The bark of the cashew tree, *Anacardium occidentale*, is known to exert various therapeutic effects in the folklore medicine. However, there is little experimental evidence to substantiate these claims. West, Garvey & Ling (1973) reported the hypotensive action of *Anacardium occidentale*. Present investigations were carried out to find the possible mode of action of its hypotensive and anticurare effect.

It is now well known that most of the clinically used antihypertensive drugs exert their pharmacotherpeutic effect via a common mode of action in spite of their different origins. Bhargava (1973, 1975) proposed that the central adrenergic, dopaminergic, cholinergic and tryptaminergic mechanisms in the central nervous system regulate the cardiovascular system, but the existence of such a central mechanism does not rule out the possibility of a peripheral component in the antihypertensive action of the drugs. The pathways followed by the central adrenergic and serotonergic nerves in the central nervous system are well suited for the regulation of the blood pressure.

Most animals which received *Anacardium occindentale* showed an initial hypotensive response immediately following the administration. This was marked and was not influenced by several experimental procedures or the drug pretreatments.

The delayed hypotensive response occurred in most of the animals between 60 to 120 min after its administration and once set, remained below the basal level all throughout the duration of the experiment. The experimental manoeuvres and drug designs did not influence the delayed hypotensive response. The hypotensive action occurred in rats, cats and dogs and did not show any initial rise in blood pressure.

In an attempt to separate the central from the peripheral and the adrenergic from the serotonergic action, 6-OHDA, FLA-63, reserpine, hexamethonium, phenoxybenzamine, propranolol, p-CPA, Lu 10-171, UML-491, DOCA, pithed rats, spinal cats and intracarotid artery administration in dogs were done. Based on these experimental results it is suggested that:

1. AO exerted an initial fall in BP in rats and dogs immediately after its administration. This appeared to be a direct depressant effect on the heart because it could not be prevented by most of the drugs and procedures used in the present investigation. After initial fall, the BP tended to recover to basal level or to even increase.

2. There was a second delayed hypotensive response in most of the animals. This usually started between 30 to 60 min, reaching a maximum in 60 to 120 min and once set, it lasted through the entire duration of the experiment up to 240 min. The exact mechanism of delayed hypotensive action is not yet known. However, the results obtained in the present investigations suggested the possibility that this was at least in part due to a central action and in part due to a peripheral one. There was some possibility that AO exerted its hypotensive effect through adrenergic and serotonergic mechanisms.

3. AO did not appear to exert any significant change in pulse pressure in most of the animals.

4. The fall in BP induced by AO was usually accompanied by a decrease in HR in most animals and this tended to last for 3 to 4 hr. The decrease in HR was marked between 120 to 240 min.

5. The rate of respiration appeared to increase in most animals after the administration of AO.

6. The FOC reduced in dogs after the administration of AO and the reduced FOC sustained through the course of

the entire experiment.

7. AO exerted an anticurare effect on the curarized skeletal muscle. This anticurare effect was mediated through the presence of potassium because it was markedly reduced in the absence of potassium from the physiological solution.

Although an effort has been made to ascertain the mechanism of hypotensive and anticurare effect of AO in the present investigations, it is desired that further research be carried out to fully elucidate its mode of action.