An Unusual Lacustrine Delta

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There are several possible approaches to the problem of land form classification. One of the most popular methods involves the gentic classification of forms that will be representative of types actually developed in the rocks and materials of the earth's surface. An attempt is made to establish the particular conditions which operate to produce similar land form types. Nature, however, is a great nonconformist and new types or variations of an old theme are not unusual in the field. Occasionally a particular land form expresses a contrary mood of nature, resulting in a truly unusual set of conditions. It is still possible to classify the type in most cases, but genesis may lie in violation of the usual explanation of origin.

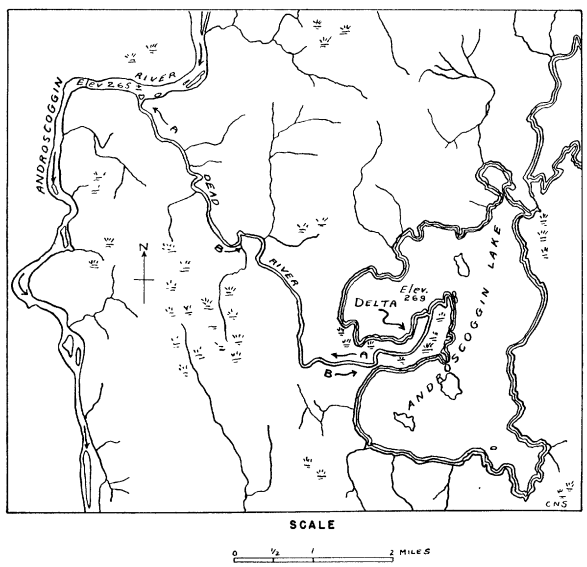

FIG. 1.

One such unique case is found in Androscoggin Lake near Leeds, Maine, about 2 miles southeast of Wayne. A lacustrine delta nearly 1.5 miles long and 0.25 of a mile wide is being built at the head waters of a stream called Dead River. Usually, one expects to find a delta at the mouth or outlet of a stream; at this locality, however, a delta is forming at the opposite end or source of the river.

Throughout much of the year the Androscoggin, one of of the larger rivers in Maine, flows south to the Atlantic Ocean. Dead River, tributary and outlet of Androscoggin Lake, flows northwest and empties into the Androscoggin River. On the sketch (Fig. 1), arrows indicate the direction of flow in the large river and similar arrows marked *A*, show normal direction of flow in Dead River. The difference in elevation between the water in Androscoggin Lake and the Androscoggin River is probably not more than 4–5 ft, and Dead River flows very sluggishly. In time of flood and high water on Androscoggin River, a considerable volume of water moves into the outlet of Dead River and the direction of flow is reversed, as indicated on the sketch map by arrows marked *B*. Water from the larger river then moves into Androscoggin Lake.

Reversal of flow on Dead River occurs generally in the spring months, when runoff is high, and large quantities of sand and silt are being carried by Androscoggin River. This load is obtained partly from the quantities of sand in the region through which the river flows. Surplus water spilling into Dead River—water laden with rock material—moves into the lake, where velocity is checked by the standing lake water and the load is dropped. This deposition normally occurs in similar fashion at the mouths of most streams where deltas are being built. In the case of the lacustrine delta at Androscoggin Lake, however, nature has played one of her pranks and heavy deposition takes place at the head or source of Dead River.

Thermostable Inhibition of Bacterial Hyaluronidases by the Serum of Normal Human Beings[1]

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Hyaluronidase, a mucolytic enzyme, and its substrate hyaluronic acid, a complex polysaccharide which is present in the intercellular substance of connective tissue, compose an enzyme system which appears to be involved in the pathogenesis of various infectious and rheumatic diseases (*2, 6*). Hyaluronidase is elaborated by various bacteria, and is present in spermatic fluid and aqueous extract of testicle. Since hyaluronidases are antigenic, there is current interest in the investigation of hyaluronidase inhibition by the serum of normal human beings and of patients with infectious disease and rheumatic fever. Specific inhibition of pneumococcus hyaluronidase by the immune serum of patients with bacteremic pneumococcus infections has been reported to be thermostable (*7*), whereas the inhibition of testicular hyaluronidase by the serum of normal, nonimmune human beings has been reported to be thermolabile (*1, 3, 4*). In contrast to the thermolability of the inhibitor of testicular hyaluronidase, this report indicates that the inhibitor of pneumococcus hyaluronidase in normal human serum is almost always thermostable, the inhibitor of staphylococcus hyaluronidase is usually thermostable, and the inhibitors of *Cl. perfringens* and streptococcus hyaluronidases may be either thermolabile or thermostable.

This study investigated the inhibition of similar test strengths of five hyaluronidases by the serum of 50 nor-

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mal human beings who were between 20 and 40 years of age. Each serum was tested immediately as it was removed from the clot, and again after it had been heated at 56° C for 30 min. The hyaluronidases used in these tests were unrefined filtrates of cultures of type 3 *Pneumococcus*, hemolytic *Staphylococcus aureus*, *Clostridium perfringens*,[1] beta hemolytic *Streptococcus* (group A),[2] and purified bovine testicular hyaluronidase. Twofold serial dilutions of each serum were titrated against the constant strength of each enzyme. Hyaluronidase inhibition was measured by the mucoprotein clot prevention test as previously described (*7*), except that fresh egg albumin was used instead of normal horse serum as the protein component of the substrate. With this modification of the test, a fourfold variation of serum inhibition titer could occur by chance, so that eightfold variation of titer was considered significant. If a 1: 3 dilution of a serum did not inhibit an enzyme, inhibitor was considered to be absent from the serum. Hyaluronidase inhibition by serum was considered thermostable if heating the serum caused no significant fall in the titer.

Before heating the sera at 56° C for 30 min, all inhibition of the five hyaluronidases by normal sera occurred in a serum dilution of 1: 48 or less, except that two sera in dilution of 1: 192 and four sera in dilution of 1: 96 inhibited the pneumococcus hyaluronidase, one serum in dilution of 1: 384 inhibited the staphylococcus hyaluronidase, and one serum in dilution of 1: 96 inhibited the streptococcus hyaluronidase. Results tabulated below reveal thermostable inhibition of the five hyaluronidases by normal sera:

TABLE 1

Hyaluronidase tested	No. of sera tested	No. of inhibiting sera before heating	No. of thermostable inhibiting sera
Pneumococcus	50	47	45
Staphylococcus	49	23	19
Cl. perfringens	50	16	10
Streptococcus	50	12	7
Testicular	48	46	2

Tests upon consecutive daily sera of four persons revealed consistent inhibition of the pneumococcus, staphylococcus, and testicular hyaluronidases. However, there was day-to-day fluctuation in the inhibition of the streptococcus and *Cl. perfringens* hyaluronidases, so that results of the inhibition of these two enzymes tabulated above are of undetermined immunological significance.

A further preliminary investigation of the thermolabile serum inhibition of *Cl. perfringens*, streptococcus, and testicular hyaluronidases was carried out. After this inhibition had been destroyed by heating the sera at 56° C for 30 min, it was completely restored in most sera and partially restored in remaining sera by the addition of complement (*5*). The complement used was 0.5 cc of a 1: 30 dilution of normal guinea pig serum, which alone did not inhibit the test strength of the enzymes.

[1] This hyaluronidase was glycerol-dialyzed. It was prepared and supplied by Dr. A. A. Tytell, Cincinnati, Ohio.

[2] Culture supplied by Dr. R. M. Pike, Dallas, Texas.

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Vitamin B_{12} and Cobalt Deficiency in Sheep

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The discovery by Rickes *et al.* (*2*) that vitamin B_{12} is a cobalt complex pointed to the possibility that this vitamin was an intermediary in the metabolism of cobalt in those species requiring this element. It has been suggested by several workers that cobalt, which is known to be required by ruminant but not by nonruminant animals, functions primarily through some unknown mechanism in the rumen, probably related to the microflora. This theory enjoys some but not conclusive experimental support.

At the time that the report of Rickes *et al.* appeared, we were engaged in studies of cobalt deficiency in lambs. Among the symptoms displayed by these lambs were loss of appetite, anemia, loss in body weight, and eventual death. Such symptoms have previously been reviewed by Russell (*3*). Among the treatments under study were the effects of cobalt administration by feeding vs. injection. It was observed that deficient lambs when fed 1 mg of cobalt per day responded quickly in improved appetite, gains in weight, and increase in hemoglobin concentration of the blood. On the other hand, deficient lambs when injected with the same quantity of cobalt showed no detectable response over a period of 7 weeks. It thus seemed possible, assuming that vitamin B_{12} is a necessary metabolite for sheep, that cobalt orally administered may be synthesized into vitamin B_{12} by the rumen flora; and that cobalt injected was incapable of this transformation, possibly because insufficient quantities of it reached the rumen Comar *et al.* (*1*). According to this postulate cobalt deficiency is essentially a vitamin B_{12} deficiency and if this vitamin is furnished the deficient animal directly by injection, a favorable response should follow. Since cobalt salts injected into deficient lambs gave no response it seemed safe to assume that the much smaller amount of cobalt present in vitamin B_{12} would not give a response *per se*.

Cobalt-deficient lambs which had previously been injected with cobalt salts were chosen for this study. All of these lambs had pronounced symptoms of cobalt deciency. They were injected with various amounts of crystalline vitamin B_{12}[1]. The injections were made in-