

Reduction of serum cholesterol and blood pressure in hypertensive patients by behaviour modification

CHANDRA PATEL, M.R.C.G.P.

General practitioner, Croydon, Surrey

SUMMARY. In a pilot study, 14 pharmacologically treated hypertensive patients were given training in psychophysical relaxation exercises, reinforced by biofeedback instruments, for six weeks. The patients were asked to practise twice a day and also incorporate the exercises into everyday activities. In spite of their unchanged drug schedule, their mean blood pressure (B.P.) was reduced from 170.6/102.5 to 147.9/89.14 ($P = < .001$) while their mean (\pm S.D.) serum cholesterol level was reduced from 241.6 ± 39.19 to 217.1 ± 38.12 mg/100 ml ($P = < .001$). I believe the possibility of one therapy which can reduce two risk factors at the same time should be explored further in a controlled study.

Introduction

The increased morbidity and mortality from coronary heart disease (CHD), particularly in men between 40–60 years of age is a cause for concern¹. The cause of this common disease is not definitely established, but in addition to heredity, widely accepted risk factors are hypertension, cigarette smoking, hyperlipidaemia, diabetes, obesity and physical inactivity^{2,3}. A high dietary intake of fat has been incriminated as the chief cause of hyperlipidaemia, but lower levels of serum cholesterol, as well as clinical coronary heart disease despite a high fat diet could not be explained on the grounds of racial susceptibility or the amount of physical activity⁴. The drug treatment of hypertension⁵ and dietary as well as the drug treatment of hypercholesterolaemia have either not reduced the incidence of coronary heart disease or produced conflicting results⁶⁻⁹.

On the other hand, undue emotional or occupational stresses are often described to precede myocardial infarction.¹⁰⁻¹² Osler¹³ described his angina patients as robust and vigorous in mind and body, and keen and ambitious men, the indicators of whose engines are always set at full speed ahead. Wolf¹⁴ described the coronary-prone person as one who not only meets the challenge by putting out extra effort, but who derives little satisfaction from his accomplishment. Kemple¹⁵ characterised him as an aggressive, ambitious individual with an intense physical and emotional drive, unable to delegate authority or responsibility with ease. Friedman and Rosenman^{16,17} described an overt behaviour pattern, characterised by intense ambition, competitive drive, constant preoccupation with occupational deadlines and a sense of time-urgency in coronary-prone persons. When classified according to their behaviour data, the coronary-prone group was found to have increased levels of blood cholesterol, reduction in blood clotting time, increase in the incidence of arcus senilis, and clinical coronary heart disease.

According to the hypothesised connection between stress and hyperlipoproteinaemia, mental stress causes increased catecholamine release. Both adrenaline and nor-adrenaline, particularly the latter, cause a rise in plasma free fatty acids by lipolysis of adipose tissue. The free fatty acids of the plasma are rapidly incorporated into the triglycerides of the liver and the plasma lipoproteins¹⁸⁻²⁰.

Increase in the release of catecholamine have been found associated with aggression, anxiety, tension and excitement²¹ and in situations like admission to hospital²², flying²³, driving²⁴, stressful interviews²⁵, performance of difficult office work under irritative conditions²⁶, anticipation of uncomfortable situations, mental arithmetic, and exposure to noise.²⁷

In addition to catecholamine, increased levels of free fatty acids and triglycerides were found in the blood during racing driving²⁸, public speaking, sauna baths, and swimming in cold water²⁹. The situations causing more prolonged stress, for example students facing

important examinations³⁰⁻³² and accountants towards the end of the financial year³³ have been associated with increased levels of serum cholesterol and a reduction in clotting time in comparison with the period of minimum stress.

Behaviour modification attained by the regular practice of psychophysical relaxation exercise, taught with the help of biofeedback instruments, not only produced significant reduction in blood pressure in hypertensive patients, but also maintained the reduction over a long term follow-up³⁴⁻³⁶.

In a preliminary controlled study it has been demonstrated that regular practice of relaxation exercises can reduce the magnitude as well as duration of blood pressure elevations in response to physical and emotional or painful stimuli, in hypertensive patients³⁷. If this is one indication of an individual coping better with stress, then other parameters of stress might also be expected to reduce. Based on this hypothesis, the effect of behaviour modification on the level of serum cholesterol has been studied in a pilot project.

Method

Patients

Fourteen hypertensive patients, five males and nine females between the ages of 36 and 74 (mean 58.5) were studied. All but one patient were controlled on antihypertensive drugs. Their average pretrial B.P. was 170.6 mm of Hg systolic and 102.5 mm of Hg diastolic. The pretrial B.P. is an average of three B.P. readings taken in each patient after ten minutes rest. All measurements were taken by an experienced nurse using a random zero machine³⁸. The patients had acted as controls in a previous study³⁶ and were familiar with the instrument, the physician, and the nurse.

Procedure

The patients lay in a supine position after 12 hours of fasting. A standard cuff was applied to the arm and pressure raised between 80-100 mm of Hg, No. 1 needle was inserted into a vein. As soon as the needle was in the vein, the pressure was released and at the same time a blood sample was withdrawn into a dry disposable syringe.

Each aliquot was put into a dry test tube and stored in a refrigerator at once. The samples were taken to the laboratory either on the same day or on the next day. The cholesterol was estimated in autoanalyser II³⁹ in a blind fashion.

The procedure was repeated similarly after six weeks of training and practice in relaxation and meditation. The training was reinforced by biofeedback instruments as described in detail previously³⁶.

Results

The results are shown in the table. Before the relaxation therapy, the cholesterol values ranged from 161.320 mg/100 ml with a mean of 241.6 and a standard deviation (S.D.) ± 39.19 . The standard error for blind duplicate in the laboratory was estimated to be ± 5 mg. After the therapy the cholesterol levels were reduced in 13 out of 14 subjects ranging from 3-60 mg/100 ml, but increased in one by 30 mg/100 ml.

The average reduction for the group was 24.5 ± 21.43 mg/100 ml ($P < .001$). The average blood pressure for the group was also reduced from 170.6/102.5 to 147.9/89.1 while the drugs were kept constant. This reduction in systolic pressure by 22.6 ± 13.78 and in diastolic pressure by 13.4 ± 8.71 mm of Hg are also highly significant ($P = < .001$). However, there is no correlation between the levels of systolic and diastolic pressures and the levels of serum cholesterol as well as between the drop in blood pressure and the drop in the cholesterol levels.

Discussion

Although both cholesterol and blood pressure were reduced significantly for the group, no conclusion can be drawn without the simultaneous study of a comparable control population. Subjects' habits regarding diet, exercise, smoking or alcohol consumption were not closely monitored, and no efforts were made to give them an insight into the relationship between the dietary habits and blood lipids apart from what they may have known already. However, their body weights did not change significantly and they denied any change in their diet during the six weeks period of study when asked in retrospect.

TABLE
EFFECT OF BIOFEEDBACK-RELAXATION AND MEDITATION ON SERUM CHOLESTEROL AND BLOOD PRESSURE

Number	Patients		Cholesterol mg/100 ml			Body weight (lbs)		Blood pressure (mm of Hg) Systolic/Diastolic		
	Sex	Age	Before	After	Difference	Before	After	Before	After	Difference
1	M	35	243	224	-19	142	143	153.3/91.3	150.3/92	-3/+0.7
2	F	39	270	210	-60	157	157	145.0/94	125.3/82	-19.7/-12
3	M	72	164	161	-3	94.25	94	141.0/91.7	117.7/73.3	-23.3/-18.4
4	F	72	250	210	-40	134	134	173.3/106.7	169.3/106.3	-4 / -0.4
5	F	43	184	154	-30	97	98	160.0/100	138 /84.3	-22 /-15.7
6	F	74	249	224	-25	154.5	154.5	164.0/99.3	140.7/82.3	-23.3/-17
7	F	72	320	290	-30	96.5	96.0	197.7/96.7	143.3/82	-54.4/-14.7
8	M	74	271	248	-23	170.5	170.0	210.0/125	173.0/102.7	-37 /-22.3
9	F	52	217	186	-31	200.5	201.5	152.3/95	145.0/90	-7.3/-5
10	F	64	225	255	+30	166.5	163	188.3/115.6	161.7/84	-26.6/-31.6
11	M	42	217	196	-21	164	164	169.0/108.3	146.7/90.3	-22.3/-18
12	F	63	273	265	-8	185.5	183.5	179.3/108	161.7/93	-17.6/-15
13	M	68	256	210	-46	189	192	178.7/99.7	160.3/87	-18.4/-12.7
14	F	49	244	207	-37	108	107.5	177.0/104.3	139.7/99	-37.3/-5.3
Mean		58.5	241.6	217.1	-24.5	147.1	147.0	170.6/102.5	147.9/89.1	-22.6/-13.39
± S.D.			±39.19	±38.12	±21.43					±13.78±8.71

S.E.M.=5.7
 t=4.328 (D.F.=13)
 P=<.001

S.E.M.=3.68/2.33
 t=6.135/5.753
 P=<.001

The first samples were taken in early October and repeated in the middle of December. Serum cholesterol levels were found to be high in winter and spring and minimal in the autumn in a group of patients with chronic heart disease.⁴⁰ The bias due to seasonal variation is therefore against the therapy in this study. The levels of biological values do fluctuate just as symptoms. Higher values tend to come down and lower values tend to go up. Such spontaneous changes are described as regression towards the mean. The majority of values were within the physiological range in this study since the patients were not selected for hyperlipidaemia. Further lowering of values in most cases is therefore more indicative of genuine reduction than the regression towards the mean.

The subjects were all hypertensive and their average blood pressures were reduced. Whether there is any relationship between the levels of BP and that of serum lipid is not known although in this study there is no quantitative relationship between the amount of BP reduction and that of cholesterol. The only patient whose cholesterol level was increased had most reduction in body weight and above average reduction in both systolic and diastolic pressures. The interpretations are further complicated by the fact that most patients were also receiving anti-hypertensive drugs which nevertheless were kept constant. On the other hand, it would seem that not one, but two risk factors can be reduced by a therapy which is at least simple, economical, and free from side-effects. In view of the poor present status⁴¹ of both primary and secondary prevention of coronary heart disease, this therapy deserves full investigation.

Acknowledgements

I thank Dr Tickner, Consultant Chemical Pathologist, Mayday Hospital, for cholesterol estimation and Professor T. R. E. Pilkington, St George's Hospital, for helpful criticisms and W. R. S. North, MRC/DHSS Northwick Park Hospital for statistical analysis. This work was supported in part by a grant from South-west Thames Regional Health Authority.

REFERENCES

1. *British Medical Journal* (1968). Editorial, **3**, 689–690.
2. Dawber, T. R., Kannel, W. B., Revotskie, N. & Kagan, A. (1962). *Proceedings of the Royal Society of Medicine*, **55**, 265–271.
3. Keys, A. (1970). *Circulation*, **1**, Suppl. I.
4. Friedman, M., Rosenman, R. H. & Byers, S. O. (1955). *Journal of Gerontology*, **10**, 60.
5. Breckenridge, A., Dollery, C. T. & Parry, E. H. O. (1970). *Quarterly Journal of Medicine*, **39**, 411–429.
6. Coronary Drug Project Research Group. (1975). *Journal of the American Medical Association*, **231**, 360.
7. Miettinen, M., Turpeinen, O., Karvonen, M. J., Elosuo, R. & Paavilainen, E. (1972). *Lancet*, **2**, 835–838.
8. Research Committee (1965). *Lancet*, **2**, 501–504.
9. Scottish Society of Physicians. Research Committee. (1971). *British Medical Journal*, **4**, 775–784.
10. Russek, H. I. (1967). *Diseases of the Chest*, **52**, 1–9.
11. Jenkins, C. D. (1971). *New England Journal of Medicine*, **284**, 244–255.
12. Jenkins, C. D. (1971). *New England Journal of Medicine*, **284**, 307–317.
13. Osler, W. (1910). *Lancet*, **1**, 697.
14. Wolf, S. G. (1958). *Circulation*, **18**, 287.
15. Kemple, C. (1945). *Psychosomatic Medicine*, **7**, 85–89.
16. Friedman, M. & Rosenman, R. H. (1959). *Journal of the American Medical Association*, **169**, 1286.
17. Rosenman, R. H., Friedman, M., Straus, R., Jenkins, K. D., Zyzanski, S. J. & Warm, M. (1970). *Journal of Chronic Diseases*, **23**, 173–190.
18. Galton, D. J. (1974). *Proceedings of the Royal Society of Medicine*, **67**, 661–662.
19. Elmadjian, F., Hope, J. M. & Lamson, B. (1958). *Recent Progress in Hormone Research*, **14**, 513.
20. Kissebah, A. H. (1974). *Proceedings of the Royal Society of Medicine*, **67**, 665–667.
21. Levi, L. (1969). In: Lader, M. H. *Studies of anxiety*. British Journal of Psychiatry Special Publication No. 3.
22. Raab, W. (1966). *American Heart Journal*, **72**, 538–564.
23. Von Euler, U.S. & Lundberg, U. (1954). *Journal of Applied Physiology*, **6**, 551.
24. Bellet, S., Roman, L. & Kostis, J. (1969). *American Journal of Cardiology*, **24**, 365–368.
25. Hamburg, D. A. (1962). *Proceedings of the Association for Research in Nervous and Mental Diseases*, **25**, 406.
26. Berman, L. & Goodall, M. C. (1960). *Federation Proceedings*, **19**, 154.
27. Raab, W. (1969). *Psychosomatic Medicine*, **30**, 809–818.

28. Taggart, P. & Carruthers, M. (1971). *Lancet*, **1**, 363-366.
 29. Taggart, P., Parkinson, P. & Carruthers, M. (1972). *British Medical Journal*, **3**, 71-76.
 30. Thomas, C. B. & Murphy, E. A. (1968). *Journal of Chronic Diseases*, **8**, 661.
 31. Dreyfus, F. & Czaczkes, J. W. (1959). *Archives of Internal Medicine*, **103**, 708.
 32. Grundy, S. M. & Griffith, A. C. (1969). *Circulation*, **19**, 496.
 33. Friedman, M., Rosenman, R. H. & Carrol, V. (1968). *Circulation*, **17**, 852.
 34. Patel, C. (1973). *Lancet*, **2**, 1053-1055.
 35. Patel, C. (1975). *Lancet*, **1**, 62-64.
 36. Patel, C. & North, W. R. S. (1975). *Lancet*, **2**, 93-95.
 37. Patel, C. (1975). *Clinical Science and Molecular Medicine*, **46**, Suppl. 171.
 38. Wright, B. M. & Dore, C. F. (1970). *Lancet*, **1**, 337-338.
 39. Levine, J., Morgenstern, S. & Vlastelica, D. (1967). *Technicon Symposium*, **1**, 25.
 40. Dunnigan, M. G., Harland, W. A. & Fyfe, T. (1970). *Lancet*, **2**, 793-797.
 41. *Lancet*, (1975). Editorial, **2**, 398-399.
-

PREVENTING DENTAL CARIES

The prospect of preventing caries by immunisation is attractive, and positive results have been obtained in experiments with anti-streptococcal vaccines in monkeys.

However, many problems must be overcome before a suitable vaccine can be developed for use in human beings. From a theoretical point of view, it is difficult to understand how the antibodies produced by the vaccine can reach the site of the lesion on the tooth's surface, and the results obtained in non-human primates cannot be explained.

None the less, they encourage the belief that immunisation against caries in human beings is practicable and research towards this end should be energetically pursued.

REFERENCE

W. H. O. Chronical (1972). **7**, 315.

DRUGS AND CHILD SAFETY

About 16,000 children a year are admitted to hospitals with suspected poisoning from medicines.

The Medicines Commission has now made several recommendations, the principal one being that non-reclosable containers (e.g. strip and blister packaging) should eventually be used for all solid-dose medicines known to present a hazard to children, but that as a matter of priority it should be compulsory for medicines containing substances which seem to be particularly hazardous to be packaged in this way.

The Commission also recommend that aspirin and paracetamol tablets promoted specially for children should be uncoloured and unflavoured and limited to a maximum pack size of 25.