ovum within its folds. One can scarcely doubt that menstrual changes in the uterine mucosa prepares it for the reception and implantation of the impregnated ovum, and when the tubal mucous membrane undergoes unusual menstrual changes it not only diminishes the calibre of the tubal bore, but it becomes a soil in which an impregnated ovum may easily implant itself.'

A further study of the anatomy of the tube teaches us that it is not a straight tube, but a convoluted one, bound down at every bend by fibrous bands beneath the serous covering, and that its mucous membrane is arranged in plications, each forming elevations with recesses or depressions between them. From this it may readily be inferred that the journey of the ovum from the ovary to the uterus is naturally a slow one, being retarded by the convolutions of the tube and the irregularities which the plications afford. Next we must remember that the growth of an ovum once fructified is rapid in the extreme, reaching in size, at the end of the second week, from three to six millimetres in diameter. Now if there be any diminution in the propulsive power of the cilia, or failure in the peristaltic action of the muscular coat of the tube, or any diminution in the calibre of its bore the result of menstrual changes in the mucous membrane, or of congenital anomalies, it necessarily follows that the ovum will be further impeded in its journey to the uterus. If to these impediments we add the further difficulties which a fructified ovum, rapidly increasing in size, presents to a canal already crippled in its efforts to perform its function, it will readily be seen that a point within the tube may be reached when the fructified ovum can no longer be propelled on its journey, arrest must take place, and an ectopic gestation initiated at that point.

Once the ovum has become arrested within the tube it is there surrounded by a zone of mucous membrane within which the chorionic villi develop, and to which the ovum becomes attached, but it is exceedingly doubtful whether there be any true decidual tissue or not. A point which is worthy of careful consideration is the rapidity with which the local blood vessels become enlarged and dilated, vessels which ordinarily are quite small become doubled, and even trebled, at a remarkably early period of tubal growth. This is the source of the greatest danger, and accounts for the violent hemorrhage and rapid death after early rupture.