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 corresponding 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 spot, might keep up general and persistent vaso-motor contraction. Still this is not very probable, nor do I know that this is the view of those who discuss "hypertonus." Must we not assume for the present that hypertrophy in the arteries is produced by the same mechanism as in the heart, namely by persistently excessive pressures on their internal surfaces? In my opinion the vice lies not in a morbid tone of the vessels, but in excessive internal pressures such as obstruction at the periphery would set up. If, then, arterial spasm be also a factor in the hyperpiesis, it seems more consistent to attribute this to the same cause as that, whatever it may prove to be, which chokes the periphery. My observations are that in some cases of rising pressures without Bright's disease arterial spasm, whether primary or consequential is manifestly present; but in others, perhaps the majority, it is not obvious. In some we have what I have called the "large, lax and leathery" artery; in others we find the "wiry" artery. The first kind may be regarded as "arterial tension," for in these cases the effects of tension are very manifest in the consequent tortuosity of the vessels; in the walls of wiry vessels this stretching effect, and indeed the sclerosis itself, is less apparent. Yet in my experience the wiry hyperpiesis is far more difficult to reduce.

However, to come to the matter of prevention; if, concerning the mechanism of persistent rise of mean arterial pressure, we are in the dark, happily there is less doubt as to the treatment of the condition. If the patient is to be saved from an apoplexy, it is only by long anticipation that the proclivity can be counteracted. It seems probable that a disposition to hyperpiesis runs in families; if so, in such families vigilance is imperative. But the tendency is too common to be regarded as one confined