Discussion

Von Storch (1938), in a review of an early trial of ergotamine tartrate for migraine, mentions perivascular pains and pains in varicose veins as a rare complication. Spasm of the saphenous vein is referred to by Clarke (1957). The observation of Russell Brain quoted above is, however, the only reference to thrombophlebitis occurring after ergotamine; he mentions it as happening after a single dose, which is confirmed by the above three cases.

The third case is particularly interesting, for the difference between this patient's two legs tends to suggest that the pain which occurs in varicose veins following ergotamine may be the first stage of a thrombophlebitis.

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SALT INTAKE AND HYPERTENSION

BY

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Hypertension was found by Dahl and Love (1957) to be associated with a high dietary intake of salt. In a group of 1,346 adults, habits of salt intake were classified as follows: (1) low intake—never add salt to the food at table; (2) average intake—add salt to food after tasting if insufficiently salty; and (3) high intake—routinely add salt to foods customarily salted before tasting.

TABLE I.—*Correlation of Salt Intake and Hypertension in* 1,346 *Adults*

	No. in Group	No. with Hypertension
Low intake	135	1
Average intake	630	43
High intake	581	61

Hypertension was significantly more frequent among those with a high intake than among the average group (Table I, $p<0.01$).

Dahl (1957) also studied the sodium excretion in hypertension, assuming that sodium is the important ion and that sodium excretion normally reflects intake with reasonable accuracy. The average sodium excretion of nine hypertensive men was 204.3 ± 27.3 mEq/24 hours, while the average excretion of 19 men with normal blood pressure was 165.7 ± 32.0 mEq/24 hours. The difference is significant ($p<0.01$). In the present work sodium excretion has been measured in a larger group of men with hypertension.

Method and Results

The 24-hour urinary excretion of sodium has been measured in 20 men with hypertension and in 20 men with normal blood pressures. Blood-pressure levels over 140/90 mm. Hg have been regarded as abnormal. The mean age of the group with hypertension was 49.8 years, and their mean blood-pressure level 218.9/127.3 mm. Hg. The mean age of the normotensive group was 56.3 years. All were out-patients, taking their normal home diet and unaware that their salt intake was being investigated. The work has been confined to men because of the greater difficulty of collecting samples in women and because of premenstrual sodium retention. Sodium was estimated by flame photometry.

The mean sodium excretion of the 20 men with hypertension was 156.3 mEq/day (S.D.=54.6). The mean excretion in the 20 with normal pressures was 154.1 mEq/day (S.D.=46.2). The difference is not significant.

In this small group little reliance can be placed on differences in the subjects' accounts of their dietary habits. It is of interest, however, that high salt intake was commoner among the men with normal blood pressures (Table II).

TABLE II.—*Salt Intake in* 20 *Men with Hypertension and* 20 *Men with Normal Blood Pressures*

	Salt Intake		
	High	Moderate	Low
Normal blood pressure	9	10	1
Hypertension	4	15	1

Discussion

The present results do not confirm the findings of Dahl and Love (1957) that hypertension is associated with a high sodium intake and excretion.

There is other evidence suggesting that sodium plays an important part in the pathogenesis of hypertension. Hypertension is extremely rare among the Australian aborigines (Hicks and Matters, 1933), the Cuna Indians of Panama (Kean, 1944), and the tribes from the Szechwan mountains of West China (Morse and Beh, 1937). A factor common to all these peoples is a low sodium intake of 1–2 g. or less a day, but many other factors, nutritional or genetic, may be responsible.

With restriction of dietary salt to less than 250 mg. a day, the blood pressure falls in most hypertensive patients and rises again if more salt is added (Kempner, 1948). Moreover, Perera and Blood (1947) found that the rise in blood pressure of hypertensive subjects resulting from deoxycortone could be prevented by restriction of sodium intake. They showed also that an increase of dietary sodium chloride from 4 to 15 g. daily produced a slight but definite rise in blood pressure in six patients with hypertension.

In rats the use of 2% sodium chloride as drinking-fluid for six weeks produces hypertension and renal hypertrophy (Sapirstein *et al.*, 1950). The feeding of salt also enhances the hypertension resulting from experimental renal ischaemia (Verney and Vogt, 1938), and salt restriction prevents production of hypertension in rats by deoxycortone injection (Braun-Menendez, 1951).

Summary and Conclusions

The sodium excretions of 20 men with hypertension and 20 men with normal blood pressures did not differ significantly. Other evidence suggests that sodium is necessary for the production of hypertension. In our society, however, hypertension cannot be related simply to dietary sodium intake.

I thank Dr. George Discombe for the sodium estimations. This investigation formed part of work awarded the Central Middlesex Hospital Research Prize for 1958.

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