SYSTEMIC HAEMODYNAMICS IN MILD ARTERIAL HYPERTENSION BEFORE AND AFTER PHYSICAL TRAINING

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SUMMARY

1. Five men with borderline, latent arterial hypertension of the hyperkinetic type were studied haemodynamically at rest and during dynamic exercise before and after a 6-week period of supervised physical training.

2. Tendencies to lower heart rate, cardiac output and arterial blood pressure, both at rest and during a standardized work-load, were observed after the training period, with significant differences between paired observations for the heart rate and mean arterial blood pressure during exercise. The systemic vascular resistance being unchanged, there was a certain trend to an increased widening of the arteriovenous oxygen difference, both at rest and during exercise.

3. The present findings from a small number of subjects indicate that physical conditioning of patients with latent hypertension of the hyperkinetic type contributes to a normalization of their circulation toward a normokinetic one, thereby also normalizing their blood pressure level and achieving a more economic energy expenditure in the cardiovascular system.

Key words: cardiac output, exercise, haemodynamics, hypertension, training.

Mild essential arterial hypertension of the borderline, latent type has repeatedly been shown to be characterized haemodynamically by a hyperkinetic circulation at rest, the high cardiac output being caused by an increased heart rate (Sannerstedt, 1966; Julius & Conway, 1968; Lund-Johansen, 1968). The increased blood flow is probably primarily directed to the striated muscles as indicated by an increased blood flow in the skeletal muscles (Amery, Bossaert & Verstraete, 1969).

Physical training generally tends to decrease the resting heart rate with a decreased or unchanged cardiac output, and leads to a decreased muscular blood flow and an increased

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widening of the arteriovenous oxygen difference during exercise (Frick, 1968; Saltin, Blomqvist, Mitchell, Johnson, Wildenthal & Chapman, 1968; Clausen, 1969; Varnauskas, Björntorp, Fahlén, Prěrovský & Stenberg, 1970; Schroeder, 1972). Thus, physical conditioning could theoretically constitute a physiological way of normalizing the haemodynamics in latent hypertension, achieving a more economical way of functioning of the circulation in these cases.

To test this hypothesis five men with latent hypertension were studied haemodynamically at rest and during exercise, before and after a 6-week period of supervised training.

MATERIAL AND METHODS

Subjects

Five men, 26–38 years of age, with mild essential hypertension of the borderline, latent type agreed to participate in the study. None of them had any signs of cerebrovascular, cardiac or renal impairment. Three patients were previously untreated. One patient had been treated with saluretics for 1 week, and another had received a β-adrenergic blocking agent for 2 months. In both cases the treatment was stopped more than 3 weeks before start of the study.

Physical training

The supervised training took place in the hospital. The patients pedalled for 1 h three times a week for 6 weeks on an electrically braked, variable-load bicycle ergometer (Elema-Schölander AB, Sweden). The exercise was divided into five periods of 12 min each. During each period the load was changed every 4 min from a low of 450 or 600 kpm/min to a high of 900–1200 kpm/min, the setting being chosen to achieve a heart rate of 150–160 beats/min at the highest exercise level. The auscultatory blood pressure, heart rate and a standard ECG-lead were recorded every 4 min during the exercise, and also repeatedly before and after the bicycling.

The patients were encouraged to do additional physical conditioning on their own, but only two of the five men did this regularly. No drugs were prescribed during the training period, and the patients were not recommended to change their eating habits.

Haemodynamic study

Haemodynamic studies at rest and during exercise using techniques described in detail elsewhere (Sannerstedt, 1966) were performed in the week before and then in the week after the training period. The studies were carried out in the morning with the patients in the fasting state and included the following functions: heart rate, intra-arterial blood pressure, cardiac output by using a dye-dilution technique with bromosulphalein as indicator, and intermittent sampling of arterial blood, and oxygen consumption by collecting expired air in a Douglas bag for subsequent gas analyses. From the data obtained, stroke volume in ml/beat, systemic vascular resistance in arbitrary units, and arteriovenous oxygen difference in ml/l were calculated.

After percutaneous insertion of catheters under local anaesthesia into a brachial artery and into the right atrium or subclavian vein, the patients rested comfortably for 30 min before resting determinations were made. Determinations were then obtained during one (two cases) or two different (three cases) exercise loads on the bicycle ergometer, the load-setting varying between 450 and 900 kpm/min. The cardiac output was measured during the last minute of the exercise periods, which lasted for 10 min. Four patients were studied in the recumbent position,
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and one patient in the sitting position. At the repeat study the protocol was in each patient identical with that used at the first examination.

No adverse effects of the training or investigational procedures were observed. On the contrary, the programme was much appreciated by the patients; this was obvious from a very strict adherence to the training hours. In one case, however, a temporary break of 2 weeks in the supervised training period had to be inserted because of intervening personal problems of the patient.

The Student t-test has been used for testing differences in the values from the two studies, and for testing paired differences within the group.

RESULTS

The average values ± SEM for some haemodynamic functions before and after the period of supervised training are seen in Fig. 1.

Even if not statistically significant, the cardiac output at rest tended to be lower after the training period (−1.0 ± SEM 0.8 l/min), and this tendency was maintained during exercise. The calculated stroke volume being unchanged, the decrease in output was mainly due to a reduction in heart rate, which was obvious especially during the period of physical exercise, when the mean decreases were 12 ± SEM 4 (P < 0.05) and 17 ± SEM 2 (P < 0.01) beats/min respectively.
The calculated systemic vascular resistance showed inconsistent changes, and accordingly the mean arterial blood pressure was slightly lower both at rest and during exercise. The mean decreases during exercise were $5 \pm \text{SEM} 1$ ($P<0.05$) and $5 \pm \text{SEM} 4$ ($P>0.10$) mmHg respectively. Also the product of the heart rate and systolic brachial artery blood pressure tended to be lower during the exercise loads; the mean decreases were $40 \pm \text{SEM} 11$ ($P<0.05$) and $34 \pm 19$ ($P>0.10$) units respectively.

On the contrary, the tendency of the arteriovenous oxygen difference was toward a more marked widening during exercise after the training period, the increases being $12 \pm \text{SEM} 6$ ($P>0.10$) and $11 \pm \text{SEM} 16$ ($P>0.10$) ml/l respectively.

All subjects had lost in weight between the two studies, the losses ranging from 0.5 to 6.2 kg with an average of 3.2 kg.

**DISCUSSION**

The haemodynamic findings in the present study are on the whole consistent with reports by others on the effect of physical conditioning in normotensive subjects with or without cardiovascular diseases, in whom a lower heart rate and a decreased or unchanged cardiac output, after training, both at rest and during dynamic exercise, have been found (Varnauskas, Bergman, Houk & Björntorp, 1966; Hanson, Tabakin, Levy & Nedde, 1968; Saltin et al., 1968; Hartley, Grimby, Kilbom, Nilsson, Åstrand, Bjure, Ekblom & Saltin, 1969). In such studies the arterial pressure has been lower (Varnauskas et al., 1966; Hanson et al., 1968) or unchanged (Saltin et al., 1968; Kilbom, Hartley, Saltin, Bjure, Grimby & Åstrand, 1969) after the period of training. In vasoregulatory asthenia, a marked reduction after training of the elevated resting heart rate has been reported (Holmgren, Jonsson, Levander, Linderholm, Mossfeldt, Sjöstrand & Ström, 1957).

Previous studies on the effects of physical training in systemic arterial hypertension are few and have mainly included a small number of patients with established hypertension. The results are inconclusive, and both decreases in the blood pressure (Boyer & Kasch, 1970; Hanson & Nedde, 1970) or no change at all (Johnson & Grover, 1967) have been reported. Our own experiences from two male patients with established hypertension, who were investigated and trained in exactly the same way as the group of men with latent hypertension, have been rather disappointing as far as positive effects on the blood pressure level are concerned. Our preliminary impression is therefore that physical training by itself will probably only achieve little in this category of hypertensive patients.

On the other hand, even if not providing any definite proof, our present findings in a small group of five men with borderline, latent hypertension of the hyperkinetic type support the proposed hypothesis that physical conditioning in such patients contributes to a normalization of their circulation toward a normokinetic one, thereby also favourably influencing their blood pressure level, and may encourage further studies in this category of patients.

In conclusion then, we believe, in agreement with the opinion of Hanson & Nedde (1970), that an increased physical fitness will benefit many patients with latent hypertension of the hyperkinetic type, helping them to achieve in a physiological way a normokinetic circulation with a more economic energy spending both at rest and during the load of physical exercise.
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REFERENCES


