abdominal sympathetic, there is still some question. It is now known, however, and daily demonstrated that the suprarenal has an active principle which is able to produce symptoms the opposite of those which characterize Addison's disease.

Adrenalin, the internal secretion of suprarenal gland, is a definite chemical body, and in doses of 1-800 of a grain produces a pronounced arterial contraction. This contraction is due to action of the secretion not on the vasomotor nerves, but on the artery through the muscle of the middle coat. This action on muscle is universal, the striped and unstriped depending on this secretion for their healthy tone.

In 1895 Shafer produced some evidence to show the *helpful* effect of adrenalin in Addison's disease. From that date to the present, Prof. Thompson, of New York, has had a patient with Addison's disease under treatment with this extract who feels the loss when he fails to take it.

An observation, first recorded by Oliver, and one of great clinical significance, is the marked diminution in bulk of the kidney, as shown by Roy's oncometer, when suprarenal extract is used. The decrease in size to one-half produces, undoubtedly, changes in secretion and elimination of urea.

Wm. Hanna Thompson, who has watched for many years the investigations into the cause of high tension pulse in many conditions of kidney disease, feels convinced that the textural change in the artery is secondary to the tension, that the continued high tension of pulse in nephritis is due to a persistent excess of adrenalin in the blood. This excess, in turn, may be supposed to be occasioned by stimulation of the adrenals by the inflammatory irritation of the neighboring great glands, the kidneys, with which the adrenals are not only topically associated, but may be functionally so as well. The inference is certainly reasonable that a high tension pulse goes with a contracted kidney, and a contracted kidney is *pro tanto* incapable of a normal excretion of urea. If, therefore, the kidney is only functionally contracted, or partially so, from the action on it of some agent in the blood and not too far sclerosed, the administration of an efficient vaso-dilator ought to be followed by an increased excretion of urea. This was the argument, and a test was applied clinically with aconite as the vaso-dilator with the result that the urea excreted increased two, three and four times the amount previous to use of aconite. With the discontinuance of this drug the urea excretion returned to former low level. Along with this there is a like improvement in the other renal symptoms, such as disappearance of casts and of albumin and general rise in the