THE INFLUENCE OF EXERCISE TRAINING ON HYPERTENSION

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I. INTRODUCTION

A. The Concept of Hypertension

1. Definition:

a. Tension or tonus that is greater than normal.

b. A condition in which the patient has a higher blood pressure than that judged to be normal.

c. In adults usually defined as pressures exceeding 140/90 mm. Hg.

2. Classification/types:

a. Benign hypertension:

Hypertension that progresses slowly.

b. Malignant hypertension:

Severe hypertension that progress rapidly, to the point of death.

c. Essential hypertension (or primary hypertension):

Hypertension that develops without apparent cause.

d. Portal hypertension:

Increased pressure in the portal venous system caused by an obstruction of blood flow through the liver.

e. Renal hypertension:

1) Hypertension accompanied by kidney disease.

2) Hypertension produced experimentally by constriction of renal arteries.

3) Caused by a humoral substance (renin) produced in an ischemic kidney.

f. Goldblatt hypertension:

Hypertension that resembles renal hypertension, produced in experimental animals by decreasing the blood flow to the kidney.

g. Renovascular hypertension:

Hypertension due to obstruction of the renal arteries.

3. Etiology:

a. Primary cause:

1) Increase in total peripheral resistance:

This results from vasoconstriction or narrowing of peripheral blood vessels.

b. Pathological causes:

1) Coarctation of the aorta.

2) Hyperthyroidism with thyrotoxicosis.

3) Patent ductus arteriosis.

4) Arteriovenous fistula.

5) Pheochromocytoma.

6) Psychogenic causes.

7) Certain forms of renal disease, particularly when limited to one kidney.

8) Adrenal tumors.

9) Primary aldosteronism.

10) Polycythemia.

c. Other factors associated with hypertension:

1) Heredity.

2) Fiber type distribution.

3) Autonomic nervous system imbalances.

4) Central nervous system changes.

5) Hormonal elevations.

6) Metabolic imbalances.

7) Amount of body fat.

9) Increased caloric intakes.

10) Increased salt intakes.

11) Degree of inactivity.

12) Age.

13) Enviromental conditions (temperature, noise).

14) Magnitude of alcohol consumption.

15) Coffee and cigarrettes.

16) Diminution in baroreflex sensitivity.

17) Increase in natriuretic hormone:

a) Inhibition of sodiun transport in cells other than renal tubules.

b) Changes in arterioles.

c) Increases sensitivity to vasoconstrictive stimuli.

4. Pathology:

a. Adaptive changes:

Cardiac hyperthrophy.

b. Degenerative changes:

1) Generalized degenerative arterial lesions:

a) Plasma filtration.

b) Retained lipoprotein fractions.

2) Tenuous vascular supply.

3) Increases pressure gradient.

4) Atheromatous lesions on coronary vessels:

Aggravate development of ischaemic fibrosis.

5) Hypertensive heart failure.

6) Charcot-Bouchard aneurysm.

5. Pathophysiology:

a. Disturbed function of one of the normal blood pressure regulatory system (neurogenic, renal pressor, renoprival, adenomedullary, adrenocortical).

b. Categories of the blood pressure regulatory system:

1) Rapid acting mechanism:

a) Change blood pressure within seconds.

b) Components of the central nervous system involved.

c) Changes made through efferent sysmpathetic nerve activity.

3) Intermediate acting mechanisms:

a) Requires minutes or hours to perform blood pressure changes.

b) Renin-angiotensin system.

c) Circulating vasoactive agents.

d) Influences that lead to shifts in the distibution of extracellular fluid between the intravascular and extravascular comparments.

4) Long-range control mechanisms:

a) Affects blood pressure in hours or days.

b) Modification of blood volume.

c) Renal excretion/reabsoption of salt and water.

d) Aldosterone and other mineralocorticoids.

c. Pathophysiology of most secondary forms of hypertension:

1) Peripheral vascular constriction and secondary plasma contraction.

2) Primary plasma volume expantion with little or no change in vascular resistance.

d. In some instances activation of one pressor mechanism leads to activation of a second:

1) Example:

Increased sympathetic nerve discharge leads to renin release by the kidney, and angiotension II formation from renin leads to aldosterone release.

e. In other instances the control mechanisms are coupled by negative feedback:

1) Example:

Volume expansion induced by mineralocorticoid excess inhibits renin release.

f. Altered function of the kidney to regulate urinary output of sodium and water in response to changes of arterial perfusion pressure.

6. Treatment:

a. Nonpharmacological:

1) Relief of stress:

Relaxation-meditation techniques.

2) Dietary control:

a) Restrictive sodium chloride to 4 to 6 g per day.

b) Restrict calories (weight loss).

c) Restrict cholesterol and saturated fats.

d) Vegeterian diets.

e) Potassium and magnesium supplementation.

3) Stop alcohol consumption and cigarrette smoking.

4) Regular aerobic/isotonic exercise.

b. Pharmacological.

c. Surgical.

B. Coronary Heart Disease and Hypertension

1. Arteriosclerosis:

a. Definition:

A generic term for thickening and hardening of the arterial wall with loss of elasticity and contractility.

2. Atherosclerosis:

a. Definition:

A form of arterioclerosis characterized by a variable combination of changes of the intima of the arteries, consisting of the focal accumulation of lipids, complex carbohydrates, blood and blood products, fibrous tissue and calcium deposits, and associated with changes in the media of the arteries.

b. Pathologic effects:

1) Coronary artery (heart) disease.

2) Aortic aneurysm.

3) Arterial disease of the lower extremity.

4) Major role in cerebrovascular disease.

5) Saphenous vein-graft atherosclerosis.

c. Genesis of atherosclerotic process:

1) Atherosclerotic plaque:

a) LDL cholesterol.

b) Smooth muscle cell proliferation.

c) Increased collagen formation.

2) Alteration of the permeability of the arterial endothelial layer, which is caused by:

a) Fibrinogen.

b) LDL.

c) Hypoxia.

d) Elevated levels of serum carbon monoxide.

e) Hypertension (via damage of endothelial cells).

3) Platelets and monocytes.

3. Risk factor for coronary atherosclerosis:

a. Not reversible:

1) Aging.

2) Male sex.

3) Genetic traits:

Positive family history of premature atherosclerosis.

4) Race/ethic origen.

b. Reversible:

1) Negative lifestyle habits:

a) Cigarrette smoking.

b) Alcohol consumption.

c) Inactivity.

d) Estrogen replacement/oral contraception.

2) Obesity.

3) Hypertension:

a) Markedly increases the prevalence and severity of atherosclerotic vascular disease especially in the presence of other risk factors.

b) Markedly increases the prevalence of atherosclerotic vascular disease in patients with diabetes.

c) Accelerates atherosclerosis in individuals with hypercholesterolemia and other risk factors.

c. Potentially or partially reversible:

1) Hyperlipidemia (hypercholesterolemia and/or hypertriglyceridemia):

a) VLDL (very low density lipoprotein).

b) LDL (Low density lipoprotein).

c) Total cholesterol greater than 200 mg/100 ml (or 8.64 mmol/l): Increased risk for coronary artery disease.

d) Ratio of high to low density lipoprotein:

Abnormal level if greater than 20 %.

2) Hyperglycemia and diabetes mellitus.

3) Low levels of high-density lipoproteins (HDL).

d. Other possible factors:

1) Emotional stress and/or personality type.

2) EKG evidence of compensatory left ventricular hyperthrophy.

II. OVERVIEW OF THE STUDIES

A. Questions to be Raised

1. Does habitual physical activity reduces blood pressure levels in normotensive persons, and lowers elevated blood pressure in hypertensive subjects? Is the size of the effect significant?
2. How much of what kind of exercise, over how long a period, is effective to decrease blood pressure?
3. What is (are) the mechanism(s) of action.
4. In what type of hypertensives do specific modes of exercise show the greatest or least antihypertensive effect?
5. What is the size and differences in the blood pressure reductions with physical training between females and males?
6. Does age affects the magnitud of the blood pressure changes with chronic endurance exercise?
7. Does the antihypertensive effect of exercise training is independent of other factors, such as weight loss, diet, psychological changes and other hygienic strategies?
8. What are the changes in blood pressure after an physical training program with the administration of medication? What is the dose-response relationship?
9. Can animal studies findings be generalized to human population?
10. Is the antihypertensive effect the result of the previous bout of exercise?

B. Problems and Limitations

1. <u>General limitations</u>:

a. design shortcomings.

b. Methodological problems.

c. Uncontrolled variables.

d. Limited population.

2. <u>Epidemiological studies</u>:

a. Clustering of hypertension in the sedentary population:

1) No randomization:

a) Is the clustering independent?, or

b) Is the clustering the result of a lack of physical activity?

b. Dificulty on the quantification of physical activity:

1) Inferred from person's occupation:

Energy expenditure tend to be uniformly low in most jobs, so there is little gradient of physical activity at work.

2) Leisure time activity ignored.

c. Self selection:

1) Less healthy individuals:

They tend to choose less physical demanding occupations.

2) Symptomatic individuals with physical active jobs:

They transfer to more sedentary work before the diagnosis of atherosclerotic disease.

3. Experimental studies:

a. Lack of a control/monitoring of possible confounding variables/factors:

1) Body composition changes (body weight/fat loss).

2) Metabolic variables (reductions):

a) Plasma insulin.

b) Blood glucose.

c) Serum triglycerides.

3) Diet:

a) Food/caloric intake.

b) Sodium/potassium intake and excretion.

4) Exercise-drug interaction:

a) Concurrent hypotensive medication.

b) Reactive changes in medication compliances in hypertensives taking drugs.

c) Changes in medication during the course of the study.

5) Therapist contact:

Relaxation and other behavioral interactions.

6) Psychological changes (personality alterations).

7) Autonomic nervous system modifications.

8) Maximum oxygen consumption ($\dot{V}O_2max$).

9) Non specific/placebo (relaxation?) effect.

10) Repeated measures.

b. Wide variation in methodology:

1) Blood pressure assessment procedure:

a) Direct methods (intra-arterial measurement):

- Most investigations using these procedures had post-training results that were either slightly elevated or similar to the pre-training values.
- Non-practical method; because of its invasive nature, it was almost always based on a single measurement (i.e., on one day).
- Therefore, actually it may be less accurate than several indirect (cuff) measurements taken over a period of time.

b) Indirect methods (stethoscope and sphygmomanometer):

These studies was usually identified with lower mean blood pressure.

c) Position of the subject:

Sitting pressure tend to show more of a decrease than standing or supine, which may show slight increases.

d) Inter-tester and intra-individual variability during rest and testing conditions.

2) Screening criteria/hypertensive classification:

a) A few studies failed to state their classification criteria.

b) Most of the investigations classified their subjects as essential hypertensives, although these studies utilized borderline hypertensive subjects.

c) Variability in the number of blood pressure recordings used for classification of subjects as hypertensives.

3) Exercise training programs:

a) No specification of the exercise regimen.

b) Lack of information about adherence to the training program:

This makes difficult the quantification of the overall training stimulus.

c) Absence of physical activity quantification.

4) Assessment of the effects of training on cardiovascular functional capacity:

Some studies did not present any documentation of the attainment of a trained state.

c. Other design and methodological shortcomings:

1) No control/comparison groups:

a) Lack of randomization of hypertensives subjects to treatment and control groups.

b) Self-and experimenter selection of the hypertensives that most likely show the greatest blood pressure decrease.

2) Inadequate control/comparison group:

a) Wait list/non exercise.

b) Only normotensives.

c) Few studies included a non-exercise hypertensive control group or a cross-over protocol with the hypertensive subjects serving as their own controls.

3) No alternate treatments or attention/placebo controls.

4) Poor control of training intensities.

5) Inadequate power:

Small number of examined subjects.

6) Exclusion of females from most studies.

7) Subject ages:

a) Older subjects more likely to show decrease.

b) Data from adolescent, young adult, or elderly population is very limited for both males and females.

8) No tracking/minimal measurements of potential parameters that can help explain the mechanisms of action:

a) Cardiac output.

b) Total peripheral resistance.

c) Plasma hormones (catecholamines, insulin, renin).

d) Sodium retention/excretion.

c) Anthropometric measurements.

9) Obsolet data analysis methods.

10) Dynamic and endurance-type of activities were most frequently studied.

d. The reductions in blood pressure, when shown, are from slight to moderate.

4. Animal investigations:

a. Failure to quantify the oxygen requirements of the training program:

1) Exercise training by spontaneously hypertensive rats (SHR) in excess of 75% $\dot{V}O_2max$ results in higher resting blood pressures.

2) Endurance trainning by SHR group at 40-60% of their $\dot{V}O_2max$ is associated with lower resting pressures.

b. Absence of information regarding mode, intensity, and length of training.

c. Lack of a documented training effect.

d. No specification on the methods of blood pressure measurements.

e. There are significant species differences in untrained oxygen consumption and cardiovascular capacity.

III. EPIDEMIOLOGICAL STUDIES

A. Cross-Sectional Studies

1. Comparison of athletes versus nonathletes:

a. See Table 1.

b. Conclusions:

These studies do not strongly establish that habitual physical activity can reduce blood pressure levels.

TABLE 1
COMPARISON OF ATHLETES VERSUS NONATHLETES

REFERENCE STUDY	SUBJECTS				PHYSICAL ACTIVITY CATEGORIES		FINDINGS		COMMENTS
	Num. of Subj.	Sex	Age (yr)	POPULATION/ ETHNIC ORIGEN	ACTIVE OR MORE ACTIVE	INACTIVE OR LESS ACTIVE	BLOOD PRESSURE	OTHER	
Kral et al (1966)	28,000			Athletes/ Prague	Athletes		< 1% had ↑BP (> 160/100 mm. Hg.)		No valid comparison population
Pyrorala et al (1967)	61			Athletes/ Finnish	Former champion Athletes	Age-Matched Controls	Lower $\bar{X}$BP in Athletes		
Saltin & Grimby (1968)	917	M		Gothenburg	Active Endurance Athletes (33)	• Inactive 10 yrs Endurance Athletes (29) • 50-yr Old Control Group (855)	No Significant Differences Between Groups in Blood Pressure.		

2. Physical activity occupational classes:

a. See Table 2.

b. Conclusions:

The studies show inconsistent relationships of activity to blood pressure and hypertension.

Table 2
PHYSICAL ACTIVITY OCCUPATIONAL CLASSES

REFERENCE STUDY	SUBJECTS				PHYSICAL ACTIVITY CATEGORIES		FINDINGS		COMMENTS
	Num. of Subj.	Sex	Age (yr)	POPULATION/ ETHNIC ORIGEN	ACTIVE OR MORE ACTIVE	INACTIVE OR LESS ACTIVE	BLOOD PRESSURE	OTHER	
Miall & Oldham (1958)	249			Workers	60 Heavy Workers	180 light Workers	Significant Lower $\bar{X}$BP in Heavy Workers		
Karvonen et al (1961)	790	M		Lumberjacks & Countrymen/ Finnish	369 Lumberjacks	421 Age Matched Countrymen	Lower $\bar{X}$ BP in Lumberjacks		
Taylor et al (1970)	3,076	M		Railroad Swithmen and Rail-clerks	1414 Railroad Swithmen	1662 Rail Clerks	Lower $\bar{X}$ BP in Railroad Swithmen No Sig. Diff. in the Prevalence of Hypertension	Lower $\bar{X}$ Work HR in Swithmen	
Taylor et al (1967)		M		Rail Workers/ Italian	Rail Workers	Rail Workers	Lower $\bar{X}$ BP in Active Group	Lower B.Wt. Lower SF	
Morris et al (1966)				Busdrivers & Conductors/ London	More Active Drivers	Less Active Drivers	Lower $\bar{X}$ BP in More Active Group	Lower B.Wt. in Active Group.	
Keys et al (1980)		M		7 Countries (15 Population)	4 Levels of Occupational Physical Activity Classification		Lower $\bar{X}$BP in Active Group (8 of the 15 Populations)	Lower B.Wt. in Active Group	In 7 Cohorts No Diff. in BP Between Active Classes
Chiang et al (1968)				Taiwanese			No Sig. Diff. in $\bar{X}$ BP According to Occupational Activity		Surveys
Berkson et al (1960)				Utility Worker Chicago					
Casel et al (1971)					Farmers	Nonfarmers			
Pantfenbarger et al (1977)		M		Longshoremen/ San Francisco	Active Longshoremen	Inactive Longsremen			
Humerfelt & Wederrang (1975)	1,680	M		Norway	Wide Range of Physical Activty		• Higher SBP in Heavy Physical Labor Group • No Diff. in DBP		No Relationship Between the Prevalence of Hypertension (> 170/100)
Rosenman et al (1977)	2,635	M		Workers (Western Collaborate Group)			Higher $\bar{X}$ BP in Heavy Workers	Higher B.Wt. in Heavy Workers	

3. Leisure-time physical activity and fitness:

a. See Table 3.

b. Conclusions:

Cross-sectional studies of leisure activity and fitness in relation to blood pressure show no consistent association and, when found, little independence of body weight.

Table 3
LEISURE — TIME PHYSICAL ACTIVITY AND FITNESS

REFERENCE STUDY	SUBJECTS				PHYSICAL ACTIVITY CATEGORIES		FINDINGS		COMMENTS
	Num. of Subj.	Sex	Age (yr)	POPULATION/ ETHNIC ORIGEN	ACTIVE OR MORE ACTIVE	INACTIVE OR LESS ACTIVE	BLOOD PRESSURE	OTHER	
Rose, G. (1969)	9,000	M		Civil Servants London	Categorized by Duration of Walking to Work		No Cross-Sectional Relationship to BP	Lower B.Wt. in more Active Group	
Montoye et al (1972)	1,700	M		Michigan, USA			• Lower $\bar{X}$ BP in Active Group (The Relationship was less marked when adjusted to Body Fatness) • Fatness Contribution > Phys. Act. Contrib.		
Epstein et al (1976)	509			Civil Servants/ Brittish			No Relationship of Leisure Activity and BP		
Kannel et al (1971)				Framingham			No Correlation of a Physical Activity & Fitness Index with BP Levels.		
Wilhelmsen et al (1976)				Western Collaborate Group			No Sig. Relationship to BP Among the More Active Exercisers		This was independent of Wt. or Wt. Gain
Cooper et al (1976)	3,000			Cooper's Institute (Dallas)	Fit	Unfit	• No Sig. Diff. BP Between Fit & Unfit • Slightly Lower $\bar{X}$ BP Associated with Lower % B. Fat in Fit Group.		
Gyntelberg & Meyer (1974)	5,249	M	mid-age	Employees/ Copenhagen			Linear, Negative & Independent Relationship of Fitness to BP		Survey
Rouse et al (1983)		M		Perth			No Relationship of Leisure Activity to BP		Survey (Questionaire-Based)
Panico et al (1986)	958	M,F	7-14	Obese School Children/ Naples	Higher Fitness	Lower Fitness	• Lower $\bar{X}$ BP with Increased Fitness & Recovery Index. • Sig. Relationship with SBP.		• Phys. Fitness Measured by Harvard Step Test Recovery Pulse Index. • Relationship Persisted after Adjust. for B. Mass Index.

B. Cohort Studies

1. See Table 4.

2. Conclusions:

a. These studies showed a relationship of activity habits or fitness to subsequent high blood pressure; yet, in Paffenbarger studies this was not demonstrated to be independent of body mass.

b. Therefore. the findings in cohorts tend to support the relationship between increased physical activity and reduced future risk of hypertension.

Table 4
COHORT STUDIES

REFERENCE STUDY	SUBJECTS				PHYSICAL ACTIVITY CATEGORIES		FINDINGS		COMMENTS
	Num. of Subj.	Sex	Age (yr)	POPULATION/ ETHNIC ORIGEN	ACTIVE OR MORE ACTIVE	INACTIVE OR LESS ACTIVE	BLOOD PRESSURE	OTHER	
Paffenbarger et al (1968)	7,685			Students/ Univ. of Penn	> 5 hrs/wk of Coll. Sports	< 5 hrs/wk of Coll. Sports	Higher Rate of Hypertension in Less Active Group (Medications or BP ≥ 160/95 mm. Hg.)	Adjustment to B.Wt. ↓Predictive Risk for Hypertension	• 20-30 yr Follow-up • Not Independent of B.Wt
Gillum & Paffenbarger (1978)	8,852	M		Students/ Harvard Univ.	> 5 hrs/wk of Coll. Sports	< 5 hrs/wk of Coll. Sports	40 % Higher Incidence of Hypertension in Less Active Group		• 20-30 yr Follow-up • Not Independent of B.Wt
Blair et al (1984)		M,F			High Fitness	Low Fitness	Significant Excess Risk (Risk Ratio, 1.5) for Hypertension in the Low Fitness Group (72% of Subjects)	Incidence of Hypertension Inversely related to Treadmill-Tested Fitness	• 4 yr Follow-up • Independent of B. Wt.

C. Final Conclusions

1. Several lines of evidence due in fact provide a theoretical basis for exercise conditioning as a way to lower blood pressure and to reduce the risk of cardiovascular disease:

   For instance, some studies have observed that active individuals have lower blood pressures.

2. However, due to methodological problems, the influence of body weight on blood pressure, and of the equivocal/conflicting results from epidemiological studies, it can not be concluded with absolute certainly that the current practice of regular physical activity prevents, control and/or reduce elevated blood pressure in the population.

IV. HUMAN EXPERIMENTAL STUDIES

A. General Considerations

1. Findings of 22 studies reviewed by Martin & Dubbert (1987):

   a. See Table 5

   b. Categories of human experimental studies:

      1) Uncontrolled studies (absence of a control group).

      2) Partially controlled studies (normotensive exercise control group).

      3) Controlled studies (wait list/nonexercise hypertensives control group).

   c. It should be noted (see Table 5) that as the studies became more controlled, the size of the blood pressure reductions decreased considerably.

Table 5
SUMMARY OF HUMAN EXPERIMENTAL STUDIES*

Features		Uncontrolled Studies	Partially Controlled Studies	Better Controlled Studies
Number of Studies		7	3	6
Number of Subjects		183	68	65
Mean Blood Pressure Reduction (mm Hg)	SBP	-19	-15	-8
	DBP	-14	-9	-6
Control Group		—	Normorensive Exercise	Hypertensive (Wait List/Nonexercise)
Confounding Variables				• Weight • Sodium Excretion

*Data from Martin, J.E. & Dubbert, P.M. The role of exercise in preventing and moderating blood pressure elevation. Biblioteca Cardiológica, 1987, 41. P. 128.

B. Overlook of the Human Experimental Literature

1. See Tables 6, 7, and 8.

2. Conclusions:

a. Most studies results/trend:

1) Moderate antihypertensive effect of exercise training on mild (borderline) to moderate hypertensives of all ages:

a) Limitation:

Evidence sources are mainly from partially controlled studies.

b. Usefulness/values of exercise training:

1) Preventive strategy for high blood pressure.

2) Nonpharmacological treatment of mild to moderate hypertensives.

3) For some patients, it can be used independently and/or with other pharmacological and nonpharmacological therapies.

V. ANIMAL EXPERIMENTAL STUDIES

A. The Rat Model

1. Normotensives rats.

2. Deoxycorticosterone acetate (DOCA) induced rats.

3. Renal hypertensive (RH) rats.

4. Okamoto-Aoki spontaneously hypertensive rats (SHR).

5. Dahl salt-sensitive (SS) and salt-resistant (SR) hypertensive rats.

6. Stroke-prone spontaneously hypertensive rats (SHR-SP).

B. Training Program

1. Mode:

a. Walking.

b. Running.

c. Swimming.

2. Length of training:

From 8 weeks to 1 year.

3. Intensity:

Not specifically defined in the majority of the studies.

C. Findings from Studies

1. See Table 9.

2. Conflicts/inconsistency among investigations:

a. Possible reasons:

1) Variability in the exercise regimen (type, intensity, and training period).

2) Temperature regulation problems.

Table 6

Uncontrolled Studies

REFERENCE STUDY	SUBJECTS				Resting Blood Pressure $\bar{x}$ mm Hg	Antihypertensive Drugs	Exercise Program		EFFECT OF TRAINING					COMMENTS
	Num. of Sub J.	Sex	Age (yr)	Description / Hypertension Classification			Exercise	Period (m = months w = weeks)	$\bar{x}$ BP change (mm Hg)	$\bar{x}$ wt change	$\dot{V}O_{2max}$ ml·min^{-1}·kg^{-1} or l·min^{-1}	Control group	Control group $\bar{x}$ BP changes (mm Hg)	
Johnson and Grover (1967)	4	?	?	Essential	147/88	No	treadmill (35 min/3x wk) Peak HR of 160 bpm	1.5 m	+8/+2	not reported	—			
Rudd and Day (1967)	19	M		Essential; elderly	155/95		walk/jog, etc. (60 min)	5.5 m	−22/−10	Not stated				
Wilmore et al. (1970)	7	M		No description	140/81		walk/jog (12–24 min 3x wk)	2.5 m	−15/−7	Not stated				
Hansen and Nedde (1970)	5	M	30-54	Mixed group	150/86	Yes★	jog (60 min/3x wk)	7 m	−16* / −11*	Not stated	Pre 2.2 ± — Post 2.9 ± — Δ +0.7 (32%) P ??	—		★Two patients PWC 130, PWC 150 increased 100-150 kpm/min * 0.001 <p<0.05
Sannerstedt et al. (1973)	5	M	26-38	borderline Young; hyperkinetic	107 MAP	No	ergometer (60 min/3x wk) Peak HR 150–160 bpm	1.5 m	−3 MAP[a]	−3.2 kg	—	—		[a] Estimated from figure Submax HR reduced
Ressl et al. (1977)	10	M	38-53	Essential	182/99	No	cycling (30 min/5x wk) 70% $\dot{V}O_{2max}$	1 m	−6 / −1	−3.0 kg	—	—		PWC 130 increased from 107 to 128 W ($P<0.01$)
Krotkiewski et al. (1979)	27	F	37	Obese Essential	134/87	No	jog, dance, gymnastics 60 min, 3x wk 10-15 bpm below HR_{max}	3 m / 6 m	−6* / −5* ; −9* / −7*	+1.2 kg (n.s.)	—	—		* 0.001 <p<0.05 Submax HR lower after training ($P<0.05$)
Roman et al. (1981)	27	F	30-69	Essential	182/113	No	walk/jog, cycle, (30 min, 3x/wk) 50-70% $\dot{V}O_{2max}$	27 m	−28* / −16* mm	—	Pre 0.89 ± — Post 1.53 ± — Δ +0.6 (67%) P <0.01	—		* 0.001 <p<0.05 Salt-restrictive diet advised

Table 6
Uncontrolled Studies (cont.)

REFERENCE STUDY	SUBJECTS				Resting Blood Pressure $\bar{x}$ mm Hg	Antihypertensive Drugs	Exercise Program		EFFECT OF TRAINING					COMMENTS
	Num. of Subj.	Sex	Age (yr)	Description / Hypertension Classification			Exercise	Period (m = months w = weeks)	$\bar{x}$ BP change (mm Hg)	$\bar{x}$ wt change	$\dot{V}_{O_2max}$ $ml \cdot min^{-1} \cdot kg^{-1}$ or $l \cdot min^{-1}$	Control group	Control group $\bar{x}$ BP changes (mm Hg)	
Cade et al. (1984)	105	M & F		mild-severe			walk/jog (40.5 min, 7 day/wk?)	3 m		1.9 lb ($\bar{x}$ loss for 60 % who lost = 6.3 lb)				* 0.001 <p<0.05 [a] Estimated from figure
				(a) 58 unmedicated	150/96)[a]				−17.4* / −14.7*					
				(b) 23 medicated	165/ 116)[a]				−28* / −21*					
				(c) 24 discontinued medication	152/ 97)[a]				−22* / −18*					

Table 7
Partially Controlled Studies

REFERENCE STUDY	SUBJECTS				Resting Blood Pressure $\bar{x}$ mm Hg	Antihypertensive Drugs	Exercise Program		EFFECT OF TRAINING					COMMENTS
	Num. of Sub J.	Sex	Age (yr)	Description / Hypertension Classification			Exercise	Period (m = months w = weeks)	$\bar{x}$ BP change (mm Hg)	$\bar{x}$ wt change	$\dot{V}O_{2max}$ ml·min^{-1}·kg^{-1} or l·min^{-1}	Control group	Control group $\bar{x}$ BP changes (mm Hg)	
Barry et al.(1966)	8	M & F	70	Elderly	147/88		ergometer, bicycle (40 min/3x wk)	3 m	$\frac{-20^*}{-5.7^*}$	n.s.		5 NT ($\bar{x}$ 152/93.6)	+3.6/−2.8	Very low intensity of training (heart rate = 130 bpm) * 0.001 <p<0.05
Boyer and Kasch(1970)	23	M	35-61	Essential	159/105	Yes	walk/jog 40-50 min, 2x wk 65% of HR reserve	6 m	$\frac{-13.5^*}{-11.8^*}$	−1.06 kg	—	22 NT (x BP not reported)	0/−6	No data provided on $\dot{V}O_{2max}$ or other training effects * 0.001 <p<0.05
Choquette and Ferguson(1973)	37	M	42	Borderline	136/90	No	calisthenics, jog, volleyball (120 min/2x wk)	6 m	$\frac{-15^*}{-8^*}$	n.s.	—	128 NT ($\bar{x}$ BP = 115/78)	$\frac{-5^*}{-2^*}$	PWC increased from 623 to 734 kpm/min ($P<0.01$) * 0.001 <p<0.05

Table 8
Controlled Studies

REFERENCE STUDY	SUBJECTS				Resting Blood Pressure $\bar{x}$ mm Hg	Antihypertensive Drugs	Exercise Program		EFFECT OF TRAINING					COMMENTS
	Num. of Sub J.	Sex	Age (yr)	Description / Hypertension Classification			Exercise	Period (m = months w = weeks)	$\bar{x}$ BP change (mm Hg)	$\bar{x}$ wt change	$\dot{V}O_{2max}$ ml·min^{-1}·kg^{-1} or l·min^{-1}	Control group	Control group $\bar{x}$ BP changes (mm Hg)	
deVries (1970)	66	M		middle-aged & geriatric	140/76		walk, jog, etc. (30–40 min/3x wk	7 w	−4/−3			32 M HT ($\bar{x}$ BP 140/76)	+1/−2	
Bonnano and Lies (1974)	12 8	M	33-58	Essential normotensives	138/92 123/84		walk/jog 40–55 min 3x wk 70–85% HR max	3 m	−13* −14*	n.s.	Pre 33.4 ± 5.1 Post 35.5 ± 5.1 Δ +2.1 (6%) P <0.005	15 HT ($\bar{x}$ BP = 150/101) 4 NT ($\bar{x}$ BP = 135/87)	−3 −11* −3 −9*	* 0.001 <p<0.05 Control group DBP decreased 11 mmHg (P<0.01)
Deplaen and Detry (1980)	6	M, F	44	Essential	169/108	Yes	walk, jog, bike, calisthenics (60 min/3x wk) 60–70% $\dot{V}O_2$ max	3 m	−1 +3	0.8 kg	Pre 31.8 ± 14 Post 35.9 ± 17 Δ 4.1 (13%) P <0.01	4 HT ($\bar{x}$ BP = 158/113)	−4/−6	Exercise and control groups decreased BP during 3 mo screening period (P<0.02)
Kukkonen et al. (1982)	13	M	35-50	Borderline	145/99		walk, jog, bike x-count ski (50 min/3x wk) 40-66% $\dot{V}O_{2max}$	4 m	−9* −11*	−1.2 kg	Pre 41 ± 7 Post 45 ± 7 Δ +4 (10%) P <0.05	12 HT ($\bar{x}$ BP = 142/104 17 NT ($\bar{x}$ BP = 136/94)	0 −7* −5* −11*	* 0.001 <p<0.05 Control group DBP decreased 7 mmHg (P<0.001)
Nomura et al. (1984)	21	M, F		mild-moderate (a) 7 M (b) 14 M & F	 152/100 153/97		 ergometer 6 min/4x day/ 7x wk same as above + salt restriction	3 w	 −5.8* −9.4* −11.4* −8.3*	 −1.8 kg −1.6 kg		—		* 0.001 <p<0.05
Hagberg et al. (1983)	25	M, F	14-18	borderline/ Essential	137/80	No	jog 40min, 5x wk 60-65% $\dot{V}O_{2max}$	6 m	−8* −5*	n.s.	Pre 43.4 ± 8.3 Post 47.6 ± 9.3 Δ 4.2 (10%) P <0.01	17 HT (BP not reported)	no sig. difference	* 0.001 <p<0.05 Those with diastolic hypertension decreased DBP by 12% (91 to 80 mmHg)
Dubbert et al. (1984)	2	M		MILD	93 DBP (S1) 96 DBP (S2)		S1: jog/ ergometer S1: jog/swim	3 w 10 w	−7 DBP −9 DBP	−1 lb +1 lb		—		

Table 8
Controlled Studies (cont.)

REFERENCE STUDY	SUBJECTS				Resting Blood Pressure $\bar{x}$ mm Hg	Antihypertensive Drugs	Exercise Program		EFFECT OF TRAINING					COMMENTS
	Num. of Subj.	Sex	Age (yr.)	Description / Hypertension Classification			Exercise	Period (m = months w = weeks)	$\bar{x}$ BP change (mm Hg)	$\bar{x}$ wt change	$\dot{V}_{O_2max}$ ml·min^{-1}·kg^{-1} or l·min^{-1}	Control group	Control group $\bar{x}$ BP changes (mm Hg)	
Hagberg et al. (1983)	6 Exp. 6 Con.	M	38	Haemodialysis	155/83	Yes	walk, jog, bike etc. (30 min/ 3–5x wk) 70–85%	4 m	−31/−9*	No Change		6 hemodialysis HT M (x BP 144/80) nonexercise	+2/−2	*Significant Reduction
Duncan et al. (1983)	56	M		44 HT Hyperadrenergic Normoadrenergic	146/94		walk/jog (60 min/ 3x wk)	4 m	hi catech −15.5* (n = 18) −8.1* / lo catech −10.3* (n = 26) −6.4*	no signif. difference	+11% Change	12 HT M ($\bar{x}$ BP 145/93)	−6.3 / +2.9	* 0.001 <p<0.05 Reductions in resting blood pressure were proportional to reductions in catecholamines.
Martin et al.	27	M		10 HT Mild HT	137/95		walk, jog, bike 30 min 3-4 x/wk >65% HR_{max}	10 w	−6.4 / −9.6*	−0.4 kg		9 HT M* (placebo exer.) ($\bar{x}$ BP 135/95)	+.9 / +.8	* 0.001 <p<0.05 *Nonaerobic <60 % HRmax

3) Early death.

4) Limitation in the model of hypertension.

3. Broad findings of long-term experiments:

a. Normotensive rats training:

Lowered average blood pressure.

b. Spontaneously hypertensive rats training:

Delay and reduction (not to normal) of blood pressure.

VI. POSSIBLE MECHANISM OF EXERCISE TRAINING-INDUCED BLOOD PRESSURE REDUCTION (See Figures 1 and 2, and Table 9)

A. Hemodynamic Changes

1. Decreased cardiac output.

2. Decreased total peripheral resistance.

B. Central Adaptations

1. Decreased sympathetic nervous system activity.

2. Increased parasympathetic nervous system activity (activation of vagal tone).

C. Altered Adrenergic Receptor Function

1. Increased sensitivity of beta-2 receptors:

a. Effects:

1) Decreased arteriolar tone, which produces vasodilatation in the coronary, skeletal muscle, pulmonary, and abdominal vicera, blood vessels.

2) Consequently, systemic blood pressure will be reduced through a decrease in the total peripheral resistance.

2. Decreased sensitivity of alpha-1 receptors (mainly to norepinephrine):

a. Effects:

1) Dilation of resistance blood vessels in skin, skeletal muscle, mucous membrane, kidneys and other tissues.

2) Total peripheral resistance and mean systemic blood pressure is reduced.

D. Endocrine-Metabolic Alterations

1. Reduced plasma catecholamines:

a. Lowered circulating epinephrine:

1) Beta-2 adrenergic effect:

A decrease in the plasma concentration of epinephrine causes vasodilatation on the smooth muscle vessels supplying skeletal muscles.

b. Decrease in serum norepinephrine:

1) Physiologic alpha adrenergic blockade:

Table 9

Summary of Studies Using Animals*

Normotensive rats
1. Studies involving more than 260 animals were examined. 2. Swimming and running exercise programs were used with male and female animals. 3. In more than 70% of the investigations, lower resting pressures were associated with the trained groups. 4. Positive and negative results were observed with both sexes and types of training programs. 5. The pressure differences between the trained and nontrained groups ranged from 5-18 mmHg.
DOCA-induced hypertensive rats
1. Studies involving more than 80 animals were examined. 2. Running was associated with lower resting pressures whereas both positive and negative results were obtained with swimming animals.
Renal hypertensive rats
1. Studies involving more than 40 animals were examined. 2. Running and swimming training programs were associated with either no marked differences or with higher resting pressures in the trained animals. 3. Mild training (40-60% $\dot{V}O_{2max}$) was also associated with increases in muscle enzymes and myocardial capillarization as well as higher pressures.
Genetically hypertensive rats (SHR)
1. Studies involving more than 400 rats were examined. 2. Swimming and running exercise programs were used with male and female animals. 3. In more than 75% of the studies, lower resting pressures were associated with the trained groups. 4. The influences of training was observed in both sexes and with both types of exercise programs. 5. The pressure differences between trained and nontrained groups ranged from 7-26 mmHg.
Salt-sensitive and resistant hypertensive rats
1. Studies involving more than 100 animals were examined. 2. Swimming and running programs were used with female animals. 3. Lower resting blood pressures were always observed with the trained rats that were salt-sensitive. This effect was also observed in the majority of studies with the trained rats that were salt-resistant. 4. The pressure differences between the trained and nontrained groups ranged from 7-55 mmHg.
Stroke-prone hypertensive rats
1. One study with more than 75 animals was examined. 2. Voluntary exercise in activity wheels was associated with lower resting pressures during the early stages of training and with higher pressures as the program continued. 3. The pressure differences between the trained and nontrained male animals ranged from 18-22 mmHg.
Non-human primates
1. One study involving 10 animals was examined. 2. A running program was used for 20 weeks. 3. At the end of the experiment, the trained animals had a resting pressure value that was 10 mmHg lower than the controls.

* Reproduced from Tipton, Charles M. Exercise and Blood Pressure In Eckert & Montoye (ED). Exercise and Health. American Academy of Physical Education Papers (No.17). Champaign, IL: Human Kinetics Pub., 1984. P. 37-38.

A reduction in norepinephrine (which acts mainly on alpha receptors as a vasoconstrictor) generates vasodilatation of arterial smooth muscles.

2. Reduced plasma cortisol:

Inhibition of the circulatory response to vasoconstriction (e.g. catecholamines.

3. Decreased fasting and stimulated plasma insulin:

a. Decreased sodium reabsorption in the distal tuboli of the kidney:

1) Effects:

a) Reduced sodium retention and increase sodioum excretion.

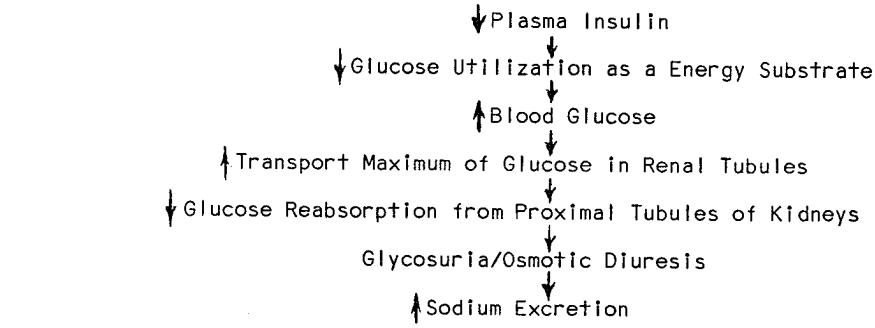
b) Reduce water retention and increase water excretion.

c) Lowering of blood volume.

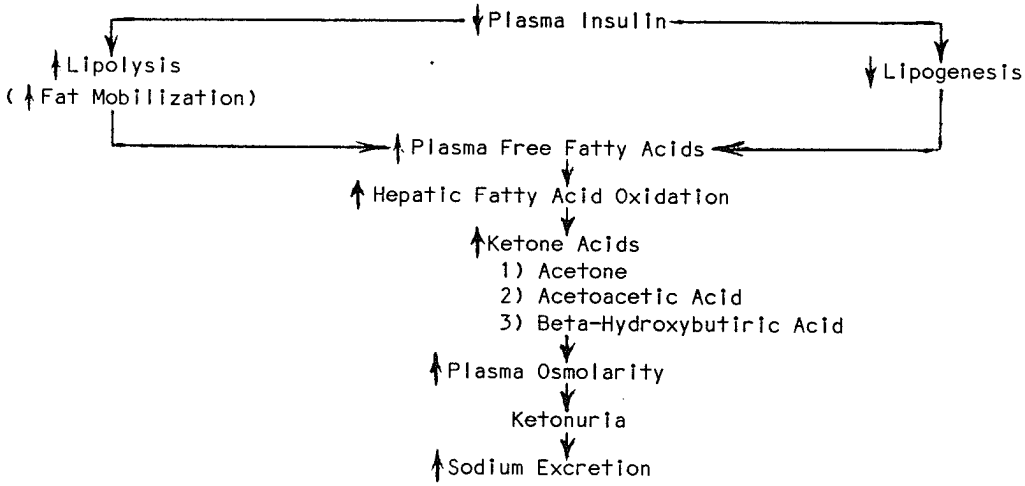
d) Eventually, decrease in cardiac output and blood presure.

2) Possible mediator of insulin-sodium reabsorption blockade mechanism.

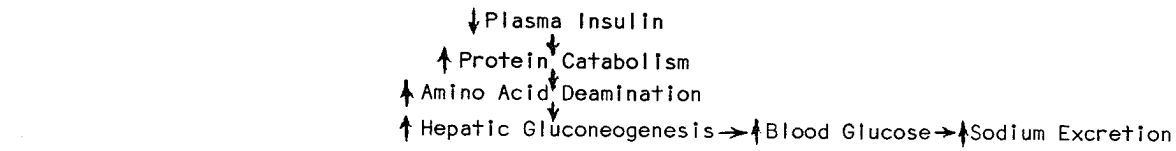
a) Via increased levels of blood glucose:



b) Via increased fat mobilization/ketone acids:



c) Via Protein catabolism:



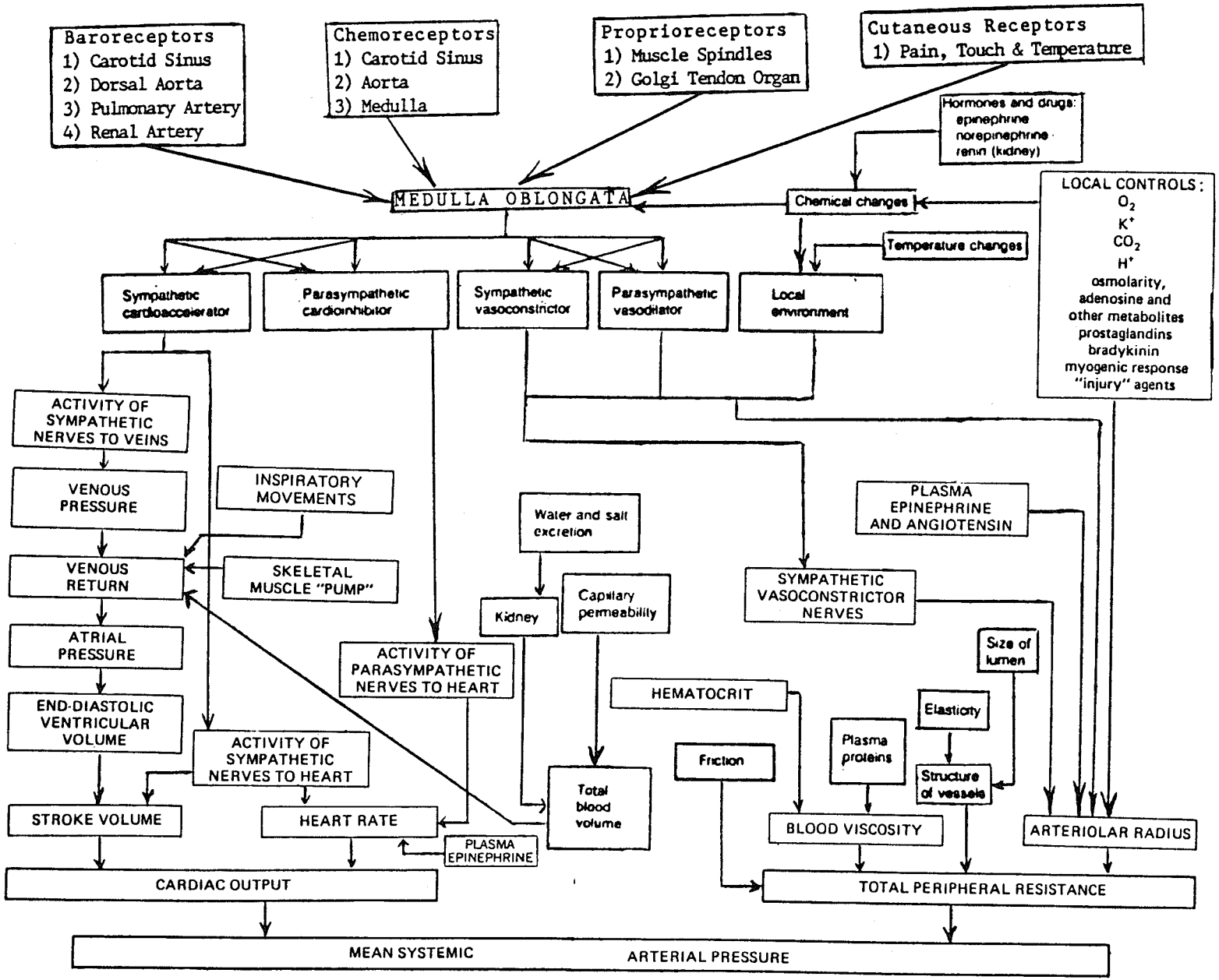


Figure 1

Factors that Determine Systemic Arterial Blood Pressure

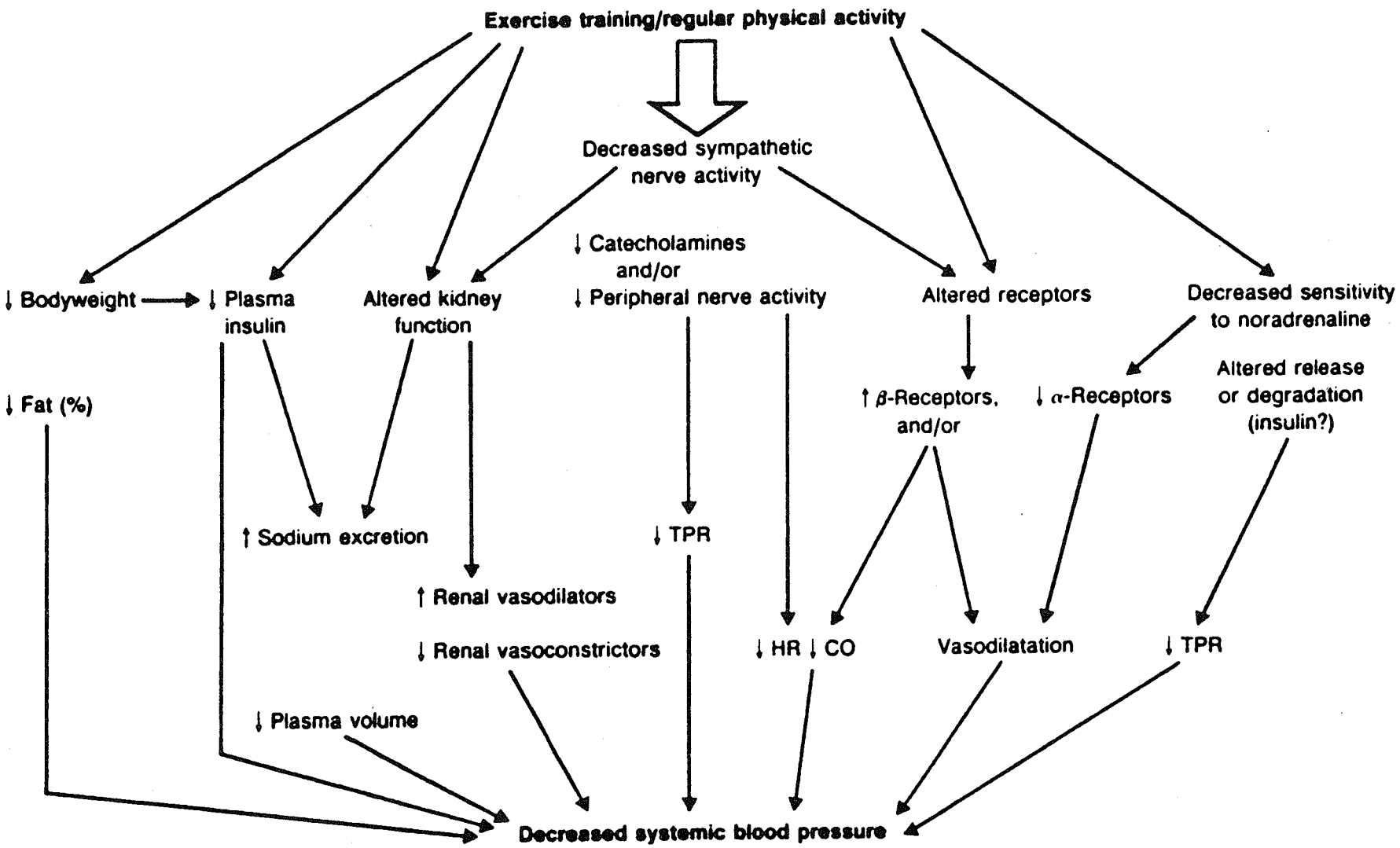


Figure 2

Possible Mechanism for the Antihypertensive Effect of Physical Training

(From Kenney, W.L. & Zambraski, E.J. Physical Activity in Human Hypertension. Sports Medicine. 1:459-473, 1984. P. 467.)

b. Decreased nervous system activity

Lowered catecholamine turnover in sympathetic nervous system synapses.

E. Nutritional Changes

1. Weight loss.
2. Reduced alcohol consumption.
3. A decrease in sodium intake, combined with increases sodium loss (as in sweating) in salt-sensitive individuals.
4. A change in either dietary fats to polyunsaturated fats (increasing vasodilating protaglandins) or high-potassium foods.

F. Behavioral Adaptations

1. Exercise-induced anxiolitic effect.
2. Practice of relaxation techniques.

G. Other Possible Mechanisms

1. Resetting of baroreflexes (decrease in baroreceptor sensitivity).
2. Exercise-mediated stress/catecholamine/renin reduction or expenditure.
3. Exercise/fitness-induced acute and chronic peripheral vasodilatation.
4. Change in myotonic tone.
5. Improvement of neuromuscular relaxation (mediated by a reduction in sympathetic overactivity).
6. Metabolic autoregulation.
9. Decreased extracellular blood volume through:

a. Sweating.

b. Increased renal function and consequently increased fluid and sodium excretion.

10. Some general effect of improved cardiovascular fitness.

TABLE 9

*Postulated Mechanisms of Reduced Blood Pressure Associated with Repeated Physical Exercise**

Hemodynamic
Decreased resting cardiac output
Decreased sympathetic nervous system activity
Decreased total peripheral resistance
Nutritional
Weight loss
Reduced alcohol consumption
Reduced sodium intake
Changes in other dietary factors (fat, potassium, fiber, etc.)
Endocrine-Metabolic
Reduced plasma catecholamines
Reduced plasma insulin
Behavioral
Relaxation techniques

*From McMahon, M. & Palmer, R.H. Exercise and Hypertension. Medical Clinics of North America. 69(1):57-70, 1985 (p. 59).

VII. RISKS FOR EXERCISE-RELATED DEATH

A. Sudden Cardiac Death

1. Possible causes:

a. Cardiovascular morbid events:

1) Cerebral hemorrhage.

2) Pulmonary edema (due to sudden rise of left ventricular end diastolic pressure).

3) Dangerous ventricular arrhythmias.

b. Left ventricular hypertrophy.

B. Evidence from Studies (Pickering, 1987)

1. Exercise-related sudden death studies:

a. Absence of any particular high incidence of hypertension.

b. Common causes of death:

1) Hyperthrophic cardiomyopathy.

2) Anomalous coronary arteries.

2. Risk for cerebral hemorrhage:

a. Death due to subarachnoid hemorrhage had been reported within 24 hours after an isometric activity.

b. Recreational exercise does not appear to place the hypertensive patient at increased risk of an exercise-related cerebral hemorrhage.

3. Patients with coartation that may develop systolic pressures of over 300 mm Hg during exercise:

They are at increased risk for sudden death during exercise caused by aortic rupture and intracranial hemorrhage.

VIII. EXERCISE AND ANTIHYPERTENSIVE MEDICATION

A. Goals/Recommendations (Rost, 1987)

1. Arterial blood pressure should be lowered not only at rest, but also during physical stress.

2. Pharmacological treatment must lower arterial blood pressure, and not influence physical performance.

B. General Concepts (Rost, 1987; Pickering, 1987; McMahon & Palmer, 1985)

1. Most antihypertensive drugs:

a. Worsen (to a varying degree) the adaptational mechanism during exercise.

b. Produce side effects that limit exercise capacity.

2. Ideal antihypertensive drug:

a. Function primarily as vasodilators:

Reduce peripheral vascular resistance without adversely affecting cardiac output, myocardial function, muscle blood flow, or exercise capacity.

C. Recommended Antihypertensive Drugs (Rost, 1987: Pickering, 1987; McMahon & Palmer, 1985; Heikki, 1985)

1. Suitable patients:

   Mild or moderate hypertenves.

2. Beta-blockers:

   a. Indication:

      Initial labile and hyperkinetic phase of hypertension.

   b. Benefits:

      1) Prevent exercise-induced arrhythmias.

      2) Lessen the increase in blood pressure during exercise by diminishing:

         a) The positive inotropic action of exercise upon the heart.

         b) Heart rate.

         c) Cardiac output.

         d) Contractility of heart muscle.

      3) Substances blocking sympathetic tone yield good results in situations raising sympathetic activity such as exercise.

   c. Disadvantages:

      1) Increased probability of arrhythmias:

         This is possible because these drugs exacerbate the increase in serum potassium that occurs during exercise.

      2) Peripheral side effects:

         a) Muscular weakness in legs.

         b) Feeling of coldness in the digits.

      3) Complains of physically active hypertensives:

         a) Fatique.

         b) Muscle aching.

         c) Reduced performance.

   d. Prefered type of beta-blocker to be used:

      1) Cardioselective beta blockers (specific beta 1-blocking properties), and/or

      2) Beta-blockers with ISA (intrinsic sympathomimetic activity).

      3) Reason to use the above beta-blockers:

         a) Less harmful:

            This is true because of peripheral and metabolic effects are milder.

         b) Therefore, these drugs impair exercise tolerance less.

      4) In practice the above reason is not always the best, because higher doses makes insufficient the degree of peripheral beta-blockade

and other problems due to the high central cardiac effect (beta-1):

Therefore, it is advisable to be combined with a diuretic and/or vasodilator (to reduce the dose of the beta-blocker).

e. Maximun target pulse rates will be decreased by beta-blockers:

1) Recommended index to equate exercise intensity:

a) Heart rate

b) Ratings of perceived exertion.

2. Calcium antagonists.

3. Diuretics.

4. Concerting enzyme inhibitors.

IX. GUIDELINES FOR EXERCISE PRESCRIPTION/TRAINING (See Table 10)

A. Candidates

1. Hypertension-prone individuals (regardless of age).

2. Mild (borderline) to moderate hypertensives:

Individuals with resting blood pressures of about 140/90 mm.Hg.

3. Obesity-related hypertension.

4. Some patients with antihypertensive medication.

B. Patients Characteristics to Consider

1. Age.

2. Primary physical condition.

3. Possible concomitant diseases.

4. Possible end-organ damage caused by hypertension.

C. Patients at Risk

1. Hypertensives with left ventricular hypertrophy:

a. Unlikely to be reversed by exercise.

b. Reversed by sympathetic antihypertensive agents (e.g., beta-blockers).

c. Should be treated pharmacologically before begining an exercise program.

2. Patients with severe hypertension:

Should receive antihypertensive drug therapy in order to reduce blood pressure before engaging in an exercise program.

D. Exercise Dosage

1. Data from studies:

a. Insufficient and to varied to suggest an appropriate amount of exerse.

b. Rely on generally accepted principles of exercise prescription (e.g., ACSM).

TABLE 10
Guidelines for Exercise in Patients with Hypertension*

1. Stress testing should be performed prior to starting an exercise program. The objectives are to observe BP control and response, incidence and type of arrhythmias, evidence of ischemia, and exercise capacity.
2. Emphasis should be on dynamic (aerobic) forms of activities, e.g., jogging, swimming, cycling, walking.
3. Exercise in lieu of antihypertensive drugs must not be considered a substitute form of therapy. Exercise and pharmacotherapy should be considered as synergistic components of treatment.
4. Gradual adaptation to training will permit better patient acceptance and will decrease the likelihood for injury. Toleration for extremes of ambient temperature is also related to a slow, progressive adaptation to the activity being performed.
5. Potassium supplements (Elixir of KCl, Slo-K) or potassium-sparing diuretics (amiloride, triamterene) may be necessary for patients receiving diuretics and who exercise in hot climates. Water should be considered the preferred fluid replacement.

*Reproduced from Lowenthal, DT & Broderman, SJ. Exercise in Renal and Hypertensive Disease. In Bove, AA & Lowenthal, DT. Exercise Medicine: Physiological Principles and Clinical Applications. New York: Academic Press, 1983. P. 293.

2. Type:

   a. Dynamic/aerobic, loading large group of muscle:

      1) Walking.
      2) Jogging.
      3) Cross-country skiing.
      4) Biking.
      5) Rowing.
      6) Swimming.

   b. Contraindicated exercises:

      1) Exercises involving isometric (static) contractions:

         a) Weight lifting.
         b) Body building.
         c) Wrestling.

3. Duration, intensity and frequency:

   Half to one hour of training at a pulse rate of 70 percent of age-predicted maximum two to three times per week (Heikki, 1985, p. 92).

4. High intensity sports are not recommended for persons whose pressure is not controlled or who have signs of target organ damage (e.g., left ventricular hypertrophy) (Pickering, 1987, p. 316).

X. SUMMARY AND CONCLUSIONS

Exercise training appears to produce a moderate blood pressure reduction in the borderline/moderate hypertension. When an exercise prescription is designed to the patient in conjuction with other nonpharmacological interventions, it may be possible to reduce or even eliminate drug therapy. Patients with more severe hypertension are generally considered not to be suitable for nonpharmacological therapy, yet, after blood pressure normalization with antihypertensive medication, an exercise program can be prescribed to the patient.

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