from that of the nonimmune elimination phase in group A (5.4 days, $p > .20$). The induction period ended after 5 days when the half-disappearance time decreased to 2.2 days. The statistical significance of the difference between the immune disappearance times for groups A (1.5 days) and B (2.2 days) was equivocal ($p = .05$), but the difference between the induction period for group A (7 days) and the posthibernation induction period of group B (5 days) was significant ($p < .001$).

To determine whether the rate of disappearance of the antigen upon awakening from hibernation was comparable to the nonimmune disappearance rate or was actually the result of physiological processes peculiar to arousal, we studied the disappearance rate of labeled squirrel serum.

In four squirrels (group C) injected with labeled serum, the half-disappearance time was 3.3 days. In five squirrels treated similarly (group D) that entered hibernation 4 days after serum injection, the half-disappearance time upon arousal from 14 days of hibernation was 3.3 days. This was almost identical with the half-disappearance time of 3.6 days for the same animals during the 4 days before hibernation and for the group C animals that did not hibernate. In group D, as in group B, there was little or no disappearance of the labeled material during hibernation. Therefore, it is safe to assume that the disappearance rate of antigen in squirrels after arousal is the reflection of a physiological state (measured by antigen disappearance) comparable with that of the nonhibernating ground squirrels.

Although there is little or no disappearance of homologous or heterologous proteins from the circulation of hibernating ground squirrels, the induction period ends 5, not 7, days after arousal (group B, Fig. 1). Since the normal induction period is 7 days (group A, Fig. 1), it would appear that some of the events that occurred during the induction period transpired while the ground squirrels were hibernating. Work is in progress to determine whether the whole or only part of the induction period can be passed in hibernation.

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18 May 1961

Development of Needle Blight Symptoms on Rooted Cuttings from Diseased White Pine Trees

Abstract. A method of rooting cuttings from white pine trees is described. Approximately equal numbers of cuttings from diseased and healthy trees were rooted by this method. After the rooted cuttings were planted in the field, they were closely watched for root growth and the development of foliar injuries.

The disease, needle blight of eastern white pine (*Pinus strobus L.*), is characterized by an orange-red discoloration of the distal portions of current-year needles. For more than 50 years it was believed that the disease affected the tips of needles first, and that this was followed by a progressive dieback. To the contrary, it was recently discovered (*1*) that the injury began in semimature leaf tissues and then spread distally to more mature tissues. Based on data with respect to the nature and occurrence of the foliar symptoms, the hypothesis was set forth that needle blight is heritable (*1*).

Support for this theory would ensue if shoots removed from diseased or healthy trees and induced to root should produce phenotypic reactions similar to their parents when grown under similar environmental conditions. The question also arose whether cuttings from needle-blighted trees could be rooted, since diseased trees possibly lack food reserves because of reduced needle and shoot growth (*2*) and death of distal ends of needles.

Cuttings were taken from trees with histories either of disease recurrence or of freedom from blighting. The selected trees were 10 to 13 years old, which is considered fairly old by forest geneticists for obtaining high percentages of successfully rooted cuttings (*3*). Most conifer cutting experiments involve seedlings 3 to 6 years old. Trees this young, however, usually do not possess sufficient shoots to provide large enough clones for further experimentation.

Twenty-five cuttings were removed from each of four trees (two diseased and two healthy) in early September 1958, after shoot growth had ceased and buds had fully developed. Lateral shoots, 3 to 5 inches long, the full length of the current growth, were pulled from the trees. The basal fascicles were not removed. The "heels" were trimmed with a sharp knife, and the bases of the cuttings were dipped in 0.2 percent indolebutyric acid in talcum powder. The cuttings were placed immediately into pots 12 inches in diameter and 8 inches high. The medium used was Perlite (*4*), a chemically inert substance with good drainage and aeration properties. About one-third of the length of the cuttings was inserted into the firmed, moistened medium, and the cuttings were spaced about 1½ inches apart with 2 inches between rows. The 25 cuttings from each tree were placed in a separate pot. Wire hoops placed in the pots formed supports for thin polyethylene plastic covers. These covers were removed once a day, and the cuttings were sprayed with water until a fine film covered the needles. The covers helped maintain high humidities about the cuttings and also allowed an exchange of gases. The pots were kept at room temperature on a bench adjacent to

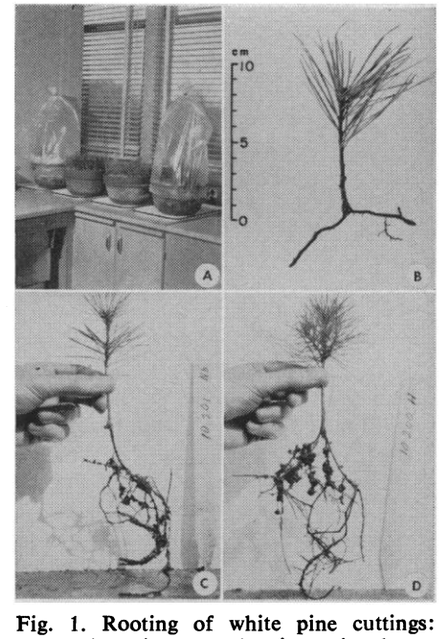

Fig. 1. Rooting of white pine cuttings: *A*, cuttings in pots showing wire hoops and polyethylene covers; *B*, cutting from a needle-blighted tree that rooted within 8 months; *C*, rooted cutting from a diseased tree 1 year after striking roots; *D*, rooted cutting from a healthy tree 1 year after striking roots.

windows with venetian blinds that could be adjusted to reduce the sunlight when necessary (Fig. 1*A*). No nutrients were added to the media before or during the rooting procedure. Once a week, the pots were immersed in tap water for 10 minutes, and then drained. The cuttings were sprayed with pesticides about once a month to combat fungi and insects.

Sample cuttings were removed periodically and examined for any evidence of rooting. The first cutting to strike roots was observed 7 weeks after potting. Mortality of both diseased and healthy cuttings was high in the first 4 months. Twenty-one cuttings rooted within 8 months (Fig. 1*B*), and eight more rooted within 12 months. From the original 100 cuttings, 29 rooted, of which 14 were from diseased and 15 from healthy trees.

The rooted cuttings were planted individually in pots, 5 inches in diameter, containing a 1:1 mixture of Perlite and surface soil from a hardwood stand. The cuttings were kept moist and partially shaded in a greenhouse. In late September 1959, nine of the rooted cuttings (six from a diseased tree and three from a healthy tree) were lined out in a nursery, located approximately 3 miles from the site of the parent trees. The cuttings were planted in the same type of media in pots, were placed side by side in the nursery, and thus were growing essentially in identical sites.

In late July 1960, the diseased mother tree developed initial needle blight symptoms. Examination of the six rooted cuttings from this tree, planted 3 miles away, revealed a simultaneous development of needle blight on the new foliage, with the injury originating in tissue of the same maturation as in the mother tree. In addition, the rate of distal spread of foliar necrosis was comparable in the cuttings and in the parent tree. The three healthy cuttings were unaffected, as was the mother tree. Examination of the extent of the root systems of the diseased and healthy cuttings 1 year after they struck roots revealed no apparent differences between the two (Fig. 1, *C–D*).

From the above observations it may be concluded: (i) that the excessive mortality of main root tips which has been found to occur in needle-blighted trees (*5*) probably is a consequence of the disease and is not primarily associated with the cause of needle breakdown; and (ii) that symptom development which occurred simultaneously in the same local area on rooted cuttings and their diseased parent tree is further evidence strengthening the hypothesis that susceptibility to the unfavorable conditions which contribute to needle blight is inherent in the individual (*6*).

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Transmembrane Action Potentials from Smooth Muscle in Turtle Arteries and Veins

Abstract. Transmembrane potential and active tension were measured in isolated segments of turtle aorta and inferior vena cava. Changes in tension were associated with action potentials, but the potentials had a different pattern in the two tissues. When the frequency of the action potentials increased, the contractions summated, resulting in a steadily maintained contraction.

The activity of smooth muscle in blood vessels has usually been estimated indirectly by measuring the diameter of intact vessels, the tension in isolated strips of vessel, the compliance in the vessel wall, or the resistance to flow. The results of such measurements have often been difficult to interpret because it was impossible to determine the relative contributions of active changes in the muscle and passive changes due to the physical properties of the wall.

A more direct way of assessing muscle activity in blood vessels would have many advantages. One method would be to measure the electrical changes accompanying activity in single cells. Unfortunately, muscle cells in blood vessels are generally very small and are usually surrounded by considerable amounts of fibrous and elastic tissue, especially in larger vessels. Consequently, the impalement of these cells with microelectrodes and the measurement of their membrane potential is very difficult. Turtle arteries and veins contain relatively little fibrous and elastic tissue, which makes the impalement of their muscle cells somewhat easier. The present report describes some relationships between changes in tension and membrane potential in the smooth muscle of these vessels.

Excised segments of the abdominal aorta and the inferior vena cava were cut open lengthwise and mounted in an organ bath filled with frog Ringer's solution at 24°C. The transverse tension exerted by the opened segment was measured with an RCA mechanoelectronic transducer (No. 5734). Single muscle cells were impaled through the intimal surface with "floating" microelectrodes (*1*).

The time courses of membrane potential and contractile tension during rhythmic activity in aorta are shown in Fig. 1. In this experiment the resting potential was about 35 mv. It can be seen that the increase in tension was just preceded by a single heartlike action potential, which consisted of a

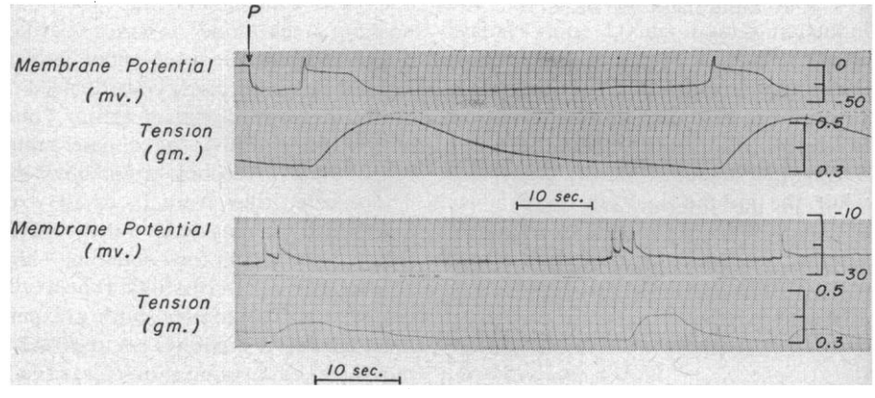

Fig. 1. Action potentials in single smooth muscle cells of turtle aorta (upper record) and turtle inferior vena cava (lower record) associated with changes in smooth muscle tension. At *P*, the arterial smooth muscle was impaled with a microelectrode.