solution contained, in millimoles per liter, Na^+, 476; K^+, 8; Ca^+, 20; Mg^{++}, 12; Cl^-, 538; HCO_3^-, 10.
3. Supported by grant B-3819, U.S. Public Health Service. This work was done at the Friday Harbor Laboratory, University of Washington. A full account is in preparation.

28 September 1962

Feather Replacement in Birds

Abstract. *During natural molt in many birds, feathers of the old generation are passively pushed out of the follicles attached to the tips of the sheaths of incoming feathers.*

Recent observations on several species of birds reported here show that when feathers are replaced in natural molt, they remain temporarily attached to the sheaths of the new feathers and are passively pushed out of the follicles during growth of the incoming new feathers.

This is known to be the mechanism for replacement of natal downs during the first molt (*1*) and is also the normal mechanism of feather replacement during all molts in penguins and cassowaries (*2*).

Natural molt in all other birds, however, has been thought to be a two-part process entailing the passive loss of old feathers (ecdysis) and the subsequent growth of new feathers (endysis) (*3*). Because artificial plucking

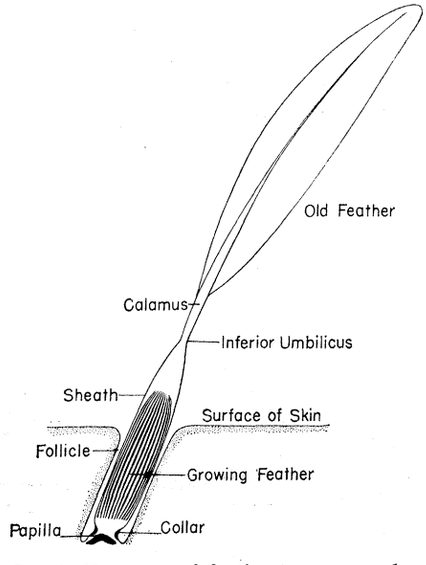

Fig. 1. Diagram of feather structures during molt; sheath and follicle below inferior umbilicus in longitudinal section.

often stimulates regeneration of a complete feather from a papilla, it has been assumed that loss of the old feather also stimulates new feather growth in molt.

During the early stages of feather replacement, the calamus of the old feather is firmly attached to the tip of the sheath of the new feather (Fig. 1). When a feather is artificially plucked after growth has begun, the break occurs at the collar, which is the region of active growth below the inferior umbilicus. As much as 10 mm of new feather sheath has been found attached to the base of a plucked primary. The outer layer of the keratinized old calamus appears continuous with that of the new sheath. The connection appears to be of primary origin. It is probably not, secondarily, induced by the pressure of growth from below. Soon after the zone of juncture emerges from the follicle and keratinization is complete, the new sheath becomes brittle and the old feather breaks off with part of the tip of the new sheath attached (*4*).

This mechanism of feather replacement occurred during all molts observed in daily examinations of captive Rhode Island Red chickens, chukar partridge (*Alectoris chukar*), and Japanese quail (*Coturnix japonica*). It was also found in postmortem examinations of wild specimens of greater scaup ducks (*Aythya marila*), a ruddy shelduck (*Casarca ferruginea*) and a house sparrow (*Passer domesticus*) (*4*). Similar observations have been reported, usually as aberrations, in a shorebird, pigeons, a parrot, and a magpie (*5*). This mechanism of feather replacement has been demonstrated in a total of eight diverse orders of birds: Sphenisciformes, Casuariiformes, Anseriformes, Galliformes, Charadriiformes, Columbiformes, Psittaciformes, and Passeriformes.

Thus, during natural feather replacement in many, and probably all birds, the loss of the old feather is brought about by the initiation of growth in the new feather which pushes the old feather passively out of the follicle. Molt in birds is consequently a single growth process actively concerned only with the production of feathers of the new generation. The new growth causes the passive loss of the old feathers.

This demonstration of the mechanism of feather replacement is of fundamental significance in studying the relationships of molts and plumages. It also offers support for the system of nomenclature for molts and plumages proposed by Humphrey and Parkes (*6*). The fundamental concept underlying the new nomenclature is that, during molt, energy is expended only in producing the incoming generation of feathers and not in shedding the old generation.

Humphrey and Parkes, therefore, name molts in terms of the incoming generation. For example pre-basic molt leads to basic plumage. Other systems of nomenclature (see *7*) do not reflect this natural relationship of molts and plumages but name molts in terms of the reproductive cycles (postnuptial molt, prenuptial molt). The new system of nomenclature has been adopted for the *Handbook of North American Birds* (*8*) recently reviewed (*9; 10*).

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Acid Phosphatase in Serum: Increase in Acute Myocardial Infarction

Abstract. *Ten consecutive cases of acute transmural myocardial infarction were accompanied by a rise of 50 to 400 percent in the serum acid phenylphosphatase. The increase in phosphatase began several hours after onset of symptoms and lasted 3 to 5 days. A similar rise was seen during the acute stages of other thromboembolic diseases. While the mechanism by which this acid hyperphenylphosphatasia occurs is not clear, platelets may play an important role.*

Acid phosphatase was first described in 1925 when it was shown that human urine contained an enzyme which hydrolyzed hexose diphosphate at an optimum *p*H of 5 (*1*). Subsequently it was found that acid phosphatase was present throughout the urogenital tract, but in particularly high concentration in the prostate (*2*). Prostatic carcinoma often causes increase in the serum acid phosphatase (*3*), and such increases constitute the major clinical significance of this enzyme. Acid hyperphosphatasia is present occasionally in other diseases (*4*) but in such cases has been regarded more as a curiosity than as an aid to diagnosis.

In the course of study on nonprostatic causes of acid hyperphosphatasia, serum acid phenylphosphatase was observed to increase during the acute stages of thrombophlebitis, phlebothrombosis, pulmonary embolism, and systemic arterial embolism (*5, 6*). This paper records a similar increase in the most important of all the thromboembolic diseases—acute myocardial infarction.

Six women and four men with acute transmural myocardial infarction were studied. The diagnosis was based upon a recent history of severe oppressive retrosternal pain that lasted several hours, characteristic electrocardiographic changes, and a transient rise in the serum glutamic-oxaloacetic transaminase activity. All of the patients were seen within the first 24 hours after onset of symptoms. Because of the necessity for making determinations at fixed intervals only patients who survived for at least 10 days were included.

Quantitative determination of serum acid phenylphosphatase (AP) and serum glutamic-oxaloacetic transaminase (SGOT) was made on every patient. In four cases, the serum glutamic-pyruvic transaminase (SGPT) was also determined. The acid phenylphosphatase was determined by the Gutman modification (*7*) of the King-Armstrong method (*8*) as described