on, the individual cells may proliferate, and our tissue becomes sodden and infiltrated by inflammatory cells, that is, by leucocytes and the new-born cells that have sprung from the "cells of the part." If the toxin is weak, the congestion, diapedesis, etc., may be slight and the reproduction of "cells of the part" correspondingly greater. May I digress to point out the aptitude of the ancient jingle, "rubor, calor, tumor dolor?" The congestion gives the "rubor;" the increased heat production of the congestion, and possibly the lessened heat radiation, by slowing of the circulation, combine to produce the "calor;" the exuded serum, the inrushing leucocytes, and the cells new-born from the original "cells of the part" together require increased space, and constitute the "tumor," and the stretched nerves are responsible for the "dolor." Any degree of this process may and does exist in the stroma of an organ as accurately as it occurs in the subcutaneous tissue.

To arrive at the condition of any inflamed organ, it is necessary to observe to what extent the damage to the parenchyma has gone, and what degree of interstitial reaction has accompanied it. The most acute irritants are prone to destroy both; the moderate irritants will probably affect the less hardy parenchyma to a greater degree, and the weakest irritants exerting their influence during long periods, while tending to destroy some cells may irritate others to reproduction, and thus preserve a continuity of parenchyma at the same time that they are causing a gradual overgrowth of the interstitial substance. Let us take a definite example, acute enteritis. The mucosal cells will be found to be in a stage of reaction, anywhere in the series, from cloudy swelling to a state bordering upon death; the connective tissue will undergo the various stages of congestion, exudation, diapedesis; during the congestion, glandular activity and exuding serum together, constitute the fluid which drains from the part, and observing this to be the most strongly marked characteristic, the condition is called *catarrhal* enteritis. Should diapedesis be so great that the leucocytes render the secretion turbid, it is called *purulent*; should the sodden, infiltrated connective tissue break down at a late stage and form an abscess, it is designated, a *phlegmonous* enteritis; should death of the mucosal cells be associated with death of the submucosal cells, there is loss of tissue, and it is called "*ulcerative*"; should mucosal cells die, remain in situ, and the exuded serum and leucocytes form lymph which also remains upon the surface, we have a mass of necrotic tissue, lymph, etc., which, being the most striking feature, earns for the condition the name of *membranous*. Thus, according to the stage, or according to the preponderance of any one feature, the enteritis may be designated catarrhal, purulent, phlegmonous, ulcerative, or membranous; yet it is essentially the same unvarying process.