those of some slow injury to the blood-vessels; but the heart is or has been hypertrophied, and the result of the conditions is rupture rather than occlusion.

Now what do we know, or what can we find out, concerning these awful visitations? For the last quarter of a century I have taught that in a large number of cases of sanguineous apoplexy the kidneys are not granular; and if in some of them they are fibrous, they do not partake of the nature of chronic Bright's disease. This I affirm on the condition of the secreting structures of the tubes, which dwindling or crushed as they may be here or there, present no foci, or traces of past foci, of degeneration or necrosis. Professor Osler has given his valuable judgment in favor of the proposition that a large number of cases of the kind we are contemplating are not attributable to chronic Bright's disease. Now, my belief is that if we can carry our analysis of causes far enough back, we shall reach a junction where we shall travel on a line of common approach to apoplexy with Bright's disease and to apoplexy without it; but for present convenience, and under the restriction of time, I must rule out the Brightian class. It is by the study in the first instance of the simpler case that we shall get back to the junction.

Now in a case of apoplexy what do we find in the damaged parts? Brain assumably healthy; heart hypertrophied; arteries spoiled: the phenomena lie then in the mechanism of the circulation. Thus, in accordance with our desire, we step back from the static point of view and enter upon the dynamic. We shall try to discover which of the variables in this function are altered? In a simple case the heart presents no primary changes, but changes altogether secondary; essentially it is not only healthy in tissue but has worked for a long time at high pressure, thus doing not less but more than its contract. Such changes as may be seen in it are compensatory, or, if morbid, evidently consequential. Then what about the arteries? These have undergone a change, call it atheroma, sclerosis—what you will, so long as we are agreed on signification—but, diseased as they are, they have not silted up, as in the cases we contemplated but to put aside, but have burst. Why have they burst? Because they have been submitted, not only to the mean pressure of age, but also to the augmenting mean pressures of a reluctant peripheral circulation. They have burst at last for the same reason that they have sustained gradual injury; namely, by the accumulation of obscure stresses, which, if we might observe and measure them, we