Observations on the California vine disease

Ormond Butler

(with plates 1–5)

I

Introduction

In the height of the summer of 1886, the grape-vines in Los Angeles and bordering counties, in California, began to show very marked and alarming symptoms of disease. The vineyards in the environs of Anaheim, then a flourishing viticultural center, were the most seriously affected. In fact, Anaheim is generally considered the birthplace of the scourge that had thus suddenly appeared and was, within the next few years, to devastate Los Angeles and Orange counties. The Anaheim disease, as the new malady was called before it received the name of California vine disease, gradually decreased in violence in each succeeding year after 1886, and, today, one may say that it is little feared by the growers. To be sure, Anaheim is no longer a viticultural center, but the vine is nevertheless extensively grown in places where the malady existed in 1886, and there are vineyards in Los Angeles County that have passed through the years of the epidemic without serious loss.

But if the California vine disease is a malady of little economic importance in 1910, it was a very grave problem to face in 1886. In August of that year F. W. Morse began an investigation of the malady, under the direction of the director of the Agricultural Experiment Station, University of California, and published, a few months later, the first description that we have of the California vine disease.

From this author we gather: that the disease first became apparent by a failure of the vines to bud, or, as occurred more generally, in a noticeable backwardness in starting, which often extended to six weeks, the foliage of the vines thus late in leafing

out soon becoming blighted; that the disease might affect the vine at any time from early spring until the autumn; that it was reputed to be, in some cases, very rapid in its action, affecting an apparently healthy vine in the space of a day. Morse himself had observed no such rapid changes, the changes noticed being only "such as one may frequently note in any vineyard where sudden variations in meteorological conditions occur, and sunburn or scald follows";[1] predominantly, however, the progression of the disease was gradual.

The malady was characterized, to continue our quotations, by a "drying up, and apparent burning of the foliage at all times, up to the time of ripening of the fruit. . . .[2] The leaves in general have the very decided appearance of sunburn. When the vineyard is seen as a whole the foliage looks withered, leaves partially dried and wrinkled, and large parts of them have become red, the affected part of each leaf assuming no regular shape or particular position. The canes having the most upright growth and those most exposed have the leaves the worst affected; low growth is less troubled."[3]

"Among the several varieties the Mission is, undoubtedly, the worst affected. . . . Other varieties such as Golden Chasselas, Sultana, Semillon and Sauvignon are affected to a greater or less extent, and in about the order named."[4]

Morse believed that the mortality of the vines was "due to more or less accidental and local peculiarities of climate, soil, moisture conditions, etc."[5]

In a report[6] submitted to the State Board of Viticultural Commissioners in 1890, Dowlen described the California vine disease with care, and added some remarks on the anatomy of the canes of affected vines which I shall have occasion to quote later. This

[1] Morse, F. W. Report of an examination into the phenomena and causes of a supposed vine disease in Los Angeles County. Report of the viticultural work during the seasons 1885 and 1886, College of Agriculture, University of California, 176, 177. 1886.

[2] *Loc. cit.*, 176.

[3] *Loc. cit.*, 177.

[4] *Loc. cit.*, 178.

[5] *Loc. cit.*, 183, 184.

[6] Report California Viticultural Commissioners for 1889–90, 57 et seq.

author did not advance any opinion as to the nature of the disease.

Pierce's important memoir[1] on the California vine disease appeared in 1892 and contains many facts and figures of interest. In describing the disease he says: "In the majority of diseased vines, although not in all varieties, the leaf presents distinct characters. They may be mentioned as *constitutional* and *localized* characters."[2]

The general, or constitutional effects are "due to a failure in the formation of chlorophyll, or degeneration of that once properly formed, in those portions of the leaf supplied by the finer spiral vessels. These general effects are found to some extent in nearly all varieties. . . .

"*The localized effects upon the leaf* are most clearly seen in the white varieties, and are especially distinct in the Muscat." The leaves become more or less covered with yellow spots "in that part of the parenchyma supplied with the finer spiral vessels. These spots are often well defined, the outline being very sharp and distinct. . . ."[3]

"The cane usually becomes bare of leaves before the wood is properly ripened. The end of the cane, being last to ripen, is most immature, and soon after the leaves fall the unripened parts turn black and become dry. This progresses more rapidly and the dying is more complete when the leaves drop early. . . . The peculiar and unequal ripening of the cane is very marked."[4]

In the roots "among the first signs of disease is a discoloration and shrinkage in diameter of the finer root fibers, the root hairs and cap. This progresses until the tissue begins to decay. . . . The root, at last becoming wholly rotted, passes into a brown, loose, amorphous mass."[5]

The fruit of diseased vines is markedly affected. "If the first attack of the disease be violent the grape will sometimes fall from

[1] Pierce, N. B. The California vine disease. U. S. Dept. Agr., Div. Veg. Path. Bull. 2. 1892.

[2] *Loc. cit.*, 41.

[3] *Loc. cit.*, 42.

[4] *Loc. cit.*, 46.

[5] *Loc. cit.*, 51 et seq.

the bunch. This dropping of the fruit is not so strongly marked and is less important than the drying of the berry upon the bunch. . . . In some cases the growth of the berry is retarded. . . . The drying of the fruit upon the vine is a leading effect of the disease and is very general in all varieties and under all conditions."[1]

Pierce states that the California vine disease has a period of incubation. He says "In the affected district it is common to find a vineyard of one variety looking perfectly healthy and the adjoining vineyard of another variety badly affected or killed by the disease. It may be that the vines are of the same age and upon like soil. When we see a sharp line of this kind," he continues, "drawn between varieties it is folly to say that the disease has affected one and not the other, for it may occur that the dying variety is found on all sides of the living one. It must be admitted, then, that the disease has produced its effect upon vines not yet showing those effects." Again, "it is also common to find a few Mission vines scattered here and there in vineyards of other varieties, they having been planted through oversight and the mixing of cuttings. Where this has been the case, these Mission vines have been singled out and killed by the disease as surely as if they were by themselves in adjoining vineyards. . . . The truth is, that all the vines have felt the same influence of the disease, but on account of difference in hardiness some show this influence earlier than others."[2] Another, and more striking feature of the malady is the overbearing of the vines "while the disease is incubating." But overbearing is not a constant symptom. Pierce notes the fact, and at the same time remarks: "That overproduction has not always been noticed is but negative evidence, and its well-attested occurrence in a reasonable number of cases is of more value than much negative evidence."[3]

The California vine disease appears also to be transmitted in cuttings.

Pierce does not come to any conclusion regarding the nature of the disease.

---

[1] *Loc. cit.*, 53.

[2] *Loc. cit.*, 57.

[3] *Loc. cit.*, 58.

In 1892 Viala and Sauvageau also published a note on the California vine disease in the Comptes Rendus, and a memoir on the Brunissure and the California vine disease in the Journal de Botanique, which was republished later in the Annales de l'École Nationale d'Agriculture de Montpellier. These authors described the anatomical features of the California vine disease very accurately, as I shall have occasion to point out later, and concluded that this malady was produced by a myxomycete very similar to the organism causing Brunissure (*Plasmodiophora Vitis*), but as it was infinitely more destructive they gave it specific rank under the name *Plasmodiophora californica*.

In recent years Ravaz[1] has advanced the opinion that the California vine disease and the Brunissure are one and the same malady, both being due to overbearing, but the description I give of the latter disease in chapter III will show that this view is incorrect. If we modify, however, Ravaz's opinion to read that the Brunissure killed a number of the vines during the epidemic of 1886 in southern California, I think that it can then be supported on grounds. But I shall not attempt to advance the arguments in favor of this modification of Ravaz's view; it would lead me too far to do so adequately, and a brief presentation is, in the nature of the subject, impossible.

II

Description of the California vine disease

A. Morphology

The California vine disease affects primarily the leaves, fruit, shoots, and canes. The arms and trunk reveal nothing of diagnostic value, and an examination of the roots is fruitful in contradictory results.

The leaves, fruit, shoots, and canes show symptoms that are constant from one variety, or species, of vine to another, the observable discrepancies being due to the fact that the virulence

[1] Ravaz, L. Influence de la surproduction sur la végétation de la vigne. Ann. École Agric. Montpellier, II. 6: 5–41. 1906.

Remarques sur le dépérissement de quelques vignes en Tunisie et en France. Progrès Agricole 44: 41–50, 71–73. 1905.

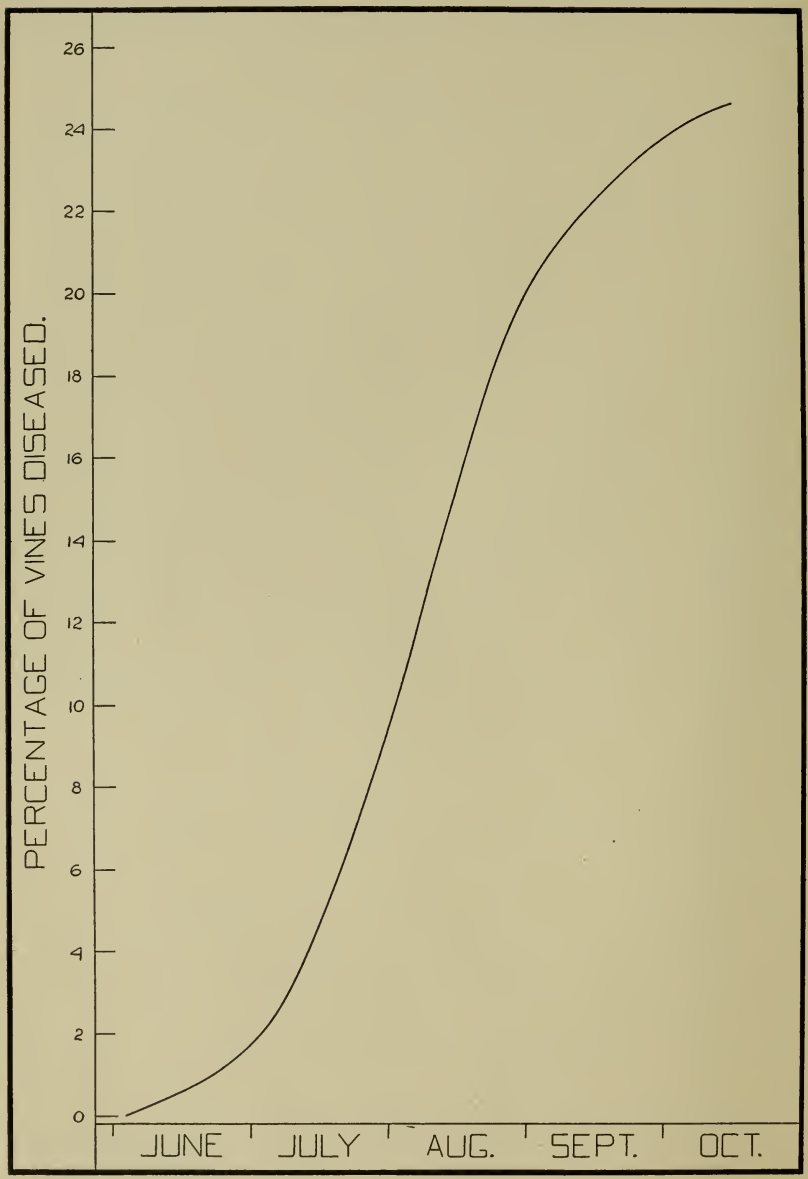


FIGURE 1.—Curve showing the general course of development of the California vine disease.

of the disease is subject to marked variations. These variations are both individual and inter-individual. The symptomatic differences between two plants are often found in replica on the shoots of a single vine. The disease, in fact, appears to work, to use a colloquialism, without rhyme or reason. It may affect a single shoot, or several shoots; one side only, or the whole of a vine. One vine may show all its characteristics, manifold though they be, and another, a part, perhaps only one, of them. The disease may never proceed beyond the first stages, a not uncommon feature, or it may develop slowly until all its characters are patent to the most casual observer; more often, however, its development is rapid.

The California vine disease, though it may appear at any time of the growing season, usually develops to a noticeable extent only when the vines are ripening their fruit, that is, at the most critical period of their yearly development. This fact is brought out very clearly in the accompanying graphic, which may be considered representative when 25 per cent. or more of the vines in a vineyard are diseased. When less than twenty-five vines in one hundred are affected, the curve may become either flatter or sharper: flatter, if the malady develops gradually throughout the growing season; sharper, if the disease develops very rapidly. As soon, however, as one quarter of the vines become diseased it tends to assume the form shown in the graphic.

The development of the disease is sporadic; and the malady always develops in the following general manner whether few or many vines are affected:

A certain number of scattering vines show the ominous symptoms; then more vines, unrelated positionally to the others, become affected, and thus, by the continued addition of diseased individuals, a vineyard becomes, to a greater or less extent, infested. There is no such thing, however, as spreading by contiguity, no "oil spots." Such a thing as a diseased center, using the term in its narrower sense, does not exist, nor is there in the different varieties of the grape-vine, if we confine ourselves to the Viniferae, any marked difference in predisposition. One cannot predict, as in the case of the powdery mildew, for instance,

that the disease will first appear in this or that variety. We only know that at *A* the disease may affect almost exclusively *x*; at *B* that *y* is worse than *x*; and at *C* that *y*, *u* and *v* are equally, or almost equally affected. But such observations as these are only good for the season in which they are made; they may not be true for the year before, nor yet in the year to come. In a word, the past is no criterion of the future: this is true whether the vines become diseased suddenly or progressively, and I mean by this latter term an intermittent addition rather than a natural sequence of symptoms.

I have said that the past is no criterion of the future. This statement, while true for the individual vine, is not necessarily true for the vineyard as a whole. When a large percentage of the vines in a vineyard are affected, not suddenly[1] but progressively, it is not infrequent that the disease reappears the following years and becomes chronic, as it were, in a large number of vines, though it is impossible to select with certainty the vines in which it will be so, and gradually, in bad cases, so weakens them that they die.

From the behavior of diseased vines, both as individuals and collectively, it appears that the California vine disease is sufficiently variable in its mode of action to be considered as possessed of two forms, differing from one another in immediate virulence. The one we may call apoplectic, from the suddenness of its action; the other chronic, from its lesser destructiveness and predisposition to recurrence. These two forms, identical symptomatically, conduce, the one rapidly, the other slowly, to the same end, the death of the vine. It is, therefore, unnecessary, in describing the disease, to state specifically which of these two forms one has in mind, the line of demarcation between them being, to all intents and purposes, but a line in point of time.

This being so, the following description determines the California vine disease, provided the characters of the affected organs are considered in conjunction with one another and not as separate and sufficient entities; for, and it cannot be too strongly urged, the disease we are considering cannot be surely and safely

[1] When a vine is affected suddenly in all its shoots it frequently dies of chlorosis the following season.

identified except in the "all together." Concretely: a vine showing but a *single* symptom of the California vine disease is, *in itself,* a doubtful subject.

Symptomatology

(*a*) LEAVES.—The young leaves—on upper third of shoots—generally show particular characters only in the case of the chronic form of the California vine disease. When a vine is affected apoplectically they either remain normal or assume the characters common to the older and adult leaves or, when the shoots are defoliated without death ensuing, form a healthy plume, as it were, at their tips. In the apoplectic form the young leaves are not indicative, but in the chronic form they are very often premonitory symptoms; the vine first showing them may become no further diseased, but it is practically certain that other vines in the vineyard, if they are all of one variety and age, will, and not only lightly, but severely. The characters shown by the young leaves are, in the case of the chronic form, then of considerable interest.

They are:

Case 1.—The leaves become pale in the intervenium, growth ceases at the periphery—sometimes also between the veins—and death ensues. The tissues not immediately affected, not having reached complete development, continue growing, the leaves becoming paler in color, more or less convex, and, according to the amount of dead tissue other than peripheral, variously distorted. (PLATE 1, FIGURES 1, 2, 3, 4.)

Case 2.—The leaves, leaves more developed than those just described but of the same coloring, do not become convex as a whole, but only in one or both wings of the petiolary sinus. This distortion is accompanied by a sinking of the tissues between the venation, and subsequent death.

In older leaves the symptoms of the disease are variable and cannot be accounted for by their position in regard to other diseased leaves. Those leaves that are still in fairly active growth may show the characters common to the young leaves described in Case 2. In other cases, and coincident with the furrowing and death of the wings of the petiolary sinus, there appear, to a

greater or less extent, between the veins of the remainder of the leaf-blade, suffused yellow or red spots, which, when their centers die, have the appearance of reddish brown maculations surrounded with aureolae of red or yellow, as the case may be.[1] (PLATE 2, FIGURE 1.) If instead of, or coexisting with, the spotting of the leaf we have a reddening or yellowing of the intervenium, the dead tissue forms strips. Marginal discoloration and death may occur, as in the adult leaves, but is not so frequent.

In fully developed leaves the leaf-blade is not deformed. The tissue between the veins, and the margin, also, very largely, becomes yellow or red, the discolored areas dying in time, the dead tissue assuming a color which ranges from *feuille-morte* to gray, according as the death has been rapid or slow. Instead, however, of beginning as a general discoloration of the intervenium, the disease may first appear as suffused greenish yellow spots, which, enlarging and becoming more definite in outline, often merge together, forming large maculations and stripes. These maculations and stripes may die to the edge of the healthy tissue itself, but are more often surrounded by aureolae, which may be red, red and yellow, or yellow alone. (PLATE 1; PLATE 2.)

The leaves near the base of the shoots sometimes show a slight variation from the characters just described. As soon as the spots appear between the veins, enlarge, and form stripes, the remainder of the parenchyma becomes chlorotic. Death in the diseased areas proceeds slowly and, when accomplished, the dead parts being soft and crumbly, the leaf is beaten by the winds into deeply incised fragments which hang together around the petiole.

Diseased leaves—this remark is generally applicable—fall sooner or later with, or without, their petioles. The fall of the leaf prior to that of the petiole occurs, so far as I have been able to ascertain, only when the intervenium becomes diseased immediately around it. The death of the parenchyma then involves the death of the apex of the leaf-stalk, and the blade becomes severed from its support.

[1] In the varieties of the grape-vine producing white fruit, the aureolæ are always yellow; but in those bearing colored fruit they may be both yellow and red on the same leaf, the predominant color varying with the variety.

(*b*) FRUIT.—The fruit may be affected at any time from setting to maturation. If the fruit is affected just after setting the whole bunch withers and falls away; if attacked somewhat later, on passing the hand over a diseased bunch, the berries will come away often with, not infrequently without, their pedicels—they may even fall of themselves. As the fruit grows older it does not fall, but may shrivel or, if nearing maturation, ripen imperfectly. This shriveling and imperfect maturation of the fruit is a feature of the California vine disease. "The drying of the fruit upon the vine," says Pierce, "is a leading effect of the disease and is very general in all varieties and under all conditions."[1]

(*c*) SHOOTS.—The effect of the disease on the shoots depends on the amount and suddenness of the defoliation, which, itself, is a measure of the quality of the attack. If the vine is affected apoplectically, the life of the shoots, or of the canes, will be more endangered than if it be affected with the chronic form of the California vine disease. The season of the year at which the vines are affected is also a factor of some importance. Vines that have suffered even complete apical defoliation on one, or several, of their shoots in early summer, that is while they are still growing vigorously, may not have them visibly damaged. This is evidenced by the fact that such defoliated shoots continue to elongate, after a period of rest, and throw out axillary foliage.

In the height of summer, and at maturation, defoliation brought about by either form of the disease is more serious. The growth of the vines has then normally ceased and their recuperative powers are low. In July, and to some extent, also, in August, defoliation is followed by a progressive dying of the shoots, the amount of death in each shoot being, as it were, a register of its defoliation. When the disease affects the vines after lignification has set in, and during maturation, the fall of the foliage leaves the canes very imperfectly formed. The shoots, instead of maturing properly, remain, to a greater or less extent, green. One side of a shoot will be mature, the other not. Maturation may have proceeded normally at the base, and be

[1] *Loc. cit.*, 53.

very disordered further up. Or again, lignification may be imperfect only around a few nodes, the internodes showing apparently normal maturation, and conversely.

If one cuts a smooth cross-section of a shoot or cane that is bearing diseased foliage, or has been defoliated, he will notice that the wood is slightly discolored. The pith is best seen in longitudinal section; it will be found discolored more or less discontinuously, or turned completely brown, if the foliage has fallen.

(*d*) SPURS, ARMS, AND TRUNK.—Externally, the spurs, arms, and trunk have the same appearance in diseased as in healthy vines. This is no longer true when cross-sections are examined.

In the spurs the woody tissue of the previous year is discolored and, often, more especially near the edge, zoned with darker lines. In the arms the zones become less marked, and disappear before reaching the trunk. The discoloration of the wood is much more persistent, but hardly descends to the roots.

Besides the characters just mentioned, the arms and trunk may show alterations due to die-back from old wounds, decay, and various other injuries interfering with the circulation. The tissues surrounding these impediments are generally affected in such a manner that their differentiation from those that have become diseased through the action of the California vine disease is impossible. The characters found in the arms and trunk, in the case of the California vine disease, are, therefore, of but very little diagnostic value, and, I am inclined to think, hardly worth the pains one must be at to find them.

(*e*) ROOTS.—The roots do not present any characteristic alterations. They are either diseased, or not diseased, according to the situation in which the affected vines are growing and the presence, or absence, of root parasites, either animal or vegetable. If the affected vines are suffering also from the *Phylloxera Vastatrix, Adoxus Vitis* larvae, or nematode worms, the roots will appear damaged; they may be decomposed by root-rot (*Dematophora Necatrix*), or dry-rot, simply die, or, a frequent occurrence, be entirely sound.

B. Histology

Technique.—In studying the anatomy of the California vine disease I have employed the usual cytological methods. The diseased tissues were prepared and placed, with rare exceptions, in the fixing fluid directly in the field.

The fixative that gave me the best results is one per cent. chromic acid. Flemming's solution does not appear superior to chromic acid as a fixative, and labors under the disadvantage of blackening the tissues to such an extent that details are masked, and decoloration of the sections in hydrogen peroxide has to be resorted to to bring them out. Even then, however, the chromic acid material gives preparations superior in sharpness and contrast. Sections made from alcoholic material give stained preparations that compare favorably with those obtained from chromic acid material. Though alcohol is not so good a fixative as chromic acid, I believe that it may nevertheless be advantageously substituted for it in many cases. The matter occluding the cell lumen in diseased tissues contains tannin, and is more or less darkened by the latter fixative, which is quite a disadvantage in all but very thin sections.

After fixing, the tissues were washed, passed into alcohol and through bergamot oil into paraffin; or, if working partly in the cold, from alcohol to chloroform, in which the paraffin was dissolved to saturation. Finally the chloroform was evaporated off at 60 degrees Centigrade.

The stain that gave me the best results for general purposes is acid fuchsin, a concentrated aqueous solution of bichromate of potash being used as the differentiator.[1] This stain, prepared by dissolving 20 grams of acid fuchsin in 100 cubic centimeters of aniline water, gave me the best results when used in the following manner:

The sections to be stained, previously fixed to the slide, were covered with warm acid fuchsin, which was kept steaming hot, but not boiling, for a few minutes. The sections were then washed and plunged into warm bichromate, washed again in water, and mounted.

[1] Cf. Zimmermann, Botanical Microtechnique, 197. 1893. [Humphrey trans.]

The various other stains or reagents that I have used were prepared for the most part after the formulae given in Zimmermann's Botanical Microtechnique.

LEAVES.—From the description I have given of the appearance of diseased foliage it will be seen, upon close examination, that leaves have always one or another characteristic in common, and may be classified in two groups: first, those leaves in which the maculations and striations occur in a field of green; and secondly, those leaves in which the spots and stripes occur in a field of yellow. The young leaves that betray a diseased state only by the convexity of their blade and a pale color naturally belong to the second group, chlorosis being the characteristic of it.

This separation of the diseased leaves into two groups, while of little value for descriptive purposes, and I made no use of it, is of considerable assistance in their microscopic examination. I found, as would be supposed, that corresponding to the observed macroscopic differences, there existed microscopic differences. These latter differences, though less pronounced perhaps than the former, and more of degree than of kind, appear to best advantage and in their truer value when considered separately from one another. I have, therefore, considered in (*a*) the anatomy of those leaves possessing diseased areas and healthy areas; and in (*b*) the anatomy of those leaves that are entirely pathognomonic.

(*a*) If one cuts a cross-section through a leaf, being careful to include tissue in various stages of disease, it will present, under the microscope, very nearly the following appearance:

The epidermal cells (of upper surface and lower surface also, but to a markedly less degree in the latter) are full of matter in the diseased areas which becomes less dense and gradually disappears as one enters the healthier tissues. This matter may be globoidal, homogeneous, or coarsely or finely granular. (PLATE 3, FIGURE 4; PLATE 4, FIGURES 3, 4, 5.) The globoidal form is infrequent, homogeneity the rule; and both forms do not go beyond the areas of marked disease. When the globoidal form is present it may pass over into the homogeneous, or disappear as the healthy tissue is approached. The homogeneous deposit,

however, invariably changes in density, and not infrequently in character, on nearing the healthy tissues.

In the palisade cells the deposit, as a rule, corresponds in density with that in the abutting epidermal cells (when there is a difference it will be in favor of the latter), but extends greatly beyond the outer limit of the matter in these cells. Homogeneity of the deposit is the rule, though it occurs now and then in granular form, the size of the granules varying from cell to cell, but not to any extent in the individual cell. The incrusting of the primordial utricle of the palisade cells is a striking and constant feature. The density of the incrustation decreases, as one passes from diseased to apparently healthy tissue. (PLATE 3, FIGURES 4, 6; PLATE 4, FIGURE 4.)

In the lacunose tissue the incrusting of the lumen is not so regular or so marked as in the case of the palisade layer. The row of lacunose cells adjoining the palisade tissue is more free from it than the others, though there is no absolute constancy in this matter. These cells contain not infrequently, though scatteringly, a few globules much smaller and constitutionally different from those in the epidermal cells, as will be shown subsequently. In the remaining cells of the lacunose tissue the deposit is either homogeneous or granular, dense or thin, corresponding, in this respect, with the variations in the palisade layer. (PLATE 3, FIGURE 4; PLATE 4, FIGURES 3, 4, 5.)

The chloroplasts are sometimes absent from the older portion of the diseased areas; when present their degeneration is marked, but, I should add, not unexceptionally, as sometimes palisade cells may be found filled with dense homogeneous matter in which chloroplasts, still containing starch, lie embedded. Degeneration, however, is the rule, but is more noticeable and has progressed further wherever the deposit in the cells is less dense; the chloroplasts may then be observed as protean plasmodium-like masses, sometimes of considerable size, from aggregation of individuals. As one progresses towards the healthier tissues, and with the decrease in density of the deposit, the chloroplasts are generally more difficult to observe, their resorption having usually progressed further. (PLATE 3, FIGURES 3, 4, 6; PLATE 4, FIGURES 3, 5.)

The degeneration of the chloroplasts, like the incrusting of the primordial utricle, proceeds further in the palisade than in the lacunose tissue. In the cells of the lacunose tissue the chloroplasts do not, as a rule, form large plasmodium-like aggregates. They become vacuolate, but usually without distending to any extent, and, in the row of cells next the palisade layer, when the lumen is free, are inclined to fragment or degenerate into oil-like bodies. (PLATE 3, FIGURES 3, 4; PLATE 4, FIGURES 3, 5.)

The vessels of the minor bundles of the leaves are occluded more or less by granular or homogeneous matter, and the same may be said of the bast, cortical parenchyma, collenchyma, and epidermis of the main veins. (PLATE 3, FIGURE 5.)

Thylloses are often present in the vessels of the main vein. (PLATE 4, FIGURE 1.)

Viala and Sauvageau, in their study of the California vine disease, remark that a "section cut through an apparently uniformly diseased area often shows breaches of continuity due to healthy starch-replete cells which may be coextensive with the diseased tissue."[1] This observation, while perfectly correct, applies only to diseased leaves taken from varieties of grape-vines bearing black grapes. In these varieties diseased centers may be surrounded directly by red aureolae,[2] and the cells in this reddened tissue are replete with starch. When therefore, the aureolae of diseased centers are contiguous, or we have an intervenar stripe dying irregularly, the prenecrotic color being red, such an irregular alternation of starch-replete and starch-free cells may occur. The presence of starch, however, is pathognomonic, and not indicative of health, as the authors just quoted believed. When, as occasionally happens, the dead tissue abuts directly on that which is still green, without apparently any intervening morbid cells, we do not find such a thing as starch-replete and starch-free cells. In fact, in section, what appeared macroscopically as a decided line of demarcation is certainly

[1] Viala, P., & Sauvageau, C. La Brunissure et la Maladie de Californie. Ann. École Agr. Montpellier, 7: 101. 1892. [Translation.]

[2] The reddening of vine leaves, so far as I have observed, is always accompanied by a starch congestion. See Ravaz, L., & Roos, L. Le Rougeot de la vigne. Progrès Agricole 44: 363–370, 392–398. 1905.

not distinctly delineated; disorganization of the cell constituents proceeds into the green tisue and starch, if present, is nowise abundant.

(*b*) Examined in cross-section, those leaves in which the appearance of the disease is followed by a progressive chlorosis of the entire parenchyma differ from those in which it remains normal, *i. e., Case a.,* rather in degree of occlusion of the cells and degeneration of their chloroplasts than otherwise.

The deposit in the epidermal cells (upper epidermis almost exclusively) is either homogeneous or granular, apparently rarely globoidal. It is never very dense, though generally homogeneous in character where the tissue is longest diseased, and becomes granular and finally disappears as one proceeds into the surrounding chlorotic tissues.

The deposit in the palisade cells is usually homogeneous and thin, or more or less granular, and proceeds well into the healthier tissues. Starch is rarely to be found. The chloroplasts are largely resorbed—those still remaining being smaller than in normal tissue and reticulate—in the surrounding chlorotic tissues, and, in the diseased spot itself, form only comparatively small plasmodium-like aggregates. (PLATE 4, FIGURE 5.)

In the lacunose tissue the cells abutting the palisade layer are the freest from deposit; they are frequently almost empty. The remaining cells do not differ much in appearance from the palisade cells; their lumen, however, is freer from deposit.

The deposit in the cells of the lacunose tissue is either homogeneous or granular.

Occlusion of the vessels of the smaller veins is not general, nor do thylloses appear to occur in the vessels of the main veins.

I have just described the general microscopic appearance of sections cut through diseased leaf tissue. I will now describe in detail the anatomy of the pathognomonic tissues with the view of determining as far as possible the nature of the catabolic processes brought about in them.

In describing the California vine disease I said that this malady may appear spontaneously as it were, or develop more or less slowly. This statement applies not only to the individual vine but to individual leaves.

We found that the leaf dies in spots or stripes, infrequently without prenecrotic coloration, the rule being a reddening or yellowing before death, even though all the stages are concurrent as it were; we found that exceptionally the disease appears as yellow maculations, isolated or running together, when death is slow and confined to the diseased areas, even though the leaf turns chlorotic—but I did not lay particular stress upon the point that when death is very rapid, the dead tissue has a somewhat glossy brick tint, that when less rapid it is more reddish brown and matte and when slow, fawn-colored. These differences in coloration of the dead tissue have, however, considerable anatomical importance.

If one examines sections through material showing the color characters mentioned above, he will obtain a conspectus of the behavior of the chloroplasts. Thin sections must be cut, owing to the opacity of the deposit in the lumen of the cells, when the chloroplasts may be well brought out by acid fuchsin,—carbol fuchsin and iron haematoxylin (the first gives the clearer preparations) do not give as sharp a differentiation. Acid fuchsin might almost be called a specific chloroplastid stain. Sections placed in it for a few minutes, and then washed in bichromate, will show the chloroplasts deep red, the cytoplasm very faint rose, the other cell inclusions being practically colorless. By means of this stain the chloroplasts may be studied without fear of misinterpretation. Carbol fuchsin and iron haematoxylin, the latter especially, did not appear to me quite so trustworthy and were soon discarded.

If we take, then, a series of sections through diseased tissues that have died with various rapidities and stain them, preferably in acid fuchsin, we shall find that the resorption, vacuolation and plasmodium-like aggregation of the chloroplastids is, to a certain extent, inversely proportional to the amount of lumen occlusion. In the tissues that have died very rapidly the deposit is homogeneous, dense, and the chloroplasts hardly show more than a slight vacuolation and some appear, in optical section, as hollow elliptical spheres; their center is not a vacuole, however, but a starch grain, as the blue color they assume on the

addition of an iodine solution readily shows. (PLATE 3, FIGURE 4.) This observation regarding the presence of starch in a few chloroplasts applies only to those cases where the prenecrotic coloring of the diseased tissues is red, but does not apply when the change of hue is not apparent or yellowish. In these cases the chloroplasts do not appear to contain starch, and their vacuolation is more pronounced, which fact would lead one to suspect that death does not really occur without some previous discoloration of the tissues, however transient it may be.

When the death of the tissues is less rapid, vacuolation and distension of the chloroplasts is marked, and one will observe plasmodium-like aggregates here and there in the cells, but in more important masses at the lower extremities. With the final occlusion of the lumen all further changes are arrested. (PLATE 3, FIGURES 3, 6; PLATE 4, FIGURES 3, 4, 5.)

When the cells die with moderate rapidity, *i. e.,* when the sections are taken from typical diseased leaves, the vacuolation and plasmodium-like aggregation of the chloroplasts seems to reach a maximum. The homogeneous matter occluding the lumen, though still dense, is light-colored.

As the rapidity of death still decreases, the tendency of the chloroplasts to run together is less marked; though still vacuolate, they distend less and their resorption progresses further and further. The density of the deposit in the cells also decreases. (PLATE 4, FIGURE 5.)

Finally we come to the stage (diseased leaves that become entirely chlorotic, the original diseased areas dying first and thus remaining distinct) when sections through an autumn leaf near its fall and a diseased leaf differ not at at all or very little in appearance; traces of chloroplasts may remain in both cases, and the lumen may also be slightly occluded by homogeneous or granular matter. (PLATE 3, FIGURE 2; PLATE 4, FIGURE 5.)

The chloroplasts up to the stage of plasmodium-like aggregation stain readily, but as their resorption progresses further, they stain less readily and, in fact, at the final stage (autumn-leaf stage may I not call it?), they hold the acid fuchsin less readily—the other cell inclusions not at all—and decolorization in potassium bichromate is unnecessary.

The facts that I have just related in regard to the behavior of the chloroplasts refer almost exclusively to the cells of the palisade tissue. In the cells of the lacunose tissue the chloroplasts become vacuolate, but remain small, as a rule, and their resorption progresses gradually. The occluding of the lumen of these cells does not occur so rapidly and is rarely so dense or dark in color, even in the most rapid cases of death, as that of the palisade cells.

I remarked, in a previous passage, that the row of cells of the lacunose tissue abutting on the palisade layer was very free, when compared to the other cells of the same tissue, from deposit. When the lumen of these cells is free from deposit the chloroplasts not only become vacuolate but fragment and, it would appear, decompose with the formation of oil-like bodies, which, when small, stain like the chloroplasts, but do not color, when larger, as vividly, if at all, in acid fuchsin, which fact leads me to believe that, if originally largely decomposing chloroplastid remnants, they grow by accretion of other proteid substances; this is brought out clearly when sections are stained with rosaniline: the smaller bodies will appear red, the others violet. They all stain, however, more vividly in safranin and eosin than the chloroplasts themselves, which would tend to show that their composition is fairly complex.

During the course of my remarks on the behavior of the chloroplasts I have frequently made mention of the homogeneous substance filling the cell lumen. The various stages of chloroplast degeneration we found to depend on the relative amount and rapidity of production of this substance. It is therefore, important for us to determine the nature and origin of the homogeneous deposit and its homologues, the globules and granular matter. This I will now attempt to do.

From my observations on the degeneration of the chloroplasts it plainly appears that the substance occluding the cell lumen is not a product of their decomposition. That from the decomposition of the chloroplasts there appears to result, in some cases, the formation of oil-like bodies is no contradiction to this statement. The latter form of decomposition is rare. Furthermore, the fact

that the decomposition of the chloroplasts is in inverse ratio to the density of the occluding matter, precludes the assumption that, in the major cases, these bodies act as nuclei of condensation, like crystals in supersaturated solutions. But if the chloroplasts play no part in the formation of the homogeneous matter, or its homologues, whence does it come? The answer to this question involves considerable difficulties, and must, at best, be largely hypothetical.

The deposits in the lumen of the cells are probably genetically related, though it would appear, from their variation in form, that their ultimate composition is somewhat different. They all behave very similarly to reagents and stains. They dissolve in Javelle water, but are not at all, or but little, affected by either hydrochloric or sulphuric acid. A concentrated solution of potash has practically no effect on the homogeneous and granular matters, but will sometimes remove the globules in the epidermis. Fixing diseased tissues in Flemming's solution is apt to make the homogeneous, granular, and globoidal matters so dark that, for staining purposes, sections taken from such material are very imperfect. Chromic acid, one per cent. solution, does not change the color of the cell occlusions to any extent, and sections taken from material fixed in it are hardly more opaque than those taken from alcoholic material.

The deposits in the cells turn black when the sections are placed in a saturated solution of iron acetate; the black color Flemming's solution imparts to them may be very largely removed by peroxide of hydrogen.

Amongst the stains, iron haematoxylin[1] is retained vigorously by the deposits, but Böhmer's haematoxylin is without effect. The homogeneous matter colors deep red in safranin, takes eosin readily, colors in erythrosin, tropaeoline 00, carmalum, rosaniline[2] (dull red), orcein and hydrochloric acid.

From these reactions of the deposits it is clear that they contain tannin (action of iron acetate, regeneration of osmic acid by

[1] I mordanted the sections in "liquor ferri sulfurici oxidati," diluted with two volumes of water, for twenty-four hours; stained in 1 per cent. solution of hæmatoxylin (Benda's) and differentiated in 20 per cent. acetic acid.

[2] An alcoholic solution of equal parts fuchsin and methyl violet.

hydrogen peroxide), proteid matter (eosin, safranin); that they contain also a gum, somewhat similar to wound-gum, would appear from their reaction to orcein and rosaniline.

The deposits in the cells are, then, complex in composition, but it is not improbable that a decomposition product of starch forms their predominant ingredient. The following facts strengthen this view:

The cell walls and cytoplasm of healthy tissues stain blue in Böhmer's haematoxylin, but pathognomonic tissues, on the other hand, do not. If Böhmer's haematoxylin and safranin are used together the healthy tissues will appear as we have just described them, but the cell walls and cytoplasm, when diseased, are red or reddish. This would prove that a decided change takes place in the cell walls of diseased tissues simultaneously with the change in the cytoplasm, but as this change is visible only upon coloration, it is not likely that the cell walls contribute materially to the formation of the deposits in the lumen. The deposits must, therefore, arise as decomposition products from one or more of the cell contents. I believe that starch is the most important contributing substance of them all, and circumstantial evidence favorable to this view is not lacking.

We have seen that the more rapid the death of the leaf the denser the occluding deposit. We have seen also that when the tissues die suddenly without apparent prenecrotic coloration, they are, nevertheless, as free from starch as tissues that become previously colored. We have seen also that reddened tissues are replete with starch and practically free from deposit, but no sooner die than occlusion becomes noticeable and starch, to all intents and purposes, absent; we have seen further that the longer the period of chlorosis before death, the freer the cell lumen from deposit, and starch, it is well known, is quite scarce in chlorotic tissues. It appears to me, therefore, that the substance (of the nature of wound-gum) found in the cell lumen, as granular or homogeneous matter, is largely derived from starch.

The deposits we find in diseased cells do not appear to contain pectic substances in any considerable amount, for Victoria blue and chloriodide of zinc do not give the typical reactions, and the

retention of methylene blue seems to be due to the presence of tannin.

In my observations, just described, on the anatomy of diseased leaves, I never mentioned that hyphae, plasmodia, and bacteria were always absent from pathognomonic tissues: such, however, is the case. In diseased tissues that have not been weathering under conditions favorable to the growth of fungi or bacteria no foreign organism of any kind is to be found. I must, therefore, deny the existence of *Plasmodiophora californica,* but in so doing I wish to impugn only the interpretation of Viala and Sauvageau, not the correctness of their observations. The facts exist as they saw them, but not as they interpreted them.

CANES.—The anatomy of the shoots of diseased vines does not reveal any facts of importance until they begin to mature. Their maturation is somewhat fantastic and the resulting canes appear interspersed with green immature tissue. This immature tissue constitutes the immature spots so characteristic of the California vine disease and will, therefore, occupy our attention almost exclusively in the following remarks.

I think it well, for clearness sake, to preface my observations on the immature spot with a brief description of a healthy cane, as seen in cross-section. We find, around the pith, a ring of wood composed of wood-fibers and large vessels interspersed radially, at equal intervals, by the ligneous medulla; beyond the wood, the cortex, corresponding to the wood fascicles, the basts containing two to three or four rows of fibrous bundles and separated by a parenchymatous and widening prolongation of the medulla; beyond the bast, and separating it from the remaining cortical tissues, the suber; beyond the suber, and capping the basts, as it were, the pericycles; the other tissues, parenchyma, collenchyma, and epidermis, have turned brown, and have more or less collapsed.

In immature spots the departure from the normal, as I have sketched it, is as striking as unexpected. Around the pith we find the ring of wood to be of unequal, instead of equal, diameter, and the wood fascicles to be of unequal development: at the center, or to one side of the center, in the immature spot, they

are very undersized and the vessels themselves fewer in number and subnormal; the phloems corresponding to these undersized wood fascicles are greatly reduced in size, free from bast-fiber bundles, which normally should be 2 or 3 in number, and covered with dwarfed pericycles. As one works towards the sides of the immature spots the wood fascicles increase in size and assume normal proportions; the phloem correspondingly increases in size, but the production of the bast fibers is slow, and at first fragmentary; the pericycles are soon of a size with those in matured tissue. There is no production of suber in the typical immature spot; it ends abruptly at the immediate edge of the matured tissues. With the production of the suber, if the cane is perfect, except for the immature spot, the bast fibers are produced normally. Starch is present in the matured tissues, but not in normal amount, if at all, in the immature spot.

If we now examine in greater detail the modifications occurring in diseased canes, we will find that the above description applies only to those canes in which the immature spot occurs solely on shoots which are not only apparently but also morphologically matured. These canes are, however, rather the exception than the rule, and we find more frequently immaturity and morphologically imperfect maturity forming, to macroscopic vision, immature spots and maturity respectively. Furthermore, if one examines a sufficient number of canes, he will find that the immature spot is not morphologically constant: it may resemble more closely a morphologically matured cane than the cane, macroscopically speaking, on which it is found. I think it best, therefore, seeing the confusion that is liable to arise in the mind of the reader, to precede all further remarks by three comprehensive definitions: he will then know exactly what I mean when I speak of an immature spot; and my dual use of the term cane will not be ambiguous.

Cane (morphologically perfect).—Tissues exterior to the endodermis brown, dead; suber strongly developed; phloem normally developed; bast fibers never absent.

Cane (morphologically imperfect).—Tissues exterior to the endodermis brown, dead; suber well developed; phloem perfectly

and imperfectly developed; bast fibers absent more or less over extended spots.

Immature spot.—Any part of the cane (perfect or imperfect) in which the cortical tissues, from epidermis to endodermis, still preserve all the characters of live tissue.

With these definitions in mind, the reader will be better able to appreciate the extent of the morphological changes that occur, and I do not pretend to cite them all, in diseased canes.

Immature spot.—(*a*) No suber is laid down in the immature spot; it stops on one side beneath the pericycle, then jumps above it and runs out to the epidermis. The phloem, at this point, contains but one bast fiber bundle; the second phloem (immature spot), contains but an imperfectly developed one, and the third (one half the normal size), none. Where the immature spot begins on the other side we have the following state of things: the suber ends beneath the pericycle, but has attempted to push through one end; there is then a break and an attempt to form suber above the next pericycle. The phloem beneath the pericycle under which the suber stops contains two rows of bast fiber bundles, but five phloems further on they have completely disappeared, and the bast itself is about one half its normal size. (PLATE 5, FIGURE 1.)

(*b*) The suber stops, as a definite layer, at the edge of the pericycle, but may continue for a while as a feeble thread. We find the following striking anomaly in some cases: the suber, upon arriving at the immature spot, jumps above the pericycle, and the cortical parenchyma, for a certain distance, intermittently even, divides in an attempt at suberization. The cell walls of the phloem are generally discolored, in some cases markedly so, especially where abnormal suberization of cortical parenchyma has occurred. Bast fibers are present; three or more bundles may be observed in each phloem, generally two, sometimes only one. The bast fiber bundles are always imperfectly developed, even when three in number. The following condition is sometimes met with: one phloem will contain three bundles of bast fibers, the next none and the next two, or the conditions shown in PLATE 5, FIGURE 3 may be observed.

(*c*) The suber is always produced. The normal number of bast

fiber bundles seems to be produced and the phloem is of normal size.

(*d*) The production of suber is scant, and occurs at about the position normally occupied by the second bast fiber bundle. The phloem is much reduced in size, and the bast fiber bundles are obsolete.

(*e*) The suber is continuous: it has not always formed immediately below the pericycle, but frequently several rows of phloem cells beneath it.

(*f*) The suber is continuous in the immature spot and lies well within the phloem. The bast fiber bundles, which are normally developed at the edge of spot, gradually dwindle down to a fragment of one, and disappear. Following the decrease in the number of the bast fiber bundles there occurs a parallel decrease in the size of the phloem; when the bundles disappear the phloem is about one half its normal size. (PLATE 5, FIGURE 2.)

Cane.—The canes on which the immature spots I have just described occurred were all morphologically imperfect. Morphologically mature tissue would interchange, by gradations, with morphologically immature tissue, and the immature spots would form a break in one or the other, or, as it were, the connecting link between the two. Where the formation of the tissues was the least perfect the wood fascicles and their corresponding phloems were much undersized, and no bast fiber bundles were formed.

I have just shown that in diseased canes the morphological variations are considerable, and the reader will naturally expect, as a consequence, considerable variation in the cell pathognomonics. These variations, though in themselves interesting, are not sufficiently important to warrant particular mention, and I shall, therefore, confine my attention to a general description, taking for type a section through a cane in which the cells show considerable disease. The part of the cane most diseased will be, as a rule, the immature spot.

Dowlen says, in describing the histology of diseased canes, that in "those canes which have one side ripe and the other side unripe, the tissues of the ripened portion are almost always well supplied with starch—some starch will always be found—whilst

in the unripened portions the tissues will be altogether devoid of starch. In the discolored areas of the woody bundles, the components of the tissues are seen either to have their walls simply stained brown or else the cell cavity is partially or wholly filled up with a black brown deposit. . . . The larger ducts and vessels are often seen to be more or less filled up with thylles, which are developed sometimes to a great extent."[1]

These observations are correct. Canes taken from diseased vines show a paucity of starch, and, when treated with 1 per cent. iodine solution, give (macroscopically) no starch reaction at all. In section taken through canes with immature spots, starch will usually be found under the microscope, generally in the matured tissues, though, contrary to Dowlen's observations, I have found it in the immature spots, and in larger quantity when suber is produced than when it is not. Its entire absence, however, I have also observed. The presence or absence of starch in the cortex depends, I believe, on the production or nonproduction of the suber. The presence of starch in the xylem, ligneous medulla and pith near the protoxylem depends also, to a certain extent, on the production or nonproduction of the cork—the relation, however, is not so apparent.

The presence, or absence, of starch also bears a very close relation to the quantity of brown granuloid, globoidal, or homogeneous matter found in the diseased cells. The production of these homogeneous substances is proportional to the amount of starch present. The freer the cells from occluding matters, the freer the sections from starch.

In a cross-section of an immature spot one will observe, suber being present, the following condition of affairs:

The pith cells encircling the protoxylem are full of starch or of starch and brown, more or less finely divided matter, which may become coarser, predominant, or even run together into a pseudo-homogeneous mass and entirely fill the cell lumen, the starch grains being perceptible only here and there as clear spots. In other cases the brown masses are more globoidal, or fill the space between the starch grains like a cement, or encom-

[1] Dowlen, E. Report of Board of State Viticultural Commissioners for 1889–90, 60. 1890.

pass the starch in a brown translucent film. Where the medullary rays begin, and in the secondary wood, this occluding matter is darker; the starch grains are blackened, or appear to lie in a blackish matrix, or the lumen of the cells may be filled with a homogeneous black mass. Sometimes before the medullary rays reach the cortex the following change takes place: the starch has largely disappeared from the cells, and the brown irregular lining of their walls appears to be due to an incrusting of the primordial utricle. In the cortex the medullary rays present the following appearance: the lumen of the cells is more or less filled with yellowish or brown homogeneous matter. The deposits are darker where the bast fibers have not been produced. Brown homogeneous matter occludes the greater number of the phloem cells. The cells in the cortical parenchyma, collenchyma, and epidermis may also be more or less occluded. Thylloses are generally present in the primary wood, and are not infrequently very numerous in the secondary wood; they contain now and then granuloids. The discoloration of the cell walls in the different tissues is, except, perhaps, in the neighborhood of the cambial layer, rather inconstant.

Outside the immature spot the occlusion of the cells is less pronounced, though of the same general character.

The matter occluding the cells in the diseased canes does not appear to differ, for the most part, from that filling the cells of the leaves. Microchemically, the gummous substance in the cells of the mesophyl does not differ from the occluding matter found in the cells of diseased canes. This statement, though generally true, needs to be qualified: the reactions characteristic of the deposits found in the leaf are more constant in the cortex, and especially in the phloem, than in the wood, in this latter tissue a great deal of browning of the primordial utricle seems to be due to death rather than to particular catabolic changes—hence the normal appearance of the starch.

Roots.—The roots may be either healthy or show, without structural modification, to a lesser or greater degree, the same character of cell occlusion I described as occurring in the cells of diseased canes, less the accompanying starch when decay is evident.

The homogeneous form of the deposits may still persist in roots in the last stages of decay. Pierce observed in very decomposed roots corroded homogeneous matter. He says: "a microscopical examination of the decaying outer parts of the vascular bundles shows that the cell lumen is filled with a translucent amber-like deposit. This persists after the decay of the cell wall, and presents rod-shaped, more or less irregular and eaten, amber-like casts."[1]

To sum up our microscopic observations in a few words: An examination of the various organs of a diseased vine—leaves, canes, fruit, roots—fails to reveal the presence of any parasitic organism. In all organs exhibiting signs of disease we find, however, a polymorphous gummous substance, containing proteid and tannic matters, that appears to be derived from starch. In the leaves chloroplastid degeneration precedes the occlusion of the cell lumen, or is arrested by it.

III

The relationships of the California vine disease

I said before entering on the description of the California vine disease that the symptoms shown by the various aërial organs had to be considered in conjunction with one another, and not singly, if this malady was to be identified with any certainty. Such a statement as this implies that the symptoms of the disease are also more or less common to other maladies; and this upon examination we find to be the case.

A comprehensive study reveals the interesting fact that the California vine disease has features in common with the diseases known as Folletage, Rougeot, Sun-scald, Brunissure, Shelling, and Tetranychosis. What their common characteristics are I shall now attempt to show, and this will be best accomplished by short descriptions of the related maladies.

Folletage.—This disease may affect a vine either in part or in its entirety, is very rapid in its action, and, as a rule, fatal. Foëx tells us that all the vines in a vineyard are sometimes

[1] Pierce, N. B. *Loc. cit.*, 53.

affected.[1] Chauzit remarks that he has seen "vineyards in which one quarter of the vines were destroyed,"[2] and Professor F. T. Bioletti tells me that it has been known to destroy three quarters of a vineyard. The symptoms of Folletage vary somewhat with the rapidity of the attack; if the vines are affected and killed within the space of a day, the leaves fade, curl, and dry; but when the attack is less severe the seared leaves will be confined more to the apex of the shoots, the lower leaves being "much discolored, either with red or yellow spots or stripes."[3] The shoots always die from the apex downward and the fruit withers and dries up more or less according to the degree of its maturity and the seriousness of the affection on the shoots upon which it is borne.

The anatomy of Folletage does not appear to differ greatly from that of Brunissure.

Folletage generally occurs only at midsummer, but may affect vines as early as May.

The accredited cause of the malady is a rupture of equilibrium between absorption and transpiration.

Rougeot[4] is a mild form of Folletage and Pierce says the following description would apply to leaves of vines affected by this malady:

"The leaves of the dark varieties of grapes show a red discoloration between the veins and at the margin. In the earlier stages this color is faint, but later on the tissue lying between the main veins becomes bright red, and still later dies and changes to dull brown. The death of the leaf usually begins at the margin, or in the center of the red stripes lying between the veins, or it may involve both regions at once. The venation of the leaf remains green in most instances, forming a symmetrical green vein system after nearly all the intervening tissue is dead, or has turned red or brown. Thus there are in these later stages three distinct gradations of color in the affected leaves: (1) A brown and more or less dried margin, or bands of brown lying between the main veins, or both; (2) a band of bright red bordering the dead brown portion of the leaf; (3) normal green

[1] Foëx, G. Cours complet de viticulture, 573. [ed. 4].

[2] Chauzit, B. Revue de Viticulture 26: 50. 1906.

[3] Pierce, N. B. *Loc. cit.*, 195.

[4] Rougeot of authors *pro parte.*

tissues outlining the main venation of the leaf. All colors vary according to the time since the first alteration took place. The petiole is not involved at once in any evident change, but later the leaf is cut off. A second variety of grape had leaves altered in a somewhat similar manner to those of the dark varieties described, but the bright colors did not prevail. There was little to be seen of a third color on these leaves. The alteration is almost directly from the normal green to a dull muddy brown, as if the base colors were yellow and black. The dead tissue occurs first at the margin, and in spots and stripes between the main veins, rarely if ever touching a large vein. Between this dead tissue and the green next the veins is sometimes a slight transitional shade of yellow, which is nearly wanting in many cases, the brown being directly joined to the green on either side of the main veins. Where the intermediate yellow line is wanting, the appearance of the leaf is very striking, and differs in color from any diseased varieties noticed in California. The pattern of the markings is, however, the same. The difference observable is a varietal one. The leaves of a variety of white grape were altered in the manner described for the Muscat of Alexandria in California. In the early stages the changes of the leaf are foreshadowed in faint yellowish spots in the parenchyma, which become more pronounced as the trouble advances. At this time the leaf may have a yellow speckled appearance. The spots are yet somewhat cloud-like and illy defined, and are rarely located upon a vein. As the discoloration becomes more marked these cloud-like spots are better defined at their margin and more and more of the parenchyma of the leaf between the veins becomes involved. As the light yellow spots enlarge the parenchyma at their center turns reddish brown and dies. Later there is a brown central stripe between the veins and at the margin of the leaf, and bordering this dead tissue is a line of half-dead yellow tissue lying next the green bordering the veins. All these markings are very distinct and well defined in the later stages of the trouble. As the death of the tissue between the veins progresses it gives to the green bands at the veins the symmetrical appearance seen on the Muscat leaf in California."[1]

Rougeot has been ascribed to the same cause as Folletage.

Sun-scald.—This malady is described as follows by Viala:

"Sun-scald appears as irregular intervenar, somewhat depressed *feuille-morte* maculations. Leaves are sometimes affected periph-

[1] Pierce, N. B. *Loc. cit.*, 186.

erally: in these cases the discoloration gradually works inwardly towards the petiole, the dead parenchyma assuming a dirty yellow or light brown color; in other leaves again the diseased tissues form sinuate intervenar bands extending from the petiole to the edge of the blade. The hairs of tomentose varieties are dry on those portions of the lower surface of the leaves that correspond to the diseased spots of the upper; they are white, agglomerated and bear a sufficiently close resemblance to the tufts of powdery mildew to have sometimes been taken for them. In some instances the leaves show small, more or less brownish spots. . . ."[1]

In severe cases Sun-scald gradates into Folletage.

Brunissure.—"The disease first appears on the upper surface of the leaves in the form of very small, very numerous yellowish brown spots, in the case of the varieties of the vine bearing white fruit, and as brown almost black punctuations, in the case of those varieties bearing colored fruit. As these spots are all very near one another, for they are separated only by the ultimate ramifications of the fibro-vascular bundles, they run together almost from the day of their inception. After coalescence has taken place they form yellowish brown or dark brown areas that cover the leaf-blades to a greater or less extent. Some cover only the space of half an inch, while others cover a quarter, one-third, the half, and sometimes even the whole of the leaf.

"These maculations appear indifferently here and there upon the blade of the leaf, now between the veins, now upon the tissues adjacent to the veins, and across the latter; now along the edges of the leaf, now at the center of the blade. In general they form between the veinlets, encroaching upon the main veins and the circumjacent tissues later."[2]

All the leaves do not become diseased at once. The basal leaves are the first to become affected, and the apical leaves, even when the shoots have ceased growing, are the last to become diseased; they may even, in mild cases, remain entirely healthy.

Brunissure has been studied by Viala and Sauvageau, Debray, Prunet, Ducomet, and Ravaz.

Viala describes the appearance of diseased cells as follows:

[1] Viala, P. Les maladies de la vigne, 470 *et seq.* 1893 [ed. 3]. [Translation.]

[2] Ravaz, L. La Brunissure de la vigne. Ann. École Nat. Agric. Montpellier II. 3: 175 *et seq.* 1904. [Translation.]

"In the first stages of the disease the parasite develops more especially in the palisade tissue; it invades the cells of the lacunose tissue later, but is found only exceptionally in the epidermal cells. One observes in sections taken through recently affected tissue that healthy cells may contain starch in goodly amount, but that in the cells that are being invaded, it is much less abundant. The starch completely disappears with the spread of the parasite throughout the cell. . . .

"Wherever the leaf blade is brown the tissues are infested, only a few cells at most remaining healthy within the diseased areas. In sections taken through tissue but slightly affected one may find that the cells of the palisade tissue are uniformly affected and those of the lacunose tissue still healthy; in the worst affected tissues, however, the infection is general; all the cells of the mesophyl are filled with the plasmodium.

"The plasmodium varies considerably in appearance. In some cases it destroys the cell contents and entirely fills the lumen; when this is the case it appears fairly dense, is non-transparent and very granular; when observed under a high power the granular structure becomes vacuolate; one might say that the plasmodium presents the appearance of a sponge. At other times it lines the cell walls to a greater or less extent; this parietal plasmodium is more or less finely vacuolate and may be compared to fine lace-work. Protoplasmic strands sometimes join the various parts of the plasmodium and may even anastomose in a more or less complete manner.

"Finally, in a number of cases, especially in tissues in an advanced stage of disease, the plasmodium breaks up into more or less spherical masses, infinite in number and size; they are sometimes so abundant that the cells are choked with them. Among these spheroids we find some that are absolutely homogeneous, refringent, and oil-like; others that contain a large central or more or less excentric vacuole; lastly, others that are finely vacuolate and apparently composed of spongoid protoplasmic matter."[1]

What is the cause of Brunissure? Viala and Sauvageau believed that the vacuolate matter was a plasmodium, *Plasmodiophora Vitis,* and the globoids, homogeneous and granular matters, products of decomposition. Debray considered that the globoids and the homogeneous (cereous) matter as well and the vacuolate, plasmodium-like masses were one and all phases in the development of an organism, *Pseudocommis Vitis.* Prunet as a result

[1] Viala, P. *Loc. cit.*, 403 *et seq.* [Translation.]

of his investigations determined that Brunissure was caused by a fungus which he referred to the genus *Cladochytrium* Nowakowski. Ducomet, on the other hand, concluded that the vacuolate, plasmodium-like bodies were degenerate chloroplasts, and the globoids, granular and homogeneous matters, products of decomposition, and his results were confirmed later by Ravaz. Both Ducomet and Ravaz claimed that Brunissure was a physiological disease; they did not agree, however, as to its cause. Ducomet believed that the disease was induced by rapid changes of temperature, a sudden rise or fall of the thermometer being causal. Ravaz argued that the malady was due to over-production, and, in support of his thesis gave a great deal of data; he further strengthened his opinion by asserting that he could produce Brunissure at will.

Shelling.—In this disease "the leaves at the outer extremities of the shoots first show a yellow discoloration which follows more or less continuously the outer margin. . . ."[1] This "yellow portion dies and turns brown," and the leaf, as a consequence, curls at the edges. In older leaves and young leaves in which foliar development is exceedingly slow the following characters will be observed: "Small irregular blotches of a dark color appear between the veins, these enlarge rapidly, . . . and coalesce to fill up the space between the veins which remain green or yellow. These changes occur so rapidly that the foliage seems to change color suddenly. The contrast between the green or light yellow veins and dark purplish brown of the intervening tissues gives a peculiar streaked appearance to the leaves. In the most serious cases they curl up, become dry and brittle, and finally drop from the vine, leaving it nearly bare."[2] The most striking feature of the disease is, however, the fall of the berries from the pedicels.[3]

[1] Lodeman, E. G. Some grape troubles of Western New York. Cornell Univ. Agric. Exp. Sta. Bull. 76: 416. 1894.

[2] Fairchild, D. G. Diseases of the grape in Western New York. Jour. Myc. 6: 96. 1891.

[3] It may be well to remark that though this feature of the disease is very striking, too much stress should not be laid upon it. The spontaneous separation of the berries from their pedicels and their separation only under the stress of a slight mechanical force are differences of degree, not of kind. *Americo* × *American direct producers* are subject as a class to shelling at

"As the season of ripening approaches, certain berries of the affected clusters fall to the ground on account of the inability of the main fibers and other connecting tissue of the fruit-stems to sustain their weight. . . . The portions of the clusters first affected are, so far as my observations go, invariably either the lower extremity of the cluster as it hangs from the cane, or, in the case of heavily shouldered clusters, the outer extremity of the stem forming the shoulder. . . . Sometimes only one or two berries may fall, but in other cases the drying and shriveling of the stem gradually extends upwards, the affected portion being plainly marked by the absence of the berries. . . . It often occurs that not a berry remains hanging upon the bunch. . . . Some clusters upon a vine seem to be more free from shelling than others upon the same plant."[1]

The development of shelling is sporadic. The disease affects vines at the height of summer and is as selective as Folletage or Rougeot. One, several, or all the shoots may be affected.

"It very commonly occurs that plants in certain portions of a vineyard shell, while the large majority of them do not. The line is sometimes so sharply drawn," our authority continues, "that the affected plant may be entirely surrounded by healthy vines; and it is not uncommon to find a healthy vine in the midst of those which shell. . . . Some clusters upon a vine seem to be more free from shelling than others upon the same plant. This seems to be due, in many cases, to its location upon the cane, but there are so many exceptions that no definite rule can be laid down. . . . Another peculiarity which may sometimes be seen, although cases of it are very rare, is the shelling of the berries upon only one portion of the vine, as for instance those borne upon the canes which spring from an arm, the difficulty thus affecting only one half of the plant. One case was noticed in which the clusters found upon one cane were the only ones which suffered, amongst all those borne by the vine."[2]

The cause of Shelling is not definitely known, though the weight of the evidence points to defective nutrition.

Tetranychosis.[3]—Young vigorously growing leaves become convex and paler than normal; they may even become somewhat

---

maturity and the Viniferae are not entirely free from the trouble. Certain *Franco* × *Americans* behave similarly.

[1] Lodeman, E. G. *Loc. cit.*, 413–415.

[2] Lodeman, E. G. *Loc. cit.*, 415.

[3] Maladie rouge.

spotted with dead tissue and dry up peripherally. Leaves that have ceased rapid growth or are already fully developed become yellowish green and more or less covered with maculations between the veins. The spots spread over the intervenium, not so much from individual development as from increase of centers. The centers of the maculations die, but a running together of the dead areas to form stripes does not seem to precede the death of the whole surrounding parenchyma. As a rule the leaves die from the edges inwards, and in severe cases maturation of the shoots is interfered with; the canes appear spotted or striped with immature tissue. The anatomy of pathognomonic leaves may be briefly described as follows:

A deposit, in the epidermal cells of the upper surface and, to a less extent, of the lower surface, occurs in the most diseased areas only. It is homogeneous to all intents and purposes, and rapidly thins out in the healthier cells. In the palisade tissue, where the cells have not collapsed, they are either full of a homogeneous deposit or with granular matter. The chloroplasts are small, vacuolate, and scarce. The cells of the lacunose tissue in the row next the palisade layer, a certain number of them at least, contain disintegrated chloroplasts, but rarely globuloids. The remaining cells contain disintegrated chloroplasts as well as granular or homogeneous matter. The vessels of the small fibro-vascular bundles are occluded, more or less, with granular or homogeneous matter. The microchemical reactions of the deposits are the same as in the case of the California vine disease.

The disease is caused by *Tetranychus Vitis*[1] and may be held in check by sulphur or the polysulphides.

We may conclude from the study of the relationships of the California vine disease that this malady, while having many points of resemblance with other diseases, is, in diagnosing specimens, likely to be confounded only with Folletage, Tetranychosis or Sun-scald. I have just shown that all these diseases have at least one salient character that differentiates them from the California vine disease, and these characters are clearly

[1] Mr. N. Banks, to whom I sent specimens for identification, could not be positive, from the material at hand, whether this *Tetranychus* was *T. Vitis* or a variety of *T. telarius*, but was of the opinion that it was *T. Vitis* Boisduval.

observed on sufficiently fresh material that includes both the apical and basal portions of the shoots.

IV

Nature of the California vine disease

When empiricists agree, their opinion may be taken as having some foundation in fact. The grape-growers of California have always considered the California vine disease as a "top disease," and the facts undoubtedly support this opinion.

Nearly every observer who has written upon the California vine disease has dwelt at length upon the foliar characteristics of this malady, and either stated implicitly or expressly that it travels downwards, not upwards. Morse observes that "scattering vines which appear not to have made good growth last year, are dried up and dead to the roots, which in nearly all cases still contain sap."[1] Dowlen is of the opinion that the disease travels downwards. "The disease always travels downwards," he says, "both in vines and cuttings." "Some cuttings were purposely planted in an inverted position, still the result was the same; the disease always started at the end which was naturally farthest from the main stem, whether that end was placed in the air or in the soil."[2] That the disease does not affect the roots is an opinion that has been largely held by viticulturists, if we may judge from a letter published in the Pacific Rural Press of October 20, 1888.[3] "The published accounts of the disease which I have seen," writes Scribner, "assert that the roots are perfectly sound." But this is not exactly his opinion, for in the very next sentence we find him saying—"In every case examined by Professor Viala and myself we found the ultimate rootlets dead often for a foot or more from their tips." Scribner does not state, however, whether the vines examined were in the first or last stages of the disease, but I am inclined to believe, from the

[1] Morse, F. W. Report of the viticultural work during the seasons 1885 and 1886, College of Agriculture, University of California, 177. 1886.

[2] Dowlen. Report of Board of State Viticultural Commissioners for 1889–90. 1890.

[3] Letter of F. L. Scribner to Benj. Pratt, of Orange, Cal.

observations of Pierce, that they were in the latter, or, at least, in an advanced stage; for this writer says: "The time when the roots become diseased is difficult to ascertain, and will probably not be known before the nature of the malady is determined. . . . At present I incline to the opinion that the extremes of the vine show the early signs of disease at nearly the same time."[1] Viala and Sauvageau observe that "the disease gradually descends towards the base of the shoots, becomes manifest in the arms, the trunk, and later reaches the roots."[2]

My personal observations are in perfect accord with those of the authorities just cited. I have observed that the roots of diseased vines are not affected at the inception of the disease and will take the longer to show signs of weakness the healthier the vine, and the greater the amount of foliage still remaining in normal function. It is evident that when a vine is affected in all its shoots with a severe apoplectic attack, disorders in the finer roots will immediately take place; the sudden die-back of the shoots almost to the spurs will be followed by corresponding death in the rootlets—but if we take a vine that is diseased only in a few of its shoots, then its roots will not be different in appearance from those of the neighboring healthy vines. Visible symptoms of disease in the roots do not precede the appearance of disease in the shoots. When the roots decay it is because they are in a weakened state and external conditions are superinducive; this is evidenced by the fact that roots will dry-rot in one soil and soft-rot in another.

The visible seat of the disease being in the foliage, then, as popular opinion vouchsafes, and our own and other investigators' observations substantiate, it will be in the study of the behavior of the diseased vines and of the anatomy of pathognomonic tissues that we will find the answer to the question: What is the nature of the California vine disease?

I shall attempt to answer this question.

In the preceding chapter I established that the California vine disease does not differ in method of attack or propagation from Folletage, and showed also that the foliar characteristics of the

[1] *Loc. cit.*, 51.

[2] *Loc. cit.*, 99.

two diseases are in a large measure similar. The importance of this similarity is enhanced when the anatomical study of pathognomonic tissues in the former malady fails to reveal the presence of any foreign organisms, but simply shows a chloroplastid degeneration inversely proportional to the rapidity of death, and an occluding of the cell lumen by a gummous product probably due to starch decomposition. When we also consider that Roze is credited by Debray[1] with having found *Pseudocommis Vitis* (Syn. *Plasmodiophora Vitis*) in leaves taken from vines attacked by Folletage it can hardly be denied that the two diseases are very closely related, for this supposed organism is the cause of Brunissure, which malady I have shown to be undifferentiable anatomically from the California vine disease. Furthermore, we know that sun-scald may gradate into Folletage and, a fact also of capital importance, that this malady is prevalent in southern California—the home of the California vine disease.

The California vine disease, Folletage, Sun-scald, and Brunissure are then very closely related. In fact, a close and comprehensive study of the anatomical features of these diseases cannot help but lead one to the conclusion that all four are due to the some functional state in the vines themselves, outwardly indicated, owing to the play of external agencies, by somewhat different symptoms. This common functional state I shall call a lowering of functional activity; and for the following reason: In the California vine disease—one might say without grave inaccuracy in all four diseases—the anatomical changes observed follow very closely those occurring in autumn leaves, preeminently is this the case when the sequence of changes is slow. Now it is well known that the changes brought about in the leaves of deciduous plants in autumn are due to a lowering of functional activity. The rest these plants require after a certain period of growth takes place normally at the end of such a period, provided conditions are not conducive to its prolongation, without the determinant intervention of external agencies. The rôle of external agencies is largely of secondary importance; they hasten or retard autumnal changes in the leaves, but they will not bring them

[1] Debray, A. Bull. Soc. Bot. France 45: 256. 1898.

about unless the plant has reached the proper stage of development. But once the plants have reached the required stage in their development then external agencies may hasten or retard autumnal changes in the foliage. In countries with a cold winter climate it is a matter of common knowledge that the weather influences very largely the brilliancy of autumn tints, their period of duration and time of development, but is not, I repeat once more, the determinant factor: the plant must first have reached a certain stage of functional activity.

The changes occurring in the autumn leaves of vines, vacuolation and absorption of chloroplasts with, in cases, a slight production of globoidal and homogeneous matters—are similar to those observed in the California vine disease; one might say that they represent a diseased area of a leaf affected by the latter malady in a very mild form. If, therefore, in certain of its forms the California vine disease becomes microscopically identical with an autumn leaf it necessarily follows that Brunissure, Folletage, and Sun-scald are also related to it. In autumn leaves, then, and in the above maladies the same cause must be paramountly active. And as the changes observed in autumn leaves are due to a decrease in functional activity, the disease we are considering, *i. e.,* the California vine disease, may be said to be due to the same cause. But the same functional inactivity need not necessarily be operative in all cases:

In autumn leaves the changes are due to a decrease of vegetative activity; in the case of Brunissure, to overbearing, as appears from the researches of Ravaz; in the case of Folletage, Sun-scald, and the California vine disease, to a rupture of equilibrium between absorption and transpiration operating upon vines weak in their power of absorbing and translocating water, and brought about by external agencies favoring transpiration.

That it is really to a weakened state of the vine that the characteristics of the disease above mentioned are due, may be deduced from the conditions favoring their development. To consider, however, only the California vine disease, these conditions taken individually could not be held responsible for its development, but when considered as factors favoring the visual

manifestation of an organic weakness, they are worthy of consideration.

The California vine disease develops with greater intensity in sunshine than in shade, in wind-blown vineyards to windward rather than to leeward, and soil fertility and texture are not without influence. I shall examine more at length these factors, and independently of one another, though in reality, they cooperate more or less.

Shade.—The effect of shade on the development of the disease is marked. Pierce notes that vines well shaded are but slowly killed; that "shade has a marked retarding influence upon the work of the disease."[1] My own observations confirm this view. Vines shaded at the time the rupture of equilibium between transpiration and absorption is brought about in the open vineyard, whether by sudden insolation or insolation plus humidity or wind, are not affected except in extreme cases.

Insolation.—The effect of excessive insolation has been observed by Morse, who remarks that "many successive vines could be found with dead spurs of last year upon the sunny side, and not infrequently a line of dead wood extended with the fiber to or near the surface of the ground. No shoots started from this side. Suckering, if it occurred at all, came invariably from the north side, where the greenest wood was always found; in fact, I saw no line of dead wood upon this side."[2]

The deleterious effect of intense sunshine following a deposition of moisture is well known to all horticulturists. Morse thinks that water of condensation is not without effect in the burning of the foliage in some cases of the California vine disease. He writes: Those vines "which are protected more or less by trees, present a scalded appearance; some leaves show three different stages: about the margin, and extending an inch or so outward, they will be perfectly red and dead; next comes a zone of light green color, followed by another only slightly lighter colored than the healthy part of the leaf. These are usually most exposed to the sun. In the early morning large drops of moisture, almost equal to that from a heavy rain, are

[1] *Loc. cit.*, 111.

[2] *Loc. cit.*, 177.

found upon these vines, and it is probable that the hot sun scalds the leaves before the water is evaporated."

"It is quite noticeable that leaves exactly similar to those found injured in the open vineyard may also be found among the scalded ones."[1]

Wind.—The effect of wind on the development of the disease I have been able to follow closely. I have observed that the disease appears under the following climatic conditions: When hot, still mornings are followed by stiff breezes, the disease may be expected to appear, and if one walks through a vineyard that has become affected from this cause, with his eye to the wind, he will observe less disease than if he walk before the wind. The vines are affected to windward, which would be expected were transpiration difficulties the cause of the disease.

Soil texture and fertility.—Pierce observes that the rapidity with which vines succumb to the California vine disease depends upon the physical condition of the soil. Dividing the soils of the state into (1) "Heavy soils, including the red and black adobe and clay soils; (2) the gravelly soils; (3) the fine loose soils, including the sandy loams and the sands and fine sedimentary deposits of the river bottoms,"[2] he finds that "If conditions of age and variety are the same, the power of any vine to resist disease is about as follows upon the three classes of soils: (1) Least resistance upon coarse gravelly soils; (2) medium resistance upon soils of a heavy and compact nature; (3) greatest resistance upon level soils which are loose and sandy but not infertile."[3]

The rôle of soil texture on the development of the California vine disease I have been able to follow particularly well in one instance. In a vineyard already old and subject to the daily blast of the trade wind, I found that the disease first appeared where the soil was heaviest, developing later where an admixture of sand and fine gravel made it more open and penetrable, and this despite the fact that the free moisture was approximately the same in both cases.

---

[1] Morse, F. W. *Loc. cit.*, 177.

[2] *Loc. cit.*, 96.

[3] *Loc. cit.*, 98.

CONCLUSION.—The facts gathered during the course of this paper point to the conclusion that the California vine disease is due to some weakness in the functions of absorption and translocation of water becoming manifest when conditions favoring transpiration are marked. To say that the disease is due to a rupture of equilibrium between absorption and transpiration does not conflict with any recorded observations. This is true whether one considers the disease from the point of view of the effect of external agencies upon its general development, or from the point of view of its development upon individual vines, or from the point of view of its symptomatology, anatomy, and relationships.

CORNELL UNIVERSITY,
ITHACA, NEW YORK.

Explanation of plates 1-5

PLATE 1

1, 2, 3, 4. Young diseased leaves of *Vitis vinifera,* var. Mission.

5. Leaf of *V. vinifera,* var. Muscat of Alexandria, showing intervenar striations and death of tissues at edge of blade.

PLATE 2

Leaves showing various stages of disease.

1, 2, 3. *V. vinifera,* var. Mission.

4, 5. *V. vinifera,* var. Berger.

PLATE 3

1, 2, 3, 4, 6. Sections through palisade tissue of leaves in various stages of disease.

5. Cross-section of a small fibro-vascular bundle.

PLATE 4

1. Cross-section of part of a fibro-vascular bundle of a main vein showing development of thylloses in the vessels.

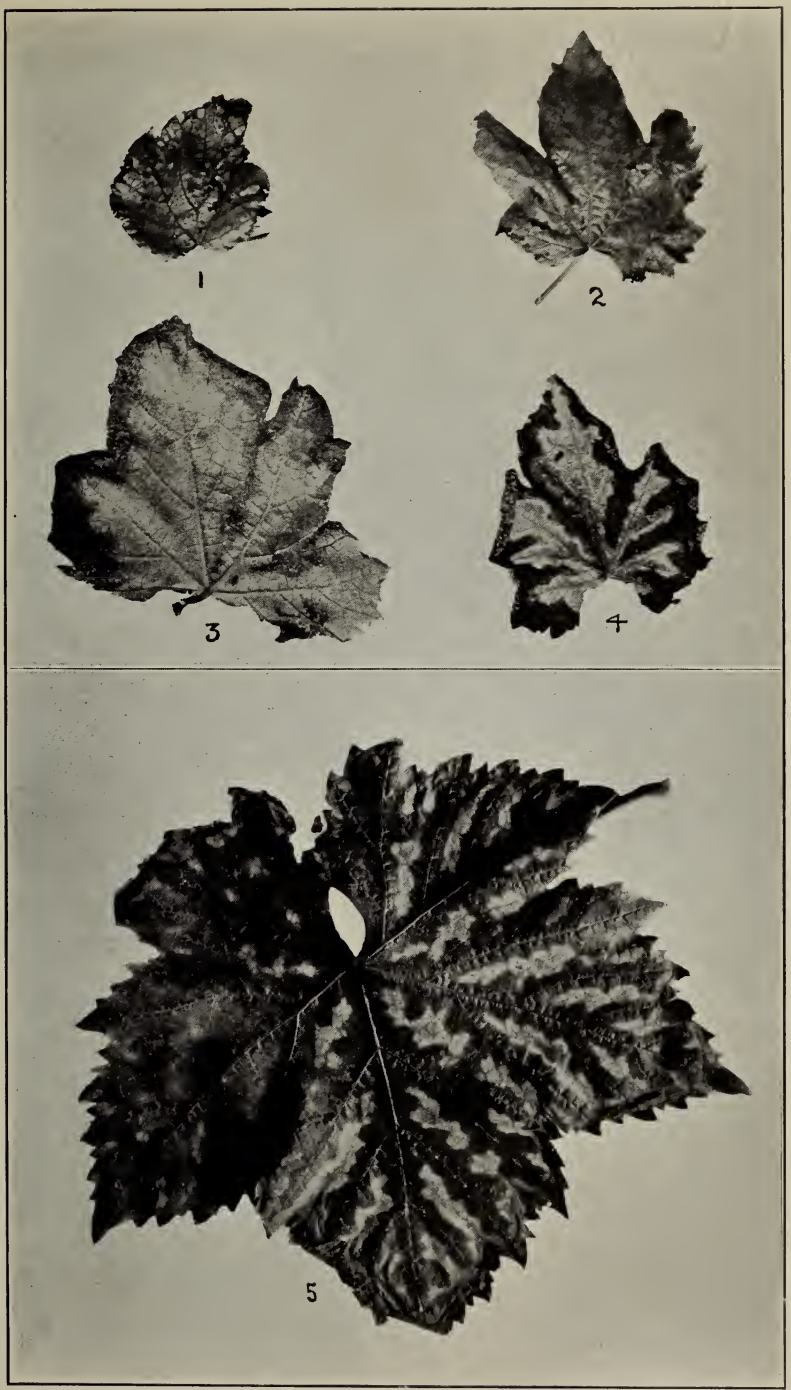
2. Section of healthy leaf showing normal appearance of chloroplastids.

3, 4, 5. Sections through palisade tissue showing various stages of disease.

PLATE 5

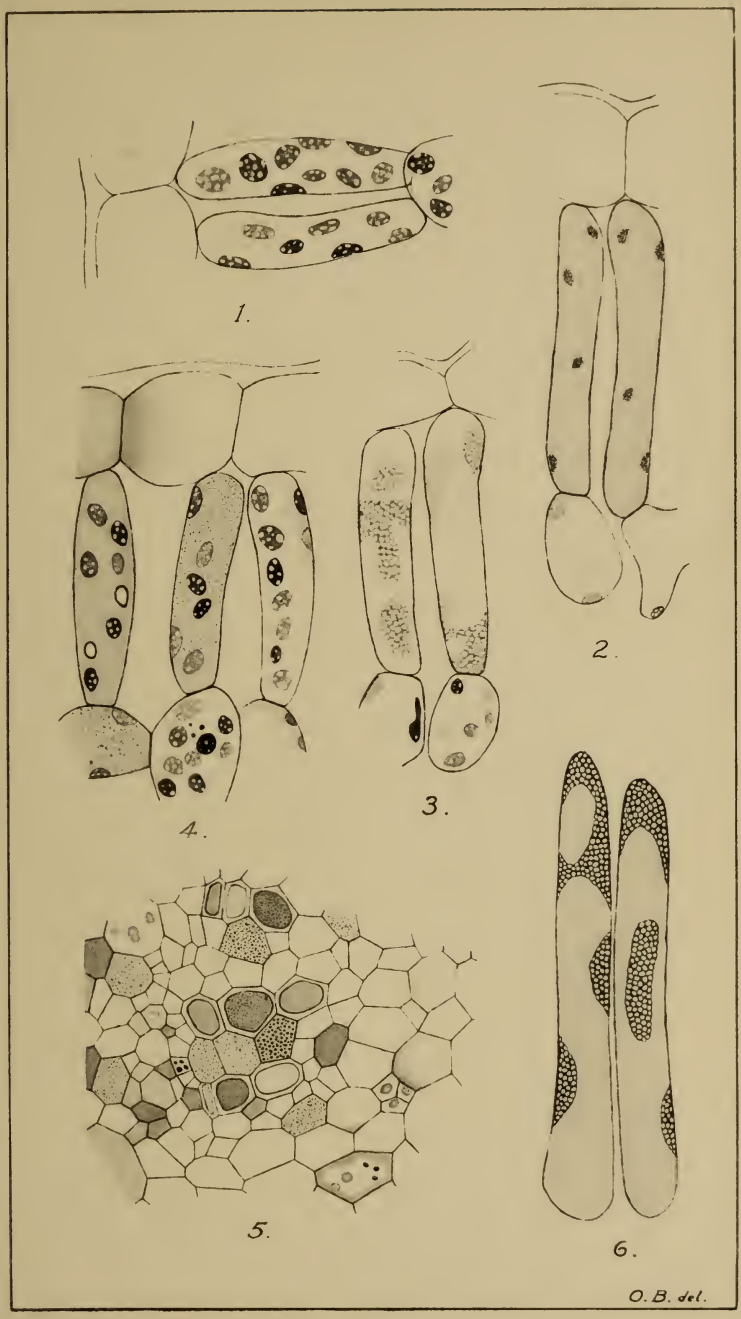
1, 2, 3. Cross-sections of diseased canes.

B, phloem; *C,* cortex; *F,* bast fiber bundles; *M,* medulla; *P,* pericyclic fibers; *R,* medullary rays; *S,* suber; *X,* xylem.



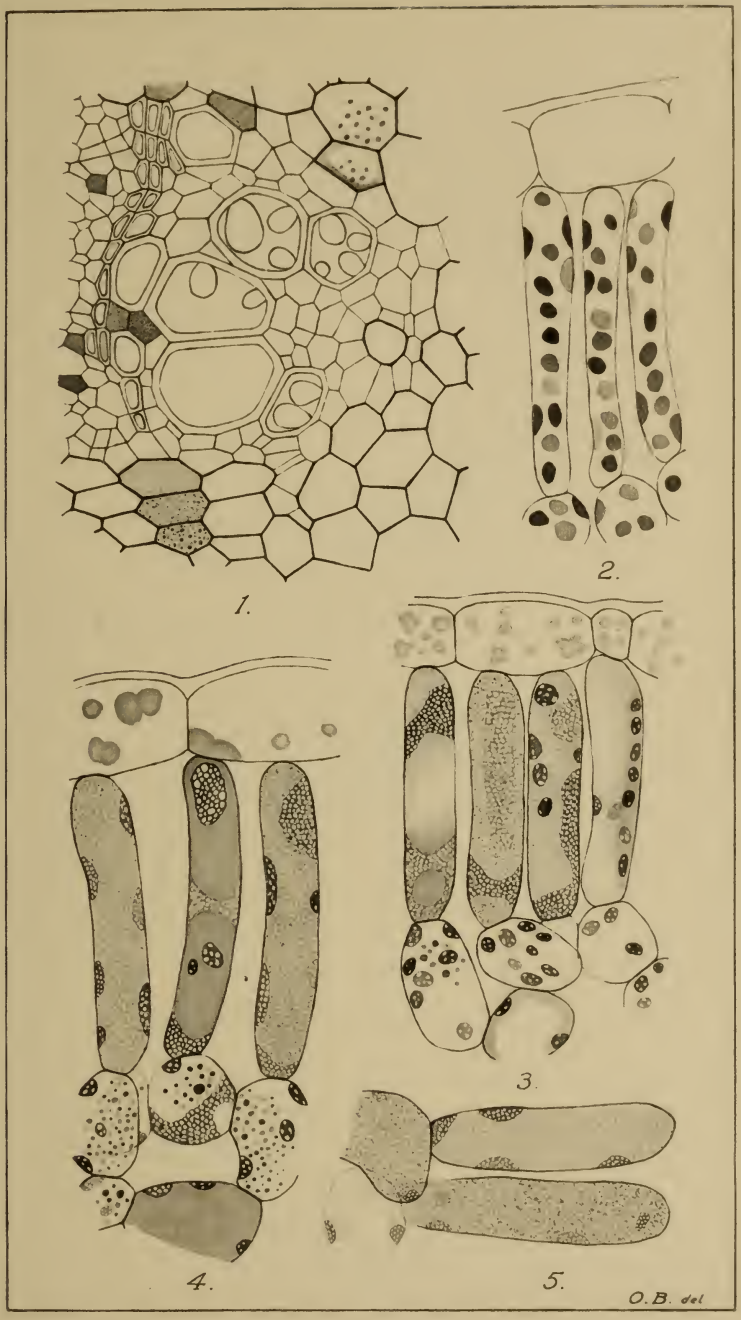
BUTLER: CALIFORNIA VINE DISEASE

BUTLER: CALIFORNIA VINE DISEASE



BUTLER: CALIFORNIA VINE DISEASE

THE WYANOAK CO. NEW YORK, N. Y.



BUTLER: CALIFORNIA VINE DISEASE

THE WYANOAK CO., NEW YORK, N. Y.

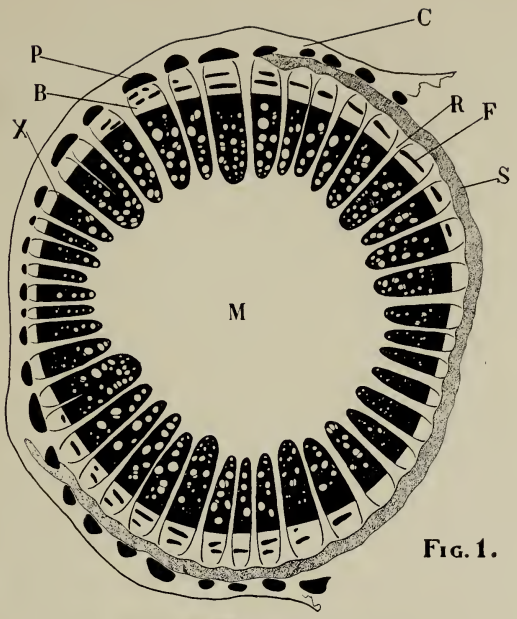


FIG. 1.

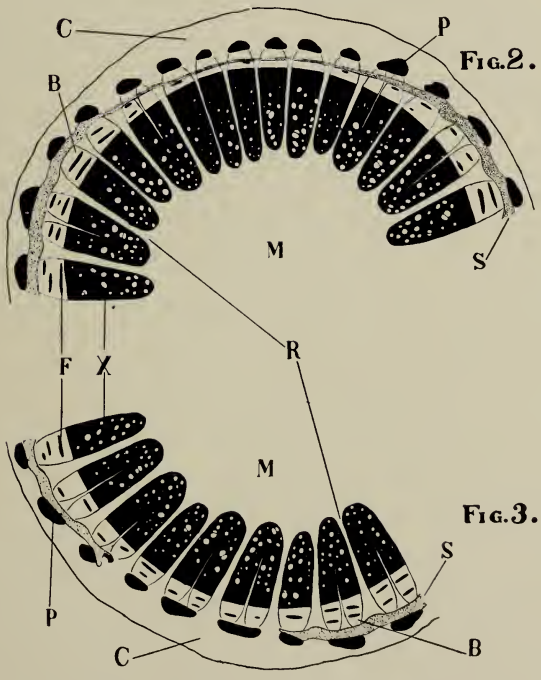


FIG. 2.

FIG. 3.

BUTLER: CALIFORNIA VINE DISEASE