PLANTS IN CARDIOLOGY

Dietary pulmonary hypertension
The idea that certain plants could produce pulmonary hypertension seemed so unlikely that I began to wonder just how this discovery had been made. The story began in Iowa in 1884 when a new disease of horses with hepatic cirrhosis was traced to the ingestion of Crotalaria spectabilis (Leguminosae) the native species of the rattlesnake plant which was grown as green manure to improve the sandy soil. In 1921 C. spectabilis was introduced from India and it caused many outbreaks of disease among farm animals in the southern United States. Lesions in cows, pigs, goats, chickens, and horses included subendocardial haemorrhage, thickened pulmonary alveoli, pulmonary oedema, anaemia, and renal and hepatic disease. Senecio (Compositae) also caused equine cirrhosis in South Africa in 1920, while in North America the tar weed Atalaya (Sapindaceae), alike clover Trifolium (Leguminosae), and Amsinckia (Boraginaceae) caused it too. A recent human epidemic of cirrhosis in India followed the accidental ingestion of Heliotropium (Boraginaceae). All these different genera and families contain pyrrolizidine alkaloids.

But none of these stories of natural or induced disease reported cardiac hypertrophy or pulmonary arterial disease. However, in 1955 Schoenfeld and Head produced pulmonary infarction in rats with Crotalaria. Then in 1961 the breakthrough came when J J Lalich in Madison showed that rats fed Crotalaria seed or its alkaloid monocrotaline developed acute pulmonary arteritis. He went on to pioneer the crucial long term study. The rats developed intimal and muscular thickening of the pulmonary arterioles, dilated pulmonary arteries and right ventricular hypertrophy. In 1967 Kay, Harris, and Heath in Birmingham were the first to measure the right heart pressure in treated rats and confirm pulmonary hypertension. Later it was shown that the British plant, ragwort, Senecio jacobaeas, sold in health stores for coughs and colds, produced pulmonary hypertension in rats. Meanwhile Bras and his colleagues had discovered that cirrhosis in Jamaican children was caused by hepatic veno-occlusive disease and they showed that the histology of their patients was identical with that of animals with Crotalaria and Senecio poisoning. Jamaican children often drank "bush-tea" made from these plants. But pulmonary hypertension has never been found in patients with veno-occlusive cirrhosis. Maybe only rats are susceptible. Donald Heath has recently looked back over his 25 years work on dietary pulmonary hypertension (Circulation Research 1991;25: 973-74).

Again, as with sweet clover disease of cattle (British Heart Journal 1991;66:181), a leguminous plant introduced from abroad for farming purposes has led via veterinary medicine to an important cardiovascular discovery.

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