importance of hypoalbuminaemia in these postural shifts. While we have not studied this point in the present work, it is likely that variations in cholesterol level due to postural changes will be greater in patients with reduced plasma albumin levels than in healthy people. In view of the relationship of plasma cholesterol concentration to plasma volume change, the recent work of Eisenberg and Wolf (1965) is of interest. These authors showed that in quiet standing the decrease in plasma volume in a hypertensive group of subjects (13.2%) significantly exceeded that in their normal group (7.3%). Though cholesterol levels were not estimated, one would expect rises commensurate with those they observed in serum protein and haematocrit. The significance of these findings lies in the fact that hypertensive patients are likely to be assessed for evidence of coronary artery disease and raised plasma cholesterol levels, and changes in these levels due to the influence of posture are likely to be greater in them than in normal subjects.

The effect of local venous hydrostatic changes on withdrawing blood from a dependent limb has been studied by Eisenberg (1963) by estimation of haematocrit and serum protein levels. The rise in concentration of these values is in accord with the rise we found over the levels in the vertical subject after increasing venous pressure. Venous compression, without reference to postural alterations, has been shown to cause significant increases in plasma protein, cholesterol, and calcium (Gerbrandy, 1960) and in plasma protein and cholesterol (Koerselman *et al.*, 1961; Page and Moinuddin, 1962). For example, Page and Moinuddin (1962) applied sphygmomanometer-cuff pressure of 100 mm. Hg and after five minutes found a rise in concentration of cholesterol of 5.4–19.8% and a 6–20% rise of plasma protein concentration.

The demand for serial estimations of blood cholesterol will not diminish in the foreseeable future. The Framingham study (Kannel *et al.*, 1964) has shown that neither the various lipid indices nor independent lipid levels surpassed the serum cholesterol level as the most important contributor to risk of coronary artery disease. If for this reason added significance is to be placed on the circulating cholesterol level, it is important to exclude the uncontrolled effect of posture. As one would expect the findings to apply equally to all protein and protein-bound constituents of the blood, ideally one should advise a period of some 20 minutes' lying down for the patient before a blood sample is withdrawn, with a minimum of venous compression.

Summary

When the posture is changed from lying to standing for a quarter of an hour a mean increase of 12.9% in the level of plasma cholesterol results in normal subjects. A similar change occurs in the haematocrit and plasma protein levels, and, by inference, in all non-filterable elements of the blood. The reverse process occurs on lying down, though it takes rather longer. The significance of these findings is discussed, especially with regard to serial readings during attempts to reduce the blood cholesterol level.

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Medical Memoranda

Hypotension after Exercise

Brit. med. J., 1966, **1**, 338–339

Ever since Bradbury and Eggleston (1925) described the syndrome of orthostatic hypotension the mechanism of functional derangement has been the subject of numerous physiological studies. In the case presented here the clinical picture was dominated by neurological symptoms due to a profound fall in blood-pressure after exercise. With variations in posture, changes in the filling pressure of the right atrium preceded alterations in arterial pressure, an order of events suggesting a disturbance of venous tone.

CASE REPORT

A 55-year-old police inspector complained of transient visual disturbances for two years. Sometimes he saw coloured lights, and on other occasions there was blurring, lack of colour, and constriction of the visual fields. These episodes were bilateral, lasted one to five minutes, and usually occurred when he rested after exertion. They were often accompanied by a sensation of unsteadiness, though he never fell or lost consciousness. Occasionally they were associated with dysarthria, and once with dysphagia. He had noticed poor sweating for 10 years, and he had been impotent for three years.

Examination revealed a dry skin, with a Parkinsonian facies. The only other abnormalities were mild rigidity of the right arm and lack of facility with repetitive movements in the right hand and foot. There was no tremor.

Routine blood and urine studies were normal, including glucose-tolerance test and W.R. There was no significant abnormality in radiographs of the skull, cervical spine, or chest. Sweating could be provoked all over the body, though the response was poor in the face, hands, and feet.

The following vascular reflexes were observed.

Posture.—When the patient got himself up from lying to standing there was a fall of 12 mm. Hg in right atrial pressure, recorded from a cardiac catheter. This was accompanied by a rather slower

drop in systemic arterial pressure from 170/80 to 70/45 mm. Hg, recorded from a cannula in the brachial artery. The pulse rose from 54 to 78/min. When he lay down again all the parameters returned to normal, but once more the arterial pressure lagged behind right atrial pressure. Similar haemodynamic changes occurred with a tilt-table, and in no instance did alteration in posture induce any symptoms.

A convenient method of simulating the effects of postural change is to apply suction to the lower half of the body as described by Ardill *et al.* (1965). When a pressure of 44 mm. Hg below atmospheric was applied to this patient's body distal to the iliac crests, the systolic pressure fell to 55 mm. Hg, though no symptoms occurred. His circulatory adjustments to this manœuvre will be reported in detail elsewhere (Bannister, Ardill, and Fentem, in preparation).

Exercise.—The response to exertion was studied by asking the patient to climb up and down a step at a rate equivalent to 500 kg.-m./min. for three minutes. When he rested his arterial pressure dropped from 90/40 to 45/25 mm. Hg. Right atrial pressure fell 12 mm. Hg, but the pulse showed little change. Within 10 seconds of stopping exercise the patient complained of the visual disturbance which had brought him to hospital. He was unsteady, but he did not lose consciousness. His symptoms cleared on lying down.

Valsalva's Manœuvre.—This showed complete block of the vasomotor reflexes.

Comment

Orthostatic hypotension has become much commoner since the introduction of powerful hypotensive drugs and antidepressants with hypotensive side-effects. Rarer causes include polyneuritis, tabes dorsalis, craniopharyngioma, and Addison's disease. Its association with Parkinsonism, as in this case, was described by Shy and Drager (1960). The incidence of orthostatic hypotension in old age is probably much higher than has previously been recognized, and this has been attributed to cerebrovascular disease (Johnson *et al.*, 1965).

Interest in the physiological defect has recently been focused on a more critical appraisal of venous tone (Bevegård *et al.*, 1962). In the patient described here changes in right atrial pressure preceded alterations in arterial pressure on lying and standing. This supports the view that a disturbance of venous tone may play an important role in the failure of arterial pressure.

Returning to the clinical features of this patient, it is notable that his symptoms occurred after taking exercise, and no discomfort could be induced by changes in posture. In 1907 Gordon found a fall of 25 mm. Hg in the systolic pressure of two healthy rugby players after an international match. With more sophisticated techniques, Holmgren (1956) recorded drops of 45 mm. Hg in four normal subjects after 2,500 kg.-m./min. on a bicycle ergometer. Presumably the fall is due to circulating vasodilators persisting after cessation of the reflex and mechanical factors which maintain arterial pressure during exercise. While a drop of 45 mm. Hg can be tolerated in a normal subject with a systolic pressure above 140 mm. Hg during exertion, in this patient the pressure during exercise ran at 90 mm. Hg, so the subsequent fall was disastrous.

I wish to thank Dr. C. J. Earl, Dr. Denis Williams, and Dr. Roger Bannister for their help. Dr. R. D. Bradley did the manometric recordings.

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Effect of Starch and Sucrose on Carbohydrate-induced Hyperlipaemia*

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Essential hyperlipaemia has been subdivided by Ahrens into two entities, in one of which the hyperlipaemia is fat-induced and in the other carbohydrate-induced (Ahrens *et al.*, 1961). Of these the latter is the more common. In carbohydrate-induced hyperlipaemia most of the dietary carbohydrate is converted into triglycerides by the liver at an accelerated rate (Farquhar *et al.*, 1964). Recently it has been emphasized that the various dietary carbohydrates have different effects on human lipid metabolism. Thus Macdonald (Macdonald and Braithwaite, 1964) found in an isocaloric exchange of 550 g. per day of sucrose for maize starch in seven normal men that there was no change in serum lipids on maize starch, but that sucrose caused an increase in serum triglycerides. It was therefore of interest to study the effect of dietary starch and sucrose in patients with carbohydrate-induced and other types of hyperlipaemia. The marked responsiveness of the lipids of these patients to such diets has already been referred to (Kaufmann *et al.*, 1965), and the details of the responses of one of these patients are here presented.

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Methods

The patient was treated as an out-patient in the dietetic kitchen of our metabolic unit while she pursued her normal daily activities. Food was prepared by trained dietitians and consisted of three meals served in the dietetic kitchen, and a small snack taken home by the patient to be eaten in the late evening. Unconsumed food was returned so that the daily caloric intake and composition of the food could be calculated. The patient was weighed daily.

The diets were prepared from natural foodstuffs, and were adjusted to the special needs and habits of the patient as much as possible. The composition of the various diets was calculated, using food composition tables (McCance and Widdowson, 1960; Guggenheim, 1964). During the experiments three almost isocaloric

Composition of Experimental Diets

Diet	Total Calories	Composition of Diet (% of Total Calories)			Composition of Carbohydrate (% of Total Carbohydrate)		
		Protein	Fat	Carbohydrate	Polysaccharides	Sucrose	Undefined
Starch ..	2,720	21	4	75	91	0	9
Unsaturated fat ..	2,640	17	45*	38	87	0	13
Sucrose ..	2,620	15	2	83	13	79	8

* 91% of fat consisted of sunflower oil.