gradually impressed itself upon us so that we view this occurrence as the usual lesion in certain forms of infection. We have hardly reached the time when all are willing to place definite forms of kidney disease in the same group. Nevertheless, an examination of human material as well as experimental studies force us to accept this view. As is true with so many forms of non-suppurative infections in which a bacteriæmia is temporarily and periodically present, many of the organs suffer unequally. The bacterial attack upon various tissues is only an incident in the disease, and it would be impossible to designate the lesion in each organ as a common or constant manifestation.

With the type of infection which dominates acute and subacute cardiac disease, we recognize organisms which are not constant in their virulence, which are sporadic in their systemic distribution and which are very uncertain in their localization in the tissues of the body. At times, during a given illness a dissemination of bacteria occurs in the blood stream for short periods of time, then the circulation is rapidly freed from the meteor-like distribution, only to be involved in a subsequent and similar reinfection from a local focus. The disease does not carry with it a constant bacteriæmia.

As, then, the hæmatogenic infection of different organs is so uncertain and unequal, the lesions arising in different cases are difficult of comparison. We have, however, found that inflammatory changes arising in the interstitial tissue of the kidney were not so uncommon in these infections. In the milder forms where the kidney was least involved and where clinical evidence of a nephritis was wanting, the lesion consisted of a lymphocytic and plasma cell infiltration in the interstitial tissue close to the interlobular arterioles. This subacute inflammatory reaction was distributed mainly about the arterioles and began in about the middle of the medulla. The inner coats of these arteries were not appreciably altered but the adventitia was quite loose and œdematous with an infiltration of lymphocytes. From the perivascular lesion the inflammatory exudate spread along the course of the vessel into the cortex, so that streaks of infiltration could be followed from the medulla to the surface of the organ. Primarily, this perivascular non-suppurative inflammation with its œdema gave a more bulky appearance to the involved areas. The tubules of the vicinity were surrounded by the exudate of cells while but little change occurred in the epithelial lining. Similarly, the capsules of the neighbouring glomeruli were not uncommonly surrounded by a similar infiltration.