SUMMARY

Whole plant of Centella asiatica has been extracted by several methods and found that the plant contains both alkaloid and glycoside. A preliminary study of the extracts have been made. Fresh squeezed juice, hot water extract and glycoside produced the hypotensive action in dogs and rats. The alkaloid obtained was found to have no hypotensive effect. The glycoside was then used for study in more details.

The yellowish powder glycoside was isolated from the dry plant of Centella asiatica. For identification of the glycoside, thin layer chromatography was performed. It showed only one spot on the chromatogram which could be detected under U.V. light. The Rf value of the glycoside was 0.3. The mixture of chloroform-methanol-water (65 : 35 : 10) was used as sovent for separation.

The intravenous administration of glycoside solution in anaesthetized dogs and rats produced a marked fall in blood pressure. The hypotensive action was recovered completely within few minutes. The heart rate was also decreased concomitantly with the fall in blood pressure.

Direct evidence for relaxation of vascular smooth muscle was obtained by perfusion the blood vessel in isolated rabbit ear. The glycoside also decreased the force and rate of cardiac contraction in isolated perfused rabbit heart. Furthermore, this potent cardiovascular action of the glycoside could not be blocked by atropine, hexamethonium, propranolol, and antihistamine. The glycoside itself

could not block the ganglion and alpha-adrenergic receptor. Therefore, it was safe to concluded that the Centella asiatica glycoside caused hypotension and cardiodepression by direct action on heart and vascular smooth muscle.

The intravenous injection of glycoside in water-loaded rat under ethanol anaesthesia produced an effect on urine flow which could devided into two phases. The first phase was a very slight increase in urine flow lasting 5-10 minutes which occured concomitantly with abrupt fall in blood pressure. The action was probably the result of an expansion of plasma volume or by increase in renal blood flow. The second phase was a prolong antidiuretic response to the drug and it was possibly mediated by the reflex release of endogenous vasopressin from neurohypophysis in response to hypotension.

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