ring in arterio-sclerosis, then the latter process must be regarded as functional. Were some irritant discovered capable of directly inducing the hyperplasia of the intima, the case might present a different facies. That such an irritant exists is, it seems to me, highly improbable. The peculiar contrast between the pulmonary and the systemic arteries in their liability to arterio-sclerotic changes is strongly suggestive of the action of differences in the circulation as explaining the contrast and not of the action of any irritative component of the blood.

I do but suggest this, and suggest it most tentatively. I shall feel rewarded if the suggestion leads to increased study into the phenomena underlying some of the commonest and most important forms of connective-tissue overgrowth. We are so woefully ignorant of the causation of such common conditions as chronic valve disease and arterio-sclerosis that I feel that, even if the views here enunciated originate strong and successful opposition, the stimulus they may have given to further investigation will be an ample reward.

The whole matter, as it appears to me, resolves itself into this: "Can we regard fibrous connective tissue as following the same laws as the higher tissues and so as undergoing hypertrophy in consequence of increased work or increased strain brought to bear upon it?" If we can, then it would seem that we can divide off an important series of fibroses from the huge class of inflammatory fibroses. If we cannot, then we must continue to regard all fibroses save the neoplastic as chronic "itides."

Provisionally, therefore, I would divide the various forms of fibrosis as shown in the diagram, namely:

A. Of inflammatory origin:

1. Replacement.
2. Productive.