DISCUSSION

Some of the conclusions reached above may be stated as follows:

The infection of plant tissues and development of disease appear to be a complicated process in which enzymatic degradation of the cell and growth of the pathogen in the host tissues are likely to be concerned. Fectinolytic enzymes and cellulases may cause degradation of the cell-wall and breakdown whereas killing of the protoplast may be due to toxins (Wood, 1967; Goodman et al., 1967; Baruah, 1969).

The physical effects of a pathogen on the host tissues remain as unexplained as the dynamics of the enzymatic reactions. The physical forces concerned may bring about changes in the constituent cells of the tissue when infection begins. Infection of a healthy cell may also be interpreted as a process by which the thermodynamic activities of the cell content are affected bringing in the resultant degradation. The process may be irreversible. The total entropy change affected in the system due to infection is the sum of the entropy exchange with the surroundings (positive or negative) and entropy formed in the system by irreversible process (dis, say) which, according to second law of thermodynamics, is always positive. The dissipation function, defined as entropy reduction per
unit time, is given by

$$\phi = T \frac{\text{d}S}{\text{d}t} \geq 0$$

and represents the degradation of free energy due to irreversible process.

Active transport, which is a universal phenomenon in cells and tissues, may determine the selective influx and outflow of substances. By its regulatory function, it may play an important role in osmoregulation and in the adoption of organisms to their environment. In case of active transport, coupling with entropy-increasing metabolic processes allows the permeation flow to proceed in an entropy reducing direction (Katchalsky, 1969). If active transportation in the cells is affected due to infection, there may be rise in osmotic pressure, probably an osmoregulation of the cells. Further, the change in elasticity of the infected tissue indicates the occurrence of turgor variation within the cells and shrinkage in their sizes.

The change in turgor pressure due to infection, as noted by the changes in elasticity of the cell in healthy and infected tissues, is also confirmed by Bustrom (1971) in another way by observing the change in elastic constant ($Y$) with changing turgor pressure. Bustrom (1971) however, states that 'the idea turgor
expands cell is unfortunate and misleading. It is wrong to assume that turgor which itself is the result of expansion causes expansion and acts as a driving force'. It appears that it is the elasticity of the cell wall that controls its turgidity.

Bustrom's (1971) earlier observation on the relationship between elastic modulus (Y) and turgor (P) in growing Pisum internodes showed that with increasing (Y) there was increase in the value of (P).

The possible correlation between cell dimension of the host cells in health and in disease in the form of a polar equation:

\[ r = \frac{ae^2}{1 + e^2} \quad (\text{Fig. 12}). \]

shows that individual cells may be affected in varying degrees by the incidence of infection. This is probably because the energy source (Adenosine Triphosphate) for each cell is different from the other and as such suffers varying effects to the same reaction. Evidence is further given of percentage change in cell sizes of tissues of different types of fruits such as guava (35.25%), pear (24.05%), mango (30.34%), apple (54.79%), orange (55.04%) and of potato (24.89%), onion (71.55%) and sugarcane (26.71%) due to infection by the specific
pathogens; cell volume (expected value) in each case is greater in case of healthy ones than in the case of infected ones - a phenomenon which can be explained in terms of Boyle's law. Evidence given of the amino acid changes during infection based on a chromatographic analysis suggests the contribution of these compounds to the disturbances physically.

In plant cell permeability is generally influenced by

(i) lipid solubility (Overton, 1911)
(ii) molecules penetrating by permease system (Cohen et al, 1967) and
(iii) ions penetrating through pores (Solomon, 1962).

(i) Overton (1911) studied the rate of penetration of numerous substances in a variety of plant cells and concluded that, in general, the rate depended on its lipid solubility. He further contended from observations based on his suggested lipid solubility - cell permeability correlation that the cell membrane might be formed of a thin film of lipid. His findings were later confirmed but the theory was found to be limited in application. It was observed that the lipid solubility rule broke down with very small molecules.
like water, methanol etc. Small molecules penetrate the cell much more rapidly than expected on the basis of their lipid solubility (Lowey et al., 1969). Cell physiologists, therefore, suggested that Overton's membrane carry small aqueous pores through which rapid penetration of small polar substances such as water or methanol takes place.

(ii) Cohen (1967), however, studying the catalysed permeability in cells suggested that the cell membrane contains 'catalysts' termed permeases. They play a specific role in negotiating the penetration of certain compounds. The presence of the penetrating substances in the medium induces the synthesis of these permeases, a phenomenon similar to the production of many enzymes.

(iii) Solomon (1960, 1962) studied, on the other hand, the active transport which plays fundamental role in the functioning of both individual cell and multicellular tissues and suggested the possible existence of a pumping system in cells working against the electrochemical potential gradient within them to regulate the osmotic forces. Briggs (1967) studied hydraulic permeability, diffusion permeability and conductance of the cell membrane in Nitella and proposed a composite membrane for the plant cell consisting of a tortuous porous wall backed by a
continuous lipid layer in which water and ions are soluble. The suggestion, however, does not rule out the possibility of aqueous process throughout along which ions can move.

Experiments carried out using healthy and infected apple tissues indicate the possible mechanism of electro-osmosis operating in which cations (\(K^+\)) and anions (\(Cl^-\)) in KCl solution move through the tissue pores when placed in an electric field. The sizes of both these ions are more or less equal, the diameters being of the order of 3.96 Å and 3.86 Å (Solomon, 1960). Evidences also support that as ions pass through the cell membrane more water molecules move with the \(Cl^-\) ions than with \(K^+\) ions. This indicates an easier path for \(Cl^-\) ions and a restricted one for \(K^+\) ions through the pores in the membrane. The differential preference in the pores suggests that they may carry positive charges. Further, since the volume flow is always towards the positive electrode, fixed charges in the membrane system may be positive. If Helmholtz - Smoluchowski model equations are applicable, the Zeta - potential is also positive. Neglecting the change in volume due to electrode process which is very small, results suggest the occurrence of electro-
osmosis in the plant cell.

The pattern of changes due to infection may thus be due to:

(i) membrane structure being affected mechanically
(ii) charge content in the pores of the membrane being also affected.

The first change, however, may not fully explain the observed phenomenon. Fitting of the data in the equation (2.6):

\[ V = \frac{FXR^2}{8\pi} \left[ 1 - \frac{2aR}{15x} - \frac{2nd^nR^n}{(n+2)(n+4)x} \right] \]

suggests the likelihood of an increase in the charge concentration in the pores of infected cells facilitating the passage of unlike ions and further obstructing the passage of like ions. This may help explain the observed increase in the volume of water electro-osmosing through the infected cells. The pore diameter of the cell due to infection may decrease and as such equations (1.2) i.e.

\[ -\frac{dP}{dE} = (b - a)C_2F. \]

may also explain the above observation.

A change of value on the left hand size (equation 2.6) indicates a corresponding change of
values of factors on the right hand side. With \( V \) therefore, \( X \) (mean counter ion concentration) may vary. As such a re-distribution of the available electrical charges may follow infection as the counter ion concentration within the cell may be adversely affected. Thus ionic imbalance in cell due, probably, to osmotic influences may occur.

The infection of plant tissues may be associated with changes in (a) elasticity of the cell wall, (b) osmotic properties and volume (A.C.V), (c) cell dimension and (d) electro-osmotic properties, thus following the physical laws not hitherto explored in plant pathology.