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 pressures 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 might avert and interpret. I put aversion before interpretation because happily in many conditions of life we can take up our guard before we know why we are the object of hostility, or even before we recognize our enemy. We do not know why in cases such as these the circulation is embarrassed: the cause of the reluctance in the periphery lies still beyond our ken. But, briefly, I may say that the cause must consist either in a narrowing of the calibres of the arteries or stream bed over a very extensive area, if not indeed universally, or in an increase of viscosity with excessive friction in the blood itself. I have been asked somewhat tartly how I demonstrate excess of viscosity, and in what it consists? My answer is, that I never said that the blood in these cases is more viscous, but that there exist the two alternatives only which I have cited—narrowing of the channels and increased friction within the fluid itself. To decide which is the cause, or, if both, the degree of each in the combination, I never pretended. But I admit that it is not easy for me to conceive a contraction of arteries in all or virtually all areas without compensatory dilatation in some of them. It has been suggested to me that in elderly persons the depressor property of the heart and vaso-dilatation may be stiffened or abolished. But a simple test will indicate that our vaso-dilator mechanism is not much abated. Let an elderly man enter a hot bath. For a