DECAY DISEASES OF STEMWOOD: DETECTION, DIAGNOSIS, AND MANAGEMENT

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1. Introduction

Three major needs exist to better relate tree biology research to practical agroforestry (Larsen 1984). The first need is to identify the specific, desired product to be yielded from the forest. Valid and appropriate management techniques to yield one product may not be appropriate to yield a different product, even from the same species of trees. The second need is to synthesize the relevant scientific and technical knowledge. Frequently, the relevant knowledge to effectively yield a product needs to be integrated across a wide range of resources. The third need is to develop a comprehensive life history of the crop tree species. Tree life history and biology includes the growth and the decay processes that are common to all mature trees. The opposing processes of growth and decay occur simultaneously within a living tree for long periods of time. However, the mere survival of damaged and diseased trees in the forest can interfere with the yield of the desired stemwood product.

For this paper, we are concerned with the production of stemwood from mature standard trees (30–75 cm in diameter at 1.4 m aboveground). The threat to stemwood production posed by the decay process will be our focus.

Pioneering research continuing over several decades links tree biology, forest pathology, and the yield and performance of wood products. The partial synthesis of this knowledge gained from experimentation and tree dissection is available to land managers to improve the production of wood products (Shigo 1984, 1986a, 1986b, 1991; Shigo and Hillis, 1973; Shigo and Shortle, 1985; Shortle, 1979b; Smith 1989). For most tree species, comprehensive life histories need to be developed from existing literature, careful record keeping, and new research.

Our purpose in this paper is not to review the extensive literature that relates specific types of decayed wood to particular decay fungi or associated microorganisms (Boyce 1961; Hepting 1971; Rayner and Bobby 1988). This paper presents general concepts and principles that may serve as a starting point to assess decay diseases that limit the utility of stemwood products from utility poles to violins, from pulpwood to veneer.

2. Infection Pathways and Decay Detection

Infections that destroy stemwood in living trees are spread by one of three pathways (Figure 1). One common pathway is the spread of root-rot fungi into the stem from below (Figure 1B) An advanced infection that has spread into the stem from the roots is generally called "butt-rot". Extensive literature is available on root-rots and butt-rots. Unfortunately, most of the emphasis is on advanced stages of decay, too late to be of much value in the mitigation of loss of product yield.

The early stages of the same root-rot infections can spread far up the stem long before any wood is visibly decayed in freshly cut logs. These early stages of the wood decay process might be called "wetwood," "discolored wood," or "incipient decay." Microbiological isolations from wood in these early stages of the decay process would likely yield bacteria and yeasts. A budding, yeast-like growth phase occurs in some normally filamentous fungi under condition of low oxygen and high carbon dioxide concentrations. These microaerophilic conditions frequently occur in the stemwood of living trees, except near exposed surfaces (Jensen 1969). Decay fungi develop poorly in wood with little oxygen (Jensen 1967). Bacteria and budding, small-spored fungi have a definite physiological advantage for growth in these low-oxygen environments. In some instances, the oxygen concentration is sufficiently low to allow for the growth of obligately anaerobic bacteria (Shigo et al. 1971). Eventually, oxygen levels increase due to surface checks, cracks, and holes bored by insects and other arthropods. As oxygen levels and activity of the decay fungi increase, the oxidative degradation of natural wood preservatives and the depolymerization of wood cell wall materials will proceed (Shortle 1979a and b; Shortle and Cowling 1978a and b; Shortle and Smith 1990). The usual progression is the replacement by oxygen-demanding, wood-destroying fungi of their microbial associates. This process is sometimes called a "succession" (Shigo and Hillis 1973).

The most reliable external indicators of stemwood decay due to the spread of root-rots are basal cracks (Butin and Shigo 1981; Shigo and Tippett 1981). The cracks generally begin between the buttress roots and

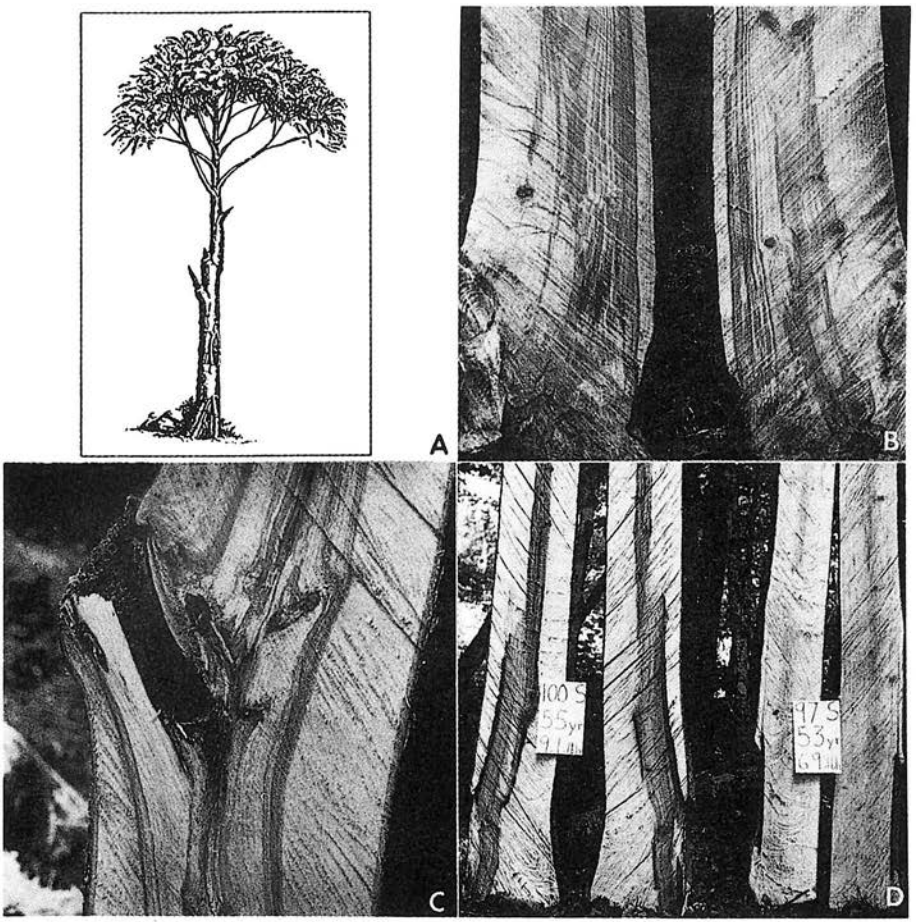

Figure 1. Detection of stemwood diseases begins with a careful examination of the tree for external indicators of internal infections (A). Root-rot infections that spread upward into stemwood are often associated with basal scars and cracks (B). Branch stubs are key entrance points for canker-rot infections (C). Stem scarring directly exposes stemwood to infection (D).

expand upward, causing considerable defect in the lower portion of the stem and the loss of product yield. Wounds often provide the opportunity for the initial infection of roots that eventually leads to stemwood cracks and decay. Wounds result from logging, grazing, road construction, fire, etc. Changes in soil drainage patterns also kill roots and expose wood to infection.

Root-rot infections that spread into stemwood are easily detected by measurements of electrical resistance (Ostrofsky and Shortle 1989; Shigo and Berry 1975). The earliest, previsual stages of the wood decay

process and accompanied by a decrease in electrical resistance (Shortle 1990; Shortle and Smith 1987). Consequently, stemwood that appears to be sound and undecayed may be, in fact, altered by the wood decay process as indicated by decreased electrical resistance. This has caused some confusion for workers who had previously focused only on the advanced, visible stages of decay. Previsual stages of the wood decay process have long been recognized to affect product performance (Hartig 1894; Hubert 1931). More recent research shows that wood in these early stages may be unsatisfactory for certain kiln-drying schedules (Shortle and Hill 1987), other wood processing steps, and in service (Ostrofsky and Shortle 1989).

Electrical methods to detect early stages of the decay process may prove to be more valuable as younger trees, in which advanced decay has not developed, become an increasingly important source of stemwood. For crop tree management, early detection of infection is far superior to waiting for the appearance of fungus fruiting bodies to indicate that stemwood decay is in the advanced stages.

The second common pathway for infections that destroy stemwood is through the stubs of stems or branches (Figure 1C). All tree stems shed branches. The successful, clean shedding of branches is an important contributor to the yield of high value stemwood. Stems that have shed small branches are not frequently associated with stemwood infections. Decay of small, residual stubs is usually limited to the branch wood. However, small-diameter branch stubs of *Tsuga heterophylla* can become infected with *Echinodontium tinctorium*. Following years of stem growth and changes in the internal micro-environment, the now encased, infected stubs can provide the inoculum base for spread of the decay fungus through the main stem (Etheridge and Craig 1976). Poorly shed branch stubs can be infection courts for canker-rot fungi. Canker-rots are frequently long-term infections of living trees (Shigo 1969, 1986a). Both root-rot and canker-rot fungi can attack both living phloem and wood. Both types of pathogen are highly effective and cause much damage of mature tree.

The third pathway of stemwood infection is through direct scarring of the stem (Figure 1D). Removal of the protective bark layer kills living cells and exposes the stemwood to infection. The living, sapwood immediately adjacent to the exposed surface responds to reform a protective layer to minimize that volume of sapwood lost to desication and inevitable infection (Hepting and Blaisdell 1936; Shortle and Smith 1990). This protective zone formed in extant sapwood is effective, but easily breached by cracks due to drying and boring insects (Shortle and Cowling 1978a; Shortle and Smith 1990). The rate of spread of infection within trees is highly variable due to genetic variability, tree energy reserves, and the

local populations of insects and pathogens that are available to exploit the exposed tissue. Predictions of amounts and rates of internal spread of infection from external signs such as fire scars (Hepting 1935) and logging scars (Shigo 1966) are generally poor. This is partly due to rates to spread being due to internal factors which are not indicated by the external signs.

The most critical variables that determine the extent of infection following scarring are elapsed time, tree defense, and tree growth. To explain the pattern of stemwood decay following the wounding of trees by fire, Hepting (1935) predicted that the vascular combium formed an anomalous layer of sapwood following wounding. This anomalous layer stopped the outer progress of decay which would eventually destroy all of the wood extant at the time of wounding. Hepting (1935) observed that this anomalous layer was produced irrespective of whether the wood exposed was mostly sapwood as in the case of sweetgum or mostly heartwood as in the case of oak (Figure 2).

Later research resulted in the development of a three-stage process model that linked wounding to the eventual decomposition of stemwood (Shigo and Hillis 1973). Following wounding, exposed sapwood frequently discolors as part of the tree response to mechanical damage (stage I). Stage-I discolored wood forms in trees such as species of maples that do not normally form a colored heartood as well as in the sapwood of heartwood-forming species such as oak. The stage-I discolored wood is similar in many properties to heartwood and is resistant in some degree to microbial decomposition. Heartwood and stage-I discolored wood both are altered by decay microorganisms and their associates in stage II of the decomposition process (Shigo and Shortle 1979). In stage II, the discolored wood becomes less resistant to further decay that culminates in stage III and the physical degradation of wood.

Depending on stand history and tree species, hollow trees are more-or-less easy to find. Contrary to the heart-rot concept (Boyce 1961), hollow tree do not result from the decomposition of non-living heartwood by saprobic fungi. Hollow trees result from the formation of barriers to the outward spread of infections, consistent with the compartmentalization concept (Shigo 1984). Compartmentalization is the boundary-setting process which tends to limit the loss of normally functioning wood after wounding. Compartmentalization boundaries occur in both extant sapwood and in wood formed following wounding. These boundaries have been referred to by various terms. Heartwood and stage-I discolored wood have been termed "protective wood" because of increased decay resistance (Jorgenson 1962). Various "protection zones" are the boundaries formed in extant sapwood (Hepting and Blaisdell 1936). These boundaries have also been termed "reaction zones" (Shain 1967).

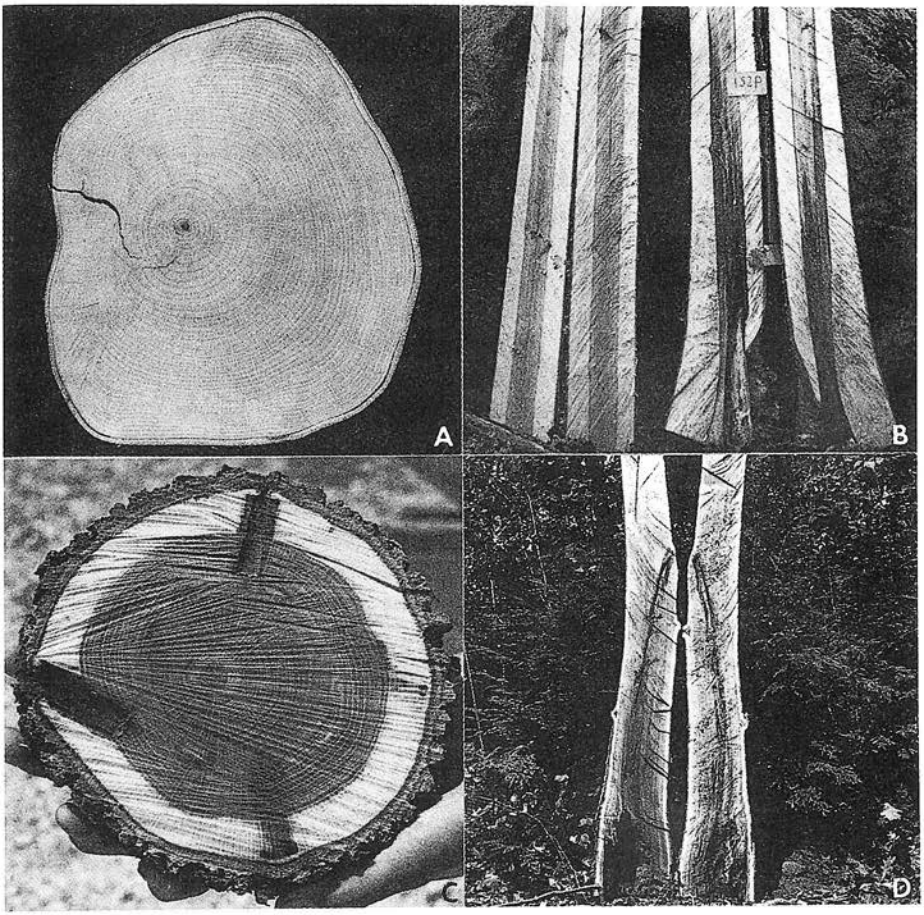

Figure 2. Tree stems in which the stemwood is mostly sapwood at maturity (A), discolor and decay when stemwood is exposed by injury to infection (B). Tree stems in which stemwood is mostly heartwood at maturity (C), discolor and decay when stemwood is exposed by injury to infection (D).

Subsequent research showed that these protective and reaction zones were physiologically distinct "column boundary layers" that formed as a result of interaction between spreading microorganisms and living sapwood (Shortle and Smith 1990). The spread of infections can also be limited by the lack of oxygen and nutrients (Highley and Kirk 1979).

The principle compartmentalization boundary that results in hollow trees is the "barrier zone" (Shigo 1984). A barrier zone is the anomalous sapwood which was predicted by Hepting (1945) to be formed by the vascular cambium after injury. The barrier zone limits the outward spread of infection and separates normal healthy wood formed after wounding

from wood present at the time of wounding. The core of wood present at the time of wounding frequently discolors and rots in complex and variable patterns (Shigo 1984, Shortle 1979a). These patterns have been confirmed by years of experimentation (Figures 3, 4).

The key factor for an agroforest manager to consider is the proportional amount of normal stemwood to the decayed core (Table 1). Injuries made to smaller, juvenile trees yield very little defect if growth rates are favorable and further wounding can be avoided. Compartmentalization tends to limit decay to stemwood present at the time of wounding. As trees grow larger and older, with less time remaining before harvest, the likelihood diminishes of producing a good yield of stemwood of high quality, no matter how effective the tree defense systems are. Unfortunately, cracks are produced in some tree species following juvenile wounding. These cracks can continue to propagate and damage stemwood through the life of the tree, irrespective of the harvest age or size of the tree (Butin and Shigo 1981).

Compartmentalization keeps trees alive. Compartmentalization does not necessarily keep trees sound and undecayed. The defense mechanisms can be so effective that trees live for many decades with many internal infections. This is good for the forest ecosystems as living, decaying trees can produce seed crops, shelter for wildlife, and nutrient-rich decayed wood for incorporation into the forest floor. Unfortunately, the survival of trees with extensive decay is not so desirable for the production of stemwood of high quality.

External indications of stemwood infections such as stem cracking, stem failure, and the presence of fungal fruiting bodies can be critical

Table 1. Estimated proportion of sound wood (%) based on the diameter of tree at harvest and the diameter of the tree at the time of a previous wounding[a]

	Diameter at harvest (cm)						
Diameter at wounding	25	30	35	40	45	60	75
5	96	97	98	98	99	99	99
10	84	89	92	94	95	97	98
15	63	75	81	86	87	96	97
20	36	56	67	75	80	89	93
25	—	31	49	61	69	83	89
30	—	—	27	44	56	75	84
35	—	—	—	23	39	66	78
40	—	—	—	—	21	44	64
45	—	—	—	—	—	36	44

[a] Estimated proportion of transverse sectional area through the horizontal plane of the wound. The vertical extent of wound-initiated discoloration and decay would depend on the nature of the wound and effectiveness of compartmentalization of the individual tree.

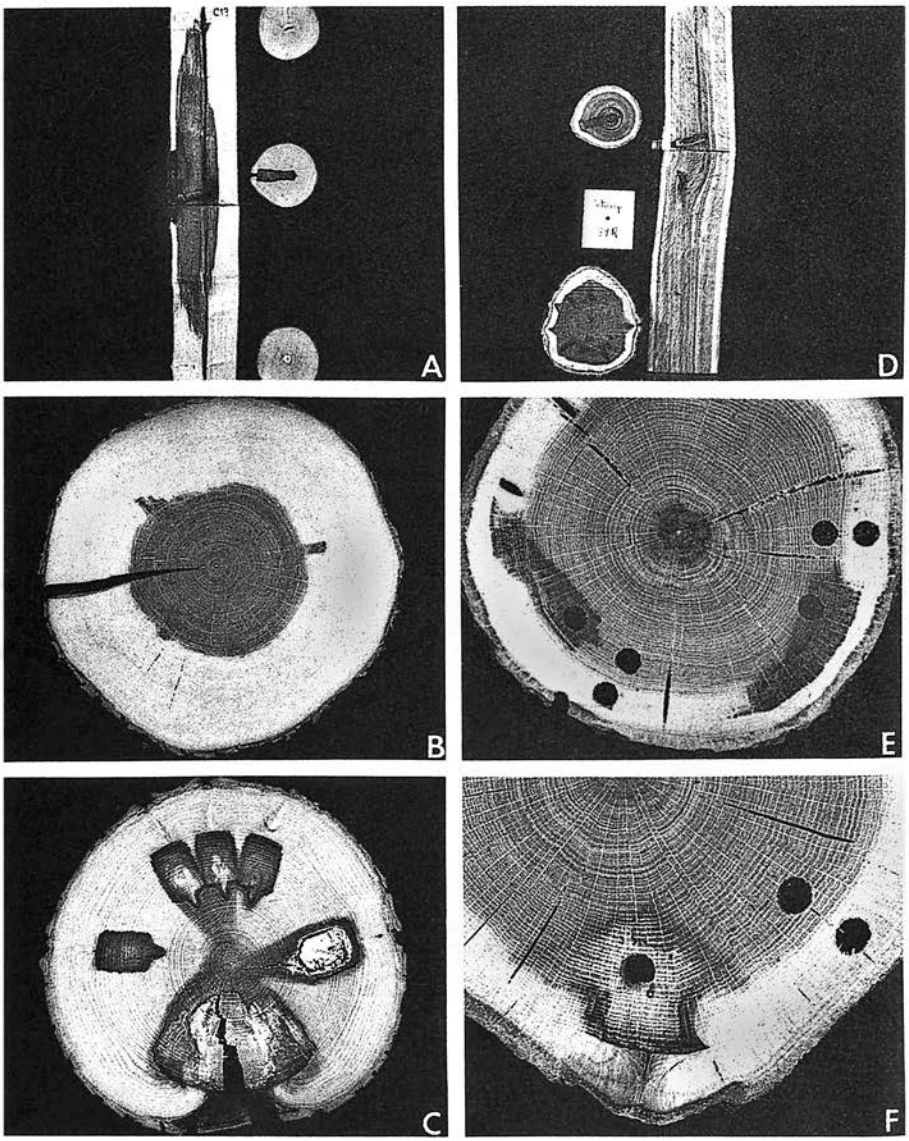

Figure 3. Trees which are predominately sapwood form discolored wood in response to injury and infection (A). Prior to alteration by microorganisms, this discolored wood (B) can be considered as protection wood as is heartwood (E). The spread of infection and decay are equivalent in both cases as confirmed by experimental injury, e.g. maple (A, B, C) and oak (D, E, F).

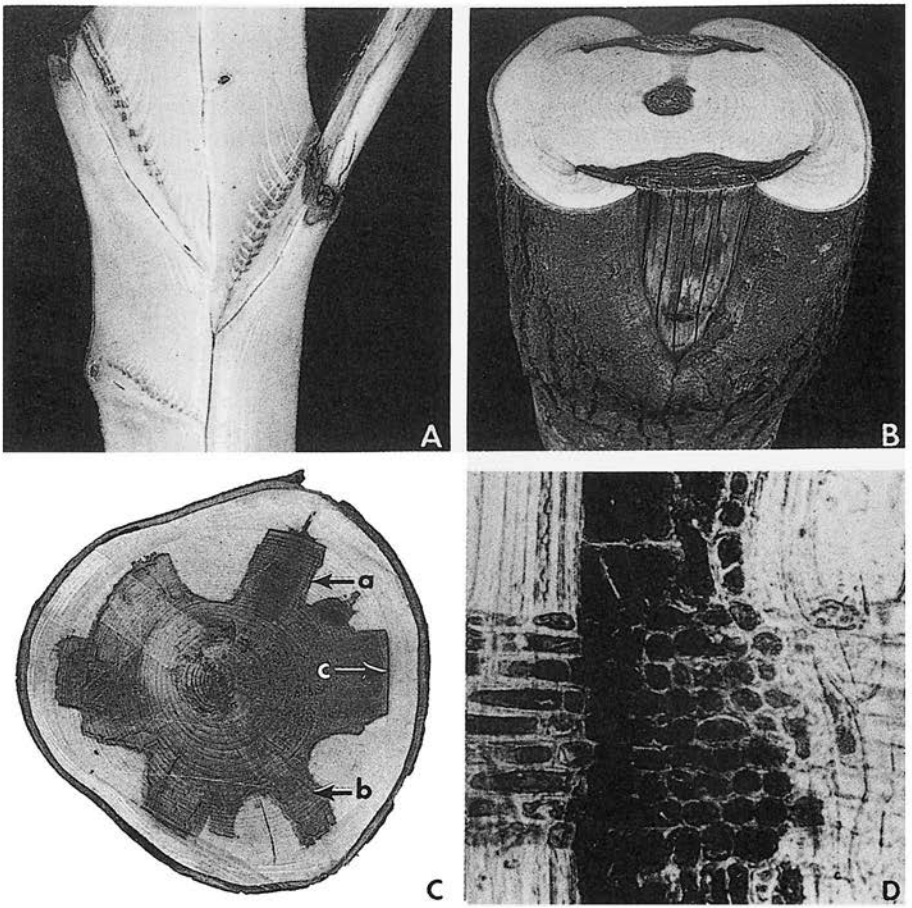

Figure 4. Localized layers of highly preserved, protective wood occurs in branch bases to prevent the spread of branch infections into stemwood (A), and they form repeatedly to separate spreading infections from live stemwood (B). If a tree lives long enough, the sapwood, protection wood and protective layers will decompose, within the bounds of a barrier zone (C, a = new protection zone, b = old protection zone, c = barrier zone). The barrier zone is anomalous sapwood formed by the vascular cambium after injury (D).

markers of stemwood infection. However, due to the potentially complex patterns of infection spread and uncertainty about the rate of vascular cambial growth away from the infection, additional methods of diagnosis are useful. Drilling a narrow hole (e.g. 3 mm in diameter) into the stem and examining the drill shavings can indicate wound-initiated discoloration. A sudden loss of resistance to torque indicates a void formed by the advanced decay of the stemwood. Electrical methods can detect internal infections and estimate relative diameter growth (Figure

5A, B) (Ostrofsky and Shortle 1989; Shigo and Shortle 1985; Shortle 1979c; Smith and Ostrofsky 1993). The measurement of internal electrical resistance will detect infected stemwood. However, the measurement will not differentiate between the early previsual stage of decay that are common to all stemwood infection and the advanced stage of decay which are highly specific to particular combinations of host and pathogen (Figure 5C, D).

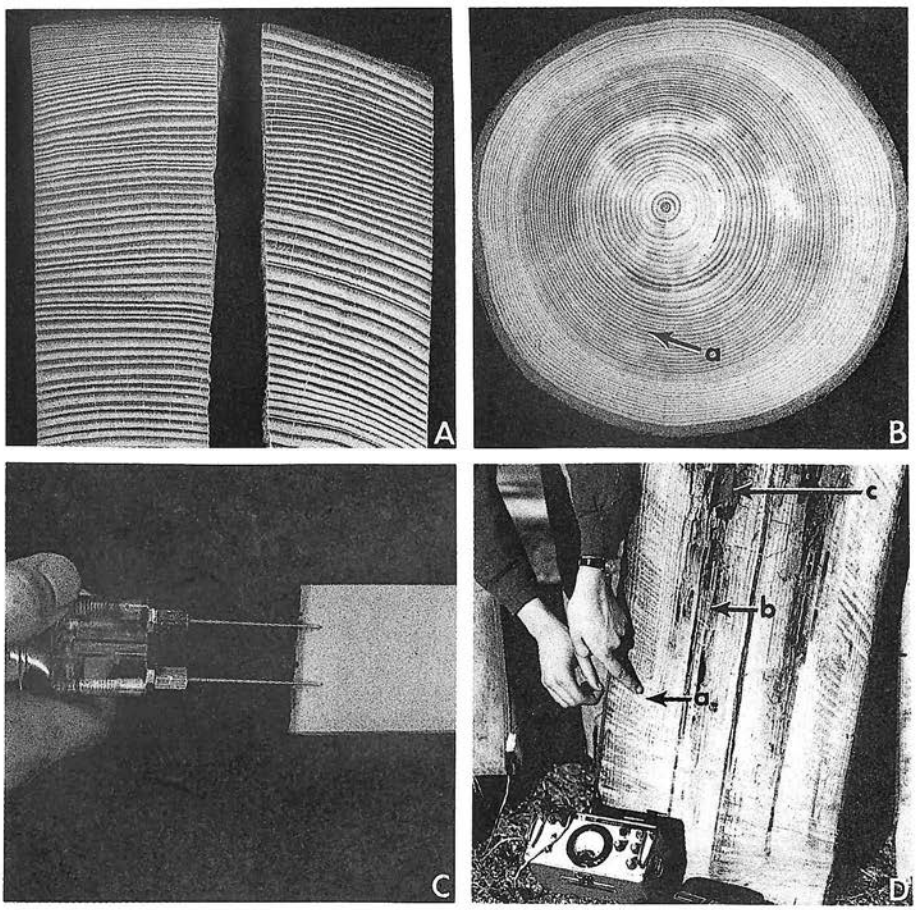

Figure 5. Variations in stemwood growth rates as seen in tree rings (A) and early stages of stemwood infection which we cannot see (B, a = previsual infection) can both be detected by an electrical technique called "Shigometry". Differences in cambial activity associated with relative radial growth rates can be detected by measurements of electrical resistance (C). Detection of all stages of decay is accomplished by the proper electrode and drill bit (D). The electrode detects previsual (D, a) and advanced (D, b) decay and reduction in the physical resistance to the turning of the drill bit detects voids (D, c).

3. Diagnosis of Stemwood Infection

Fungi that decay stemwood in living trees belong to one of three basic trophic groups (Hepting 1935): strong facultative pathogens, weak facultative pathogens, and obligate saprotrophs. Live wood and bark are infected by strong facultative decay fungi. These fungi infect intact tree tissue directly and do not require wounding of the tree prior to infection. These infections cause root-rots (for example *Heterobasidion annosum*, *Phellinus weirii*, and species of *Armillaria*) and canker-rots (for example, *Inonotus obliquus* and *I. glomeratus*) (Fig. 6). Forest managers need to consult the extensive literature available on the specific pathogens that threaten the production of their individual crop.

Unlike the strong facultative pathogens, the weak facultative pathogens (for example, *Trametes versicolor, Chondrostereum purpureum*) require wounding to initiate the infection process. In the absence of stem cracks, the infection does not spread beyond wood present at the time of wounding. The activity of weak facultative decay pathogens is enhanced by stem cracking (Fig. 6E, F), insect activity (Fig. 7A, B), and poor management practices (Fig. 7C, D). The weak facultative pathogens are frequently involved in combination with obligate saprobes. The obligate saprobes (e.g. *Gloeophyllum trabeum, G. saepiarium*) frequently decay both heartwood in infected stems and wood in service taken from the same tree species.

4. Forest Management

Specification of the desired crop and the management alternatives to be practiced often result from answering an iterative cycle of the questions: "what is desirable?" and "what is possible?". For example, even if edaphic and climatic conditions were ideal to grow teak or Norway spruce, the presence of stumps or roots infected with *Rigidiporous lignorum* or *Heterobasidion annosum*, respectively, would make foolhardy the cultivation of those tree species. If stemwood is the product, growth and decay processes need to be regulated to produce a high-value product. If non-wood products are to be produced, maintenance of tree defense systems that extend longevity will be a major goal of management. In the eastern United States, recent approaches applied to hardwood woodlots provide a practical lesson in stand management to yield a greater volume of a more valuable produce (Perkey, Wilkins, and Smith 1993). Elements of these approaches center on choosing management alternatives that favor the yield of the specified crop. After determining what the forest crop will be, the stocking density of the agroforest needs

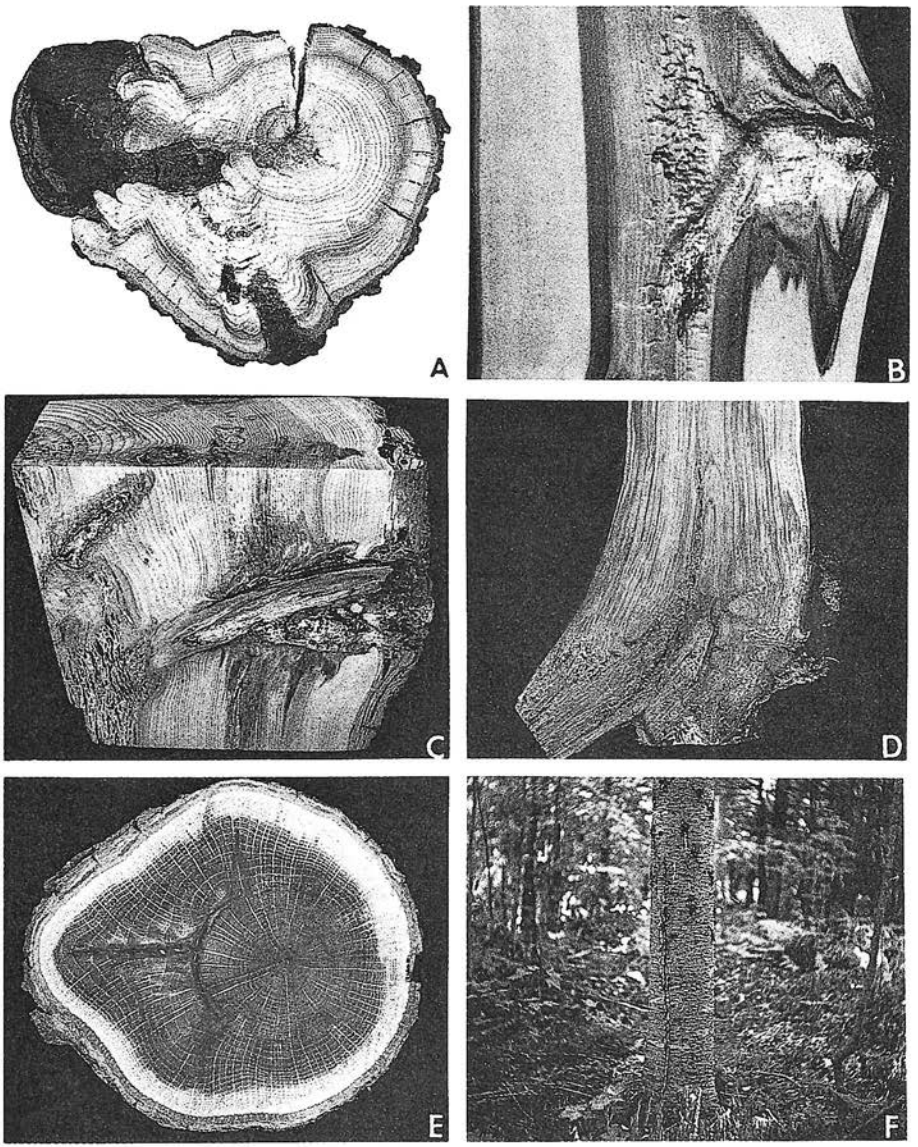

Figure 6. Compartmentalization, the system of protection wood, protective wood, and barrier zones, is least effective against canker-rot and root-rot fungi which attack both live wood and bark, e.g. *Phellinus everhartii* in oak (A), *Inonotus glomeratus* in maple (B), *Phellinus pini* in pine (C), and *Heterobasidion annosum* in pine (D). Cracks following injury and insect activity also reduce the effectiveness of compartmentalization e.g. basal cracks in oak (E) and fir (F).

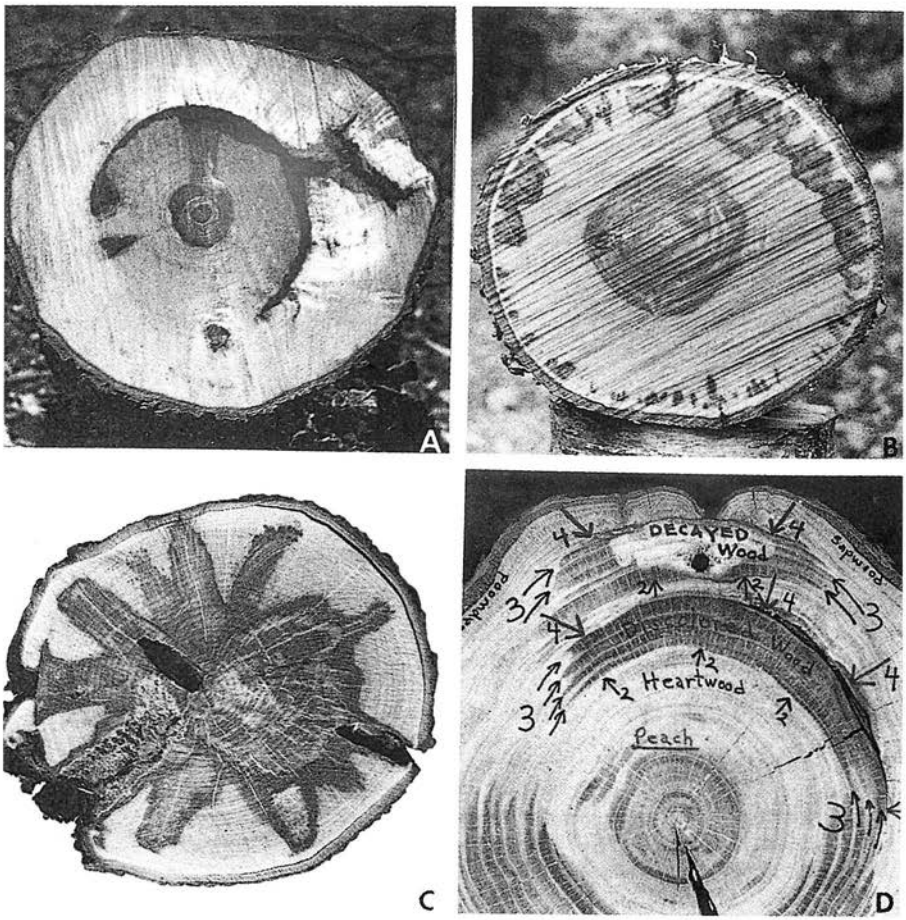

Figure 7. Insects that bore through live stemwood, such as sugar maple borer (A) and ambrosia beetle (B) can spread stemwood decay, especially in trees with declining vigor. Repeated wounding, such as improper tapping of sugar maple (C), basal injuries in orchard and plantation trees (D) and grazing, not only degrades stemwood, but shortens tree life as well.

to be critically assessed. Density is not merely the number of stems or basal area per hectare of forest, but the number and size of stems with desirable characteristics. The prudent thinning of a stand should be based on the desired mixture of tree species and the form of stems and branches. Early detection and removal of "problem trees" can greatly improve the quality of yield at the time of harvest. Trees may become problems due to poor stem form, branch architecture, poor branch shedding, wounds, etc (Shigo 1991). Avoidance of mechanical damage requires management. Repeated damage to the same stems over time

must especially be avoided as the cumulative loss can be greater than the additive effects of individual wounds. The forest manager may find it profitable to apply arboricultural treatments such as the proper pruning of branches to individual high value trees to increase the yield of knot-free stemwood (Shigo 1991).

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