

VULNERABILITY OF XYLEM TO CAVITATION AND EMBOLISM

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CONTENTS

WATER RELATIONS AND THE VULNERABLE PIPELINE.....	19
EARLY EFFORTS TO DETECT CAVITATION AND EMBOLISM	21
ACOUSTIC DETECTION OF CAVITATION.....	22
<i>Audio (Low-Frequency) Detection</i>	22
<i>Ultrasonic Detection</i>	22
<i>Physics of Sound Propagation</i>	24
HYDRAULIC DETECTION OF EMBOLISM.....	25
CAVITATION AND EMBOLISM IN NATURE.....	26
MECHANISMS OF EMBOLISM FORMATION.....	27
<i>Water Stress-Induced Embolism</i>	27
<i>Embolism Formation by Winter Freezing</i>	29
<i>Pathogen-Induced Embolism</i>	29
EMBOLISM REPAIR.....	30
VULNERABILITY OF XYLEM TO WATER STRESS-INDUCED EMBOLISM.....	31
HYDRAULIC ARCHITECTURE AND SUFFICIENCY OF TREES	33
SUMMARY.....	35

WATER RELATIONS AND THE VULNERABLE PIPELINE

The evolution of cell walls allowed the plant kingdom to solve the problem of osmoregulation in freshwater environments; confining protoplasm inside a rigid exoskeleton prevented cell rupture as a result of osmotic inflow of water. The cost of cell walls for plants was a loss of motility. In contrast, in the

animal kingdom osmoregulation involved the evolution of a vascular system that bathed most cells in isosmotic blood plasma; this avoided rigid walls and permitted cell and organismal motility. Cell walls also placed constraints on the evolution of long-distance transport systems. Tissues were too rigid to evolve a heart pump mechanism. Instead plants evolved two novel transport systems. One is a positive pressure system that moves concentrated, sugar rich sap in the phloem from leaves to growing meristems. Phloem transport uses a standing-gradient osmotic flow mechanism similar to that found in some animal excretory organs, but it is unique in that it occurs at very high pressures (up to 3 MPa) and requires two standing-gradient systems in tandem, one of which works in reverse.

The other transport system distributes water from the soil throughout the plant via the xylem. It is even more extraordinary in that it requires water in the xylem conduits to exist under negative (sub-atmospheric) pressures, typically of -1 and -2 MPa, and sometimes as low as -10 MPa. This means that water must remain liquid at pressures well below its vapor pressure. In this "metastable" state, nucleation of vaporization, or *cavitation*, must be prevented if continuity of the water column in the xylem conduits is to be maintained. Cavitation results in a primarily vapor-filled conduit that eventually fills with air. A conduit in this air-filled state is *embolized* and is not available for water conduction. Thus, plants depend for their water supply on an inherently vulnerable transport system.

Our current understanding of the mechanism of xylem transport is based on the cohesion theory usually ascribed to H. H. Dixon (18). Evaporation from cell wall surfaces in the leaf causes the air-water interface to retreat into the finely porous spaces between cellulose fibers in the wall. Capillarity (a consequence of surface tension) tends to draw the interface back up to the surface of the pores and places the mass of water behind it under negative pressure. This negative pressure is physically equivalent to a tension (a pulling force) that is transmitted to soil water via a continuous water column; any break in the column necessarily disrupts water flow.

Xylem transport can occur by this mechanism because of the special properties of water and the structure of the xylem. Hydrogen bonding promotes cohesion between water molecules and allows water to remain liquid under tension. Calculations of the theoretical tensile strength of water indicate that tensions in excess of 100 MPa would be needed to induce cavitations in the bulk phase (1, 37, 40). Xylem tissue includes a network or matrix of conduits (vessels and tracheids) with rigid walls that do not collapse when the water is under tension. Xylem conduits are water filled from inception and contain no entrapped air bubbles that could nucleate cavitation. Their walls are extremely hydrophilic, decreasing the likelihood of cavitation at the wall-water interface. If a cavitation occurs, or if a conduit is punctured, the

resulting vapor or air bubble does not expand beyond the confines of a single conduit because of the surface tension effects at pit membranes between conduits (5). Built-in pathway redundancy insures that water conduction can continue despite limited numbers of cavitations. The optimal design of the xylem argues for strong selection during evolution for resistance to cavitation.

Here we summarize our current knowledge of cavitation and embolism, and evaluate its biological significance in plants. Obviously plants have evolved to survive the threat to their water supply that cavitation imposes. One of the questions we address is how they have done so and what sacrifices were involved. We also consider under what environmental conditions cavitation occurs, and how the consequent disruption of water transport influences the water relations and ecophysiology of plants.

EARLY EFFORTS TO DETECT CAVITATION AND EMBOLISM

The cohesion theory of sap ascent did not initially meet with widespread acceptance. Although there was experimental evidence that negative pressure had to exist in the xylem (e.g. 7), many people argued that cavitation events would be all too common an occurrence if water was under tension for long periods. Paradoxically, however, the demonstration that cavitation events occur can be the most powerful proof of the cohesion theory. From the turn of the century the quest was on to determine the vulnerability of xylem to cavitation events—i.e. to determine how the number of cavitation events increases with tension in plant tissues.

The difficulty of studying the problem in the xylem itself led some to use model systems. In glass vessels, cavitations occurred at tensions of 4–20 MPa and were accompanied by an audible click (6, 18). Because vulnerability to cavitations is determined mostly by the quality of “adhesion” at the wall-water interface (74), many of the earlier attempts to measure the vulnerability of water in artificial vessels are irrelevant to the ultimate issue of when cavitations occur in plants.

In 1915 Renner (41) and Ursprung (70) were the first to watch cavitation in plant cells under the microscope; in the annulus cells of fern sporangia cavitations occur at tensions of ca. 30 MPa. More recently, Milburn (30) observed cavitations in ascospores at tensions of 1.8–7.1 MPa. Peirce in 1936 (39) may have been the first to demonstrate the occurrence of cavitation events in the xylem of plants. He used liquid nitrogen to freeze stem segments of trees in situ. He then excised the stems and while they were still frozen tried to blow air through them. He could not blow air though branches collected in early spring during times of ample water supply, but he could later in the year when transpiration was high. This suggests that some of the

xylem conduits must have been embolized prior to freezing and that therefore some cavitation events must have occurred. A similar technique has been used recently (9) to detect embolism in soybean roots.

When negative pressures in plants were actually measured with the thermocouple psychrometer (49) and the pressure bomb (46) in the 1950s and early 1960s, the cohesion theory gained wide acceptance. Given the lack of suitable quantitative studies of cavitation, plant physiologists gradually concluded that if the cohesion theory was correct, cavitation must be relatively rare. However, the validity of the cohesion theory and the commonness of cavitation events are not mutually exclusive.

ACOUSTIC DETECTION OF CAVITATION

Audio (Low-Frequency) Detection

The study of cavitation made an important step forward in 1966 when Milburn & Johnson found that it could be detected in plants by acoustic means (34). A cavitation event causes a rapid relaxation of a liquid tension that produces an acoustic emission (AE) of energy. These AEs can be detected using audio (low-frequency) acoustic transducers and amplifiers. While suspended in air, a *Ricinus* leaf with its petiole attached to an acoustic transducer produced a total of 3000 AEs while it wilted. This number is approximately the number of vessels one might expect to find in such an organ, but an exact count was not made. AE production could be stopped or slowed by adding a drop of water to the cut end of the petiole. The AE production rate could be increased or decreased by any one of several means of increasing or decreasing the rate of transpiration.

Milburn and others have reported audio-range AEs in a wide variety of species (15, 16, 31, 32, 34, 35, 71) and have collected a considerable amount of circumstantial evidence that the audio AEs are indeed correlated with cavitation events. Cavitated vessels soon become embolized and emboli are slow to dissolve (see the section on Embolism Repair, below), so we would expect that once a plant has been stressed to a xylem pressure potential (Ψ_x) of, say, -1 MPa then even if tension were partially released, few new AEs would be detected until Ψ_x drops below -1 MPa again. This appears to be confirmed by Milburn and others (31; 33, p. 165; 38). If emboli were refilled by vacuum infiltration, AE production at Ψ_x above a previous stress level was restored (31).

Ultrasonic Detection

More recently Tyree (62, 64, 65) adapted from engineering a more powerful technique for the acoustic detection of cavitations using ultrasonic frequencies typically between 50 and 1000 kHz. The techniques were well-established

methods for the detection of incipient structural failure in solids. As solids begin to fracture under tension forces, there are rapid strain relaxations in the vicinity of the cracks. The potential energy released by this strain relaxation is propagated away from the crack in the form of AEs containing a broad range of frequencies. The energy contained in the lower-audio-range frequencies (< 15 kHz) is usually higher than in the ultrasonic range (> 15 kHz), but engineers choose to work with the ultrasonic frequencies because these can be amplified selectively while lower-range frequencies that might be confounded by audio-range noise can simultaneously be filtered out. Commercial equipment already existed, or circuits could be easily fabricated, that could count individual AE events at rates up to 1000 sec^{-1} and simultaneously measure the energy and frequency range of the AEs. Automatic monitoring has obvious advantages over Milburn's method, which usually involves aural detection by an observer using earphones. Automatic detection increases the speed of counting, reduces subjectivity in threshold detection, and eliminates the boredom of listening to experiments for many hours. The ultrasonic technique also permits accurate AE detection despite low-frequency noise from various experimental manipulations of the specimen.

Circumstantial evidence comparable to that obtained by Milburn and coworkers for audio AEs relates ultrasonic AEs to cavitation events (20, 27, 38, 44, 62). The following three experiments provide particularly strong evidence for the correspondence:

1. AEs could arise from any structural failure in the liquid or solid phases of stems. When plants dehydrate the xylem cell walls are under radial compressive stress while the water is under tension. In a pressure bomb, however, radial wall compression and water tension can be independently modified. Inside a pressure bomb ultrasonic AEs arise only when xylem water tension exceeds a threshold value of a few tenths of a MPa (64). This suggests that AEs arise at the time of rapid release of tension in xylem water.

2. The ultimate effect of cavitation events in xylem conduits should be a loss of hydraulic conductance of the cavitated stems. Thus the first measurable AEs should correspond in time with the first measurable loss of hydraulic conductance (see the section below on Hydraulic Detection of Embolism). This has been demonstrated in stems of *Thuja*, *Tsuga*, and *Acer* (63). In principle any cell with walls rigid enough to resist collapse under negative pressure ought to be capable of cavitation. In conifers, where most woody cells are conducting elements, the cessation of AE activity ought to correspond in time with the loss of the last vestiges of hydraulic conductance, and this has been verified (63; Figure 1). In hardwoods, however, AE activity persists long after the loss of most of the measurable hydraulic conductance (56, 63; Figure 1), presumably because of cavitation events in wood fibers, parenchyma, and ray cells.

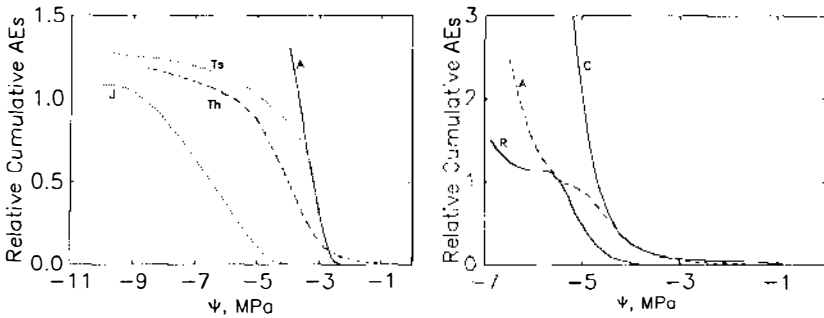


Figure 1 Vulnerability of various species to cavitation measured as relative cumulative acoustic emissions versus water potential. The left-hand graph gives data for conifers: J = *Juniperus virginiana*; Th = *Thuja occidentalis*; Ts = *Tsuga canadensis*; A = *Abies balsamea*. The right-hand graph gives data for hardwood species: R = *Rhizophora mangle*; A = *Acer saccharum*; C = *Cassipourea elliptica*.

3. If AEs correspond to cavitation events then there ought to be a one-to-one correspondence between the number of AEs counted during the dehydration of a sample and the number of rigid-walled cells in that sample, provided (a) none of the cells are initially embolized; (b) all cells emit sufficiently strong AEs; and (c) the sample is small enough so that AEs are not lost by signal attenuation before they reach the transducer. A one-to-one correspondence between AEs and the number of cells in small samples of *Thuja* and *Tsuga* wood has been demonstrated by two independent laboratories (28, 65) using quite different transducers, amplifiers, and counting equipment. Sandford & Grace (44), however, reported only about 16% of the expected AEs from *Chamaecyparis* wood. Either one or more of the above conditions were not met or their custom-built amplifier system had a much lower signal-to-noise ratio, resulting in a less efficient AE counting system.

Physics of Sound Propagation

Initially there was some hope that the energy and frequency composition of individual AEs might be used to determine the origin of AE events (31, 44, 62, 65). For example, it has been suggested that more elastic (potential) energy is stored in large cells deformed by negative sap pressures than in small cells, so AE energy may indicate the size of the cell that has cavitated. It has also been suggested that large cells may emit lower-frequency AEs when they cavitate than do small cells. Ritman & Milburn (42) have recently argued that large conduits produce only low-frequency audio AEs when they cavitate, but we do not agree with their conclusions. Unfortunately, the physics of AE propagation through plant tissues and of the interaction of an AE with a detection system is complex (69), and our level of understanding of these factors prevents reliable interpretation of underlying events.

Because the velocity of sound propagation through water-saturated wood is about the same as in dry wood and greater than in water (8, 69), it follows that sound propagation is through cellulose rather than water. The rate of signal attenuation in cellulose is a function of tissue hardness (lignification?), being approximately 1, 10, and $> 20 \text{ dB cm}^{-1}$ in hardwoods, softwoods, and herbs, respectively (69 and M. T. Tyree, unpublished). But even in samples cut very small so that attenuation ought to be negligible and so that all cell sizes are about equal, there is an inexplicably wide range of AE amplitudes (65 and M. T. Tyree, unpublished). Consequently, AE amplitude or energy cannot be used to determine what kinds of cells have cavitated.

There is also good reason to believe that the frequency composition of AEs as received by the transducer contains little relevant information, because the transducers commonly used tend to ring on after being struck by an AE, much as a bell does when struck by a clapper. AEs of known duration (e.g. a $0.5 \mu\text{sec}$ pulse caused by a pencil-lead break) will cause the transducers commonly used to ring on for $50\text{--}500 \mu\text{sec}$. In addition, the wood through which the sound travels enhances or reduces selected frequencies contained in complex AE waveforms (69).

The properties of the transducer probably dominate. For example, Sandford & Grace (44) reported beats in amplitude of AE signals; similar beats were observed by us in 1983 (M. T. Tyree, unpublished observations). Sandford & Grace suggest that these beats could represent interference of coincident AEs of slightly different frequency. In our opinion they are more likely to arise as waves bounce around inside the transducer, alternately interfering and reinforcing, especially if the transducer has closely spaced harmonics for sound propagation along different axes. Similarly, subtle increases in the average frequency of AEs from woods as they dry probably reflect the effects of water content on the mode of AE transmission through the wood rather than the change in vibrational mode at the source.

HYDRAULIC DETECTION OF EMBOLISM

Cavitation is important biologically because embolized conduits reduce the hydraulic conductivity of the xylem. Sperry (50) introduced a method that quantifies embolism by how much it reduces hydraulic conductivity. In essence, the hydraulic conductivity of the xylem of excised plant segments is measured before and after the removal of air embolisms by a high-pressure treatment (see 52). In this way the cumulative effect of all cavitations that have occurred (and have not been repaired) is measured. Although this method is destructive, it has advantages over the acoustic technique. In addition to directly assessing the impact of cavitation on water transport, it can measure cavitation caused by a variety of factors (such as winter freezing)

over long periods (e.g. months). It is well adapted to long-term studies of cavitation in the field.

CAVITATION AND EMBOLISM IN NATURE

Much effort to date has been devoted to the development of reliable methods for detecting cavitation and embolism. Most of this work has involved artificially stressed plants and has not directly addressed the important issue of how common these processes are in nature and what environmental conditions cause them to occur.

The limited information available indicates that cavitation is common in nature, occurring as a result of water stress and winter freezing. Extensive water stress-induced cavitation apparently occurs daily in some herbs and in corn. Using the acoustic method, Milburn & McLaughlin (35) and Tyree et al (66) studied cavitation in *Plantago major* and *Zea mays*, respectively. In both cases, enough AEs occurred during a single day to cause a significant disruption of water flow (perhaps by half for corn). In both species, nightly root pressure apparently served to refill embolized vessels. Using the hydraulic method, Sperry (50, 51) found that embolism occurred in the small palm *Rhapis excelsa* during natural drought. Embolism was confined to the leaves (petioles) and accounted for up to an 84% reduction in hydraulic conductivity. Circumstantial evidence indicated that embolized vessels could be refilled during a prolonged rain when xylem pressures approached atmospheric pressure. Root pressure was not observed.

The only long-term study of embolism in the field has recently been completed, using the hydraulic method, for a stand of sugar maple saplings growing in northern Vermont (53). Embolism during the growing season was confined to the main trunk and had reduced hydraulic conductance 31% by summer's end. The summer was wet; during a dry year this value could be much higher. During the winter, embolism increased until there was over 80% loss of conductance in the upper twigs, with many twigs 100% embolized. Main trunk xylem suffered 60% loss of conductance. Gradual spring recovery from these high winter levels was associated with positive xylem pressures generated in the root and stem. This phenomenon of winter embolism and spring recovery by positive root pressures has also been documented for wild grapevines (54); it may be general for many temperate woody species.

Few as these studies are, they indicate that embolism and recovery may be important processes in the water relations and ecophysiology of plants. It is reasonable to speculate that if nightly recovery did not occur in herbs, their growth and productivity would diminish. Similarly, if spring recovery from winter embolism did not occur in woody plants, the health and vigor of the

plants would suffer. The varying abilities of species either to withstand embolism or to recover from it may significantly affect the ecological distribution of some plants. As the severity of winter increases along altitude or latitude gradients, those woody plants that avoid or reverse winter embolism may be at an advantage. Future research on embolism must concentrate on its occurrence in nature and its impact on the growth and productivity of plants.

MECHANISMS OF EMBOLISM FORMATION

Water Stress–Induced Embolism

What is the mechanism linking water stress to embolism? A common hypothesis is that large conduits are more vulnerable to cavitation than small ones. This correlation comes from extensive anatomical surveys using both systematic and floristic approaches. Although some detailed interpretations have been questioned (2–4), it has been concluded that arid zone plants (hypothetically less vulnerable to cavitation) also tend to have smaller conduit sizes (10–14). Recent experimental work indicates that although this correlation exists within an individual, it does not necessarily hold among species (19, 28, 63; see the section on Vulnerability of Xylem to Water Stress–Induced Embolism, below). Thus, the mechanism of embolism formation is not directly related to conduit diameter.

Pickard (40) reviewed two possible mechanisms of cavitation: bubble formation in bulk liquid (homogeneous nucleation) and bubble formation at an interface between water and a solid (heterogeneous nucleation). There is ample theoretical and experimental evidence to indicate that homogeneous nucleation cannot occur in plants in the range of Ψ_{xs} in xylem lumina (1, 37, 40). Pickard discussed two kinds of heterogeneous nucleation that could occur at the range of Ψ_{xs} found in plants: (a) nucleation at hydrophobic cracks and (b) meniscal failure at a pore.

A submicroscopic air bubble can remain in a stable state at the base of a hydrophobic crevice. As the xylem pressure becomes progressively negative the shape of the air–water interface changes until it reaches an unstable volume and it buds off into the bulk solution, nucleating a cavitation. For a crevice of conical shape, Pickard has calculated that this will occur at -1 MPa when the radius of curvature is $0.14 \mu\text{m}$.

The walls of all xylem lumina are porous, and when the pore vents onto an air space a concave meniscus must form to balance the negative pressure in the fluid. The radius of curvature needed to sustain a negative pressure, P , is given approximately by $r = -2T/P$ where T is the surface tension of the solution. As P declines so does r , and when r drops below the radius of the pore, r_p , then an air bubble will be sucked into the lumen of the xylem conduit, nucleating a cavitation. The pore radius needed to cause nucleation at

–1 MPa is also about 0.14 μm , a value indistinguishable from that of the hydrophobic crack mechanism.

Although Pickard discounted the meniscal failure mechanism in favor of the other, Zimmermann has recently resurrected the concept (74) under the name of the “air seeding hypothesis.” There is a simple experimental method of distinguishing the two mechanisms. If the meniscal failure model is correct then the positive air pressure needed to blow air through the largest water-filled pores should be the same in magnitude but opposite in sign to the Ψ_x needed to cause cavitation. If cavitations are caused by seeding from hydrophobic cracks then there should be no such correspondence.

There is considerable evidence that water stress-induced embolism occurs by air seeding at pores in the intervessel (or intertracheid) pit membranes. Of course, this mechanism can only occur if some vessels are embolized to begin with, but this happens frequently by such prosaic events as herbivory and mechanical damage to stems and leaves. In part, the air pressure required to force air through hydrated stems longer than the longest vessel (and hence through intervessel pits) is of the same magnitude as the tension required to induce embolism in dehydrating stems (15, 54, 56). Additional evidence is that increasing the permeability of pits to air by changing the surface tension of the xylem sap, or by other means (see the section on Pathogen-Induced Embolism, below) also increases the vulnerability of the xylem to embolism (15, 54). Thus, rather than conduit diameter, it is the pit membrane pore diameter that determines a conduit's vulnerability; the larger the pore, the more vulnerable the conduit.

The fact that larger conduits also tend to be more vulnerable within a species apparently results from a correlation between conduit size and pit-membrane pore size that can be explained on a developmental basis. Pit membranes are primary walls laid down at the time of cell division. The rapid cell expansion following cell division tends to stretch and reorient the cellulose fibers in the wall, increasing the spacing between the fibers and therefore increasing the effective porosity. More primary wall is laid down during the early phases of cell expansion, and this tends to reduce the porosity. Large cells are formed in spring when water is plentiful, and this promotes higher cell turgor and more rapid cell expansion. At the same time the demand for carbohydrate reserves is also highest, so the rate of primary (and secondary) wall formation is lowest. The net result will be bigger pit-membrane pores in early spring than in summer. In summer, water is limited, causing slower cell expansion, and carbohydrate reserves are higher, permitting more rapid primary (and secondary) wall growth; thus the cells have smaller pit-membrane pores and are less vulnerable to cavitation. In addition there is a genetic component to the rates of wall formation and cell expansion. For example, cells of the same size but from different species can have different vul-

nerabilities to cavitation (see Vulnerability of Xylem to Water Stress–Induced Embolism, below).

Although the air-seeding phenomenon explains how cavitation is nucleated, it is not the complete explanation of embolism formation. Once the vessel cavitates, it is initially filled with water vapor and only a little air. Embolism occurs as the vessel becomes air filled. The time required for a vessel to embolize fully following cavitation is likely to be less than 10^3 sec. The justification for this is as follows: Air will diffuse from surrounding tissue and come out of solution in the water vapor void. From air solubility tables it can be calculated that an annulus of water about 1 mm thick will contain about 14 times as much air as needed to fill the void contained in a 20- μ m-diameter vessel in the center of the annulus. From a special solution of Fick's law it can be shown that the average time, t , required for an air molecule to diffuse a distance, x , is given by $t = x^2/2D$ where D = the coefficient of diffusion of air molecules in water (about 2×10^{-9} m² sec⁻¹). As the air pressure builds up in the void some gas molecules will diffuse out while others move back in. But after about 4 times the time it takes air molecules to diffuse 1 mm, the void ought to be near equilibrium with the air in surrounding tissue; using the above value of D and $x = 10^{-3}$ m yields $4t = 10^3$ sec.

Embolism Formation by Winter Freezing

The winter embolism observed in sugar maple (53) and grapevine (54) is most easily explained as a result of freeze-thaw cycles; several studies have shown that when xylem is frozen while under tension, extensive embolism develops after thaw as air bubbles forced out of solution during freezing expand and nucleate cavitation (45, 47, 72). A curious exception occurs in certain gymnosperms (24, 58).

Another explanation is that embolism is formed by sublimation of ice from frozen vessels. This would lead to large air bubbles on thawing that would remain stable regardless of whether tension was immediately present. This mechanism may have been most responsible for the embolism observed in the sugar maple study, because it occurred primarily during very cold (-25 to -30°C) and sunny weather when the main stems probably remained frozen. In addition, the embolism was localized to the south sides of the trunks exposed to the sun where sublimation would be enhanced (53). In all likelihood, both the sublimation and the freeze-thaw mechanism play a role.

Pathogen-Induced Embolism

The role of embolism in vascular disease has not received much attention. Although it has been known for some time that vascular diseases induce water stress in their host by reducing the hydraulic conductivity of the xylem (17), embolism as a cause for this has been virtually ignored. The blockage has

generally been attributed to vascular occlusion by material or structures of pathogen or host origin. In the one study that has addressed the role of embolism, evidence was found that it preceded any occlusion of vessels by other means in Dutch Elm disease (36).

Assuming that the basic process of air seeding would be responsible for pathogen-induced embolism, we can speculate on the one hand that it is caused simply by increased water stress induced by the pathogen. Water stress could arise from modification of stomatal behavior (as in fusicoccin diseases, 59), limited occlusion of the xylem, or interference with root uptake. On the other hand, embolism could be caused by a lowering of the threshold for air seeding. This could happen through pathogen-induced changes in sap chemistry. For instance, compounds could be produced that lower the surface tension of the sap. Millimolar concentrations of oxalic acid drastically reduce the air seeding threshold in sugar maple (53) and balsam fir (J. S. Sperry and M. T. Tyree, unpublished); this compound is produced by many pathogenic fungi (57).

EMBOLISM REPAIR

Assuming that the sometimes drastic reduction of xylem transport caused by embolism poses a serious problem for the continued growth of a plant, the embolism repair mechanisms that exist are critical to plant health. The dissolving of air in embolized vessels requires that the xylem pressure must return to positive values or at most a pressure only slightly below atmospheric. Air bubbles are inherently unstable in water at atmospheric pressure even if the water is already saturated with air, provided the body of water is itself in contact with air. Surface tension puts the air bubble in such a body of water under pressure. This pressure equals $2T/r$, where T = the surface tension and r is the radius of the bubble. If r is in microns then this pressure in kilopascals is $140/r$; a bubble about the radius of a vessel ($10\ \mu\text{m}$) thus contains air at 14 kPa above atmospheric pressure. Because the solubility of air in water depends on the pressure of the air at the air-water interface, this air will dissolve in the water. Bubbles contained in *Sphagnum* hyalocyst cells ($r \sim 10\ \mu\text{m}$) have been observed under the microscope to dissolve in < 1 min (29). Bubbles in glass capillary tubes (which can dissolve only from their ends until they are smaller than the glass tube) can dissolve in 10 min to 100 hr for bubble radii of 30 and $375\ \mu\text{m}$ (21). Bubbles under positive pressure will no doubt dissolve at much faster rates.

As previously mentioned, root pressure generates positive xylem pressure that helps to repair embolism. This would be particularly effective for repairing embolism in herbaceous plants. How embolism might be reversed in tall plants is not so clear. Perhaps larger root pressures occur during spring than

previously suspected. Springtime reversal of embolism has been documented in maple trees (53), but this species is capable of producing stem pressure as part of a springtime sap flow mechanism unique to the genus (60). Spring reversal of embolism in wild grape is driven by rather large spring root pressures (> 0.1 MPa; 54). In grape the vessel and pit membranes are dry during winter, and vessels are very long (several meters). While in the dry state, root pressure drives up the column of water in these long vessels from the ground and literally expels air through the dry pit membranes and out of the vine at leaf and inflorescence scars. Under laboratory conditions a pressure of just a few kilopascals restored about half the hydraulic capacity of dry grape stems. The remaining bubbles apparently dissolve over the next day or so well in advance of leaf flush. Once the pit membranes are wet they are capable of preventing air seeding at xylem pressures below -1 MPa (54).

VULNERABILITY OF XYLEM TO WATER STRESS-INDUCED EMBOLISM

Of ultimate significance to the cohesion theory of sap ascent and to a general understanding of plant water relations is some measure of the vulnerability of the xylem to water stress-induced embolism. The emphasis in the following studies is not directly on water stress-induced embolism in the field, but on the theoretical limits imposed on water transport by embolism. These studies help answer questions concerning the evolution of the xylem and how it may have constrained the evolution of plant water relations in general.

Vulnerability can be defined by the relationship between Ψ_x and embolism; this is termed a "vulnerability curve." In Figure 1 acoustic vulnerability curves are plotted as relative cumulative AE count versus Ψ_x . Relative cumulative counts have to be computed in order to compare between replications and species. Because of the high AE attenuation rate the maximum number of detectable AEs varies with sample geometry within replications in the same species. Because of the dependence of attenuation rate on wood hardness the maximum number of detectable AEs also varies between species, even if the geometries are identical. All the curves in Figure 1 have been normalized such that a relative count of 1 occurs at the Ψ_x when 95% loss of hydraulic conductance is reached, as determined by parallel experiments.

In conifers (left-hand panel) AEs stop at about the time of 95% loss of maximum hydraulic conductivity. One expects this because most cells in conifer wood are tracheids. In vessel-bearing trees (right-hand panel) many more AEs occur after loss of 95% conductance than before. This is probably the result of cavitation events in the numerous wood fibers, ray cells, and parenchyma cells found in vessel-bearing trees.

Vulnerability curves expressed as percentage loss of hydraulic conductance

versus Ψ_x are shown in Figure 2 for selected conifers (left-hand panel) and hardwoods (right-hand panel). Loss of hydraulic conductance by embolism is much more meaningful in terms of plant water relations than knowing how many cavitations (AEs) have occurred. This is because AEs can occur in many cells that contribute little to the capacity of plants to conduct water from roots to leaves.

Examination of Figures 1 and 2 shows first that the cohesion theory clearly survives the test of cavitation. Vulnerability to cavitations will not totally incapacitate xylem tissue in the normal range of water potentials experienced by all tree species examined to date (56, 63). However, this is not the same as saying water stress-induced embolism is not a significant threat to plant survival. We argue in the last section of this review that xylem is a highly vulnerable pipeline and that woody plants operate at their theoretical limit of hydraulic sufficiency.

Second, vulnerability is not correlated with whether a conduit is a hardwood vessel or a softwood tracheid; nor does it correlate with conduit diameter. Although the conifers shown all have tracheids of about the same size (J. S. Sperry and M. T. Tyree, unpublished), there are significant species variations in vulnerability curves; *Juniperus* is the least vulnerable and *Abies* is the most vulnerable. Among the hardwoods there are also significant species variations in vulnerability. *Cassipourea* is the most vulnerable and *Rhizophora* is the least vulnerable, even though the vessel diameters of the two species are the same (56). As explained in the section on Mechanisms of Embolism Formation, above, these differences in vulnerability result strictly from differences in air seeding at interconduit pit membranes (55, 56; J. S. Sperry and M. T. Tyree, unpublished).

In general, the vulnerability of a species correlates with the xylem pressures it experiences in nature. For instance, the mangrove *Rhizophora* experiences

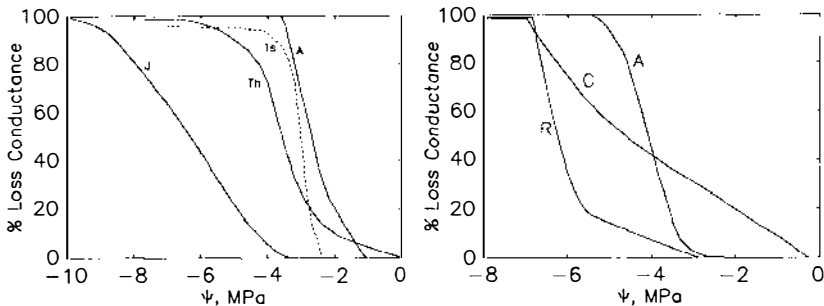


Figure 2 Vulnerability of various species to embolism measured as the percentage loss of hydraulic conductance versus water potential. Species in right- and left-hand curves as in Figure 1.

Ψ_x at least as low as -4.0 MPa and is much less vulnerable than its inland relative *Cassipourea*, which seldom has Ψ_x lower than -1.5 MPa (56). There are trade-offs between vulnerability and hydraulic sufficiency of stem tissue that must relate to the ecology of species. On the one hand, smaller pores confer resistance to cavitation; on the other hand, they may reduce the hydraulic conductivity of the xylem. Thus, the safer the xylem, the less efficient it may be in water conduction.

HYDRAULIC ARCHITECTURE AND SUFFICIENCY OF TREES

Another aspect of the vulnerability of trees to embolism is the degree of redundancy built into the xylem. In other words, how much embolism is too much? Valuable insights can be drawn from recent models of water transport in whole trees based on the vulnerability of their xylem to embolism, and on their “hydraulic architecture” (68).

Hydraulic architecture is a term coined by Zimmerman (73) to describe how the hydraulic conductivity of the xylem in various parts of a tree is related to the leaf area it must supply. This is quantified for a segment of the tree by the Leaf Specific Conductivity (LSC), defined as the absolute conductivity of the segment (k_h = flow rate per unit pressure gradient) divided by the leaf area supplied by the segment (73). The principle utility of this definition of LSC is that it allows a quick estimate of pressure gradients in stems. If the evaporative flux, E , is about the same throughout a tree, then the xylem pressure gradient, dP/dx , in any branch can be estimated from: $dP/dx = E/LSC$.

Generally the LSC for minor branches is 10–1000 times lower than for major branches (22, 23, 51, 67, 73). Thus most of the water potential drop in the xylem occurs in the small branches, twigs, and petioles. In these axes, water potential drops are 10–1000 times greater per unit length than in the main trunk. Prior to Zimmermann’s classic work, Hellkvist et al (25) had found, in Sitka spruce, that the main resistance to water transport from soil to leaves resides in the branches. From their data we calculate that on the order of 65% of the total water potential drop occurs in the xylem; roots contribute 20% and leaves 14%. These results explain why trees are able to survive severe, but localized, damage to their trunks—e.g. a double saw-cut (48), or 50% blockage by fire restricted to a few feet of trunk (26). Although such damage to trunks causes a decreased conductivity, it does not cause much of an overall drop in water potential because trunk LSC is very high to begin with, and the damage is localized to a short distance.

Embolism can occur throughout the tree, and by decreasing xylem conductivity over long distances it can substantially influence water status. Accord-

ing to Zimmermann's "segmentation hypothesis" (74), in times of water stress, embolism will preferentially occur in minor branches where LSCs are lowest and consequently xylem tensions are greatest. The effects of the embolism resulting from these cavitation events could drastically worsen the water balance of the tree. As a consequence, peripheral parts of the tree would be sacrificed and the trunk and main branches remain functional. This hypothesis has received support from work on palms (50, 51) and on a woody shrub (43).

We have recently obtained support for the segmentation hypothesis from modeling studies based on woody plants from diverse taxa and environments (61, 68). In this work we examine the "hydraulic sufficiency" of branches of trees to supply water to their leaves. The hydraulic sufficiency depends on both the hydraulic architecture of the tree and the vulnerability of its xylem to water stress-induced embolism. The model is specifically focused on the conditions required to generate "runaway embolism," whereby the blockage of xylem conduits through embolism leads to reduced hydraulic conductance requiring increased tension in the remaining vessel to maintain water flow to leaves and generating more embolism and tension in a vicious circle.

We examined the water relations, hydraulic architecture, and xylem vulnerability of four diverse species; two were tropical (*Rhizophora mangle*, a mangrove, and *Cassipourea elliptica*, a moist forest relative) and two were temperate (*Acer saccharum* and *Thuja occidentalis*). There were great differences among these species in hydraulic architectures, maximum transpiration rates, specific hydraulic conductances of stem tissue, and water relations. Despite these differences the model predicted for all species that: 1. embolism occurs more in minor than in major branches (thus supporting the segmentation hypothesis); 2. xylem tensions could lead to 5–30% loss of transport capacity without adverse effects; 3. if embolism caused more than 5–30% loss of transport capacity, then runaway embolism occurs leading to catastrophic xylem dysfunction (blockage) in a patchwork fashion throughout the crown; and 4. after catastrophic failure of selected minor branches, an improved water balance (less negative Ψ) in surviving minor branches results from dead-shoot leaf loss.

Examples of the predictions of the model are shown in Figure 3. In the vertical axis we have plotted average Ψ of minor shoots bearing leaves in a large branch system. The horizontal axis is the average evaporative flux, E , from leaves required to produce the computed Ψ . The range of Ψ s predicted agrees with field observations. The maximum E observed in the field for each species is indicated (*) near the horizontal axis. Based on the minimum observed Ψ and the vulnerability curves in Figure 2 one might wrongly conclude that xylem embolism might never exceed 5–30%. This model predicts that if stomatal regulation did not limit the maximum E then runaway

embolism would occur, leading to shoot dieback. The improved water balance of surviving shoots can be seen in the gradual increase of Ψ versus evaporative flux, E , in the dashed line when E exceeds the threshold value marked (*). We conclude that these species are operating at the limit of hydraulic sufficiency.

The implication of these results is that xylem structure and vulnerability to embolism place important constraints on the water relations, morphology, and physiology of trees. Specifically the model shows that stomatal regulation and xylem physiology must function and evolve as an integrated unit in order to prevent catastrophic dysfunction. Up until now it has been presumed that the primary role of stomatal regulation was to prevent desiccation damage to the biochemical machinery of the photosynthetic system. It is now clear that another important role of stomatal regulation is to prevent catastrophic xylem embolism while pressing water conduction through stems to their theoretical limit of hydraulic sufficiency. Trees must evolve mechanisms to keep an appropriate balance for carbon allocation between leaves, which increase evaporative demand, and stems, which supply the demand for water evaporated from the leaves.

SUMMARY

Cavitation and embolism occur in plants in response to water and freezing stress. Water stress causes embolism by air seeding at pit membranes between xylem conduits. Freezing of xylem sap forces air out of solution that nucleates cavitation during subsequent thaw, and embolisms can form in frozen vessels

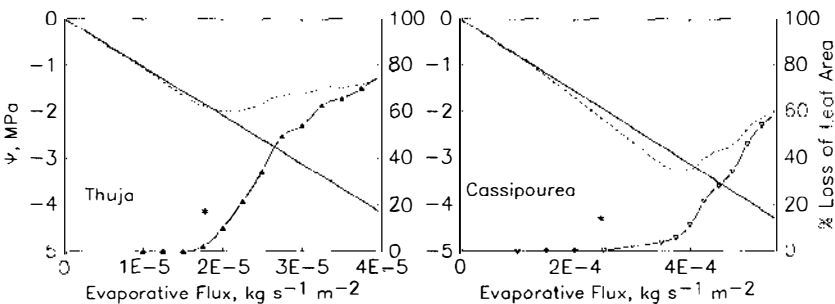


Figure 3 Results of a model that calculates the dynamics between water stress development in two species of trees and the loss of hydraulic conductance of minor stems due to embolism. Solid line: average water potential (Ψ) of all minor shoots versus evaporation rate assuming no embolism occurs. Dashed line: average Ψ of all surviving minor shoots assuming loss of conductance by embolism. When evaporation rate exceeds the maximum observed in each species under field conditions (given by "*" near the x-axis), runaway embolism occurs causing loss of leaf area (curves with triangles) in a patchwork fashion. See text for more details.

by sublimation. Repair of embolism by positive xylem pressures has been observed on a nightly basis in herbs, and during the spring in trees and woody vines. Modeling studies imply that vulnerability to water stress-induced embolism has played a major role in the evolution of the xylem and of stomatal regulation. It appears that embolism may be an important factor in the ecophysiology of some plants; specifically in their tolerance of low water availability and freezing temperatures. However, we cannot know the full biological significance of embolism until we know more about its occurrence under natural conditions and how much it reduces growth and productivity.

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