It is typically assumed that the vascular cambium (or any tissue) is killed at a threshold temperature (e.g., 60°C). In fact, the vascular cambium is killed at some combination of exposure time and temperature as surface flames pass a tree and heat transfer into the stem causes a rise and fall of tissue temperatures. Here, I use two temperature-dependent rate-process models to describe data on tissue impairment at elevated temperatures. At fixed temperatures, one of the models describes a simple negative-exponential decline through time in tissue viability (no-lag model) and the other includes a mechanism by which a lag, commonly seen in data, occurs before the onset of rapid rates of tissue impairment (lag model). The temperature-dependence of tissue-impairment rates is exponential and the models assume that temperatures are high enough and last for a short enough time that cellular acclimation and repair processes play an insignificant role. Thus, the models are appropriate for the rapid heating of the vascular cambium during forest fires. I apply the models to data on mortality within populations of aspen (*Populus tremuloides*), Englemann spruce (*Picea englemannii*), Douglas-fir (*Pseudotsuga menziesii*), and lodgepole pine (*Pinus contorta*) live bark cells showing that differences among species are small (a result also reflected in other studies). As a demonstration of how the thermal tolerance models can be used, I show how an exponential dependence between tissue temperatures and rates of impairment combined with the relatively rapid rise and fall of stem vascular cambium temperatures during forest fires cause an inward propagation of a tissue-necrosis threshold in tree stems. A staining technique to quantify the threshold is described. Given appropriate thermal tolerance parameters, the modeling approach described here can be applied to any tissue heated relatively rapidly in fires and is well suited for coupling with a stem heating model for predicting stem death as described by Jones and Webb (Stem Mortality in Surface Fires: Part III, Linking Stem Heating with Tissue Response for Planning Prescribed Burns).