A fair assumption from these facts seems to be, that the sense of injury experienced by the free extremity of the odontoblast is communicated to the nerve filaments, with which its central extremity is associated, and by these transmitted to the brain.

Whatever may be the precise *modus operandi* by which it is affected, it would seem perfectly clear, from the anatomical structure of dentine, that sensation is conveyed through, or by, the contents of the tubules, and that sensation in dentine is confined to these contents.

Though all dentine is more or less sensitive, there is a vast difference in the normal sensibility of the teeth in different individuals.

This variation is dependent on age, temperament, sex, quality of tooth tissue, and other causes, and is so great that what would be hyperæsthesia in one patient would not reach the standard of normal sensibility in another.

Ordinarily, in the discussion of the treatment of this painful condition, this fact has been overlooked. Methods of treatment which, in cases of exalted sensibility, as a pathological condition, have been entirely satisfactory, have, in apparently similar cases, proved useless and disappointing, because the condition was normal and not pathological.

Up to comparatively recent years the commonly accepted cause of hyperæsthesia of the dentine seems to have been inflammation. This theory is defended at considerable length by Dr. Taft. In the light of our present knowledge of the minute structure of dentine, as revealed by the microscope, his argument cannot be considered very exclusive. Nor has any treatment, scientifically based on the inflammatory theory, ever produced satisfactory results. Another, and more plausible suggestion, was that the dental pulp was really the seat of the exalted sensibility, and that the contents of the tubules were merely the passive instruments or agents to transmit the external impression to this central organ. Rational treatment based on this hypothesis would be the administration of such therapeutic agents as, acting on the nervous or circulatory systems, or both, should lower this exalted sensibility. The observed result of the use of nervous or arterial sedatives for this purpose has not tended to confirm the correctness of the theory.

Dr. Louis Jack has discussed the subject in the second volume of "American Dentistry," and concludes that "it may be considered clearly established that dentinal sensibility is attributable to the state of the tubular contents, and that it is excited into extreme manifestation by some physical irritation of the fibrillæ." The doctor has only considered this sensitiveness as associated with dental caries, and attributes the physical irritation to the disintegrating process by which caries are developed. It is well known, however, that this condition is not confined to teeth affected by caries, and, consequently, is not always occasioned by the disintegration of dentine.