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*).

GEORGE E. WATSON
Division of Birds, Smithsonian Institution, Washington 25, D.C.

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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

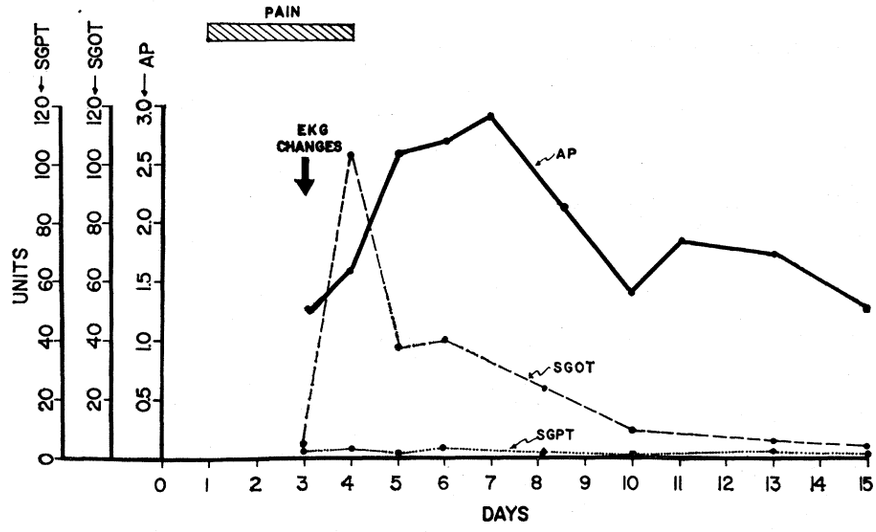

Fig. 1. Acute myocardial infarction. A 54-year old Caucasian male with an acute transmural anteroseptal myocardial infarction. AP, serum acid phenylphosphatase in Gutman-King-Armstrong units; SGOT, serum glutamic-oxaloacetic transaminase in Sigma units; SGPT, serum glutamic-pyruvic transaminase in Sigma units.

elsewhere (*6, 7*). The two transaminases were determined by the Sigma technique (*9*). The upper limit of normal for acid phenylphosphatase in our laboratory is 1.6 units (*5*), a value which approximates that originally reported (*7*). The upper limits of normal for the two transaminases are 40 and 36 units respectively (*9*).

In all ten patients, there was an increase of acid phenylphosphatase to 2.3 to 3.9 units throughout the first 3 to 5 days of the illness, with a return to the normal range of 0.6 to 1.5 units thereafter. Figure 1 illustrates a typical case. This acid hyperphenylphosphatasia was noted as early as 6 hours after onset of symptoms, and amounted to a rise of 50 to 400 percent above baseline values. Such a rise is certainly modest when compared to the occasional values of over 1000 units that have been reported with metastatic prostatic carcinoma (*10, 11*). On the other hand, approximately a quarter of all cases of metastatic prostatic cancer have acid phenylphosphatase values of less than 5 units and half have values less than 10 units (*11*), so that the increases of this serum enzyme in myocardial infarction do not compare so unfavorably after all.

Several possible sources for this acid hyperphenylphosphatasia have been considered:

1) Erythrocytes and platelets, and to a lesser extent leukocytes, are rich in acid phosphatase (*12*), and autolysis of these cells within the coronary thrombus might be expected to release their contained enzymes into the blood stream. On the other hand, the thrombus is very small indeed and is hardly likely to contain enough cellular elements to raise the acid phenylphosphatase 50 to 400 percent. Moreover, the acid hyperphenylphosphatasia of thromboembolism appears to be only roughly proportional to the size of the thrombus, so that a huge clot filling the entire superficial femoral vein may give not much more of an increase than a tiny coronary thrombus.

2) Heart muscle contains acid phosphatase (*13*), and infarcted myocardium could release this enzyme into the blood stream. This is not the major source of the acid hyperphenylphosphatasia of myocardial infarction since this enzyme is not increased in rheumatic myocarditis or the idiopathic myocardiopathies; nor is it increased in pneumonia, tuberculosis, and bronchogenic carcinoma, or cellulitis of the leg. However, thromboembolism of these same organs (heart, lung, limb) does increase the enzyme (*5, 6*).

3) Probably accompanying every case of myocardial infarction there is some degree of transient hypotension, with or without shock, and this leads to some parenchymal damage of most body organs. Since prostate, liver, spleen, kidney, hemotopoetic, and nearly all other tissues contain acid phosphatase (*13, 14*), the enzyme could be released from these tissues into the blood stream as a result of such damage. However, thrombophlebitis and phlebothrombosis are not associated with hypotension or significant injury to extravascular tissue, and yet these diseases are associated with acid hyperphenylphosphatasia.

4) Many forms of stress and injury, and therefore perhaps myocardial infarction too, cause splenic contraction with sudden rise in the number of circulating platelets; any associated tissue necrosis may cause a more gradual and sustained thrombocytosis by stimulating marrow thrombocytogenesis (*15*). If, in addition, the platelets are broken down at an accelerated rate (as has been said to occur in thrombo-embolic phenomena), there may be marked increase in acid phosphatase from platelets released into the serum without a significant increase in the blood platelet count. Increase in the number of blood platelets would be expected to further increase the acid phenylphosphatase, as serum contains many platelets, even after centrifugation at the conventional speeds of 1500 to 3000 rev/min. Thus, platelets might have a central role in the acid hyperphenylphosphatasia of thrombo-embolism, a role in keeping with the key position of these cells in blood clotting and, perhaps, atherogenesis (*16*). That myocardial infarction in the presence of longstanding thrombocytopenia (*17*) did not result in an increased AP is compatible with such a hypothesis (*18*).

MYRON R. SCHOENFELD
Medical Service, Lincoln Hospital, New York 54, New York

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