Compartmentalization of Decay in Trees

Animals heal, but trees compartmentalize. They endure a lifetime ofinjury and infection by setting boundaries that resist the spread of the invading microorganisms

by Alex L. Shigo

Trees have a spectacular survival record. Over a period of more than 400 million years they have evolved as the tallest, most massive and longest-lived organisms ever to inhabit the earth. Yet trees lack a means of defense that almost every animal has: trees cannot move away from destructive forces. Because they cannot move, all types of living and nonliving enemies—fire, storms, microorganisms, insects, other animals and later man—have wounded them throughout their history. Trees have survived because their evolution has made them into highly compartmented organisms; that is, they wall off injured and infected wood.

In that respect trees are radically different from animals. Fundamentally, animals heal: they preserve their life by making billions of repairs, installing new cells or rejuvenated cells in the positions of old ones. Trees cannot heal; they make no repairs. Instead, they defend themselves from the consequences of injury and infection by walling off the damage. In a word, they compartmentalize. At the same time they put new cells in new positions; in effect, they grow a new tree over the old one every year. The most obvious results of the process are growth rings, which are visible on the cross section of a trunk, a root or a branch.

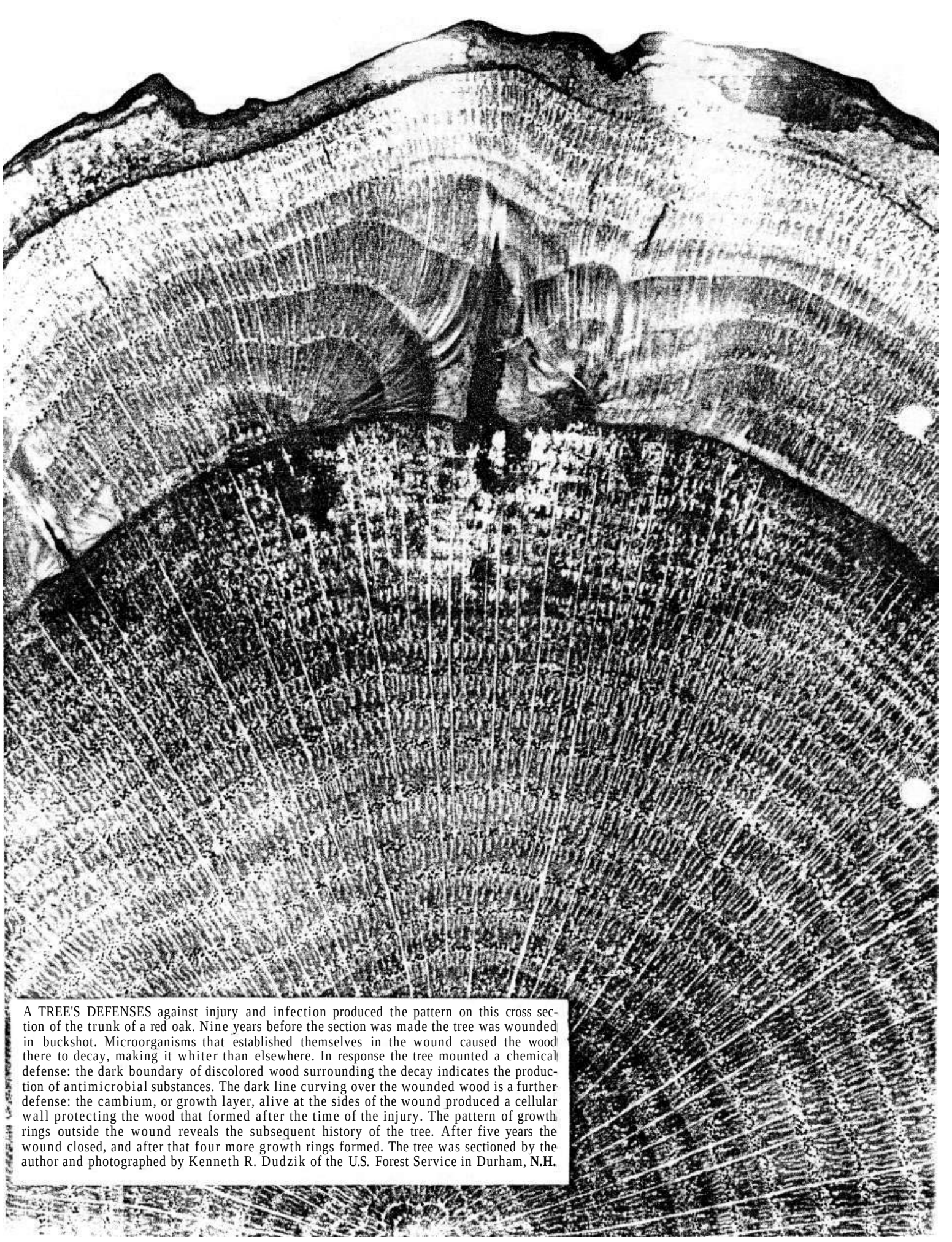
The defenses wielded by trees suggest a new view of tree biology, one in which the role of tree pathology is given full recognition. Trees have been guided through evolution by their need to defend against attack while standing their ground.

To understand disease and decay in trees it is essential to understand normal tree function and growth. Trees, together with grasses and algae, are the earth's major energy trappers. In particular, forests, which cover about a tenth of the planet's surface, trap about half of all the energy entering the biosphere. The energy, which enters as solar radiation, serves to transform carbon dioxide and water into" carbohydrates, the chemical form in which energy is stored. In turn the carbohydrates power growth, maintenance, reproduction and defense.

Trees themselves—that is, the conifers and the hardwoods (angiosperms and gymnosperms)—are perennial, woody, compartmented, shedding plants. In addition most trees are long-lived, massive and tall. Their internal structure follows a characteristic pattern. The generation of new cells in the tree is the function of the vascular cambium, a thin cylindrical layer found in the trunk, the roots and the branches. In the outer direction the cambium lays down phloem, or inner bark, the layer that transports liquids downward in the tree. (Specifically, it carries substances produced by photosynthesis in the leaves.) In the inward direction the cambium lays down concentric layers of xylem, or wood, which transports water and water-soluble substances upward.

The inward cell production is worth a close examination. On a broad scale the cambium lays down one inward layer per year; these are the annual growth rings, prominent on a cross section of the tree. (In tropical trees, however, growth rings are indistinct.) Viewed on a finer scale the cambium forms two basic types of cells: those with their long axis perpendicular to the axis of the trunk, a root or a branch and those with their long axis parallel to the axis of trunk, root or branch. The perpendicular cells become ray parenchyma, which form radial partitions in the wood. Meanwhile the longitudinal cells fill the compartments between the rays.

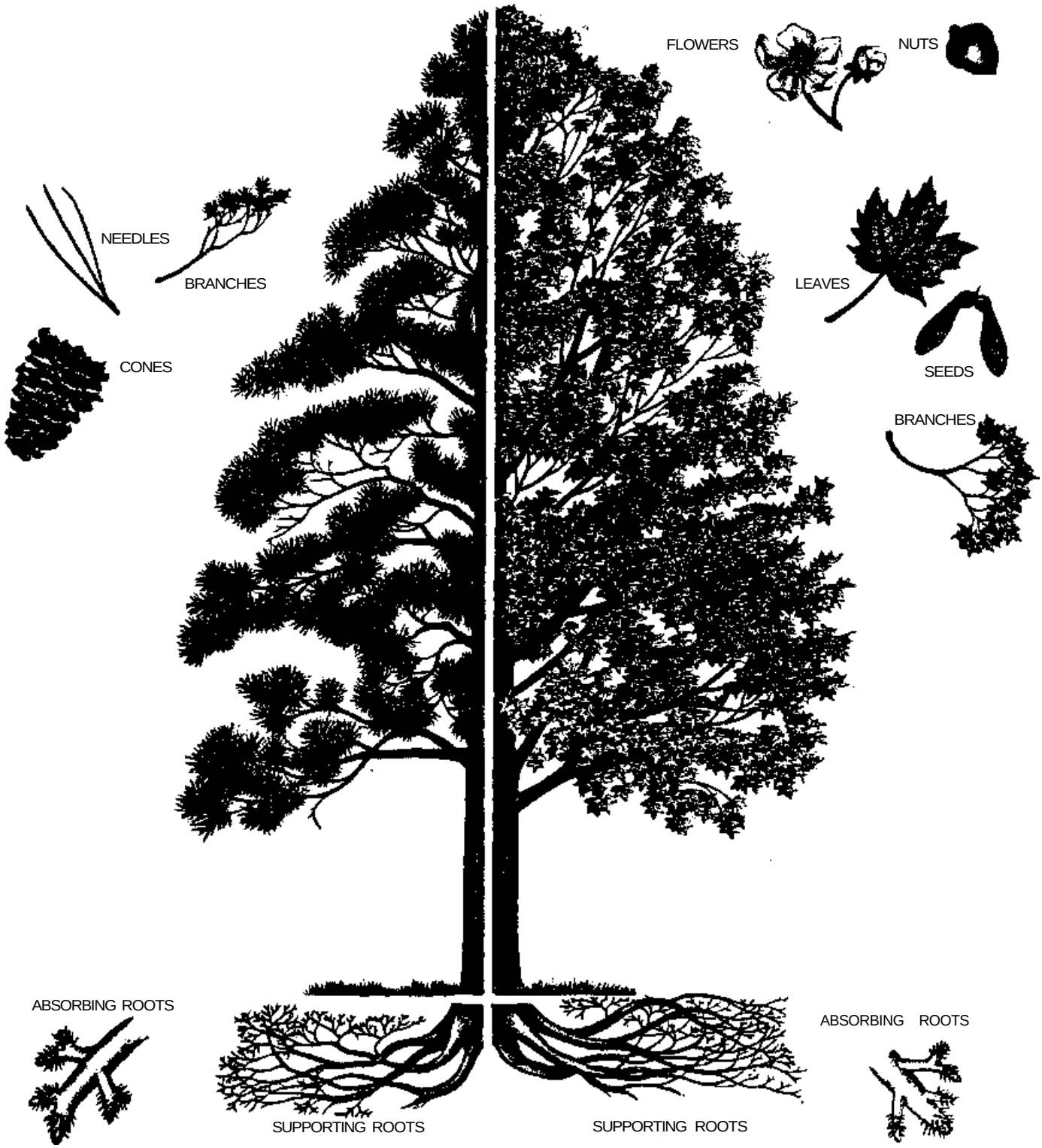
The longitudinal cells are of three varieties. In some the living contents die in a few days or weeks, leaving only a tubular cell wall. In hardwoods such cells are called vessels; in conifers they are called tracheids. They serve to transport liquids. In others the cell wall is thick. Such cells, called fiber cells or fiber tracheids, provide mechanical support to the wood. Finally, the cells called parenchyma retain their living contents behind a thin cell wall. (In contrast, vessels and tracheids have a hollow interior under a thick cell wall.) Parenchymal cells store nutrients and other materials. In them the cytoplasm can remain living for years, indeed sometimes for more than a century. The complex interlocking of the various cells in wood combines with the tough cell walls themselves to give



A TREE'S DEFENSES against injury and infection produced the pattern on this cross section of the trunk of a red oak. Nine years before the section was made the tree was wounded in buckshot. Microorganisms that established themselves in the wound caused the wood there to decay, making it whiter than elsewhere. In response the tree mounted a chemical defense: the dark boundary of discolored wood surrounding the decay indicates the production of antimicrobial substances. The dark line curving over the wounded wood is a further defense: the cambium, or growth layer, alive at the sides of the wound produced a cellular wall protecting the wood that formed after the time of the injury. The pattern of growth rings outside the wound reveals the subsequent history of the tree. After five years the wound closed, and after that four more growth rings formed. The tree was sectioned by the author and photographed by Kenneth R. Dudzik of the U.S. Forest Service in Durham, **N.H.**

wood tissue its strength. The interlocking design continues into the molecular construction of the cell walls, and even into the individual cell-wall molecules: cellulose and lignin.

At any given time in the life of the tree the more recent layers of wood, the ones in which the parenchyma still retain their living-contents, form the tree's sapwood. In many trees the older layers, nearer the axis of the tree, form a district called heartwood, which often is darker in color. Some of the darkness is due to the deposition of substances known collectively as extractives. Heartwood has a high degree of mechanical self-support; thus it continues to serve the tree. On the other hand, heartwood has no capacity to store nutrients and transport substances.



COMPARTMENTS OF A TREE enable it to resist the spread of infection after an injury; they also enable it to wall off parts of itself when the parts reach a genetically programmed stage of senescence. At the left a composite tree is shown. The left side of the composite is a loblolly pine, typical of conifers; the right side is a sugar maple, typical of hardwoods. Certain compartments, shown in the drawing, are shed when their function is completed. At the upper right the trunk of the pine appears in cross section, revealing more

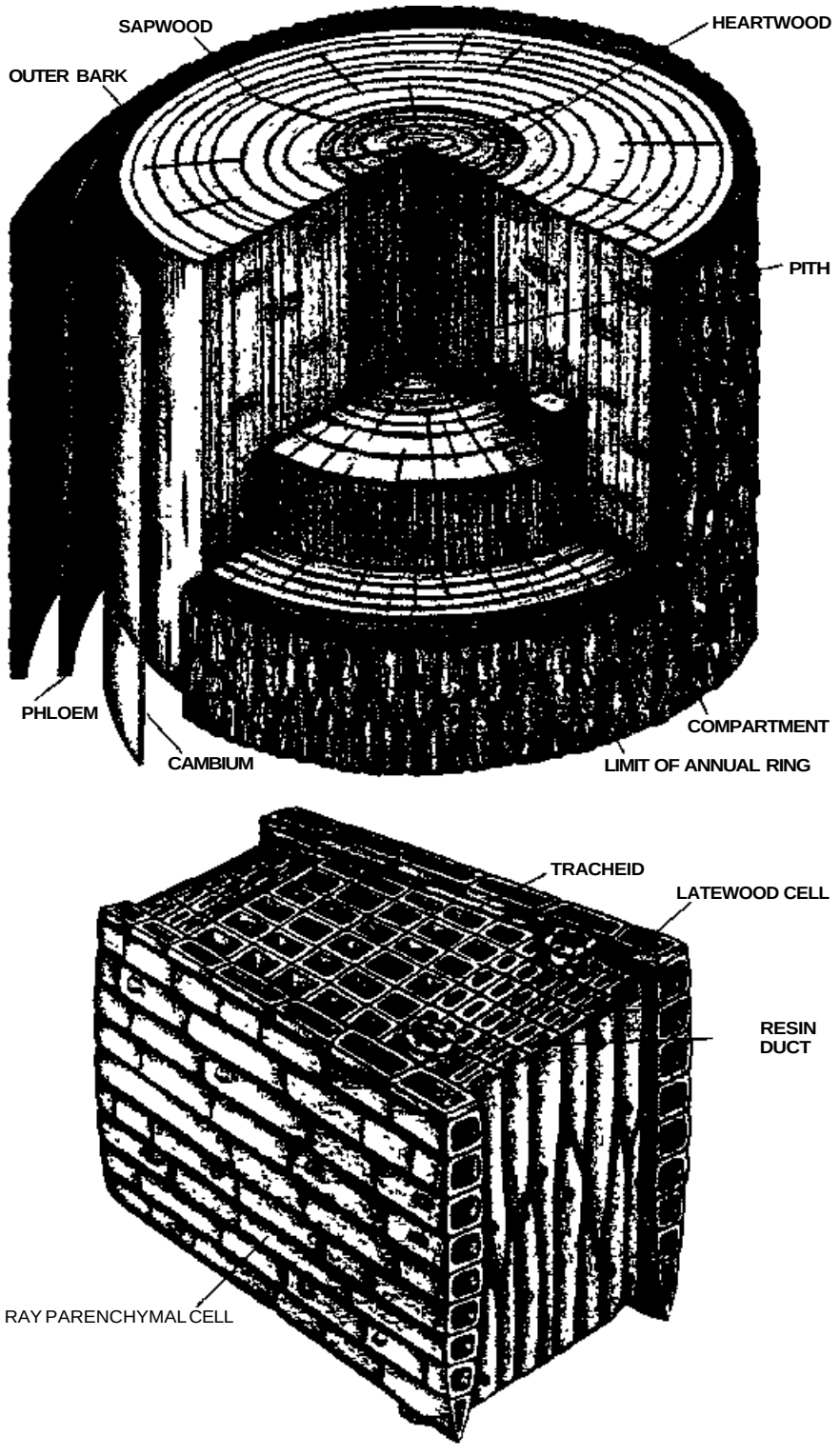
The study of tree anatomy suggests a number of ways in which trees can be viewed conceptually. First, trees arc tissue generators. In essence the germination of a seed is the activation of a cell generator. It has the capacity to proliferate cells, but always in new locations, the tree has no capacity to re store or regenerate tissues already in place. The generator is the cambium Second, wood is a highly ordered at rangement of different types of cells in different stages of aging Third, a tree- is a hierarchy of compartments. With in the trunk, the roots and the branches UK largest compartments are the annual rings. Then come groups of cells in each ring, compartmented by rays, and next the individual cells.

The study of how trees respond to injury or infection suggests a further concept Trees respond by compartmentalizing, they attempt to wall off the injured or infected region. They neither kill nor arrest the activity of microorganisms in the compartments that get walled off. Nor do they respond in specific ways to specific microorganisms; the compartmentalization comes in response to the fact of the injury.

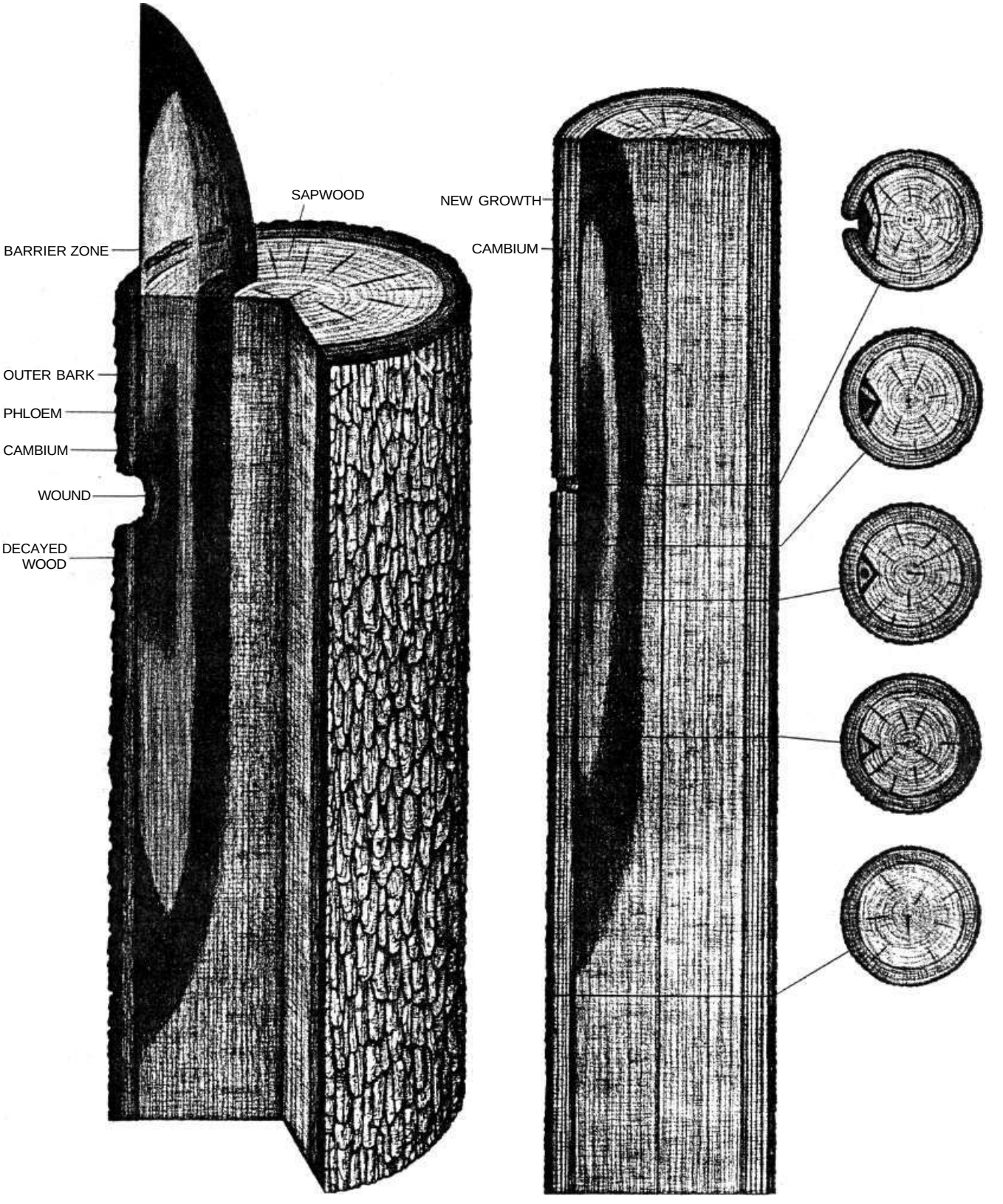
Broadly speaking, the tree makes three responses to injury and infection In the first of them the boundaries of compartments already in place are strengthened to resist the spread of destruction For the most part the strengthening is achieved by chemical means. In sapwood the metabolism of the living parenchymal cells changes in ways that alter the contents of the cells. In heartwood enzymatic reactions take place in the otherwise nonliving tissue.

The details are poorly understood. Still, under normal circumstances the cells in wood devote their biochemical pathways to the storage of chemical energy in the form of carbohydrates. Usually the molecules are in a reduced form their content of electrons is relatively great. After injury to the wood the biochemical activity in the cells surrounding the injury is shunted into new metabolic paths, so that molecules tend to be oxidized; that is, protons, or hydrogen ions, are attached. On a broad scale the cellular contents undergo a chemical process much like the tanning of leather. Thus molecules such as gallic acid and tannic acid appear. They share the property of being rich in phenol (six-carbon rings bearing hydroxyl, or OH, groups). The phenols occupy the interior of the cells; they also impregnate the cell walls. The phenols discolor the wood in shades of red, green or blue, depending on the details of the chemical pathways, which are determined by the genetics of a given species of tree. More important to the tree's defenses, phenol compounds tend to be antimicrobial.

In the second response the tree makes to injury and infection the tree creates a new wall by anatomical and chemical means. First the cambium changes the pattern by which it generates new cells In the wake of the injury few pipeline cells are produced Fiber cells too are made in lessened quantity. On the other hand, parenchyma, or



compartments. The cambium generates phloem, or inner bark, and xylem, or wood. The latter is compartmented by annual rings; in turn the rings are compartmented by partitions called parenchymal rays. At the lower right is a wood compartment It includes parenchyma, or energy-storing cells; tracheids, or cellular tubes, and thick-walled latewood cells.



RESPONSES TO A WOUND take two forms: the strengthening of existing compartments and the creation of a wall to protect the cambium. The sectioned maple trunk at the left includes an infected wound. A succession of microorganisms *(green)* has become established in the sapwood (the layers of living wood under the cambium), and at the center of the infection the wood has decayed *(brown).* At the margins of the infection parenchymal cells are producing antimicrobial substances *(red),* so that the walls already present in the tree are being strengthened against the infection. In addition cellular tubes above and below the infection are being plugged. (In some trees the ballooning of parenchymal cells constricts the tubes.) The trunk at the right shows the tree in a subsequent growing season. The wall created by the cambium has protected new growth. Meanwhile the internal defenses have compartmentalized the infection.

cells that retain their living contents, are made in increased quantity. Now, however, they are smaller, and their metabolic activity is altered so that their chemical contents resist microorganisms. The new wall, or barrier zone, is the cause of many of the defects found in wood products. For example, it can cause the wood in a living tree to separate along a circle. The defect is known as ring shake.

The third response the tree makes is to continue growing. Trees survive after injury and infection if they have enough time, energy and genetic capacity to recognize and compartmentalize the injured and infected tissue while generating the new tissue that will maintain the life of the tree. To an extent, therefore, an injured or infected tree resembles a heavily compartmented ship or an old type of submarine under attack. When the ship is hit by a torpedo, the crew rushes to seal off the damaged area. The faster the action of the crew and the stronger the walls circumscribing the damaged compartment, the smaller the spread of damage. But after the damage is contained the damaged compartment or compartments are no longer accessible. At this point the analogy ends. The tree survives by growing what amounts to a new tree (with a new set of compartments) over itself during the next growing season.

It must be said that the tree's reaction zones (its chemically strengthened boundaries) are not absolute: they may retreat, rapidly or slowly, from the infection, as certain microorganisms overcome the chemical defense. It should also be said that phenols are poisonous not only to microorganisms but also to the tree. In effect, the tree poisons part of itself in an attempt to stall an invasion. The tree survives because while it is strengthening barriers it is also creating a new tree.

Remarkably, the capacity of a tree to shed parts of itself is much like the response the tree makes to injury and infection. That is, the shedding is an aspect of compartmentalization. In particular, needles, leaves, reproductive structures and absorbing roots that have fulfilled their genetic program are walled off from the tree. (Absorbing roots are the fine, nonwoody roots that absorb substances from the soil.) Twigs, branches and large roots may also be walled off, say in the wake of an injury or an infection, or after the twig, branch or root has reached a particular stage of senescence. (It is hard to know just what stage; the genetic programming of aging in trees is poorly understood.) The foregoing requires a qualification: trees do not actively cast off parts of themselves. Wind, snow, ice, animals and other agents cause the walled-off parts to fall away from the tree. Meanwhile walled-off absorbing roots are digested by soil microorganisms.

I turn now to the other side of the strife between the tree and its enemies. I shall begin with the wounding of a tree, say by an animal. The wound provides new space and nutrients for an array of invading organisms including insects, nematodes, bacteria and fungi. While they compete among themselves for the new space and nutrients, the living cells in the sapwood underlying the wound are reacting to the invasion by undergoing a variety of biochemical changes leading to the production of phenol-based chemical defenses.

Microorganisms attack tree wounds in several ways. Certain bacteria and fungi infect inner bark and stay there, creating the diseases known as annual cankers. Other microorganisms invade wounds and remain in the wounded sapwood tissue, creating so-called wound rots. Still other microorganisms infect inner bark, become established and then infect wood. By the annual repetition of the process they create perennial cankers. Finally, some microorganisms attack a wound, first infecting sapwood and then infecting inner bark. Again the process repeats itself each growing season. These are the so-called canker-rot fungi. They are notably insidious. When the canker rot has progressed from wood to bark, it forms a hard pad of material, similar to a wedge, that kills the subjacent cambium. The tree responds by reactivating its compartmentalizing defenses. The fungus in turn invades the new wound, from which it grows another wedge. The seesaw activity can continue until the tree is girdled.

The first microorganisms to succeed in invading the tree are termed pioneers. They may simply be able to tolerate the chemical alterations brought on in the wood by the wounding of the tree. In some cases, however, their genetic makeup renders them capable of digesting the chemical defenses. Typically, but not always, the pioneers are bacteria, along with certain species of fungi. Notable among the latter are the Hymenomycetes, which cause decay in trees, and the Fungi Imperfecti and Ascomycetes, which for the most part do not themselves cause decay.

A crucial point about the infection is that microorganisms establish themselves in a particular sequence. When the pioneer microorganisms surmount the inhibitory chemicals, they pave the way for other invaders, ones that would have succumbed to the tree's defenses. Thus the invasion takes the form of a succession of organisms, in a pattern essential for the survival of the invaders. To be sure, no microorganism "eats poison" to help its successors. Each organism acts in a way that furthers its retention of space and energy. Hence some of the pioneers may actually create or preserve conditions that inhibit infection by aggressive wood-decaying fungi. Such pioneers may ultimately be the basis for the biological control of decay in trees. Studies of certain fungi, such as species of *Trichoderma*, already show that the fungi thrive in wound-altered sapwood but do not detoxify the chemicals that keep out decay-causing fungi.

Suppose a succession of invading microorganisms is successful at digesting the wound-altered sapwood. Their success does not necessarily doom the tree. For one thing, the tree is generating new cells around the old ones. If the tree can generate new cells faster than old ones are being digested, the tree has a good chance of survival. The death of a tree, if it happens, can be mechanical or biological. The tree will die if the trunk breaks. Alternatively, it will die if the cambium—the cell generator—is killed. Further still, it will die if so much of the tree's tissue has been walled off during a lifetime of defense against injury and infection that the remaining compartments are insufficient to store the tree's energy reserves.

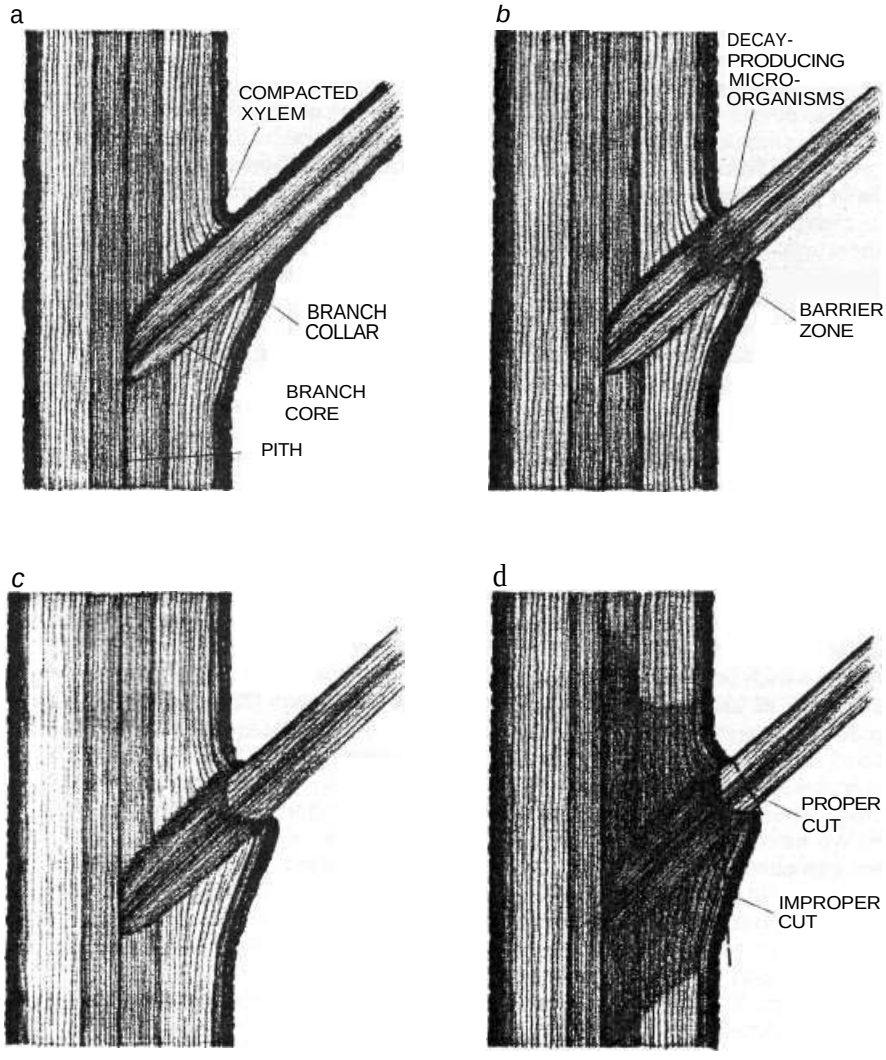
The new understanding of trees as compartmentalizing organisms did not arise at once. Indeed, it came as a contradiction of earlier notions, some of which were developed soon after the underpinnings of modern biology were established a century ago. It seems a trite thing to say, but trees are fundamentally different from animals, and much of the failure to understand trees derives from unconsciously confusing the two.

Before 1845 the preferred explanation of life was that it originates spontaneously (that is, by spontaneous generation) from inorganic material. Much was known about microorganisms; in particular the association of decayed wood and fungi had been recognized. The idea, however, was that decay gives rise to the fungi. In the wake of Heinrich Anton De Bary, Louis Pasteur and the development of the germ theory, which attributes disease to minute, harmful organisms, the German tree pathologist Robert Hartig reversed the idea by proposing that fungi give rise to decay. Hartig showed that the sporophores, or fruiting bodies, found in tree wounds and the mycelia, or strandlike organisms, found in decayed wood represent different life

stages of the same fungal organism. The observation, and the reversal of the hypothesis, set the stage for the science of tree pathology.

Subsequent studies of decay were made by investigators interested primarily in the deterioration of lumber. Accordingly the studies relied on the removal of wood from trees, followed by laboratory investigation of changes in the tissue. The studies centered on heartwood, or in any case wood darker than sapwood. The strategy underlying the studies was justified on the ground that the heartwood in a tree is dead, unresponsive tissue. To a degree the studies succeeded: chemists and pathologists came to understand how fungi (in particular their enzymes) digest wood. Still, the processes in the living tree were not considered. Although heartwood is dead by animal standards, it is reactive to injury and infection. Some texts still state that decay in trees cannot be considered a disease because only dead heartwood gets infected. Decay in trees is in fact the major disease of all trees worldwide.

Early investigators were not able, therefore, to recognize the defenses activated by injury and infection, which alter wood when the tree is wounded. In a living tree most wounds do not produce decay because the microorganisms infecting the wound are faced by wood that has changed. In contrast, the decay-producing fungi attacking lumber face no countering force from the wood. The microorganisms simply compete among themselves.



DEATH OF A BRANCH follows a course much like the reaction of the tree to injury or infection. The living branch *(a)* is separated from The trunk by an external feature called the branch-bark ridge and an inner partition of compacted xylem along the angle of the ridge. The swollen base of the branch is called the collar. As the branch dies *(b)* it is invaded by decay-producing microorganisms. The tree marshals chemical defenses in a protection zone in the collar. The sequence is essential for the shedding of the branch. In most cases the decay stops at the collar. Sometimes, however, the decay invades the rest of the core of the branch (c). If the dead branch is pruned improperly *(d).* so that the cut is through the collar, the protective zone is removed and the sapwood in the trunk is opened to an invasion.

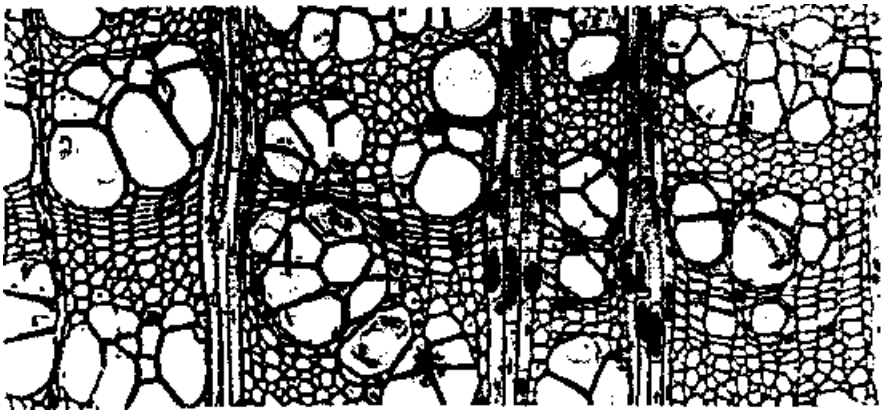
The crucial technology that enabled investigators to develop a new understanding of decay in trees was the chainsaw. By the 1940's the device was powerful enough so that an individual operating it in a forest could easily cut longitudinal sections through trees, exposing columns of discolored and decaying wood under the growth rings the tree had developed after the injury. Before 1940 trees had been dissected, but most of the cuts had been crosscuts. Only a few hardy investigators, such as the pathologist George H. Hepting, working in the Mississippi Delta in the 1930's, proceeded otherwise. Hepting, using crosscut saws and axes, was able to see what others saw later: that throughout long stretches of trees the wood generated after a wound was not invaded by the fungi infecting the wound itself.

In the 1960's and 1970's 1 myself had the opportunity to disect thousands of trees, first in the northeastern U.S. and then in Europe, India, Puerto Rico and Australia. I was impressed on the one hand by the orderly patterns of discoloration and on the other hand by the orderly succession of the invading microorganisms. My colleagues and I then made experiments in living trees. Still later, biochemical studies were undertaken by Walter C. Shortle, my colleague at the Forest Service of the U.S. Department of Agriculture in Durham, N.H.

In an effort to define a commonality among the responses a tree makes to injury and infection and account for the patterns of decay and discoloration in wounded trees, my colleagues and I have devised a model we call CODIT (an acronym for Compartmentalization of • Decay in Trees). The first part of the model represents the responses the tree makes at the time of the injury. In essence the tree strengthens walls that are already in place in the wood, at least in large part. Wall 1 resists the vertical spread of infected wood, wall 2 the inward spread and wall 3 the lateral spread. The second part of the model represents a response the tree makes later. In essence the tree raises a wall not in place at the time of the injury: the cambium generates wall 4, which separates the infected wood from newly forming, healthy wood.

Wall 1 does not really exist as an anatomical entity before the infection. It is primarily a plugging wall, which resists the vertical spread of infection by anatomical and chemical means. The tree has vertical plumbing, consisting, as I have noted, of vessels in hardwoods and tracheids in conifers. In the wake of an infection the tubes must be plugged; the tubes are an easy

way for invaders to spread. Trees plug the lubes in several ways. The cells surrounding the tubes (called contact parenchyma) can balloon into the bore of the tubes. Encrustations can develop at the ends of the tubes. Granular or crystalline material can fill the tubes. Pits between tubes can close. Sometimes air bubbles develop, impeding the transport of liquids. The wall-1 defense is relatively weak, but then the tree cannot survive if it plugs all its vertical plumbing. Moreover, the vertical spread of infection is relatively unimportant: the entire core of a tree can be infected, yet the new trees formed by the cambium in subsequent years will keep the tree alive.



CROSS SECTION OF SAPWOOD from an American elm includes a wall, or barrier zone, created by the cambium in response to an injury. Within the zone the parenchymal cells are smaller. Moreover, the parenchymal rays are swollen (three rays cross the field of view), and many of the ray parenchymal cells have dark inclusions consisting of antimicrobial substances. The larger cells throughout the cross section are vessels, the hardwood's equivalent of tracheids. The injury that provoked the tree's defenses was an infection by the fungus that causes Dutch elm disease. Elms react to the infection by walling off the fungus. In doing so, however, they wall off some of their capacity to store reserve energy.

Wall 2 and wall 3 exist before the infection: they are formed by the annual rings, which resist the inward spread of infection, and by the parenchymal rays, which resist the lateral spread. After the infection the walls are chemically strengthened. In part, however, wall 2 is anatomical. In some trees, such as the maple (but not, for example, the elm), the end of each season of growth is marked by the production, on the part of the cambium, of a final layer of what are called marginal or terminal parenchyma in hardwoods and thick-walled latewood cells in conifers. The cells form a barrier of cellular masonry at the perimeter of each growth ring. Wall 2 is moderately strong; wall 3 is the strongest of the three. If the latter fails, decay can spread like an opening fan. This is the cause of hollow trees, used for protection and dwelling by many animals. The tree itself can survive, with full crowns of healthy leaves, owing to the work of the cambium in growth seasons subsequent to the injury.

The crucial resistance to the infection is the resistance to its outward spread—in particular the defense of the cambium against destruction from within the tree. Here wall 4, the cambium's wall, comes into play. It is quite weak in the sense of strengthening the structure of the tree but quite strong in the sense of being a barrier against microorganisms, a barrier isolating tissue that is outside the injury and therefore formed after the injury. Indeed, it is impervious to most of the fungi and bacteria that inhabit wood or bark. Recent work by R. B. Pearce, P. J. Holloway and Jill Rutherford at the University of Oxford establishes that the cells of the wall-4 barrier are lined with suberin, the fatty acid that gives outer bark its resistance to microbial invaders. (The invaders almost never have enzymes capable of acting on suberin.)

My colleagues and I have also invented a device that detects decayed wood in living trees without damaging the trees. The device capitalizes on a circumstance of decay: as wood de--cays, its content of electric-charge carriers (chiefly potassium ions) increases, so that the electrical resistance of the tissue, measured in ohms, decreases. Our device therefore consists of a battery-operated pulsed-current generator, a probe and an ohmmeter. To test for decay, a hole 3/32. of an inch in diameter is drilled eight to 12 inches into the tree. The probe is slowly inserted. A sudden decrease in the electrical resistance encountered at the tip of the probe (the tip lacks insulation) signals decay. The device is also being employed to detect decay at the groundline in utility poles.

My colleagues and I are now collaborating with geneticists in an effort to learn which individuals within a given species of tree have the best capacity for compartmentalizing. (The capacity is under strong genetic control.) Armed with our new concepts of tree biology we are also reexamining tree diseases. We have found, for example, that elms compartmentalize the wood infected by the fungus that causes Dutch elm disease. The defense can lead to starvation as tissues that normally store energy get walled off. Moreover, we are devising corrections in many of the standard tree-care procedures, such as pruning.

Much of the misunderstanding that underlies improper tree care comes about because concepts developed to explain animal biology are applied, almost unconsciously to trees. In many ways trees are treated like animals or. worse, like people. Dressings are put on wounded trees in an effort to stop decay and promote healing, much as a parent dresses a cut on a child. Cavities of decay are cleaned out beyond the decay and into healthy wood, much as a dentist cleans out *a* decayed tooth. Branches are pruned flush with the trunk, and in some countries the bark of the trunk at the base of the branch is scribed in the shape of a diamond; the subsequent callus, or scar, is considered a sign that the tree is healing well.

None of these treatments is beneficial to trees; indeed, all of them can be harmful. No scientific data show that any substance applied to a tree wound will stop decay. Dressings are primarily cosmetic. (Perhaps too they are psychological medicine for the owner of the tree.) The cleaning of a cavity past the decay and into healthy wood promotes further decay. It is in fact one of the worst things one can do to a tree. The cavity exists because the tree has walled off the decay. Finally, improper pruning enables decay to take hold in injured cells in the trunk. Around the base of every branch is a swelling known as a collar. Within it is the protective zone of the branch, that is, the place where the branch's chemical defenses are established. The collar should not be injured, much less removed, by pruning.

Decay in trees is a natural process. In some cases it can be regulated: it can be quickened, slowed or stalled. When it cannot be influenced, it can at least be detected, nondestructively, in living trees. Moreover, it can be predicted, in both rate and configuration. Furthermore, trees can be selected for their resistance to the spread of decay. Scientific forestry is approaching a new understanding of trees and how they survive under many pressures.