way these infections throw into the blood toxines, or derange the metabolism of the body so as to induce the various changes in vascular sclerosis. Syphilis, according to Bromwell and Diver, cause a general arteritis, including the vasa vasorum.

(*b*) Certain agents introduced into the system have been said to cause arterial sclerosis. Among these may be mentioned lead, caffeine, theobromine, purin bodies, theina, adrenalin, glycæhæmia, mercury, alcohol, digitalis, ergot, and especially nicotin. These may act in two ways: first as poisons and irritants they act on the vessels, inducing arteritis; and secondly, by causing and keeping up prolonged high tension, which is admittedly a cause of sclerosis. The part played by alcohol is in dispute, but I think the consensus of opinion is on the side of it being a cause, notwithstanding the work of Cabot. The faulty metabolism present in gout is undoubtedly a cause; but this again resolves itself to the causes of gout, which are pretty much the same as those causing arteriosclerosis.

(*c*) Lately, much attention has been paid to the influence of the various glands of the body, such as the suprarenals, the hypophysis cerebri, the thyroid and the kidneys. There is now no doubt that the thyroid gland principle reduces arterial tension, and that the active substances of the adrenals raise it. The adrenalin does more than raise the arterial tension, and, in this way, cause sclerosis of the arteries. In addition to this, by acting as a toxic agent on the arterial walls and setting up an arteritis, it causes degeneration and calcification. It has been shown that adrenalin acts on arteries with vasomotor nerves, but the recent experiments of Barr and Hunter also show that it acts directly on the muscle fibres of the vessels. It would, therefore, contract the cerebral, coronary, and pulmonary arteries where the nerve supply is either absent or very slightly in evidence.

The high tension in myxœdema is no doubt due to the lack of the active principle of the thyroid gland. High tension may result, therefore, from defect, as well as from an excess of glandular activity.

It has also been proven by Batty Shaw that when the kidneys are inflamed an extract is given off from them that enters the blood and causes high tension. Here we have an explanation for the high tension in nephritis, and the arterial changes that are so constant in chronic Bright's disease. We can all recall the stop-cock theory of Sir George Johnston, but it failed to carry conviction to the minds of many pathologists. If Batty Shaw and others are correct in the view that the diseased kidneys send into the blood a powerful pressor agent, we can at once understand why the arteries sclerose in chronic Bright's disease. We must wait a little yet, but I think this is the true explanation. What I say here