On the present evidence, the most parsimonious interpretation is in favor of reserpine's depressing temporarily both performance and learning—that is, that the drugged animals were functionally impervious to conditioning and extinction events, had to "start from scratch" once the drug had worn off, but subsequently responded normally to such events. Insofar as the reserpine groups, when tested after the gross effects of the drug had dissipated, differed from the controls in their rate of conditioning or extinction, they required more conditioning trials and fewer extinction trials, although these differences are far from being statistically significant. If, with a larger N or more refined technique, such differences were to become significant, explanation might follow one of several courses.

Examples of such possible explanations include the following: slight amounts of reserpine (or a metabolic product) might be active in the organism long after its gross effects had disappeared; in extinction, the reserpine animals have a longer time to "forget" the conditioned response, if they are impervious to the extinction events; the "baseline level of anxiety" might remain lower even after the drug has been completely metabolized.

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New Theory of Interference in Clotting Mechanism by Abnormal Plasma Proteins

The present communication postulates a new type of defect in the clotting mechanism. This consists of the production by the body of globulins that have the ability to combine with and precipitate prothrombin and accessory factors from the blood plasma. The consequence is that the normal clotting mechanism is unbalanced. This results in the following changes: (i) a reduced concentration of clotting factors in the circulating blood, causing hemorrhages, and (ii), localized concentrations of prothrombin and accessory factors, causing thrombi.

Clotting defects, both hemorrhages and thromboses, are known to occur in association with clinical conditions in which abnormalities in the plasma globulins are well recognized, such as macroglobulinemia (*1*), multiple myeloma (*2*), purpura hyperglobulinemia (*3*), and cryofibrinogenemia (*4*). In addition, there exist clinical conditions such as carcinomatosis (pancreatic and so forth) (*5*), thrombophlebitis (*5*), pulmonary infarction, coronary occlusion, abdominal thrombosis, and postoperative thrombosis in which the causes of the thromboses have been the subject of investigation for many years without elucidation of their etiologies. Such cases now require reinvestigation in the light of our present theory.

This theory is based on the demonstration (*6*) that the precipitation of euglobulin, by dilution of plasma, takes out with it prothrombin and factor VII (stable factor). In one case (L. S.), the euglobulin consisted of macroglobulins with a major ultracentrifuge sedimentation component at $S_{20} = 20$ and two minor components at $S_{20} = >20$. In a second case (R. B.), euglobulin and cryoglobulin precipitates were obtained, both of which precipitated out prothrombin and accessory factors (factor VII), the euglobulin to a greater extent than the cryoglobulin. The euglobulin contained no macroglobulins, contrary to expectations. Both cases had increased plasma viscosity, which showed an anomalous rise with decreased temperature. Cryoprecipitability could be elicited in one case (R. B.) by prefreezing the plasma sample and in both cases by reducing the salt concentration of the sample. Macrogobulin precipitated under these conditions (L. S.) can be redissolved by the addition of albumin or minute traces of sodium carbonate but not by gamma globulin.

Both cases had hemorrhagic tendencies and reduced prothrombin activity. Case L. S. also had an extensive thrombus of the iliac vein. Precipitation of the euglobulins from both cases caused a marked reduction of both prothrombin and factor VII in the euglobulin-free plasma. The prothrombin and factor VII could be demonstrated in the solution of the precipitated globulins. The addition to and precipitation of macroglobulin from normal plasma removed prothrombin and factor VII from the normal plasma.

In addition to the afore-mentioned results, we have found that cold precipitable material is obtainable from normal and pathological plasmas. This material contains both fibrinogen and small amounts of prothrombin. Hence, the cold precipitation of fibrinogen takes down with it prothrombin. Studies of cryoglobulins are in progress. It is highly probable that prothrombin combines easily with many globulins in addition to the accessory factors required in the clotting mechanism. The ease with which prothrombin is adsorbed by barium sulfate is well known. Slight changes in the plasma globulins that permit precipitation, therefore, would sequester prothrombin from the circulating plasma.

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6. This work was aided by a grant from Leukemia Research, Inc., Los Angeles, Calif. A more detailed report is in preparation.

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Pollen from Leda Clay of Maine

Marine clays of late glacial age occur widely in eastern Maine from below sea level to 400 ft or more above. These clays occasionally contain a well-preserved molluscan fauna of northern affinities, suggesting that, during the time of their deposition, the water was colder than it is at present. Because of the presence of species of *Leda,* these clays are frequently referred to as Leda clays, although their affinity is closer to the later *Saxicava* phase of the marine sediments in southeastern Canada.

One of the most productive fossil localities of the Leda clay in Maine occurs at Goose Cove on Mount Desert Island. The fauna from this locality has been described by Blaney and Loomis (*1*), who note that the assemblage has strong affinities with the present molluscan fauna of Labrador.

I had occasion to examine the clay from this locality for microfossils. The sample from which the analysis was made came from highly fossiliferous beds about 10 ft above high tide near the head of the cove. The microfossils were separated by a bromoform-acetone mixture (*2*) with a specific gravity of 2.3. A large number of pollen grains and spores, together with some marine and brackish-water sponge spicules and a few Foraminifera, were recovered. Table 1 shows the result of the pollen-spore analysis.

The pollen content of the clay is very similar to the pollen assemblages that I found in late glacial clays of southeastern New England, but it differs in several