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 viscuous, 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 short time at first he will find the radial artery contracting; let him continue however to observe, and in two or three minutes he will find the artery beginning to dilate, until it is largely distended; and a coresponding afflux of blood takes place to the surface. This is not dilatation of the splanchnic area, it is true; but if vaso-dilator mechanism does not rust up in one area, it probably does not in other areas..

It is alleged that in the elderly the arteries become refractory because of sclerosis, whereby their walls grow sluggish or stiff. This explanation, by the way, is inconsistent with that which attributes excessive arterial pressures to arterial contraction over large areas. And in any case to attribute high pressure to sclerosis, and to overlook the large class of cases in which arterial degeneration is manifested without rise of pressure is bewildering. Again, by some writers increase of arterial pressure is explained as a "hypertonus" of the arteries, a resuscitation surely of that older pathology which used to attribute disease to "hypertrophy of the heart"? It is conceivable, of course, that a morbid state of the vaso-motor centre, due to some persistent irritation in the