UNIVERSIDAD DE MONTEMORELOS

DIVISION DE POSTGRADO E INVESTIGACION

TERAPIA DE ESTILO DE VIDA PARA PACIENTES EXTERNOS Y SU RELACION CON: COLESTEROL SERICO, PRESION ARTERIAL Y OBESIDAD

PROYECTO PROFESIONAL

PRESENTADO EN CUMPLIMIENTO PARCIAL DE LOS REQUERIMIENTOS PARA OBTENER EL GRADO DE MAESTRIA EN SALUD PUBLICA

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RESUMEN

El Proyecto trata de cambios del estilo de vida y regresión de hipertensión, hipercolesterolemia y obesidad. Discutimos en la sección I. la necesidad de control de los factores de riesgo de enfermedad cardiaca coronaria sabiendo que esta es la causa #1 de muerte en los países desarrolladas. En la sección II. vemos la posibilidad de manejar estos factores de riesgo con cambios de estilo de vida en vez de drogas o cirugía.

En la sección III. hacemos un repaso de la literatura médica de los efectos del cambio de estilo de vida en los factores de riesgo descubriendo las cosas que bajan el nivel de colesterol sérico son: proteínas y grasas de plantas en vez de productos animales; menos grasa saturada, grasa total y colesterol dietético; mas esteroles de plantas y fibra soluble; menos aceites hidrogenados; mas comidas de plantas íntegras, grasa de plantas íntegras monoinsaturadas; comiendo más temprano en el día en vez de más tarde; menos azúcar refinada; mas ejercicio (de baja intensidad como también de alta intensidad); menos café y cafeína, nicotina, cigarrillos y alcohol; mas luz ultravioleta del sol; y resolviendo problemas de: respiración, sueño, estrés, mentales, y sociales como espirituales.

Las cosas que bajan la presión arterial son: dieta vegetariana pura; menos sodio dietético; mas proteína de plantas en vez de productos animales; menos grasa saturada, grasa total y colesterol dietético; menos calorías; comidas ricas en minerales y fibra; mas ejercicio; mas luz ultravioleta del sol; menos alcohol;

resolviendo problemas de respiración, sueño, estrés, mentales, sociales como espirituales.

Las cosas que bajan el riesgo de obesidad son: menos calorías y calorías de grasa y proteína y mas calorías de carbohidratos complejos; menos sodio en la dieta; vegetarianismo, ejercicio; menos drogas que puedan aumentar el riesgo de E.C.C y otras enfermedades (alcohol, nicotina y cafeína), y resolviendo problemas de respiración, sueño, estrés, mentales, sociales como espirituales.

Nuestro estudio (IV.) trata de 97 pacientes que entraron a un programa de de estilo de vida y escuelas de cocina vegetariana pura de 4 semanas con charlas acerca de estos pricipios. Los cambios del estilo de vida reflejan los principios del mensaje de salud de la Iglesia Adventista del Septimo Día. En resumen los cambios son explicadas en la palabra ADELANTE (Aire puro, Descanso, Ejercicio, Luz solar, Agua, Nutricion vegetariana estricta, Temperancia, y Esperanza en Dios). Los principios están basadas en la Biblia como en los escritos de Elena G. de White y también apoyados en literatura científica.

Uno de los cambios fue una dieta vegetariana pura de: frutas, legumbres, cereales integrales, leguminosas, nueces y semillas. La dieta incluye comidas de plantas enteras en vez de productos refinados. Por ejemplo, pan integral en vez de pan blanco; arroz integral en vez de blanco; aguacates, nueces, aceitunas y soya en vez de aceites o productos hidrogenados; frutas en vez de azúcar blanca.

La dieta no tiene grasa ni proteína de productos animales entonces no tiene colesterol y es alta en esteroles de plantas (solamente los productos animales contienen colesterol). Es baja en grasa saturada y rica en grasas poliinsaturadas y monoinsaturadas de plantas enteras. La dieta es alta en vitaminas, minerales y fibra soluble e insoluble. También es alta en carbohidratos complejos y baja en grasa, proteína y sal (como 75% CHO, 15% GRASA Y 10% PRO). Las comidas principales fueron en la mañana y en la tarde y casi nada en la noche.

Se recomienda caminar al aire libre come ejercicio disfrutando del sol. La meta fue de 6-10 kilómetros diarios. Otros ejercicios similares en tiempo y intensidad como los ya mencionados fueron permitidos.

Solo lo necesario de todas las drogas fueron permitidas (como insulina para los diabéticos). No se permitió nicotina, alcohol, y cafeína, tampoco productos que contienen estas drogas (el cigarrillo, café, te, cola, chocolate, etc.)

Se dieron consejos para ayudar con problemas de sueño como el manejo de estres, dormir temprano, evitar comidas abundantes en la noche, y resolver problemas emocionales como espirituales.

Pacientes fueron animados para desarrollar una plena confianza en Dios con oración, meditación y lectura diaria de la Biblia. Se recomendaron otros principios a realisarse diariamente como: beber 8-10 vasos de agua y pasar tiempo en el sol y aire libre. También como aumentar la respiración profunda con postura correcta y ejercicio.

Medimos el colesterol, la presión y el peso antes y después del programa y obtuvimos resultados muy significativos. Las gráficas del apendice demuestran los resultados con los niveles de confianza. La No.1 demuestra los cambios de presión arteral en los 43 pacientes con una presión ≥ 140/90 mm de Hg. Los cambios fueron una **reducción de presión sistólica de 17 puntos o 11.3%**, y **diastólica de 6 puntos o 6.9%** ($p = 0.000$). La No. 2 demuestra los cambios en colesterol en los 61 pacientes con niveles ≥ 200 mg/dl. Los cambios demuestran una **reducción de 35 puntos o 14.5%** ($p = 0.000$). La No. 3 demuestra la presión arterial de los 21 pacientes con niveles de130-139/85-89 mm. de Hg. Vimos una reducción de **8 puntos o 6.2% en la sistólica y 4 puntos o 4.9% en la diastólica** ($p = 0.020$ y 0.077). La No. 4 demuestra el colesterol de los 15 pacientes con niveles 180-199 mg/dl. Hubo una reducción de **19 puntos o 9.9%** ($p = 0.021$). La No. 5 demuestra la presión de todos los pacientes. Vimos una reducción de **10 puntos o 7.4% en la sistólica y 3 puntos o 3.7% en la diastólica** ($p = 0.000$). La No. 6 demuestra el colesterol de todos los pacientes. Vimos una **reducción de 27 puntos o 12.6%** ($p = 0.000$). La No. 7 demuestra el peso de todos los pacientes en libras. Fue una reducción de peso de 8 libras ($p = 0.000$). El tratamiento con el estilo de vida en un programa de 4 semanas fue muy barato (< $100 US c/u) y efectivo en el manejo de los factores de riesgo para la enfermedad cardiaca coronaria.

Aceptado por la División de Postgrado de la Universidad de Montemorelos, en cumplimento parcial de los requistitos para alcanzar el grado de Maestría en Salud Pública.

SINODALES

Dr. J. William Santana _____

Dr. Seiji Matsumoto _____

Mtro. Wilfredo Caldera _____

Aprobado por la Comisión

Fecha 27 de Agosto, 1996

Concedo autorización a la Escuela de Maestía en Salud Pública de la Universidad de Montemorelos para reproducir este proyecto, ya sea en fora parcial o total y únicamente con propósito educativo; con el entendiemiento que de ninguna manera se utilizará para fines lucrativos de alguna persona o institución.

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Fecha

THIS PROJECT IS DEDICATED TO:

The One True God and His Son Jesus Christ "That Thy way may be known upon earth, Thy saving health among all nations." Psalms 67:2

To all who seek healing in the Great Physician, that in Him they "might have life, and that they might have it more abundantly." John 10:10
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TABLE OF CONTENTS

INTRODUCTION

In a day and age of economic crisis and shrinking budgets, we sometimes find that cost gains precedence over quality in the realm of health care for the patient. However, the recent trend in medicine towards lifestyle change gives hope of a low-cost, high- quality system of disease treatment and prevention.

In the past decade or so, research in prevention has focused on the effect of changing various lifestyle factors such as diet and exercise. Most would concede to the benefits of these remedies in the prevention of disease, but some recent studies in this area have explored the idea that "lifestyle medicine" can not only be used to prevent disease but to treat and to reverse it as well.

One such disease which has been the focus of many lifestyle medicine studies is coronary heart disease. Despite some improvement in mortality rates in past years, coronary heart disease remains the #1 killer in the United States as well as in many other industrial nations of the world. It is the leading cause not only of mortality with over half a million deaths annually, but also of morbidity costing the nation over $56 BILLION in combined health-care costs associated with the disease (Circulation, 1994, 1558).

Autopsies of young Americans (average age = 26) in the Korean and Vietnam wars showed that 12-20% had coronary arteries that were 50% blocked or more (Ibid.). This makes the estimate of 56.5 million Americans with cardiovascular disease very believable (American Heart Association 1994). This should urge us to seriously consider early intervention with the management of risk factors.

Two common procedures for the treatment of severe coronary heart disease are percutaneous transluminal coronary angioplasty (PTCA) and coronary artery bypass grafting (CABG).

The estimated costs for a PTCA are as follows: $2,200 for standard stress perfusion imaging and physician interpretation, $9,000 for coronary arteriography, and $15,000 for the PTCA surgery itself gives us a total of about $25,000 NOT including clinical fees. (Circulation, 1994, 1566).

Sadly, after these costly procedures, only 15-25% of patients undergo intensive cholesterol-lowering and risk-management procedures in order to prevent further disease. (Op. Cit. 1565). This makes it easy to believe the 40% restenosis rate for PTCA in 6 months (Op. Cit. 1566).

Examining the CABG procedure, one study (Am J Cardiol 1995 June 1;75(16):1092-5) examined the coronary risk factors of 136 recipients before and 6 months after their operations. They found that despite standard post-operative counseling, mean systolic and diastolic blood pressures significantly increased and a substantial number of patients (59%) continued to exhibit hypertension at 6 months. No significant changes in plasma lipid concentrations were achieved. And at 6 months, one third of the patients exceeded recommended levels for triglycerides, 85% for total cholesterol, and 92% for low-density lipoproteins. In addition, 34% had high-density lipoprotein levels < 40 mg/dl.

This follow-up of post-operative patients is simply a sad commentary of the U.S. population as a whole. An assessment of several major risk factors for coronary heart

disease among Americans reveals that: 60% are not exercising regularly, Over 50% have a serum cholesterol >200mg/dl, 33% are obese (BMI > or = 27.2), 25% are hypertensive (+140/90 mm Hg), and 25% are smokers.

The prevalence of these risk factors is alarming. More alarming is the fact that even when elevated, these risk factors seldom give blatant warning signals. In fact, up to 60% of the 1.5 million heart attacks per year occur without warning symptoms sufficient to incur medical evaluation or treatment (Circulation 1994, 1566). 1/3 of all heart attacks will be the first and last for their unfortunate victims. 1/2 of these will die before even reaching the hospital (Amer Heart Asso 1994).

Perhaps the "warning signals" we should watch for in heart disease are the presence of elevated risk factors! Improving these risk factors would greatly reduce the number of lives claimed each year from this preventable disease. Investigators at Harvard (N Engl J Med 1992; 326:1406-1413) showed the reductions of risk associated with improvement of risk factors: Quitting Cigarette Smoking (50-70% decrease within 5 years), Decreasing Blood Cholesterol (2-3% decrease for each 1% drop in cholesterol), Decreasing High Blood Pressure (2-3% decrease for each 1 mm Hg drop in diastolic pressure), Becoming Physically Active (45% decrease for those who maintain active lifestyle), and Maintenance of Ideal Body Weight (35-55% decrease for maintaining ideal weight vs. obesity).

NON-PHARMALOGICAL MANAGEMENT OF CARDIAC RISK FACTORS

One can easily see the futilitiy involved in performing the standard surgical proceedures for coronary artery disease without sufficient reduction of involved risk factors. The question arises to find an effective, low-cost treatment to reduce the risk factors of coronary heart disease.

As we have mentioned, reduction of elevated total serum cholesterol, blood pressure, as well cessation of smoking and implementing an exercise program are accepted as the four major factors in reducing risk of death from coronary heart disease (J Am Coll Nutr 1995 Oct;14(5):491-6). These risk factors including obesity play a major role in heart disease as well as in many other chronic degenerative diseases.

Three risk factors that can easily be screened for are serum cholesterol, blood pressure and obesity. Progress can also be easily and economically monitored for these three values. Of the countless ways to improve these risk factors, the goal would be to find a simple, rapid and cost-effective way to reduce these numbers to ideal levels.

Recent research suggests looking to lifestyle rather than the laboratory to find the answer to this dilemma. One study (Nutr Health 1994;9(4):237-53) pointed out that our intensive animal raising for meat, eggs and dairy products, as well as our excessive consumption of refined foods has caused many of the chronic degenerative diseases we find in our society today. They state that the answer can be found in tackling the root problem and not by "dispensing designer drugs or opening more hospital beds."

Another author affirms that non-pharmacological management has the potential to reverse mild hypertension, and to diminish fatal and non-fatal heart attack and stroke rates (J Hypertens Suppl, 1994, S71-81). They recommend lowering blood pressure, weight control, alcohol moderation, salt restriction and exercise as measures that appear the most effective in reducing risk of heart disease. Smoking cessation, and diets low in total and saturated fats and rich in fruit and vegetables are also recommended to substantially reduce atherosclerotic and thrombotic disease.

Other researchers (Am J Surg 1995 Apr;169(4):374-8) agree that low-fat diets and exercise are also effective for reducing cholesterol, lowering blood pressure, and preventing heart disease without the side effects associated with surgery and drug therapy. They state that such lifestyle changes are critical to reducing the incidence of heart disease in this country.

Researchers in Spain mentioned that the correlation between diet rich in saturated fat and plasma cholesterol was the first milestone in the field of prevetion of cardiovascular disease followed by the improvement in cholesterol levels and subsequently in CHD mortality. Smoking cessation, physical exercise and control of blood pressure they mention to be effective in reducing risk (Rev Esp Cardiol 1995;48 Suppl 5:3-12).

French authors also relate the effectiveness of lifestyle therapy to the reduction of various risk factors. They discuss the relative risks contributed by each factor and the benefits of their reduction after myocardial infarction. These benefits are comparable to

those observed in primary prevention. However, because of an overexposure to the risk, they say the absolute gains are five times greater.

The impact of diet they mention as one of the most important: in addition to the limitation of polyunsaturated fats and global calorie intake, especially in cases of central obesity, the increase in dietary alpha-linolenic acid and in omega-3 (which are rich in a vegetarian, plant-food diet), has been shown to reduce the risk of myocardial infarction and mortality by up to 70%.

After infarction, the risks of an ex-smoker decrease rapidly by half and become comparable to those of non-smokers in 2 to 3 years. Physical exercise reduces cardiovascular mortality by 20-25% and contributes to better control of risk factors.

The management of some psycho-physiological factors (reaction to stress, hostility) also gives encouraging results. A 10% reduction in total cholesterol leads to a 20% or more decrease in coronary events and a 10% reduction in mortality with a marked "dose-response effect inciting to the reduction of its level to under 200 mg/dl."

The progression of atherosclerosis can be delayed; early lesions, with the greatest risk of rupture and thrombosis, are stabilized and may even regress. A low HDLC concentration should lead one to attempt a rapid reduction of LDLC and control of smoking, obesity and sedentarity. "Its association with hypertriglyceridemia, glucose intolerance, hypertension and central obesity defines the syndrome of insulin resistance which multiplies cardiovascular risk" (Arch Mal Coeur Vaiss 1995 Aug;88 Spec No 3:51-7).

Other researchers mention that the presence of hypertension among men aged 35-64 years increases the risk fourfold. Other independent risks include obesity, a high ratio of total to high-density cholesterol. They give recommendations on sodium intake, cessation of smoking, weight control, exercise as well as other regimens. (J Hypertens Suppl 1994 Jul;12(4):S73-87)

A study of 453 patients showed that weight loss, smoking cessation, and aggressive blood pressure control would be important to potentially improving the increased cardiovascular mortality (Diabetes 1996 Jan;45(1):79-85).

Further research lists a number of risk factors for atherosclerosis, including hypertension, and dyslipidemia. But interestingly they also mention that when possible, thiazide diuretics and beta blockers should be avoided for treating hypertension, because they increase insulin resistance and, in diabetic patients, adversely affect glycemic control. They recommend exercise, weight loss, and cessation of smoking (Postgrad Med 1993 May 15;93(7):99-103, 106-7).

There is an abundance of research suggesting that a change in lifestyle and not advanced technology, drugs or surgery is the answer to controlling risk factors for chronic degenerative diseases. This management does not require long, expensive stays in the hospital either. In fact, the management of these risk factors using outpatient behavioral counseling was found to be a cost-effective strategy for the initiation and maintenance of CVD risk factor reduction in 4 different worksites (Health Educ Q 1995 Feb;22(1):9-19).

In this study we will be presenting the results of 4-week, outpatient lifestyle modification program based in East Pasco Medical Center in Zephyrhills, Florida. The program included a strict vegetarian diet and an exercise program as well as other lifestyle changes that have been shown in medical literature to be effective in improving the risk factors of hypercholesterolemia, hypertension and obesity.

In the following section we will examining various studies showing the effects of specific lifestyle changes on serum cholesterol, blood pressure and obesity.

LITERATURE REVIEW OF LIFESTYLE CHANGE AND ITS IMPACT ON RISK FACTORS

DIET AND RISK FACTORS

Diet and Serum Cholesterol

Beginning with animal studies, we find that dietary L-Arganine has a dramatic antiatherogenic effect in hypercholesterolemic rabbits (J Cardiovasc Pharmacol 1995 May;25(5):710-6). In another study, dietary plant proteins have a beneficial effect on plasma lipids as compared with animal proteins. Isoflavones in plant proteins showed improvements to rhesus monkey's VLDL and LDL concentrations of 30-40%, HDLC concentrations of 15% for females and TC/HDL ratios of 20% for males and 50% for females in 6 months (J Nutr 1996 Jan;126(1):43-50)..

In humans with a hyperlipidemic syndrome (Nutr Rev 1993 Feb;51(2):44-6) vegetarian diets based on soy-protein appeared to be effective in treating the hyperlipidemia.

Another study demonstrated (Clin Nephrol 1993 Dec;40(6):315-20) that a soy diet obtained a significant decrease of hyperlipidemia . The addition of a moderate amount (5 g/day) of fish oil in a randomized cross-over design had no further beneficial effect. They found the dietary manipulation with a vegetarian soy regimen confirmed the beneficial effects on hyperlipidemia in nephrotic patients. Such effects persisted and even improved after 4 months of diet therapy. This may suggest that **Plant proteins lower serum cholesterol, but animal proteins raise it.**

This correlation seems also to be true among plant and animal fats as well as proteins. Thirty-six young, male, Syrian hamsters were fed with test diets containing plant oils and fish oil with and without 0.5% cholesterol for 6 weeks. However, the fish oil group had significantly higher plasma cholesterol than the plant oil groups (J Formos Med Assoc 1995 Dec;94(12):724-31). This may suggest that **Animal fats (even fish, an unsaturated fat) raise cholesterol, but plant fats lower it.**

In other studies, we find the results of the standard dietary counseling on humans (Arch Intern Med 1996 Feb 12;156(3):305-12). 44 hypercholesterolemic men and women (low-density lipoprotein cholesterol [LDLC] level, 3.36 to 5.69 mmol/L [130 to 220 mg/dL]) who were consuming a high-fat diet (> 33% of total calories from fat) were randomly assigned to traditional NCEP (National Cholesterol Education Program) Step I diet (<300mg of dietary cholesterol per day, 30% or less calories from fat, and 8-10% of calories from saturated fat). The results were that after 8 weeks of treatment, total serum cholesterol and LDLC were reduced by 8 and 10% respectively).

Examining a study using the Step II diet we find slightly better results (Am J Clin Nutr 1996 Feb;63(2):234-41). The effects of the NCEP Step 2 diet (< or = 30% of energy as total fat, < 7% of energy as saturated fat, and < 200 mg cholesterol/day). A diet similar to the diet currently consumed in the United States was first consumed (35% of energy as total fat, 14% of energy as saturated fat, 35 mg cholesterol/MJ) for 6 wk and then patients were switched to the NCEP Step 2 diet for 24 wk. Decreases in plasma concentrations of total cholesterol (-14%), LDL cholesterol (-15%), and HDL cholesterol were found (-11%). This may suggest that **Lowering saturated and total fat and dietary cholesterol lowers serum cholesterol.**

We find that the standard counseling gives improvement, but the improvement seems to come over a number of weeks and does not appear to be drastic enough for significant reduction of risk factors.

Dietary cholesterol, saturated and total fat seems to be the emphasis of the standard dietary counseling. We found that in a large study supported this (J Am Coll Nutr 1993 Dec;12(6):676-84). Of 8,679 individuals, dietary cholesterol ($p = 0.038$) was an independent positive predictor of serum total cholesterol. In one study that fed cholesterol to humans currently on a strict vegetarian diet they found that serum cholesterol levels were very difficult to raise and (Am J Clin Nutr 1994 Jun;59(6):1325-31) suggested the cause being the interference of high plant sterol intakes with cholesterol absorption. This may suggest that **Dietary cholesterol raises blood lipids; plant sterols lower them.**

A controlled study in India (J Hum Hypertens 1993 Feb;7(1):33-8) of 145 hypertensives , 72 patients were assigned to take a soluble fiber and a potassium-rich diet while the other patients were used as controls. Increased intake of soluble dietary fiber (47.8 +/- 11.5 vs. 9.5 +/- 0.85 g/day) was associated with a significant decrease in serum total cholesterol (7.9%), triglycerides (7.0%) and an increase in HDLC (4.6%) with a mild decrease in the ratio of total cholesterol/HDLC in intervention patients as compared to controls. This may suggest that **Diets high is soluble fiber reduce serum cholesterol.**

In a Scottish study, researchers stated that (Br J Nutr 1995 Nov;74(5):661-70) trans-fatty acids produced during hardening of oils has been associated with higher cholesterol levels and increased risk of heart disease.

Another article confirmed this finding (Mayo Clin Proc 1995 Jan;70(1):69-79). They stated that the atherogenic potential of hydrogenated polyunsaturated fats is approximately equivalent to that of saturated fats. This may suggest that **Hydrogenated oils (margarines, shortenings, etc.) raise serum cholesterol.**

One study examined the role of monounsaturated fat found in a whole plant food and its effects on serum cholesterol (Arch Inst Cardiol Mex 1995 Jul-Aug;65(4):342-8). Scientists examined the effects of the avocado on plasma lipid concentrations a two-diet trial involving dyslipidemic patients. A diet rich in monounsaturated fatty acids (DRMA) using the avocado as their major source (30% of the total calories were consumed as fat, 75% of the total fat from the avocado), with restriction of saturated fat and less of 300 mg of cholesterol per day was evaluated. Patients were later assigned a low-saturated fat diet without avocado (DRSA). The three daily meals were eaten at a clinical unit. Diets

were four weeks in duration and they were assigned in a crossover design. In some patients both DRMA and DRSA significantly reduced total cholesterol and LDL-cholesterol levels. On other patients DRMA produced a mild reduction on triglyceride levels while DRSA increased them. On HDL-cholesterol concentrations DRMA produced a significant increase in all patients while DRSA did it only in some. They concluded that the avocado is an excellent source of monounsaturated fatty acids in diets designed to treat hypercholesterolemia with some advantages over low-fat diets with a greater amount of carbohydrates. This may suggest that **Diets that include modest amounts of whole-plant food monounsaturated fatty acids lower serum cholesterol.**

These positive findings are not only true of the **fats** in whole plant food but a vegetarian diet in general as well. In facts, after only 3 weeks of a vegetarian diet, some researchers found (Plant Foods Hum Nutr 1993 Jan;43(1):55-61) that serum total cholesterol fell from 6.61 +/- 0.50 to 4.83 +/-0.35 mmol/l ($p < 0.0001$) (That takes the serum levels from "High" cholesterol or ≥ 200 mg/dl to normal values!). Also, atherogenic index, reflecting the balance between low and high density lipoproteins, was reduced by 31% (from 5.74 +/- 0.79 to 3.97 +/-0.60, $p = 0.02$). They concluded that the results suggest that vegetarian diet may have a beneficial influence on the serum level of several lipoprotein-related coronary risk factors.

A controlled study in China (Am J Clin Nutr 1993 Sep;58(3):354-9) made a comparison between the hemostatic and lipid profiles of 55 young Chinese Buddhist vegetarians (23 men, 32 women) and 59 Chinese medical students (20 men, 39 women)

aged 20-30 y. They also demonstrated that there are major beneficial effects of a modern Buddhist vegetarian diet on blood concentrations of cholesterol.

In Europe, a number of studies were done on vegetarians. In one study, (Cor Vasa 1993;35(6):224-9) lipid parameters (cholesterol or C, HDLC, LDLC, triglicerides TG, atherogenic index AI) were estimated in four age groups of vegetarians, 82 males and 80 females, aged 15 to 60 years. The period of consumption of vegetarian food was 1.4 to 1.9 years for adolescents (15-18 years old) or 2.4 to 5.4 years for adults (age groups 19-29 years, 30-39 years and 40-60 years). Lacto-vegetarians constituted one half of females and one third of males. Low levels of TG and C (in the lower half of the reference range), low calculated values of LDLC and AI, as well values of HDLC in the upper region of the standard risk zone or over 1.4 mmol/l (reduced risk level) in males and females were found in **all age groups** and were mentioned as the positive factors of vegetarian nutrition in the prevention of atherosclerosis.

In another study, (Bratisl Lek Listy 1993 Dec;94(12):621-5) 41 vegetarians were examined, namely university students in the age of 18-24 years, 36% of which were lacto-vegetarians and 64% lacto-ovo-vegetarians, 22 men and 19 women. Low values of cholesterol and triglycerides, and an HDLC close to the value 1.4 mmol per liter, as well as a low atherogenic index were some favorable factors in the prevention of atherosclerosis for vegetarians.

In a comparitive study (Ann Nutr Metab 1994;38(6):331-5) of 59 healthy vegetarians aged 19-30 years when compared to non-vegetarians, no incidence of obesity, low levels of cholesterol, LDLC, atherogenic index or triglicerides, HDL cholesterol

levels on the margin of 1.4 mmol/l (boundary level between standard and reduced risk) as well as a higher plasma content of polyunsaturated fatty acids and a higher 18:2/18:1 ratio (P/S ratio) were all found in the vegetarians. All these factors were all favorable consequences of vegetarianism with respect to atherosclerosis prevention.

In a much larger study (Bratisl Lek Listy 1994 Aug;95(8):344-8) authors studied 183 vegetarians of ages 19-60 (4.2 yrs on diet). The detected values of lipid parameters were evaluated as favorable for vegetarians (low values of cholesterol, triglicerides, atherogenic index, LDL-cholesterol, and HDLC was 28-33% (vs 24-26% in nonvegetarians).

A US study using Seventh-Day Adventists examined blood pressure and blood lipids among vegetarian, semivegetarian, and nonvegetarians (Am J Clin Nutr 1994 Jan;59(1):103-9) and found that independent of differences in WHR (waist to hip ratio), the VEGs had significantly lower concentrations of serum total cholesterol (STC), LDL-C, triglycerides, STC/HDL-C, and LDL-C/HDL-C than the NONVEGs. The SEMIVEGs had lipid values intermediate to the VEG and NONVEG groups. Among this group of SDAs, a vegetarian diet was associated with lower cardiovascular disease risk factors than is an omnivorous diet.

A German study (Fortschr Med 1995 Jun 10;113(16):239-42) examined the effects of a vegetarian life style on health and concluded that a vegetarian diet has a positive effect on various risk factors for coronary artery disease. These include lower average body weight, lower total and LDLC levels, and lower blood pressure. Another finding was that vegetarians have roughly 30% reduction in overall mortality.

An interesting study showed that **when** meals are eaten has an effect on blood cholesterol synthesis (Arctic Med Res 1995 Jul;54(3):134-44). The results suggested that meal timing and sleep/wake cycles are more important factors than insulin and glucose-dependent insulinotropic polypeptide in controlling the rhythms of whole body cholesterol synthesis.

Another study considering this effect of meal timing on diurnal rhythm of human cholesterol synthesis (Am J Physiol 1995 Nov;269(5 Pt 1):E878-83) showed that the mean amplitude of the cholesterol rhythm was significantly greater when meals were eaten later (233 +/- 35%), compared to earlier in the day (109 +/- 15%). Their findings indicate that the 24-h variation in cholesterol synthesis is strongly dependent on meal timing.

In both of these studies, there was greater cholesterol synthesis when meals were eaten much later in the day (afternoon and evening) vs. earlier (morning and afternoon). This may suggest that **Eating meals later in the day increases serum cholesterol.**

A hospital-based study designed for weight loss also found that when a low-calorie diet was used, less medication was needed for hyperlipidemia due to reduction of serum cholesterol (Am J Med Sci 1994 May;307(5):325-8). This may suggest that (perhaps due to lower incidence of obesity) **A diet with fewer calories reduces serum cholesterol.**

One study emphasized the role of oxidation of lipids in producing harmful effects (Mayo Clin Proc 1995 Jan;70(1):69-79). They present information about risk factor clustering and the oxidation hypothesis of atherosclerosis attempting to synthesize these

facts into a clinically relevant approach to patients with or at risk for coronary artery disease (CAD).

The levels of both HDLC and remnants of triglyceride-rich lipoproteins and the inherent susceptibility of the LDLC particles to oxidative modification may be as important as the total or LDL cholesterol levels. LDLC must undergo oxidative modification by means of oxygen free radical processes before it becomes atherogenic.

Patients with high levels of oxidative stress include those with risk factor clustering or insulin resistance (or both). Such patients are characterized by hypertension, truncal obesity, hypertriglyceridemia, depressed HDLC levels, and increased insulin levels. They also have increased levels of triglyceride-rich remnant lipoproteins and LDL particles that are characterized by their small dense nature and pronounced predisposition to oxidative modification.

They suggest that biologic antioxidants seem to be promising therapy for the prevention of atherogenesis. They mention that a healthy **diet of fresh fruits, vegetables, and whole grains** is beneficial because it **improves the lipid levels** and provides high levels of natural antioxidants. **The atherogenic potential of hydrogenated polyunsaturated fats is approximately equivalent to that of saturated fats.** They also mention that monounsaturated fat is inherently resistant to oxidation and may be protective against CAD. This may suggest that **Whole plant foods are recommended to improve lipid levels.**

One study that helps to summarize dietary recommendations to lower serum cholesterol stated that (Orv Hetil 1993 Apr 11;134(15):787-96) diet is **fundamental** in

the treatment of primary hyperlipoproteinemias. The author outlines nutritional factors affecting serum lipoproteins as have been mentioned. In the treatment of hypercholesterolemia, the most effective is the reduction suggested by the author is the reduction of dietary saturated fatty acids, it being twice as effective as the reduction of dietary cholesterol, which is necessary as well. The proportion of the saturated, poly- and monounsaturated fatty acids is recommended to be 10-10-10 percent of the overall calorie intake. Such a composition of diet has no disadvantageous effect on the level of HDL cholesterol. Of carbohydrates the consumption of **sugar** and alcohol should be **limited** in the presence of hypertriglyceridaemia and/or obesity. Water soluble dietary fibers may have a cholesterol-lowering effect. The reduction of overall calorie intake is indispensable in hyperlipoproteinemias with obesity. As a result of the diet a 10-20 percent decrease in the level of cholesterol and--in some cases--the normalization of hypertriglyceridemia can be expected. They conclude that persons with or at high risk for CAD should be identified early and aggressively treated with a program that involves lifestyle changes, and alterations in dietary intake. This reaffirms previously mentioned principles as well as recommending that **Refined sugar should be limited to reduce serum lipids.**

Diet and Blood Pressure

One study gives a good overview of hypertension as well as giving some dietary recommendations to treat it (J Am Coll Nutr 1995 Oct;14(5):428-38). They point out that hypertension is the most common chronic disease in the United States (50 million

Americans) and, untreated, results in disability or death due to stroke, heart failure or kidney failure. Fortunately, the results of hypertension can be avoided to a large extent by proper treatment.

Many treatments for hypertension are expensive, especially those using new drugs. Some treatments are also somewhat self-defeating because of the side-effects that they carry. For example thiazides and loop diuretics have negative effects on serum lipids. Also, Beta-blockers tend to lower HDL and raise triglycerides (Ann Int Med 1991; 114:224-237). The standard drug therapy seems to improve one risk factor (hypertension, and even then only 20% of hypertensives have blood pressures less than 140/90 mm Hg) but worsens another risk factor (lipid levels). This is a serious problem since 40% of hypertensives also have high blood cholesterol levels (> 240 mg/dl)! However, one cost-effective treatment found to be effective in some cases is the restriction of dietary NaCl intake.

The study also considers the role of dietary NaCl in the genesis, therapy and prevention of hypertension. They mention that most people can eat as much NaCl as they like; they have good kidneys which, within about 24 hours, excrete the NaCl as fast as it is taken in and nothing happens to blood pressure. But a few, especially those with kidney disease, do not excrete it as fast as it is taken in and blood pressure rises. They are "salt sensitive". Once hypertension is established, the proportion who are "NaCl sensitive" is much higher.

About 60% of people with hypertension respond to a high NaCl intake with a rise in pressure and to NaCl restriction with a fall in pressure and reduction in the need for

antihypertensive medication. These are the same people that respond to diuretics with a fall in blood pressure.

Evidence suggests that very early some hypertensives have a subtle kidney defect which causes them to excrete NaCl and water more slowly, e.g., even before they become hypertensive, black and elderly subjects excrete intravenously administered NaCl more slowly than white and young subjects. How does NaCl retention raise blood pressure? One possibility is that the NaCl retention causes water retention which releases a digitalis-like substance that increases the contractile activity of heart and blood vessels.

Another theory mentioned is that the sodium itself penetrates the vascular smooth muscle cell, causing it to contract. "Salt sensitive" hypertension also responds to increased potassium and calcium intakes, perhaps in part because they increase NaCl urinary excretion (Ibid.)

Another study affirms these findings (J Assoc Acad Minor Phys 1994;5(4):147-51). They mention that although there have been indications since the beginning of this century that blood pressure might be influenced by dietary factors, this has been generally ignored, and the mainstay of hypertension treatment has been the use of pharmacologic antihypertensives. Attention is now being focused, however, on dietary management of hypertension because of the high cost of drug therapy, the adverse reactions associated with some antihypertensives, and the fact that hypertensives treated only by pharmacologic means remain at risk for target-organ damage.

They reveal that the medical literature is replete with evidence that **vegetarian and low-sodium dietary patterns are associated with lower blood pressure levels**.

This implies that if many people could adopt vegetarian and low-salt dietary habits, the prevalence of hypertension would be significantly reduced.

These studies give helpful suggestions and are based on many clinical trials such as one (N Z Med J 1995 Jul 14;108(1003):266-8) that found that after 3 months the 181 participants achieved significant reductions of up to 7 mm Hg in systolic blood pressure for salt restriction alone ($p = 0.03$).

Another (Nat Med 1995 Oct;1(10):1009-16) showed that addition of salt within the human dietetic range caused a highly significant rise in systolic, mean and diastolic blood pressure. They also found that the change reversed completely by six months after cessation of salt. In light of this evidence we might safely say that **Low-sodium diets reduce blood pressure.**

An animal study in France (Clin Exp Hypertens 1995 Oct;17(7):1009-24) showed that dietary L-arginine (an amino acid more abundant in plant foods than animal products) lowers systolic blood pressure in DOCA-salt hypertensive rats, probably through vascular action. It appears that **Plant proteins lower high blood pressure.**

One study showed that, in addition to other factors, a high fat diet raised the BP in 156 men (Hypertension 1996 Feb;27(2):303-7). Perhaps due to raising blood lipids **High-fat diets raise blood pressure.**

In a previously mentioned study (Am J Med Sci 1994 May;307(5):325-8), researchers using a low-calorie diet demonstrated that, in addition to weight loss, medication usage for hypertension was reduced among the patients with reduced obesity. Maybe due to reduction in obesity, **Low-calorie diets help to reduce blood pressure.**

In a trial in India (J Hum Hypertens 1993 Feb;7(1):33-8) of 145 hypertensives, the 72 intervention patients were assigned to take a soluble fiber and a potassium-rich diet which achieved a 7.5/8.5 mm Hg net decrease in mean systolic and diastolic pressures compared with controls.

Also other (Ann Epidemiol 1995 Mar;5(2):108-18) epidemiological studies have found that dietary patterns characterized by high intakes of certain minerals and fiber are associated with low blood pressure when dietary patterns were consumed for 8 weeks. This suggests that **Diets high in minerals and fiber lower blood pressure.**

Standard dietary recommendations have also been helpful to manage hypertension (Am J Clin Nutr 1996 Feb;63(2):234-41). The effects of switching to a NCEP Step 2 diet from the diet similar to the diet currently consumed in the United States showed a beneficial effect on diastolic blood pressure as well as lowering cholesterol after 24 weeks on the NCEP program. This may imply that **Reduction of total and saturated fat as well as dietary cholesterol helps to lower blood pressure.**

Other dietary factors have been shown to affect blood pressure as well (Am Coll Nutr 1993 Jun;12(3):262-9). One study mentions that the vegetarian diet has been associated with lower blood pressure (BP) and examined whether or not long-term adherence (at least 5 years) to a plant-based diet demonstrated this among blacks in America. They also examined the findings in whites. Anthropometric characteristics, nutrient intake, and resting systolic and diastolic BP were measured in older black vegetarians (n = 27, age = 69.3 +/- 1.7 years), black nonvegetarians (n = 37, age = 65.4 +/- 1.2 years), white vegetarians (n = 85, age = 66.7 +/-1.0 years), and white

nonvegetarians (n = 54, age = 65.2 +/- 0.9 years). Older black vegetarians were significantly leaner and exhibited lower average systolic BP (131.4/76.8 mm Hg) and less hypertension than the black omnivores (141.6/76.2 mm Hg), but had significantly higher average BP than either dietary group of older white adults (vegetarians: 120.9/66.7 mm Hg; nonvegetarians: 122.8/67.6 mm Hg). These data suggest that long-term adherence to a vegetarian diet by Americans may afford some protection against hypertension.

Another study (Cor Vasa 1993;35(6):224-9) examined 162 vegetarians, 82 males and 80 females, aged 15 to 60 years. As well as reduced blood lipids, low blood pressure was mentioned as a positive factors as well.

Another study examining (Bratisl Lek Listy 1993 Dec;94(12):621-5) 41 vegetarians aged 18-24 years found that low values of blood pressure was one of the favorable factors in the prevention of atherosclerosis among vegetarians.

One comparative study examined blood pressure and blood lipids among vegetarian, semivegetarian, and nonvegetarians (Am J Clin Nutr 1994 Jan;59(1):103-9). They found that only 16% of the VEGs were confirmed to be hypertensive compared with 35.7% of the SEMIVEGs and 31.1% of the NONVEGs.

Another study listed various effects of a vegetarian lifestyle on health (Fortschr Med 1995 Jun 10;113(16):239-42) and concluded that the vegetarian diet has a positive effect on various risk factors for coronary artery disease one of which was lower blood pressure.

One study from the University of Moscow helps to conclude our section on diet and blood pressure (J Cardiovasc Risk 1994 Oct;1(3):249-54). They reiterate that high

salt intake is a risk factor for essential hypertension in man. In looking for the cause of this phenomena, they found that there is evidence that, in hypertension, intracellular sodium content and univalent cation transport across erythrocyte membranes are changed. It has been proposed that a low-sodium diet has an antihypertensive effect; this may be related to changes in cation fluxes across plasma membranes. In the study, sodium and potassium fluxes and the composition of fatty acids were studied in the erythrocytes of people who had eaten a low-sodium vegan diet for many years ($n = 9$) and in those of controls who had consumed a mixed diet ($n = 11$) to investigate the dependence of blood pressure on dietary factors. They found that both systolic and diastolic blood pressures were lower in vegans than in controls. They found that the passive permeability to sodium ($P < 0.05$), Na+, K+ cotransport ($P < .001$) and the intracellular content of exchangeable sodium ($P = 0.076$) were decreased in the erythrocytes of those who had consumed the low-sodium diet compared with the controls.

They concluded that results show that levels of intracellular sodium and Na+, K+ cotransport activity, which increase in patients with hypertension, decreased in those consuming a low-sodium vegan diet. The study suggests that the risk of essential hypertension was diminished in the vegan participants. This and the previously cited studies support that **A vegetarian, particularly a vegan diet is associated with lower blood pressure.**

Diet and Obesity

Perhaps the first thing that comes to mind when weight-loss is desired is diet. With all the thousands of possible regimens, it is a challenge to find a diet that will be not only effective in weight-loss but in managing the underlying causes of obesity. We will seek to search the medical literature for some principles of diet as they pertain to reducing obesity and maintaining ideal body weight.

One a study examined the effects of a low-calorie diet behavior modification program in weight loss and weight maintenance (Am J Med Sci 1994 May;307(5):325-8). This intervention involved medical supervision as well as group behavior-modification classes, which emphasized long-term lifestyle changes, along with nutrition education. The study was conducted in 1984 and again in 1991, with unselected consecutive patients who had completed this hospital-based, weight-control program 12 to 18 months earlier. Average weight loss was 23.2 kg +/- 1.1 kg, 61% of patients kept off at least 50% of the weight they had originally lost. In addition, medication usage for hypertension, hyperlipidemia, and diabetes was reduced among the patients with reduced obesity. Perhaps an almost obvious lesson is that **Eating fewer calories reduces obesity.**

One study demonstrated the relationship between diet composition and body mass index (Br J Nutr 1995 Dec;74(6):765-73). They assessed the dietary patterns of sixty-four young men and women and found that no differences were found in energy intake between NW (normal weight) and O (obese) adolescents. However, O subjects derived a greater proportion of their energy from proteins (19.8% v. 16.4% for NW subjects) and fats (45.4% v. 38.7% for NW subjects), and less from carbohydrates (34.6%

v. 44.6% for NW subjects). Also, O subjects consumed significantly larger amounts of cholesterol. So we find that it seems to be not only calories that determines weight gain or loss but the **type** calories consumed as well. **Diets lower in fat and protein and higher in carbohydrates help to reduce obesity.**

This seems to make sense as one considers the caloric density of food. Starch or complex carbohydrates has greater volume per calorie than fat or protein. One can think of two slices of bread compared to a pat of butter. They each have the same calories but different volume and fiber contents. Thus, complex carbohydrates tend to give "fill you up" easier leading to greater satiety with less calories.

Also the thermic effect of food in fat vs carbohydrate to should be considered. Per hundred calories of carbohydrate, the body uses 23 calories to digest, absorb, and process the food. Fat however only requires 3 calories per hundred to go from your plate to your waist.

One study using a low-sodium diet (Eur J Obstet Gynecol Reprod Biol 1995 Jul;61(1):63-4) examined, besides the effect on blood pressure, the effect on weight gain and body fat storage. Interestingly, a reduction in sodium intake also caused a significant reduction in the intake of energy, protein, carbohydrates, fat, calcium, zinc, magnesium, iron and cholesterol. However, the reduced intake of calcium, zinc and magnesium in the subjects on the low-sodium diet did not result in significant changes in circulating total calcium, ionized calcium, parathyroid hormone, zinc, alkaline phosphatase or magnesium. They also found that reductions in weight and fat mass gain were more pronounced (3.4 kg ($P = 0.003$) and 1.3 kg ($P = 0.15$), in subjects on the low-sodium diet

vs. those who were not. It appears that through decreased calorie consumption

Low-sodium diets help to reduce weight and fat mass gain.

One study (Fortschr Med 1995 Jun 10;113(16):239-42) concluded that a vegetarian diet has a positive effect on various risk factors for coronary artery disease including lower average body weight.

Another study (Bratisl Lek Listy 1993 Dec;94(12):621-5) in which 41 vegetarians were examined found that no case of obesity was one of many favorable factors in the prevention of atherosclerosis.

A similar study (Cor Vasa 1993;35(6):224-9) examining 162 vegetarians found that among many beneficial results of vegetarianism, absence of obesity was an additional positive factor as well.

A larger study of vegetarians (Bratisl Lek Listy 1994 Aug;95(8):344-8) in which the authors studied 183 vegetarians (Ages 19-60, 4.2 yrs on diet) found that among other favorable factors in the prevention of atherosclerosis a vegetarian diet is associated with the absence of obesity. Again it appears that **A vegetarian diet reduces risk of obesity.**

EXERCISE AND RISK FACTORS

Exercise and Serum Cholesterol

A recent medical article (Bull Acad Natl Med 1995 Oct;179(7):1417-26; discussion 1426-8) pointed out that a gradual decrease in energy output for adults in the Western countries has been observed through the 20th century. The mechanization in industrial societies results in a decline of customary physical activity and consequently in

the potentially vicious spiral of inactivity leading to deconditioning and thence, via loss of physiological capacity, to a further reduction in activity.

A sedentary lifestyle has injurious effects on the health of individuals and especially increases the risks of cardiovascular disease. Furthermore there is a supposed relation between inactivity and hyperlipidemia. In contrast, many papers have shown that high levels of physical activity have been associated with a diminished occurrence of hypertension, coronary artery disease, etc.

Research in Japan (Nippon Koshu Eisei Zasshi 1994 Jan;41(1):46-55) agreed that atherogenic index elevated with a lifestyle of limited or light physical activity in addition to other factors.

One study in Austria showed that (Cardiovasc Risk 1995 Aug;2(4):316-22) exercise decreases postprandial lipidemia and, in turn, increases levels of HDLC. They demonstrated the favorable effects of exercise on lipoprotein metabolism.

A study (Chest 1996 Jan;109(1):52-6) of 314 patients with recent cardiac events showed promising results. A 3-month (36-session) formal, outpatient phase 2 cardiac rehabilitation and supervised exercise training program. After cardiac rehabilitation, there were significant improvements in lipid levels: LDL-C (-4%; $p = 0.07$), HDL-C (+6%; $p < 0.001$), and LDL-C/HDL-C ratio (-10%; $p < 0.01$). Given these conclusions, one may safely suggest that **Exercise helps to improve blood lipids.**

One question to ask is: What intensity of exercise is desirable for improved lipid profiles? One medical study examining the effects of 4-week ergometry training at an intensity of 30% versus 50% of maximum performance found an interesting answer.

The authors (Wien Klin Wochenschr 1995;107(6):195-201) investigated the effects of bicycle ergometer training (BET, weekly training time: 120 min, consisting of 8 units of 15 min). In a center for cardiovascular rehabilitation, two groups of 15 patients with coronary heart disease and/or hypertension and/or hyperlipidemia underwent a four-week training period.

All patients avoided other training exercises, they all had the same physiotherapy such a massage and the same diet of 800-1200 kcal/day. Current medication was kept constant. The main differences between the groups was the intensity of the training regimes: 30% versus 50% of the individual maximal physical working capacity (PWC), as determined by symptom-limited bicycle ergometry and controlled by an individual training heart rate. The higher intensity of training led to a highly significant increase of 16% in PWC ($p < 0.001$), whereas the group with the lower training intensity improved by only 5% ($p < 0.05$). The main effects were reduction of body weight (both groups of about 6%), lipid profiles (in cholesterol and LDLC levels in both groups), heart rate and blood pressure (significant decrease in both groups). But there were **no significant differences between the two groups**. The more intensive aerobic training, which was more effective on PWC, **did not reveal better results on fat metabolism, heart rate or blood pressure**. So although PWC was improved with higher intensity, in terms of lipid profiles, **Low-intensity (as well as high-intensity) exercise is effective in reducing serum cholesterol and blood pressure.**

Exercise and Blood Pressure

A French study examined physical training and blood pressure in a recent article (Bull Acad Natl Med 1995 Oct;179(7):1471-80; discussion 1481). They found that BP rises during exercise and lowers in the post-exercise period. Regular physical training results in a significant lowering of BP at rest as long as the training is continued. Moreover, exercise training results in a BP lowering during exercise which is greater in hypertensive patients than in normotensive subjects. A favorable effect is observed also on ambulatory blood pressure.

The mechanisms of training-induced changes of BP are not sufficiently known. The exercise training seems act on systemic vascular resistance, plasma catecholamine, PGE2 and taurine levels, and the renin-angiotensin-aldosterone system.

They conclude that adequate physical training can reduce BP, it can be considered a non-pharmacological treatment of hypertension.

A previously mentioned study (Bull Acad Natl Med 1995 Oct;179(7):1417-26; discussion 1426-8) emphasized that the sedentary life has injurious effects on the health of individuals and especially increase the risks of cardiovascular disease. In contrast, they showed that high levels of physical activity have been associated with a diminished occurrence of hypertension, coronary artery disease as well as other chronic degenerative diseases.

A study in New Zealand (N Z Med J 1995 Jul 14;108(1003):266-8) in which subjects walked for 40 minutes three times per week and blood pressures were assessed at three and six months found favorable results. The 181 participants who completed the

study found that significant reductions of up to 7 mm Hg were attained in systolic blood pressure at 3 months for brisk walking ($p = 0.04$).

A German study previously mentioned (Wien Klin Wochenschr 1995;107(6):195-201) investigated the effects of bicycle ergometer training in a center for cardiovascular rehabilitation. The 30 patients with coronary heart disease and/or hypertension and/or hyperlipidemia underwent a four-weeks training period. After 4 weeks they found significant decreases in heart rate and blood pressure. It appears that **Exercise helps to reduce elevated blood pressure.**

Exercise and Obesity

Perhaps the next thing after diet that comes to mind in terms of weight loss would be exercise. A recent medical article (Bull Acad Natl Med 1995 Oct;179(7):1449-57; discussion 1458-9) discussed exercise. They mentioned that physical exercise induces a mobilization of energy substrates in order to furnish fuel for metabolic pathways. A part of this regulation is under hormonal influences. A decrease in plasma insulin and an increase in catecholamines play a central role. It results in enhanced lipolysis (or fat burning)!

Another article in the same journal (Op. cit., 1417-26; discussion 1426-8) implicated the sedentary life as having injurious effects on the health of individuals and specially increasing the risks of cardiovascular disease. They support the relation between inactivity with obesity as well as other risk factors. They also mention that

those whose lives are characterized by high levels of physical activity show less risk of coronary heart disease.

Other articles support the correlation (Mayo Clin Proc 1994 Oct;69(10):997-9) that when combined with other lifestyle changes, exercise is important for the overall reduction of body weight and subsequent maintenance of weight loss.

Another study (Am J Epidemiol 1995 Jun 15;141(12):1117-27) concluded that the steps to obtaining reduction of obesity includes change in lifestyle, nutritional education and modification and increase in exercise. These changes are important for long-term success.

Many studies affirm these principles (Eur J Clin Nutr 1995 Nov;49(11):824-31). A sample of 358 male subjects who participated in phase 1 of the Quebec Family Study was examined. The results indicate that the adherence to a lifestyle characterized by sedentariness and other lifestyle habits promotes fat gain, particularly in the trunk area.

Another article (Chest 1996 Jan;109(1):52-6) examined 116 obese (body mass index [BMI] ≥ 27.8 kg/m2 for men and ≥ 27.3 kg/m2 for women; mean, 31.2 +/- 3.2 kg/m2] and 198 (mean BMI, 24.6 +/- 2.1 kg/m2) nonobese patients with recent cardiac events. A 3-month (36-session) formal, outpatient phase 2 cardiac rehabilitation and supervised exercise training program was implemented. After cardiac rehabilitation, there were modest **reductions in** the prevalence of **obesity** (116 patients [37%] vs 104 patients [33%]) and severe obesity (BMI ≥ 35 kg/m2 [3.5 vs 2.5%]) and BMI (-3%; $p < 0.0001$). **Exercise helps to reduce obesity.**

Sunshine and Blood Pressure

Perhaps a less-thought-of remedy for chronic degenerative diseases is sunlight. A recent article (Bone 1995 Aug;17 (2Suppl):107S-111S) helps to explain some the mechanisms by which sunlight is beneficial. They mention that vitamin D is absolutely essential for the maintenance of a healthy skeleton. In deficiency of vitamin D, children develop rickets and adults worsen their osteoporosis and develop osteomalacia. Casual exposure to sunlight is main source of vitamin D for most people.

During exposure to sunlight, ultraviolet B photons photolyze cutaneous stores of 7-dehydrocholesterol (a pathway for cholesterol metabolism) to previtamin D3. Previtamin D3 undergoes a thermal isomerization to form vitamin D3. Increased skin pigmentation, changes in latitude, time of day, sunscreen use, and aging are a number of factors that have an influence on the cutaneous production of vitamin D3. Once vitamin D3 is produced in the skin or eaten in the diet, it must be hydroxylated in the liver and kidney to 1,25-dihydroxyvitamin D3 [1,25(OH)2D3].

Today it is now recognized that a wide variety of tissues and cells, both related to calcium metabolism and unrelated to calcium metabolism, are target sites for 1,25(OH)2D3. This substance stimulates intestinal calcium absorption and helps stem cells to mobilize calcium stores from bone.

Non-calcemic tissues that possess receptors for 1,25(OH)2D3 respond to the hormone in various ways. Of great medical interest is that it is a potent antiproliferative and prodifferentiation mediator. As a result, 1,25(OH)2D3 and its analogs can be used in treating clinical disorders like as rheumatoid and psoriatic arthritis; type I diabetes;

hypertension; cardiac arrhythmias; seizure disorders; cancers of the breast, prostate, and colon; some leukemias and myeloproliferative disorders; chemotherapy-induced hair loss; and skin rejuvenation as well as skin diseases like psoriasis and ichthyosis.

Animal studies have shown favorable results (Hypertension 1994 Jun;23(6 Pt 2):894-8). Deendothelialized rings of rabbit aorta relax after exposure to UV light because of release of a relaxing factor that is similar if not identical to nitric oxide. Furthermore the results suggest that UV irradiation induces relaxation in aortic smooth muscle that is greater in **hypertensive** than normotensive rats and is greatly enhanced after addition of inhibitors of nitric oxide production.

One study in Germany examined the behavior of lipids, other serum parameters and cardiovascular functions in ultraviolet therapy (Hautarzt 1994 Oct;45(10):702-7). Using ultraviolet light similar to that found in sunlight during a period of 3 years, four controlled clinical studies in 34 patients with hypercholesterolemia and 40 healthy persons were conducted. The results revealed an increase of HDL cholesterol at the end of the UV treatements, increase of serum 25(OH)D3 levels, decrease of serum triiodothyronine, decrease of serum lactate, decrease of resting pulse rate, decrease of recovery pulse rate, and decrease of systolic blood pressure. It appears that the UV portion of **Sunlight helps to reduce blood pressure as wells as improve lipid profiles.**

LEAGAL DRUGS AND RISK FACTORS

Coffee and Serum Cholesterol

Many studies have examined the effect of coffee consumption on serum cholesterol levels (J Manipulative Physiol Ther 1993 Jun;16(5):327-35) and have found that serum cholesterol levels increase with increasing coffee consumption.

Another study (Fam Med 1993 Mar;2(3):317-22) found that large intake of coffee may increase total cholesterol levels; urinary and bladder cancer are associated with coffee consumption as well. More than four cups per day may be associated with increased risk of **acute myocardial infarction**. Also, there appears to be an association between ingestion of coffee and incidence of duodenal ulcer and ulcerative colitis. Increased coffee consumption by pregnant women appears to decrease fetal birth weight. Fetal heart rate, respiration, and both maternal and fetal anemia are increased with coffee consumption.

Other research (Psychosom Med 1994 May-Jun;56(3):260-6) showed that after adjustment for other risk factors, higher **caffeinated beverage intake was associated with higher LDLC and a higher total/HDLC ratio, both indicative of greater coronary disease risk**.

A large study (Clin Epidemiol 1995 Oct;48(10):1189-96) of 2109 healthy nonsmokers aged 25-65 found that a 2 mg/dl total cholesterol increase was associated with an increase of one cup of regular coffee per day ($p < 0.001$). It appears that **Coffee may raise serum cholesterol.**

Smoking/Nicotine and Serum Cholesterol

Perhaps one of the most beneficial things one can do for their own health is to stop smoking. Many studies have examined the impact of smoking on coronary heart disease. One study (J Clin Endocrinol Metab 1995 Jul;80(7):2181-5) found that cigarette smoking has been associated with glucose intolerance and dyslipidemia.

Young healthy male volunteers smoking more than 10 cigarettes/day for more than 5 yrs (n = 14) were compared with nonsmokers (n = 13) matched for age, sex, body mass index, alcohol consumption, physical activity, as well as family history for hypertension, diabetes, obesity, and coronary heart disease. Some interesting differences were found between the two groups: total cholesterol (4.87 +/- 0.15 vs. 4.27 +/- 0.16 mmol/L; $P < 0.02$), low density lipoprotein cholesterol (3.05 +/- 0.19 vs. 2.43 +/- 0.16 mmol/L; $P < 0.02$), and apolipoprotein B concentrations (1.09 +/- 0.11 vs. 0.83 +/-0.03 mmol/L; $P < 0.03$) were **all higher in smokers** than in nonsmokers.

In addition to higher total cholesterol, in one study (Hum Genet 1996 Feb;97(2):156-62), the HDL cholesterol decreasing effect of smoking was demonstrated.

Other researchers performed a prospective study of the effects of changes in smoking habits on a number of lifestyle related variables (J Clin Epidemiol 1995 Sep;48(9):1159-66). They examined 987 employees in Israeli industry who underwent health screening and were re-examined an average of two and a half years later. Those who had quit smoking showed increases in both serum HDL cholesterol (HDL-C) and a slight decrease in serum triglycerides.

Some researchers (QJM 1995 Jul;88(7):503-8) examined 1048 subjects aged 25-69 years. Compared with non-smokers, current smokers had higher plasma concentrations of LDLC by 0.2 mmol/l or 6%, total/HDLC ratio by 0.5 or 13%, triglyceride by 0.14 mmol/l or 13%, and waist:hip ratio by 0.02 or 3%, but smokers demonstrated lower HDL cholesterol by 0.13 mmol/l or 9% and HDL2 cholesterol by 0.07 mmol/l or 13%. The study concluded by stating that cigarette smoking is associated with adverse changes in lipoprotein levels.

Even in a study (Prev Med 1995 Jul;24(4):357-62) that examined smokeless tobacco users, ages 18-65, who tried to quit tobacco using nicotine gum, they found that triglycerides were higher for the nicotine gum group than the placebo group ($P = 0.031$), with triglycerides decreasing between Weeks 4 and 8 in those who had quit chewing the smokeless tobacco. **Smoking and nicotine are associated with higher total cholesterol and lower HDL cholesterol.**

Alcohol and Serum Cholesterol

In an animal study (Biophys Acta 1996 Jan 19;1299(2):160-6), it was found that fatty acid ethyl esters are a family of non-oxidative metabolites of ethanol present in many tissues after ethanol consumption.

Another study showed that (Endocr J 1995 Oct;42(5):705-12) in adult wistar rats, free cholesterol and diacyl glycerol were increased with alcohol consumption. Similar research (Tijdschr Geneeskd 1996 Jan 6;140(1):34-6) showed the effects of chronic ethanol exposure in raising total cellular cholesterol level in rats.

In humans, the findings (Minerva Gastroenterol Dietol 1995 Sep;41(3):237-45) seem to be similar. The differences that appear between wine consumers (SE = 29) and abstemious males (NE = 51) among other factors were triglycerides which were higher in wine consumers.

Another study (Eur J Clin Invest 1995 Jun;25(6):390-5) concluded that due to the fact that wine raises plasma TG, it also causes changes in plasma cholesterol metabolism and lipoprotein composition. It appears that **Alcohol contributes to raising serum cholesterol levels.**

Alcohol and Blood Pressure

There are a number of ways that alcohol is believed to raise blood pressure. One article (Clin Exp Pharmacol Physiol 1995 Sep;22(9):655-7) discussed one possibility: Changes in membrane fluidity have been proposed to contribute to the pathogenesis of ethanol-induced hypertension possibly through changes in membrane lipid patterns. In a controlled trial they documented ethanol-induced changes in blood pressure and composition of membrane lipids in ethanol-fed rats. Systolic blood pressure increased significantly in the ethanol-treated rats (9.3 mm Hg, s.e.m. 2.9) compared with the control group (-1.3 mm Hg, s.e.m. 2.6).

In an article examining humans, (Alcohol Alcohol 1995 Sep;30(5):581-9) portal hypertension, which is more common in Europe and the USA, is associated with alcohol. Also, some researchers mentioned (Acta Med Austriaca 1995;22(3):43-5) alcohol abuse as the cause of "resistant hypertension".

In another study (Addiction 1995 Nov;90(11):1479-85) examining alcohol and hypertension, 330 ambulatory care patients were assessed using a detailed interview including laboratory tests. After 2-3 years, 250 (76%) subjects were reassessed and they found alcohol was implicated in high blood pressure. This was supported by further research (Minerva Gastroenterol Dietol 1995 Sep;41(3):237-45) in which the differences appear between wine consumers (SE = 29) and abstemious males (NE = 51) were examined. They found that and arterial pressure was higher in SE ($p < 0.05$). It appears that **Alcohol consumption is associated with higher blood pressure.**

Smoking/Alcohol/Caffeine and Obesity

As has already been mentioned (J Clin Endocrinol Metab 1995 Jul;80(7):2181-5) cigarette smoking has been associated with increased upper body fat deposition, as estimated by the waist to hip ratio. In fact, in a study (QJM 1995 Jul;88(7):503-8) of 1048 persons aged 25-69 years, compared with non-smokers, current smokers had higher waist/hip ratio by 0.02 or 3%.

This association may be contested by many who point out that since nicotine is a stimulant, it would aid the obese in losing significant amounts of weight. However, one study (Health Psychol 1995 Mar;14(2):116-23) examined the effect of smoking on Resting Energy Expenditure (REE) immediately, 10 minutes, and 30 minutes after smoking. They found that obese subjects showed a 3.9% and a 0.7% increase but then the REE showed a 0.8% **decrease from baseline** during this postsmoking phase. They

concluded that the results have potential implications for discouraging obese persons from taking up smoking and intervening among those who already smoke.

Similar results (Int J Obes Relat Metab Disord 1994 May;18(5):329-32) were discovered with caffeine and other stimulants in a weight-loss program. Weight loss (7.8 +/- 0.63 kg) and loss of fat mass (6.2 +/- 0.49 kg) did not differ significantly between two groups (one group with stimulants including caffeine, and one group without the drugs).

A study (Minerva Gastroenterol Dietol 1995 Sep;41(3):237-45) comparing wine consumers (SE = 29) and abstemious males (NE = 51), found that daily caloric consumption (kcal/day) were higher in wine consumers ($p < 0.05$)

Another study confirmed these findings (Eur J Clin Nutr 1995 Nov;49(11):824-31) with a sample of 358 subjects who participated in phase 1 of the Quebec Family Study. Their results indicate that the adherence to a lifestyle characterized by high-alcohol intake as well as other lifestyle factors promotes fat gain, particularly in the trunk area. **Drugs like nicotine, caffeine and alcohol do not have positive effects on obesity.**

Breathing Disorders and Risk Factors

One article (Chest 1993 May;103(5):1336-42) found that upper body **obesity, hypertension**, and impaired glucose tolerance **occur more frequently** than expected **in patients with breathing disorders**. They concluded that there is increased cardiovascular risk associated with Obstructive Sleep Apnea (OSA).

Another similar study examined the impact of obstructive sleep apnea and sleepiness on metabolic and cardiovascular risk factors (Int J Obes Relat Metab Disord 1995 Jun;19(6):410-8). They examined 3034 participants and found that self-reported loud snoring and observed breathing pauses (high likelihood of OSA) was associated with increased frequency of dyspnea, admissions to hospital with chest pain, **myocardial infarction**, **high blood pressure**, fasting insulin, fasting **triglyceride** (women only), uric acid (women only) after adjustment for body fat distribution and other potential confounders.

A study in Israel (Isr J Med Sci 1995 Sep;31(9):527-35) found that OSA may be a major cause of **hypertension**--responsible for about 30% of all cases. Successful treatment of OSA by any means has been shown in most studies to cause significant reductions in blood pressure at all times of the day. **Breathing disorders like OSA contribute to cardiac risk.**

Sleep Disorders and Risk Factors

A recent study (J Cardiovasc Risk 1995 Jun;2(3):247-54) showed that men with myocardial infarction reported more problems with sleep also women with myocardial infarction reported significantly **more problems with sleep than their controls**. The study concluded that **sleep problems are related to myocardial infarction** independently of conventional biomedical risk factors.

Another article (Sleep 1993 Aug;16(5):457-66) examined 19 adults (mean age 40 years; 58% female) and found that 44% were overweight (i.e. > 20% excess weight) from

sleep-related eating. They also found that nightly sleep-related binge eating (without hunger or purging) had occurred in 84% of patients. **Sleep disorders are related to increased cardiovascular risk.**

Mental/Spiritual Components and Risk Factors

With all the aspects of our lives that can change cardiac risk, one cannot ignore the mental/spirital component of man. In recent research (Biol Psychol 1995 Aug;41(1):69-81) the relationship between habitual anger coping styles, especially anger expression in a socially assertive manner and serum lipid concentrations was examined in 86 healthy subjects.

Habitual anger expression was measured by the Muller Anger Coping Questionnaire (MAQ) on four scales: Aggression, Controlled Affect (not "exploding" but addressing problems in an assertive manner), Guilt, and Social Inhibition.

A positive correlation between serum cholesterol and Aggression was found, as was a negative correlation between LDL/HDL ratio and Controlled Affect. This study demonstrated that unfavorable lipid profiles are linked to a predominantly aggressive anger coping style, whereas favorable lipid profiles are associated with more socially assertive anger coping (Controlled Affect). Thus the expression of anger in a socially assertive manner can be seen as health promoting.

A larger study (Health Psychol 1995 Nov;14(6):563-9) examined personality predictors of hypertension incidence in 838 community-residing men from the Normative

One of these examined the effect of short-term cardiovascular conditioning and low-fat diet on myocardial blood flow and flow reserve (Circulation 1995 Jul 15;92(2):197-204). They assessed 13 volunteers before and upon completion of a 6-week program of cardiovascular conditioning and a low-fat diet. There was a dramatic difference in serum cholesterol (217 +/- 36 versus 181 +/- 26 mg/dL), and LDL cholesterol after 6 weeks of lifestyle therapy (140 +/- 32 versus 114 +/- 24 mg/dL).

A similar study in Germany (Wien Klin Wochenschr 1995;107(18):555-66) implemented a program including physical activity, nicotine abstinence and also included a diet containing less fat but more unsaturated fatty acids, less salt, less cholesterol, more proteins, more dietary fiber and more complex carbohydrates. In some cases, they mentioned that such dietary modifications can result in a 25% decrease of LDL-cholesterol.

Other research (Diabetes Care 1994 Dec;17(12):1469-72) desired to investigate the effectiveness of an intensive diet and exercise program for reducing risk factors associated with macrovascular complications as well as other conditions. They examined a total of 652 patients. Serum total and low-density lipoprotein cholesterol were reduced by 22% and triglycerides by 33%. The ratio of total to high-density lipoprotein cholesterol was reduced by 13%.

Another study gave health care advice to (BMJ 1995 Apr 29;310(6987):1105-9) 681 subjects aged 30-59 years, with at least two cardiovascular risk factors in addition to moderately high lipid concentrations: total cholesterol $\geq$ 6.5 mmol/l on three occasions,

triglycerides < 4.0 mmol/l, and ratio of low density lipoprotein cholesterol to high density lipoprotein cholesterol > 4.0. Most of the 577 subjects were men.

In the group receiving intensive health care advice, total cholesterol concentration decreased by 0.15 mmol/l more (95% confidence interval 0.04 to 0.26) than in the group receiving usual advice. The overall Framingham risk dropped by 0.068 more (0.014 to 0.095) in the group receiving intensive advice, and most of the risk factors showed a greater change in a favorable direction in this group than in the group receiving usual advice. The results from questionnaires completed at the group sessions showed that the subjects improved their lifestyle and diet.

One study (Am J Clin Nutr 1994 May;59(5 Suppl):1124S-1129S) examined the health effect of being a Seventh-day Adventist (Christians that endorse a healthy lifestyle characterized by: a diet with more plant foods and less animal products, salt, sugar and oil; daily exercise outdoors, sufficient water-drinking; avoiding alcohol, nicotine and caffeine; getting proper rest and having trust in God) by computer-linking the official church rosters with National Health Registries.

Total cholesterol was 0.86 mmol/L lower in the Seventh-day Adventist men ($P < 0.001$) and 0.48 mmol/L lower in women ($P < 0.001$). Total mortality was significantly lower only in Seventh-day Adventist men (standardized mortality ratio (SMR), 82; $P < 0.001$), especially in cardiovascular mortality. Entering the church at an early age had a large effect decreasing on later mortality. The study supports previous health studies of the Seventh-day Adventist population. They concluded that an early establishment of a

healthy lifestyle seems to be of decisive importance in the risk of later disease. We may conclude that **Comprehensive lifestyle changes can lower serum cholesterol.**

Lifestyle and Blood Pressure

It has been suggested (Geriatrics 1995 Nov;50(11):39-45 quiz 46-7) that the right combination of diet and lifestyle changes can help to control hypertension and reduce cardiovascular risk. For optimal results, they suggest giving the patient as much informed choice as possible in the selection of therapies and the setting of goals. They warn to proceed cautiously when it is necessary to add pharmacologic therapy, whatever agent is chosen.

In one outpatient clinical study (Am J Cardiol 1995 Jul 13;76(2):8A-20A) involving lifestyle change, exercise, wt loss, and alcohol reduction in two groups of patients (one lifestyle changes only, the other lifestyle + drug therapy), significant reductions in blood pressure were obtained for both groups between admission readings and readings taken at 12 and 24 months ($p = .0001$). The non-pharmacologic group, on the average, required fewer clinic visits (i.e. less follow up). Interventions targeted toward lifestyle behaviors, a common element in the treatment plans for both groups, appear to have therapeutic benefit.

Researchers in Japan (Am Heart J 1996 Feb;131(2):313-9) used exercise and a mild hypocaloric intake with patients. M-mode echocardiography was performed to estimate the (left ventricular mass) LVM in the subjects. This is an extremely important risk factor to assess since one article (J Hypertens Suppl 1994 Jul;12(4):S73-87)

delineating the risk factors for cardiac failure mentioned that although the presence of hypertension or diabetes among men aged 35-64 years increases the risk fourfold, but electrocardiographic evidence of left ventricular hypertrophy in this group **increases the risk for cardiac failure 15-fold, independently of the presence of hypertension!**

After the 12-week intervention, systolic, diastolic, and mean BP were significantly reduced by 13, 9, and 11 mm Hg, respectively, in the HT group. LVM was significantly reduced from 176 +/- 26 gm to 159 +/- 26 gm ($p < 0.05$) in the HT group and from 167 +/- 33 gm to 145 +/- 34 gm ($p < 0.02$) in the NT group. These results suggest that weight reduction in obese subjects by mild exercise and mild hypocaloric intake can lead to a reduction in LVM, regardless of whether the subjects have normal or high blood pressure thus reducing risk of cardiac failure. We find that **Comprehensive lifestyle changes can reduce hypertension.**

Lifestyle and Obesity

Research supports (Mayo Clin Proc 1994 Oct;69(10):997-9) the fact that composite lifestyle changes are important for the overall reduction of body weight and subsequent maintenance of weight loss. The management (Am J Epidemiol 1995 Jun 15;141(12):1117-27)
of obesity includes change in lifestyle, nutritional education and modification and increase in exercise. They emphasized that these changes are important for long-term success.

One trial (Am Heart J 1996 Feb;131(2):313-9) using mild exercise and mild hypocaloric intake found that, after the 12-week intervention, the mean reductions in body weight was 4.9 kg ($p < 0.005$) and 4.6 kg ($p < 0.0005$) for hypertensive and normotensive patients respectively.

Another study (Am J Clin Nutr 1996 Feb;63(2):225-33) including 41 healthy, moderately obese (120-140% of ideal body weight), elderly subjects (65.6 +/- 3.3 y) participating in 24-wk diet or diet + exercise programs were studied. Daily energy need (DEN) was estimated from basal energy expenditure and self-reported activity.

The diet + exercise group (n = 16) reduced their daily energy intake (DEI) by 2092 kJ from their DEN and expended 837 kJ/d in walking and resistance exercise. The two diet-only groups (n = 13 and n = 12) reduced their DEI by 2092 and 2929 kJ. Loss of body weight was significant for all groups between baseline and 12 and 24 wk (baseline: 79.3 +/- 7.6 kg; 12 wk; 75.1 +/- 7.7 kg; 24 wk; 72.8 +/-8.0 kg). 24 wk of diet or diet + exercise significantly reduced body weight in these groups. **Lifestyle changes reduce obesity.**

Lifestyle and Cholesterol, Blood Pressure and Obesity

A number of studies have implemented comprehensive lifestyle changes in various settings and have found improvement in all three of these cardiovascular risk factors. This is a critical medical need. In fact, one country examining trends in chronic degenerative diseases (Angiology 1996 Jan;47(1):43-9) found that HTN, chol, Ht dz and prevalence of other risk factors and CVD were much higher in 1991 than in 1960 for men

of the same age group. They mention as possible causes dietary and lifestyle changes that have taken place in their country during the last thirty years.

Fortunately, national lifestyle change programs (BMJ 1995 Nov 11;311(7015):1255-9) to reverse these trends have also shown promise for the future. One nation examined a large number of adults adults aged 25-74 years residing in geographically defined clusters. 5080 were assessed in 1987 and 5162 in 1992. After a nation-wide promotion for lifestyle change, significant decreases were found in the prevalence of hypertension (15.0% to 12.1% in men and 12.4% to 10.9% in women); cigarette smoking (58.2% to 47.2% and 6.9% to 3.7% respectively); and heavy alcohol consumption (38.2% to 14.4% and 2.6% to 0.6% respectively). Moderate leisure physical activity increased from 16.9% to 22.1% in men and from 1.3% to 2.7% in women. Mean population serum total cholesterol concentration fell appreciably from 5.5 mmol/l to 4.7 mmol/l ($P < 0.001$). They concluded that lifestyle intervention projects can be implemented with positive effects in developing countries.

Many suggestions have been made as to what lifestyle interventions should be included in risk factor managment (J Natl Med Assoc 1995 Aug;87(8 Suppl):642-6). Researchers mention some steps individuals can take to lessen the risk of stroke or coronary heart disease include: If obese and hypertensive, patients should reduce total calories and lose weight. Substitute olive oil or canola oil for vegetable oils to increase intake of monounsaturated fatty acids. They suggest the Mediterranean diet as well as a diet with higher amounts of fiber. Start a program of aerobic exercise or increase the current amount. Reduce salt intake. Reduce alcohol consumption. Stop smoking. If

hypertensive with a tendency to over respond to stress, try relaxation as well as other techniques, which according to some studies may produce a modest reduction in blood pressure in some individuals with mild hypertension.

Now we will examine some of the results of lifestyle trials in various in/outpatient settings and age groups examining the extent to which comprehensive lifestyle change can reduce the risk factors of coronary heart disease.

In one health promotion program (J Cardpulm Rehabil 1995 Jan-Feb;15(1):65-72), researchers demonstrated that lifestyle changes can be implemented with success when subjects are young (**before** they develop diseases; a success story for prevention). A multi-ethnic, multi-cultural sample (n = 54) of 10th grade males and females participated in a study of cardiovascular health promotion and coronary risk factor reduction.

Intervention consisted of a 10-week health-promotion curriculum of classroom education in physical activity, nutrition, smoking cessation, stress management, personal problem solving, and an exercise program of walking and running. A non-intervention control group served as a basis for comparison. Classroom and exercise sessions met on alternate days.

Following intervention, a significant treatment effect ($P = .007$) was observed in lowered total cholesterol, and significant group improvements ($P < .01$) were observed in diet habits, percent body fat, and cardiovascular health knowledge. They concluded that health education, behavior modification, and regular aerobic exercise lowers cholesterol, improves health behavior and increases health knowledge.

We find more dramatic results in patients with heart disease. One project examining the effect of short-term cardiovascular conditioning and low-fat diet on myocardial blood flow and flow reserve demonstrated impressive improvement in blood flow, but also in cardiovascular risk factors as well (Circulation 1995 Jul 15;92(2):197-204).

They mention that exercise conditioning reduces resting myocardial oxygen demand by lowering systolic blood pressure and heart rate. Lower myocardial oxygen demand at rest would be expected to be associated with a decrease in resting myocardial blood flow and, consequently, an increase in myocardial flow reserve as the ratio of hyperemic to resting blood flow.

Myocardial blood flow at rest and after dipyridamole-induced hyperemia (0.56 mg/kg iv.) was quantified with [13N] ammonia and positron emission tomography in 13 volunteers before and upon completion of a 6-week program of cardiovascular conditioning and a low-fat diet. Exercise capacity and serum lipid profiles were also assessed at the start and finish of the program. Eight normal volunteers of similar age not participating in the conditioning program served as a control group.

Cardiovascular conditioning lowered the resting rate-pressure product (8859 +/- 2128 versus 7450 +/-1496, $P < .001$), serum cholesterol (217 +/- 36 versus 181 +/- 26 mg/dL, a change from "high" to normal), LDL cholesterol (140 +/- 32 versus 114 +/- 24 mg/dL), and triglycerides (145 +/-53 versus 116 +/- 33 mg/dL, all $P < .05$).

In the control group, no changes in resting rate-pressure product, or serum cholesterol levels were achieved. They concluded that short-term cardiovascular

conditioning together with a low-fat diet offers a protective effect to patients with coronary artery disease.

A similar study (J Cardpulm Rehabil 1995 Jan-Feb;15(1):47-64) again using a small number of subjects (18) was designed to assess the **long-term** effects of a low-fat diet and intensive physical exercise. The subjects were non-selected, fully employed patients with symptomatic coronary artery disease. Results were compared with 18 patients on standard medical care.

In the intervention group at 1 year, serum lipoproteins were brought to **ideal levels**, exercise-induced myocardial ischemia was significantly reduced, and progression in coronary atherosclerosis was retarded.

After more than 5 years, patients in the intervention group showed a significant reduction in lipoprotein levels compared to initial levels (total cholesterol, 248 [179-299] vs 214 [173-272] mg/dL, $P < .01$; low density lipoprotein, 152 [83-216], vs 146 [121-197] mg/dL, $P < .005$; triglycerides; 151 [80-303] mg/dl, vs 98 [46-182] mg/dL; $P < .005$) and body mass index (26 +/- 2.9 vs 25.4 +/- 3.3 kg/m2; $P < .05$).

Exercise induced myocardial ischemia, measured by 201 thallium scintigraphy, also decreased by 29% (41 degrees +/- 36 degrees vs 29 degrees +/- 29 degrees). Coronary atherosclerosis, assessed by angiography and digital image processing, progressed at a slower pace in light of a 21% increase in physical work capacity (169 +/- 40 vs 205 +/- 50, $P < .01$) and a 28% increase in maximal rate pressure product (25 +/- 6 vs 32 +/- 4, $P < .004$).

In contrast, patients in the control group showed only poorly controlled coronary risk factors (total cholesterol, 243 [179-306] vs 269 [178-304] mg/dL; low density lipoprotein, 151 [79-229] vs 196 [107-238] mg/dL, P < .0005; body mass index 25.7 +/- 2.5 vs 27.5 +/- 3.5 kg/m2, P < .01), whereas their physical work capacity tended to deteriorate (165 +/- 45 vs 142 +/- 62 Watts)

They concluded that usual care is **insufficient** in controlling risk factors of coronary artery disease. However, intensive physical exercise and low-fat diet remain an effective form of treatment **after more than 5 years.**

Thus we see the immediate and long-term effects of lifestyle change. Even though many patients are disposed to disease by their family histories, they are not bound by the laws of genetics, but the decisions they make have a tremendous impact on their quality of life. One study (Atherosclerosis 1994 Aug;108 Suppl():S143-56) examined this fact regarding the effects of a traditional lifestyle on Pima Indians compared to their genetically similar "americanized" counterparts.

Measurements of weight, height, body fat (bioimpedance), blood pressure, plasma levels of glucose, cholesterol, and HbA1c were obtained in 19 women (36 +/- 13 years of age) and 16 men (48 +/-14 years of age) and compared with sex-, age- and diabetes status-matched Pimas living in Arizona (10 Arizona Pimas for each Mexican Pima or 35 vs 350).

Mexican Pimas (with the "traditional" lifestyle characterized below) were lighter (64.2 +/- 13.9 vs. 90.2 +/- 21.1 kg, P < 0.0001; with lower body mass indexes (24.9 +/- 4.0 vs. 33.4 +/- 7.5 kg/m2, P < 0.0001) and lower plasma total cholesterol levels (146 +/-

30 vs. 174 +/- 31 mg/dl, $P < 0.0001$) than Arizona Pimas (or the "americanized" genetic counterparts).

Also, only (11%) of women and (6%) of men had NIDDM of the "traditional" pimas, contrasting with the prevalences of 37% and 54% in female and male Arizona Pimas, respectively.

They stated that this preliminary investigation shows that obesity, and perhaps NIDDM, is less prevalent among people of Pima heritage living a "traditional" lifestyle than among Pimas living in an "affluent" environment. These findings suggest that, **despite a similar potential genetic predisposition** to certain disease conditions, a traditional lifestyle, characterized by a diet including less animal fat, more complex carbohydrates and by greater energy expenditure in physical labor, may protect against the development of **cardiovascular disease risk factors**.

As we examine studies with greater numbers, we find similar results (Prev Med 1995 Sep;24(5):509-17). One assessed a sample of 535 healthy premenopausal women, ages 44-50, for an ongoing, 5-year, randomized trial to reduce total dietary saturated fat and cholesterol, prevent weight gain, and increase physical activity levels.

Changes in CHD risk factors after the first 6 months of treatment were analyzed comparing 253 intervention and 267 assessment-only control participants. The intervention group showed significant reductions in total cholesterol (-0.34 mmol/liter), LDLC (-0.28 mmol/liter), triglycerides (-0.04 mmol/liter), weight (-4.8 kg), waist-hip ratio (-0.008), systolic blood pressure (-3.5 mm Hg), diastolic blood pressure (-2.2 mm Hg), serum glucose levels (-0.06 mmol/liter), and HDLC (-0.06 mmol/liter) and

significant increases in physical activity (+383 kcal). As might be expected, no significant changes were observed in the control group.

They stated that the six-month results suggested that participants were receptive to the preventive approach to CHD risk reduction and were successful in making positive lifestyle changes.

Amazingly, these reductions do not have to take months or years when certain principles are followed. One lifestyle program achieved rapid reduction of serum cholesterol and blood pressure in just twelve days (J Am Coll Nutr 1995 Oct;14(5):491-6).

Their study was conducted to demonstrate the effectiveness of a strictly vegetarian, very low-fat diet on cardiac risk factor modification. Five hundred men and women, participants in an intensive 12-day live-in program, were studied. The program focused on dietary modification, moderate exercise, and stress management at a hospital-based health-center in St. Helena, CA.

They found that during this short time period, cardiac risk factors improved: there was an average reduction of total serum cholesterol of 11% ($p < 0.001$), of blood pressure of 6% ($p < 0.001$) and a weight loss of 2.5 kg for men and 1 kg for women. This strict, very low-fat vegetarian diet free from all animal products combined with lifestyle changes that included exercise and weight loss was an effective way to rapidly lower serum cholesterol and blood pressure.

A larger study (Prev Med 1995 Jul;24(4):378-88) involved a 4-year, randomized clinical trial (N = 902). Their lifestyle intervention program focused on the effect of weight loss on blood pressure and blood lipid levels.

Participants all took part in a lifestyle intervention program to reduce weight, reduce sodium and alcohol intake, and to increase physical activity. Substantial changes from baseline levels were achieved for all lifestyle intervention variables. Mean weight change was -10.5 lb (-5.6%) at 1 year, -8.5 lb (-4.5%) at 2 years,-7.4 lb (-4.0%) at 3 years, and -5.7 lb (-3.0%) at 4 years.

At 4 years, 70% of participants remained below baseline weight and 34% maintained a weight loss of 10 lb or greater. Mean change in urinary sodium excretion was -12.5 mmol/8 hr (-23%) at 1 year, -10.7 mmol/8 hr (-20%) at 2 years, -8.4 mmol/8 hr (-16%) at 3 years, and -4.6 mmol/8 hr (-9%) at 4 years. Alcohol intake declined by 1.6 drinks/week among drinkers at 4 years. Reported leisure physical activity increased by 86% at 1 year and remained 50% above baseline at 4 years. Beneficial changes in blood pressure and serum lipids were associated with these changes.

They concluded that these results supported a role for lifestyle interventions as an initial treatment for stage 1 hypertension and demonstrated that such interventions can be successfully implemented in the clinical setting.

A large outpatient study was conducted using health counseling regarding diet, exercise, etc. to control risk factors (BMJ 1995 Apr 29;310(6987):1099-104). 2205 men and women were randomly allocated an initial health check in 1989-90 and a

re-examination in 1992-3 (the intervention group), 1916 additional men and women were randomly allocated an initial health check in 1992-3 who served as controls.

All subjects were aged 35-64 at recruitment in 1989. Mean serum total cholesterol was 3.1% lower in the intervention group than controls (difference 0.19 mmol/l, 95% confidence interval 0.12 to 0.26); in women it was 4.5% lower ($P < 0.0001$) and in men 1.6% ($P < 0.05$). Self-reported saturated fat intake was also significantly lower in the intervention group. Systolic and diastolic blood pressures and body mass index were respectively 1.9%, 1.9%, and 1.4% lower in the intervention group ($P < 0.005$ in all cases). There was a 3.9% (2.4 to 5.3) difference in the percentage of subjects with a cholesterol concentration ≥ 8 mmol/dl.

They point out that the benefits of health checks were sustained over three years. The health counseling help to reduce risk factors by changing the lifestyle. The benefits of systematic health promotion in primary care are real.

Thus, in recent literature such as we have reviewed, we find that (Am Heart J 1994 Dec;128(6 Pt 2):1344-52) the association between abnormal serum lipoprotein level (as well as other risk factors) and coronary atherosclerosis has been established by extensive clinical, experimental, and epidemiologic evidence.

Recent angiographic trials in patients with coronary artery disease have shown that coronary atherosclerosis can be prevented and reversed by managment of risk factors. This results in fewer future coronary events in intervention groups versus controls. Most of these types of studies have involved the standard drug therapy to

reduce the risk factors for coronary heart disease, but as we have reviewed previously, there are a few that have used only lifestyle changes to bring about desired results.

These lifestyle trials have involved patients before a heart attack as well as after. As we examine a sampling of these studies, we find that three controlled, primary prevention studies used approaches such as diet, smoking cessation as well as other comprehensive lifestyle changes aimed at lowering serum cholesterol to prevent future coronary events. They all had a long period of follow-up and discovered that there was significant differences between the trial and control groups

Not only serum cholesterol but other risk factors as well were reduced in the lifestyle group compared to no change in the controls (Ibid). **After periods of 5 to 10 years, reductions of 20% to 45% in coronary events were observed in the intervention groups** compared with controls (that would amount to substantial savings for insurance companies compared to normal surgical methods).

A summary of four trials involving patients after heart attacks reveals a similar profile. Three of the four were controled and all used similar lifestyle interventions including a low-fat diet. These changes were also aimed at lowering in serum cholesterol as well as other risk factors (Ibid).

Serum cholesterol levels fell by 14% to 24% in treated patients compared with 4% to 9% in controls. Although these studies involved a small groups of patients (36-90) and were for only short periods of time (1-3 years), they all demonstrated dramatic clinical effects on risk factors and regression of coronary atherosclerosis.

With an understanding of what lifestyle interventions influence the reduction of coronary risk, we undertake our present trial.

THE WELLNESS CHALLENGE PROGRAM TO IMPROVE CARDIAC RISK FACTORS

SUBJECTS

97 patients (aged 53.4yrs +/- 12yrs; 21 male, 76 female) participated in 4-week, hospital-based outpatient program at East Pasco Medical Center in Zephyrhills, FL. Most of the patients were patients from East Pasco County of Florida of the Caucasian race and were either self-referred or sent to the program by the recommendation of a physician. Most were interested in learning about reversal of chronic degenerative diseases like: heart disease, cancer, stroke, diabetes, arthritis, etc. Those choosing to drop out of the program before the 4 weeks were up were eliminated from the study. Drop-out rate was roughly 20% of initial patients. In other words those no longer attending the lectures or cooking schools, or not coming for any before/after blood testing, etc. were not included.

LIFESTYLE THERAPY

Lifestyle therapy revolved around the acronym NEWSTART (Nutrition, Exercise, Water, Sunshine, Temperance, Air, Rest and Trust in God).

Dietary modifications included an unrefined strict-vegetarian diet consisting of fruits, vegetables, whole grains, beans and nuts. The diet emphasized whole-plant foods in place of refined, processed or packeged products. For example, whole wheat was

recommended in place of white bread; brown vs. white rice; avocados, nuts, olives and soy vs. cooking oils or hydrogenated spreads; and fruit vs.white sugar; etc. The diet contained no animal protein or fat, and thus was naturally free of cholesterol and high in plant sterols (since only animal products contain cholesterol). It was low in saturated, but high in poly and monounsaturated whole-plant food fats. The diet was high in vitamins, minerals and both soluble and insoluble fiber. The diet also was high in complex carbohydrates and low in fat, protein and salt (app.. 75% CHO, 15% FAT, and 10% PRO). Larger meals were consumed in the morning and the afternoon and very little at night.

Walking outdoors (where an abundance of sunshine and fresh air could be obtained) was the recommended exercise. The goal was 3-5 miles per day. However, other exercises were permitted that would equate to approximately the same time and intensity of the walking goal.

All drugs (legal or illegal) were not a part of the program unless medically needed (insulin for diabetics, blood pressure medications, etc.) This excluded all nicotine, alcohol and caffeine or products containing those substances (cigarettes, chewing tobacco, coffee, teas, colas, chocolate, etc.)

Suggestions were given to improve sleeping problems such as stress management, going to sleep earlier, avoiding large meals late at night, dealing with emotional problems, etc.

Patients were encouraged to develop a firm trust in God by regular prayer, meditation and Bible reading. Other recommendations included drinking 8-10, 8oz

glasses of water daily, spending time in the sunshine and fresh air, as well as things to encourage deep breathing like correct posture and exercise.

97 patients successfully completed a 4 week lifestyle change program during four separate months. The patients were all volunteers and paid the hospital a modest fee to participate in the program (< $100). Patients of varying age and sex attended. These patients completed before/after testing for blood pressure, weight, and blood cholesterol levels.

Patients were given personal history sheets to complete to assess past/present health problems and current level of physical activity (See page 74 in Appendix). Patients were also given personal diet, exercise and water diaries to monitor and encourage compliance with the program (See pgs. 75-76)

These hand-outs were collected periodically and compliance was evaluated to be moderate to high. In other words, all patients had changed some to most lifestyle factors during the 4 weeks of the study.

Meetings were held 2-3 times a week for the four weeks of the program. Lectures about the eight lifestyle changes, various chronic degenerative diseases, and other nutritional and health-related topics were given (See pg. 77 for a sample lecture). Emphasis was placed on why, how and to what extent lifestyle change affect health. Articles from health journals were given to all the patients to understand the logic of certian dietary and lifestyle changes (See pg. 78 for a sample). Some articles gave practical tips on how to adhere to dietary changes in "real world" situations (See pg. 79 for a sample).

Vegetarian cooking schools (unrefined, total vegan food as previously described) were also given in conjunction with the lectures. The first meeting of each 4-week session, the patients received recipes to use at home during and after the program (See pg. 80 for a page of sample recipies).

CHOLESTEROL, BLOOD PRESSURE AND WEIGHT ASSESSMENT

A 12-hr fasting blood draw was performed before beginning the program and the last day of the four-week intervention. Total cholesterol levels were measured by standard medical techniques at East Pasco Medical Center.

Blood pressure was measured with the patient seated by a machine with accuracy of about $\pm$ 1 mm of Hg or with a standard sphygmomanometer by a trained health professional. Weight was taken with indoor attire exluding shoes and rounded to the nearest pound.

STATISTICAL ANALYSIS

Patients were divided into 3 groups for analysis of serum cholesterol: those $\geq$ 200mg/dl, from 180-199mg/dl, and an analysis of all patients.

Patients were divided into 3 groups for analysis of blood pressure: At or above 140/90 mm of Hg, between 130/85 - 139-89 mm Hg, and an analysis of all patients. An analysis was done for all patients for change in weight and was measured in pounds.

Before/After means were compared and analyzed using the 2-tailed T-test.

RESULTS

PATIENTS WITH RISK FACTORS (SEE PGS. 81-83)

Those patients who had diagnosed risk factors (high blood pressure, high cholesterol, etc.) at the onset of the program received the greatest benefit from the 4 weeks of lifestyle change.

The 43 patients with blood pressures ≥ 140/90 had an average initial systolic blood pressure of 151 mm Hg. After the program, the average was found to be 134 mm Hg, a drop of 17 mm Hg. or 11.3% this was very significant ($p = .000$). Diastolic pressure dropped from 87 to 81 mm Hg or a 6 mm Hg or 6.9% improvement. This change was also quite significant ($p = .000$)

The 61 patients with Serum cholesterol levels ≥ 200 mg/dl also displayed similar dramatic improvements. Fasting serum Cholesterol levels dropped from 242 to 207 mg/dl, a 35 pt or 14.5% drop. This was found to be extremely significant ($p = .000$)

SUBJECTS WITH HIGH-NORMAL RISK FACTORS (PGS. 84-86)

The 21 patients with high-normal blood pressures (130-139/85-89 mm Hg) showed some improvement with 4 weeks of lifestyle change. Systolic pressures dropped from 130 to 122 mm Hg or improvement of 6.2% . Diastolic pressures dropped from 82 to 78 mm Hg improving 4.9%. ($p = .020$ and .077 respectively).

The 15 patients with elevated serum cholesterol also experienced some improvement. Initial levels dropped from 192 to 173, and improvement of 9.9% with $p = .021$.

ALL PATIENTS (PGS. 87-90)

As a whole, the group of 97 patients had significant improvement in the 4 weeks of lifestyle change. Systolic blood pressures dropped from 135 to 125 mm Hg improving 7.4% ($p = .000$). Diastolic blood pressure fell from 81 to 78 mm Hg an improvement of 3.7% with $p = .000$.

Fasting serum cholesterol levels dropped from 215 to 188 mg/dl improving 12.6% ($p = .000$). Average weights dropped from 188 to 180 lbs a loss of 8 lbs or 4.3% ($p = .000$).

DISCUSSION

The lifestyle changes implemented in this study, were very effective in lowering serum cholesterol and blood pressure as well as achieving modest reductions in weight. This study contributes to previous findings that demonstrate the effectiveness of lifestyle intervention to improve the risk factors of coronary heart disease.

During the 4 weeks of lifestyle change, the group as a whole experienced improvement in Blood Pressure, Serum Cholesterol levels and Weight. The most significant changes were in those patients with elevated risk factors.

Perhaps a difference in this study to its antecedents would be that this outpatient program displayed significant results over a short period of time at **relatively low cost as well**. Costs included wages for staff: lecturers, cooking school instructors, lab techs for blood work, etc. These costs were partly deferred by the inscription fee (< $100) for the program.

The results demonstrated by the outpatient program were slightly less dramatic than some of those achieved in the previously mentioned trials (see literature review). Perhaps three similar programs that might be mentioned for comparative purposes are those conducted by John McDougall M.D., Dean Ornish M.D., and Nathan Pritikin.

McDougall demonstrated an 11% reduction in serum cholesterol and a 6% reduction in blood pressure and a weight loss of 2.5 kg for men and 1 kg for women in about 2 weeks (J Am Coll Nutr 1995 Oct;14(5):491-6). Dean Ornish showed a 20% reduction in serum cholesterol in about 3 1/2 weeks (JAMA 1983; 249:54-59). The Pritikin program achieved a 23% reduction in serum cholesterol over a period of 3 weeks (Arch Intern Med 1991; 151:1389-1394) Unfortunately, all of these programs were also accompanied by a 19, 16 and 17% **reduction** in HDL cholesterol.

It is **very** interesting to note that our achievements for all patients of 13% reduction of serum cholesterol and 6% reduction in blood pressure as well as an 8 lb weight loss were very similar to McDougall's achievements mentioned above. **However,** our reduction of cholesterol was 2% greater and our weight loss **was** statistically significant. Also, **the cost** for our program was greatly reduced not only for our hospital but for the patients as well (patients were charged less than $100 each for the entire program as opposed to some programs that charge over $10,000 for less than 2 weeks). This outpatient option could help to make effective lifestyle therapy more accessable to those who are unable to pay higher prices for health care.

This benefit of cost-effectiveness may help to answer the problem of a shrinking economy and rising health costs. In a day and age where both insurance companies and

hospitals are trying to survive by cutting costs, lifestyle change seminars are cost-effective and address the diseases that claim the most lives every year in the United States and other Industrialized nations (Heart Disease, Stroke, Cancer, Diabetes, etc.)

In addition, in an outpatient program like ours, patients comply to lifestyle changes **in the real world**: to cook healthfully from their **own** grocery store, set time for exercise in their **own** busy schedules, etc. This could be compared to lifestyle change at live-in programs: all meals are cooked for you, there are no "temptations" of other non-health foods, there is strong support in an exercise program, patients are away from all stresses of work and home, etc.

Outpatient programs perhaps give promise of a greater post-program compliance level. If patients can change habits while still in their own environment and in their own home, these changes are more likely to be permanent in the long run.

CONCLUSION

It appears that our 4-week outpatient program gives encouraging results in the reduction of coronary risk factors at a very low cost. An outpatient program has the advantages of practicality in that habits are changed in the "real world." Patients also do not have to leave their jobs or other commitments.

These attributes give greater access to the majority of society that perhaps is "locked" into their work scedules and cannot afford to pay much money to improve asymptomatic risk factors. Many question why they should spend much money for something that is not currently causing them pain. Despite the obstacles preventing them

from changing their lifestyles, whether internal or external, statistics show that the majority of industrialized society desperately needs these risk factor reductions.

Despite all the "temptations" to deviate from the program, all patients in our study made positive changes that were reflected in the improved levels of serum cholesterol, blood pressure and weight.

As we have mentioned before, positive lifestyle change can equate to reduction of risk of death from heart disease, we find that our results can reflect significant benefits for patients, especially those who are at high-risk.

For example, the 15% reduction of cholesterol in our patients with high cholesterol would be reflected in a 30-45% reduction of risk. The 6 pt drop in diastolic blood pressure in the hypertensive patients would represent an additional 12-18% reduction. Achieving ideal weight through compliance of the program would yield an additional 35-55% reduction in risk. Maintaining physical activity would contribute 45% to risk improvement (N Engl J Med 1992; 326:1406-1413). One can only imagine the tremendous heath benefits that can be achieved if these lifestyle changes can be maintained.

Future experimentation in outpatient lifestyle change may include follow-up on compliance rates and continued monitoring of risk factors such as blood Pressure, serum cholesterol levels, weight, etc. Such continued compliance rates are not presently known but could be assumed to be good given the previous rational of habits changed in the "real world" as mentioned above.

Other future research may include: conducting lifestyle seminars with other cultures, races, religions, and educational levels, in different countries, and in different settings (perhaps a country villa as opposed to a hospital). Including this information (race, religion, educational level, etc.) in the initial registration for may help to discover various correlations in initial risk factor levels and amount of improvement obtained in individuals with certain characteristics compared to others (perhaps certain people groups may benefit more than others from lifestyle therapy). Other more exact measurements of improvement of heart disease risk may be considered for future before/after testing as well, such as a coronary arteriogram. Other research may include monitoring improvement of other common chronic degenerative diseases like diabetes (perhaps measuring before/after blood glucose levels), arthritis, and cancer along with their respective risk factors.

BIBLIOGRAPHY

AACN Clin Issues 1995 Nov;6(4):515-25

Acta Med Austriaca 1995;22(3):43-5

Addiction 1995 Nov;90(11):1479-85

Alcohol Alcohol 1995 Sep;30(5):581-9

Am J Clin Nutr 1994 May;59(5 Suppl):1124S-1129S

Am J Epidemiol 1995 Jun 15;141(12):1117-27

Am J Cardiol 1995 Jul 13;76(2):8A-20A

Am Heart J 1994 Dec;128(6 Pt 2):1344-52

Am Heart J 1996 Feb;131(2):313-9

Am J Clin Nutr 1996 Feb;63(2):234-41

Am J Clin Nutr 1996 Feb;63(2):225-33

Am J Med Sci 1994 May;307(5):325-8

Am J Clin Nutr 1994 Jan;59(1):103-9

Am J Clin Nutr 1994 Jun;59(6):1325-31

Am J Clin Nutr 1993 Sep;58(3):354-9)

Am J Physiol 1995 Nov;269(5 Pt 1):E878-83

American Heart Association. Heart and Stroke Facts: 1994 Statistical Supplement.

Dallas TX: Author, 1994.

Angiology 1996 Jan;47(1):43-9

Ann Epidemiol 1995 Mar;5(2):108-18

Ann Int Med 1991; 114:224-237

Ann Nutr Metab 1994;38(6):331-5

Arch Intern Med 1996 Feb 12;156(3):305-12

Arch Fam Med 1993 Mar;2(3):317-22

Arch Intern Med 151:1389-1394, 1991

Arch Inst Cardiol Mex 1995 Jul-Aug;65(4):342-8

Arctic Med Res 1995 Jul;54(3):134-44

Atherosclerosis 1994 Aug;108 Suppl():S143-56

Biochim Biophys Acta 1996 Jan 19;1299(2):160-6

Biol Psychol 1995 Aug;41(1):69-81

BMJ 1995 Apr 29;310(6987):1105-9

BMJ 1995 Nov 11;311(7015):1255-9

BMJ Apr 29;310(6987):1099-104

Bone 1995 Aug;17(2 Suppl):107S-111S

Br J Nutr 1995 Nov;74(5):661-70

Br J Nutr 1995 Dec;74(6):765-73

Bratisl Lek Listy 1994 Aug;95(8):344-8

Bratisl Lek Listy 1993 Dec;94(12):621-5

Bull Acad Natl Med 1995 Oct;179(7):1449-57; discussion 1458-9

Bull Acad Natl Med 1995 Oct;179(7):1417-26; discussion 1426-8

Bull Acad Natl Med 1995 Oct;179(7):1471-80; discussion 1481

Cardiology 1993;82(2-3):100-21

Chest 1996 Jan;109(1):52-6

Chest 1993 May;103(5):1336-42

Circulation 1995 Jul 15;92(2):197-204

Circulation 1995 Jul 15;92(2):197-204

Circulation 1994 Sept; vol 90, No 3.

Clin Exp Pharmacol Physiol 1995 Sep;22(9):655-7

Clin Nephrol 1993 Dec;40(6):315-20

Clin Exp Hypertens 1995 Oct;17(7):1009-24

Cor Vasa 1993;35(6):224-9

Diabetes Care 1994 Dec;17(12):1469-72

Diabetes Care 1994 Sep;17(9):1067-74

Endocrinol Metab Clin North Am 1995 Sep;24(3):643-55

Eur J Clin Invest 1995 Jun;25(6):390-5

Eur J Clin Nutr 1995 Nov;49(11):824-31

Fortschr Med 1995 Jun 10;113(16):239-42

Geriatrics 1995 Nov;50(11):39-45 quiz 46-7

Hautarzt 1994 Oct;45(10):702-7

Health Psychol 1995 Nov;14(6):563-9

Hum Nutr 1993 Jan;43(1):55-61

Hum Genet 1996 Feb;97(2):156-62

Hypertension 1994 Jun;23(6 Pt 2):894-8

Hypertension 1995 Nov;26(5):820-7

Hypertension 1996 Feb;27(2):303-7

Int J Obes Relat Metab Disord 1994 May;18(5):329-32

Int J Obes Relat Metab Disord 1995 Jun;19(6):410-8

Isr J Med Sci 1995 Sep;31(9):527-35

J Manipulative Physiol Ther 1993 Jun;16(5):327-35

J Assoc Physicians India 1994 Jan;42(1):24-6

J Clin Epidemiol 1995 Sep;48(9):1159-66

J Hum Hypertens 1993 Feb;7(1):33-8

J Cardiovasc Risk 1994 Oct;1(3):249-54

J Am Coll Nutr 1995 Oct;14(5):428-38

J Natl Med Assoc 1995 Aug;87(8 Suppl):642-6

J Cardiovasc Risk 1995 Jun;2(3):247-54

J Cardpulm Rehabil 1995 Jan-Feb;15(1):47-64

J Cardpulm Rehabil 1995 Jan-Feb;15(1):47-64

J Formos Med Assoc 1995 Dec;94(12):724-31

J Cardiovasc Risk 1995 Aug;2(4):316-22

J Am Coll Nutr 1993 Dec;12(6):following table of contents

J Cardiovasc Pharmacol 1995 May;25(5):710-6

J Am Coll Nutr 1993 Dec;12(6):676-84

J Assoc Acad Minor Phys 1994;5(4):147-51

J Clin Endocrinol Metab 1995 Jul;80(7):2181-5

J Nutr 1996 Jan;126(1):43-50

J Cardpulm Rehabil 1995 Jan-Feb;15(1):65-72

J Clin Epidemiol 1995 Oct;48(10):1189-96

J Am Coll Nutr 1995 Oct;14(5):491-6

JAMA 249:54-59, 1983

Mayo Clin Proc 1995 Jan;70(1):69-79

Mayo Clin Proc 1994 Oct;69(10):997-9)

Minerva Gastroenterol Dietol 1995 Sep;41(3):237-45

N Z Med J 1995 Jul 14;108(1003):266-8

N Engl J Med 326:1406-1413, 1992

Nat Med 1995 Oct;1(10):1009-16

Nippon Koshu Eisei Zasshi 1994 Jan;41(1):46-55

Nutr Rev 1993 Feb;51(2):44-6

Orv Hetil 1993 Apr 11;134(15):787-96

Prev Med 1995 Sep;24(5):509-17

Prev Med 1995 Sep;24(5):509-17

Prev Med 1995 Jul;24(4):378-88

Psychosom Med 1994 May-Jun;56(3):260-6

Sleep 1993 Aug;16(5):457-66

Tijdschr Geneeskd 1996 Jan 6;140(1):34-6

Wien Klin Wochenschr 1995;107(6):195-201

Wien Klin Wochenschr 1995;107(18):549-54

APPENDIX

THE WELLNESS CHALLENGE

PERSONAL HISTORY

NAME:____

ADDRESS:_

___

AGE:_____

HOME PHONE:_

PLEASE ANSWER THE FOLLOWING QUESTIONS:

	YES	NO	
1.	( )	( )	HEART TROUBLE OR CARDIOVASCULAR DISEASE?
2.	( )	(✓)	PAINS IN HEART OR CHEST?
3.	( )	(✓)	PAINS IN LEGS?
4.	(✓)	( )	ARTHRITIS?
5.	( )	(✓)	STROKE?
6.	(✓)	( )	HEART PALPITATIONS OR IRREGULAR HEART BEATS?
7.	(✓)	( )	HIGH BLOOD PRESSURE?
8.	( )	(✓)	DIABETES?
9.	(✓)	( )	FAINTING OR DIZZINESS?
10.	( )	(✓)	LUNG DISEASE OR SHORTNESS OF BREATH?
11.	(✓)	( )	PROBLEMS WITH BONES OR JOINTS WHILE EXERCISING?
12.	(✓)	( )	BACK PROBLEMS?
13.	( )	(✓)	HAVE YOU EVER HAD MAJOR SURGERY?
14.	( )	(✓)	DO YOU SMOKE?
15.	( )	( )	HAVE YOU QUIT SMOKING?_____HOW MANY YEARS MONTHS?_____
16.	( )	(✓)	DO YOU EXERCISE REGULAR? very mild exercise
17.	( )	(✓)	ARE YOU TAKING ANY MEDICATIONS PRESENTLY?
18.	( )	(✓)	ANY CONDITIONS THAT MAY HINDER YOUR ABILITY TO EXERCISE?

PLEASE EXPLAIN ANY ABOVE QUESTIONS ANSWERED YES:_____

a minor heart blockage with some premature ventricular beats; left shoulder somewhat stiff, used to faint on occasion but not for years, disintegration of some lower back discs

INDICATE YOUR PRESENT ACTIVITY LEVEL IN HOURS OF EXERCISE PER WEEK:

1. ( ) HAVE NO EXERCISE PROGRAM
2. ( ) EXERCISE 1-2 HOURS PER WEEK
3. ( ) EXERCISE LESS THAN 1 HOUR/WEEK
4. (✓) EXERCISE 2 OR MORE TIMES/WEEK

186.1/4 152/84

THE WELLNESS CHALLENGE
DIET DIARY

DAY:

WATER INTAKE(8oz) 1 2 3 4 5 6 7 8 9 10

BREAKFAST
TIME:

DINNER
TIME:

SUPPER
TIME:

SNACKS
TIME:

THE WELLNESS CHALLANGE

DATE	ACTIVITY	DURATION	RATE	COMMENTS

FATS LECTURE

Fats, or lipids, are the most concentrated source of energy on the diet. They furnish more than twice the calories per gram as carbohydrates and protein. One gram of fat yields about 9 calories. Carbohydrates and protein yield 4 calories per gram.

1) FUNCTIONS OF FATS IN THE BODY:

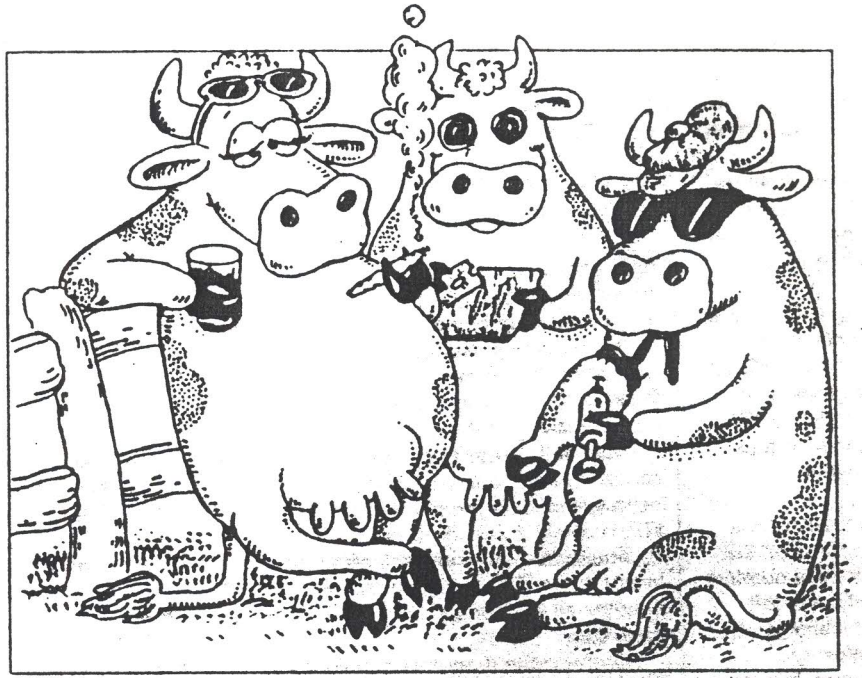
a) *Fats act as carriers* for the fat-soluble vitamins, A, D, E, and K.
b) *Fats are essential* in the formation of various hormones and immune cells.
c) *Fat deposits surround, protect, and hold in place organs*, such as the kidneys, heart, and liver.
d) *A layer of fat insulates the body* from environmental temperature changes and preserves body heat. It also rounds out the contours of the body.
e) *Fats prolong the process of digestion*, creating a longer-lasting sensation of fullness after a meal.

2) DESCRIPTIONS OF VARIOUS FATS AND FAT COMPONENTS:

a) *Fatty Acids* are the substances that give fats their various flavors, textures, melting points, etc. There are 2 types of fatty acids:
   (1) Saturated fatty acids—have all the hydrogen they can hold. This makes them hard at room temperature. These are not necessary for our health and are actually the most dangerous.
   (2) Unsaturated fatty acids (mono and polyunsaturates)—are not saturated with hydrogen. They are liquid at room temperature. The 2 "essential" unsaturated fatty acids are linoleic and linolenic (we must get them in our food). Unsaturated fatty acids are necessary for normal growth and healthy blood, arteries, nerves, and tissues. They may also be necessary for the transport and breakdown of cholesterol.
b) *Triglycerides* are made up of 3 fatty acids attached to a carrier of glycerol. 95% of fats found in nature are triglycerides. Too many triglycerides allow cholesterol to be deposited onto the coronary artery walls.
c) *Cholesterol* is a fatty, waxy substance necessary for good health and is a normal component of most body tissues, especially of the brain, nervous system, liver, and blood. It is needed to form sex and adrenal hormones, Vitamin D, and bile, which is needed for the digestion of fats. It also plays a part in lubricating the skin. It is not necessary to take in any extra cholesterol, as your body makes its own. The body wraps cholesterol in packages called lipoproteins. Two important lipoproteins are:
   (1) LDL (Low density lipoprotein)—appears to promote the deposit of cholesterol in the cells, including the cells that line the artery walls.
   (2) HDL (High density lipoprotein)—seems to pick up excess cholesterol from the tissues and take it to the liver , where it is reprocessed, then excreted into the bowel.

Cows on Drugs?

BY LISA Y. LEFFERTS



Dairy cows get sick. It's a fact of life. Dairy cows also are worth money. So when they become ill, farmers want to get them back "on line" as quickly as possible.

Most of the time, that means treating the cow with an antibiotic or other drug and then waiting a few days or a week until the residues of the medication "clear" the animal's system.

But not all farmers wait as long as they should. (For some drugs, nobody even knows how long to wait.)

So how can we be certain that drug residues haven't somehow found their way into the container of milk we just bought?

Don't ask the Food and Drug Administration. The FDA's official test for drugs in milk can't detect most of the medications that farmers customarily use—either legally or not—to treat sick cows.

It's clear that potentially harmful drugs (including some carcinogens) could be getting into the milk supply. And it's also clear that nobody's systematically looking for those drugs.

That doesn't mean you should stop drinking milk. But it bothers us that the FDA doesn't know *how* safe our milk is. What's worse: what it *does* know, it would rather *you* didn't.

Testing, Testing. The FDA doesn't routinely test milk. Instead, it has an agreement with the states to do their own testing.

But the only thing the official FDA test (which most states use) is good for is detecting penicillin. The problem is: there are dozens of other drugs that farmers use to nurse sick milk cows back to health.

Are traces of these drugs turning up in milk? The FDA, despite its assurances, doesn't have a clue.

Last winter, we decided to look for ourselves. We used a testing method that is more comprehensive and more sensitive than the one recommended by the FDA. Independently, *The Wall Street Journal* decided to do the same.

The results became front-page news: residues turned up in four of the 20 milk samples we had collected in the Washington D.C. area, and in 19 of the 50 samples collected by the *Journal* in ten cities around the country. The drugs that showed up were sulfas, penicillin, streptomycin, and erythromycin.

The FDA Makes Its Moo-ve. Only four days after *The Wall Street Journal* article broke, the FDA announced that it planned to test milk too.

Great, we thought. The FDA is finally getting serious about drug residues in milk. We should have known better.

"A nationwide survey of milk has found no residues of any antibiotics, including sulfa drugs," the FDA proudly announced a month or so later.

But a close look at the FDA's "clean milk" survey shows that the results weren't so "clean." When the FDA used the same testing method that we did (the Charm II assay), 51 percent of its milk samples showed traces of animal drugs.

So how did it conclude that there were "no residues"?

Simple. The FDA said that the Charm II assay was unreliable. In other words, our "positives" were "false," and our tainted milk was really drug-free.

Milking the FDA for the Truth. But the day after the FDA released its "clean milk" survey, the House Subcommittee on Inter-Governmental Relations and Human Resources held hearings on animal drugs in milk.

Under sharp questioning by subcommittee chairman Ted Weiss of New York, the FDA admitted that it discounted the Charm II results simply because the FDA was unable to confirm those results using other tests—tests that were *less sensitive* to some drugs and *that had never before been used on retail milk*.

That's like denying what you see under a microscope because you can't also see it with a magnifying glass. In fact, the tests that the FDA used "do not check for certain drugs picked up by the Charm II assay," testified Joseph Settepani, the FDA chemist who first blew the whistle on the FDA's sloppy regulation of animal drug residues in milk.

MENU GUIDE
for dining out
THE NEWSTART® WAY

Appetizers

Fresh Fruits and vegetables as "crudites", or juices.
Avoid sour or sweet cream and seasoned butter or oil.

Soups

Clear consomme or broth with noodles or vegetables.
Avoid cheese, cream soups, egg soups, and onion soup.

Salads

Green and tossed salads and salad bars.
Avoid cheese, dressings, gelatin, cole slaw, potato salad, and Waldorf salad prepared with mayonnaise.

Main Dishes

Pasta with vegetables and/or Maranari sauce,
Chinese and Japanese vegetable and tofu dishes,
bean burrito, beans and rice, vegetable platter, rice pilaf.
Avoid meat, fish, poultry, eggs and entrees prepared with cream sauce, cheese, or those sauteed, stirfried or deep fried.

Fruits

As much as you like.
Avoid cream or whipped toppings.

Vegetables

Plain, as much as you like.
Avoid corn or polyunsaturated oil and sauces.

Bread

All sandwhich bread, breadsticks, hard rolls, French and Italian breads, Syrian pita bread, wafers, and toasts.
Avoid biscuits, croissants, butter rolls, and muffins.

Desserts

Fresh fruit and frozen fruit ices.
Avoid cake, cookies, pies, ice cream, etc.

Food Descriptions/Preparations

Tomato sauce, steamed, in broth; in its own juice, poached or garden fresh; roasted, stir-fried.
Avoid buttery, buttered or butter sauce; sauteed, fried, pan-fried, or crispy; creamed, cream sauce, or in its own gravy; au gratin, Parmesan, in cheese sauce, or escalloped; au lait, a la mode, or au fromage; marinated, stewed, basted, or casserole; prime, hash, pot pie, and hollandaise.

Clip Out

DELICIOUS MILLET

1 qt water
1 t salt
1 C millet grain☆
3/4 C chopped dates
1/2 C toasted, chopped almonds* (opt.)
1/2 C unsweetened, shredded coconut (opt.)☆

Bring water and salt to a boil. Add millet and bring to boil again. Cover, reduce heat, and simmer 45-60 minutes. Just before serving, stir in remaining ingredients. Serves 4.

*Toast almonds in a 350° F oven for about 8 minutes.

COMPANY OATMEAL

3 C water
2 C rolled oats
3/4 C raisins
1 large apple, peeled, sliced
1 t vanilla
1/2 t salt
1/8 t coconut extract (opt.)
1/8 t coriander (opt.)☆
Pinch cardamom (opt.)☆

Mix all ingredients together and bake at 350° F for 1 hour, or simmer in a saucepan for 20 minutes. Serves 4.

CROCKPOT BREAKFAST

2 qt water
1 C pearl barley or wheat berries
1 C chopped, mixed dried fruit
1 C raisins
1 t salt
1/2 t coriander (opt.)☆
1 C toasted, chopped almonds* (opt.)

While crockpot is heating up on high setting, boil water. Add barley and bring to boil again. Pour into crockpot and cover. Lower heat to low setting, and leave on all night. About 15 minutes before serving, stir in remaining ingredients, except almonds, and place in a serving dish. Sprinkle almonds over top. Serves 6-8.

*Toast almonds in a 350° F oven for about 8 minutes.

HOT CRACKED WHEAT CEREAL

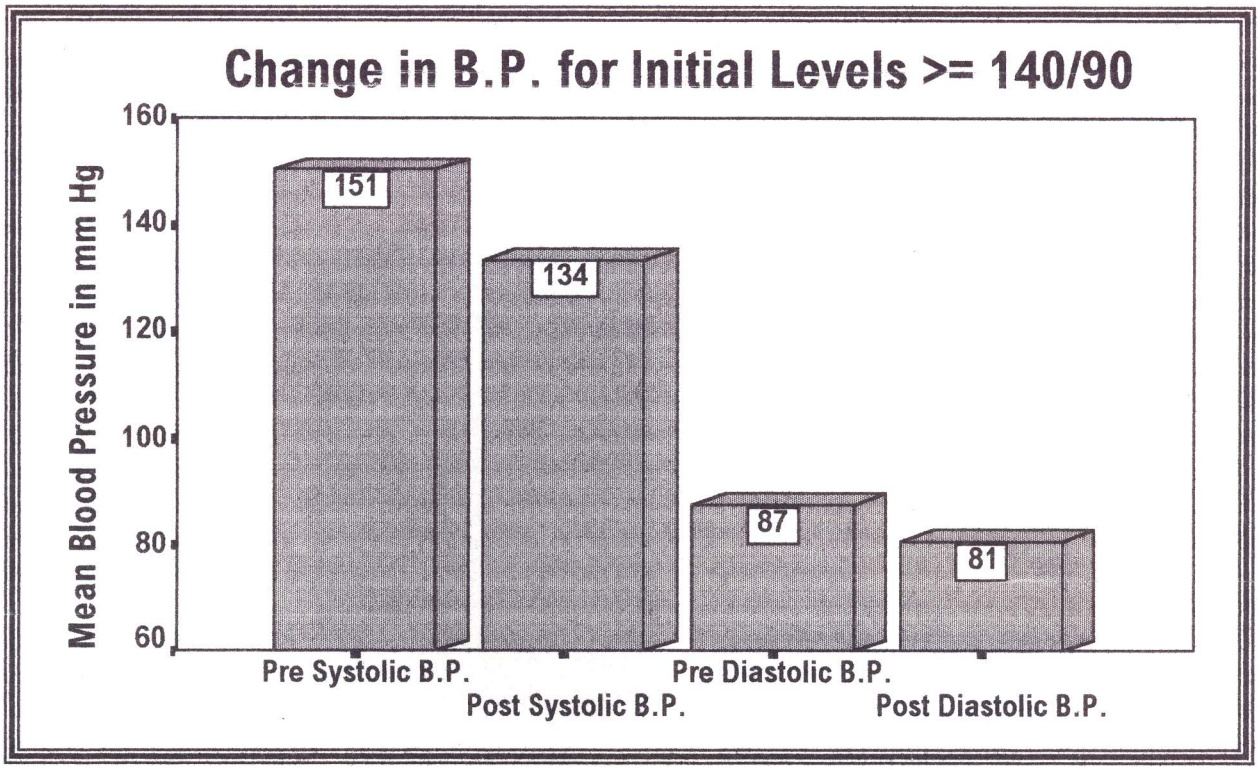
1 C cracked wheat
1/4 C unsweetened, shredded coconut (opt.)☆
1 qt water
1/4 C sesame seeds
8 pitted dates, chopped (opt.)
1 t salt

Dextrinize (toast) cracked wheat and coconut by stirring constantly in a dry pan over medium heat for several minutes, until lightly browned. Place in a saucepan and add remaining ingredients. Bring to a boil. Reduce heat and simmer for 15-20 minutes. Sprinkle with chopped nuts, if desired. Serves 4 to 6.

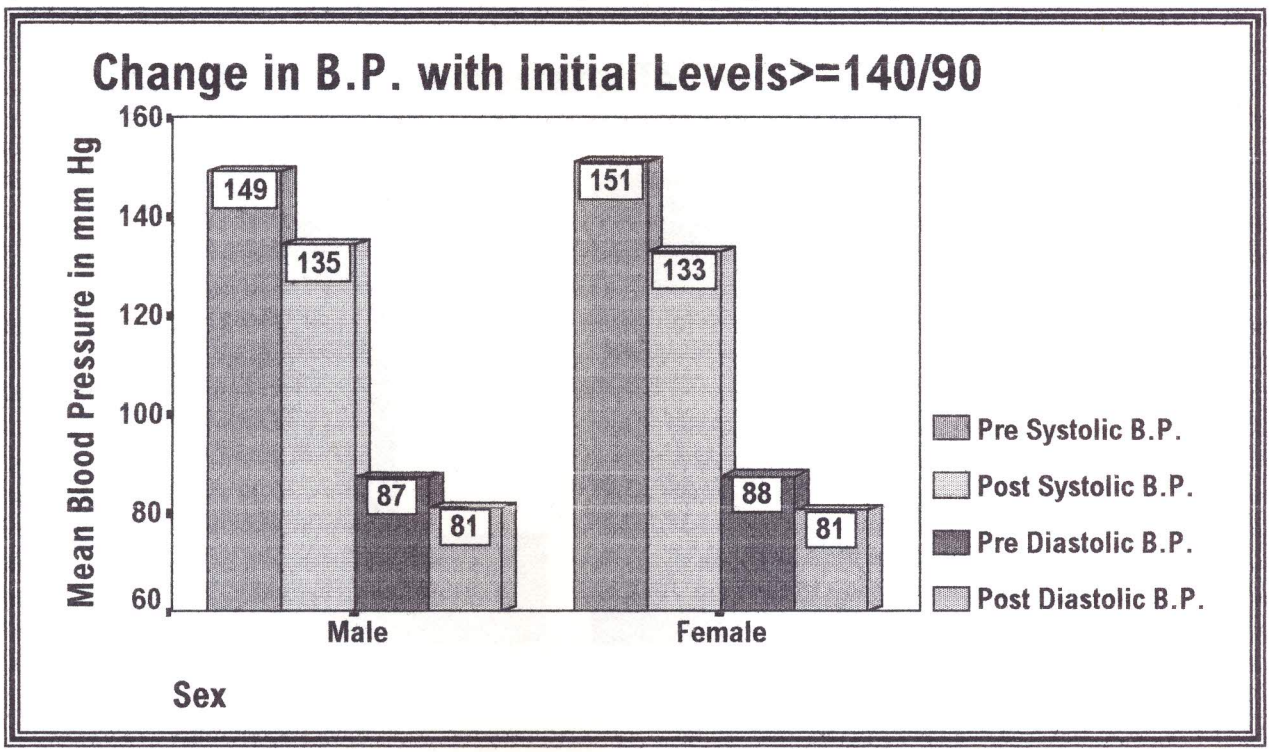
CREAMY MUESLI

2 1/2 C rolled oats
3 C apple, orange, or pineapple juice
2 bananas
1 t vanilla
1/2 t salt
1/4 t maple extract (opt.)
1/2 C raisins
1 C shredded apple

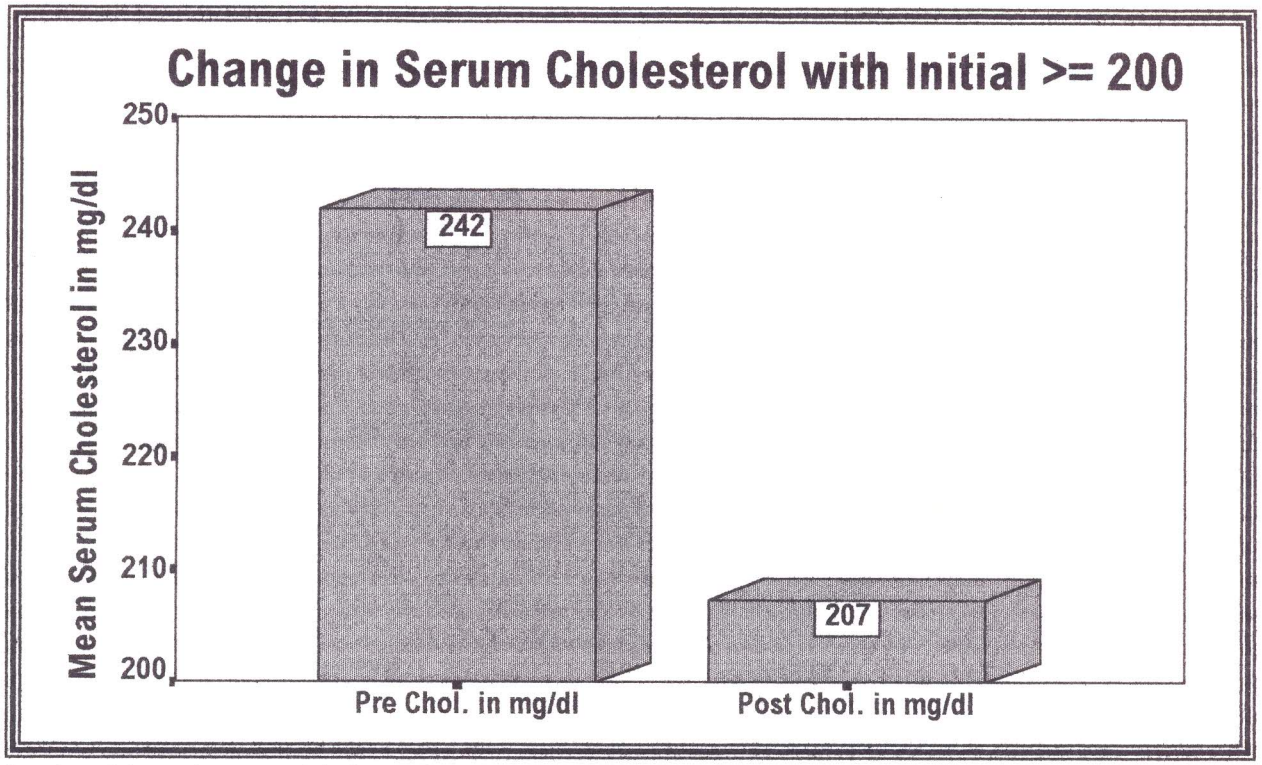
Dextrinize (toast) oats on a baking sheet at 300° F for 20 minutes, or until very lightly browned. Watch carefully to prevent burning. Place in a bowl. Blend next 5 ingredients until smooth. Stir into oats and add raisins. Cover and refrigerate overnight. Just before serving, shred apple and stir in. Serves 4.



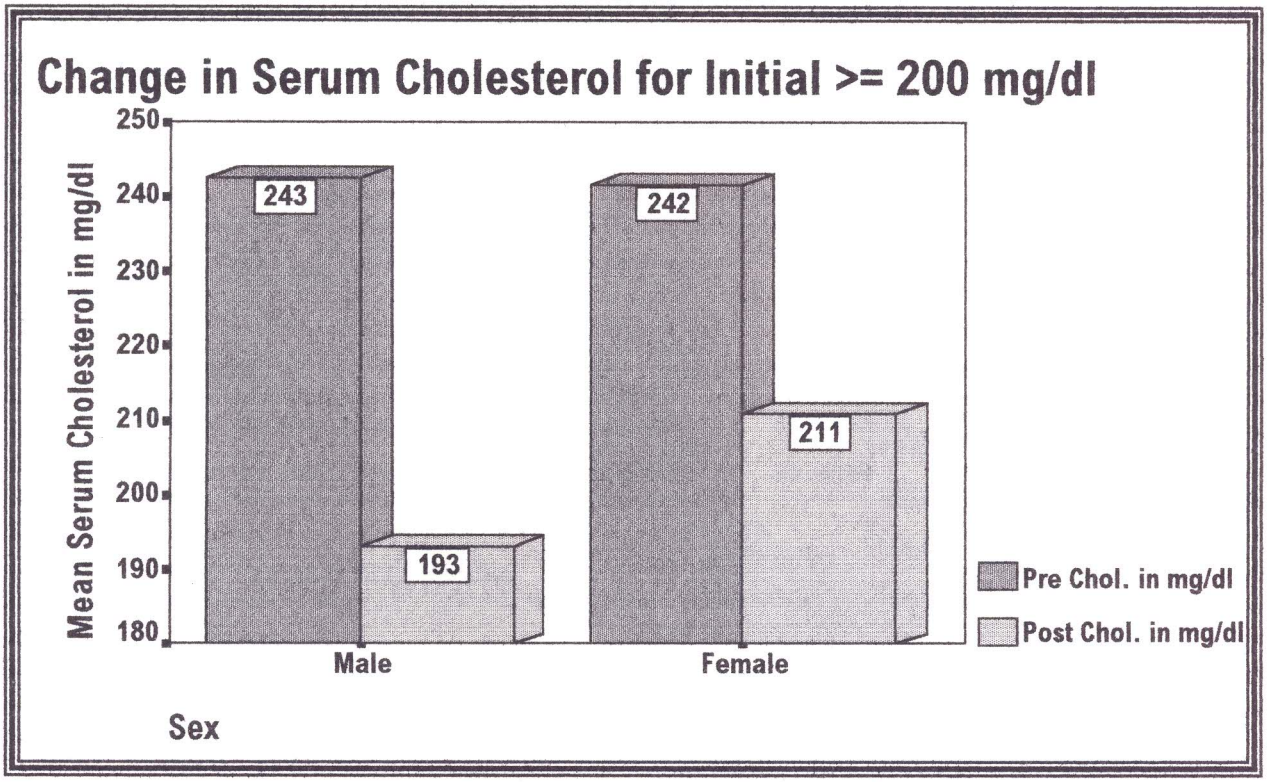
The 43 patients whose initial blood pressures were ≥140/90, systolic pressures dropped an average of **17 points or 11.3%**, and diastolic pressures dropped **6 points or 6.9%** after 4 weeks of lifestyle change($p = 0.000$).



This was broken down as an average **14 point or 9.4%** drop in systolic pressure and a **6 point or 6.9%** drop in diastolic for men, and an **18 point or 11.9%** and a **7 point or 8.0%** drop in the respective pressures for women.



In the 61 patients with initial serum cholesterol levels of ≥ 200 mg/dl, 4 weeks lifestyle change lowered values an average of **35 points or 14.5%** (p = 0.000)



These changes were seen as a **50 point or a 20.6%** drop for men, and a **31 point or 12.8%** drop in women.

CHANGE IN SERUM CHOLESTEROL FOR PATIENTS WITH INITIAL LEVELS >= 200 mg/dl

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_CHOL	Pre Cholesterol	61	.627	.000	242.0000	35.802	4.584
PST_CHOL	Post Cholesterol				207.4262	35.827	4.587

Paired Differences					
Mean	SD	SE of Mean	t-value	df	2-tail Sig
34.5738	30.948	3.962	8.73	60	.000
95% CI (26.646, 42.502)					

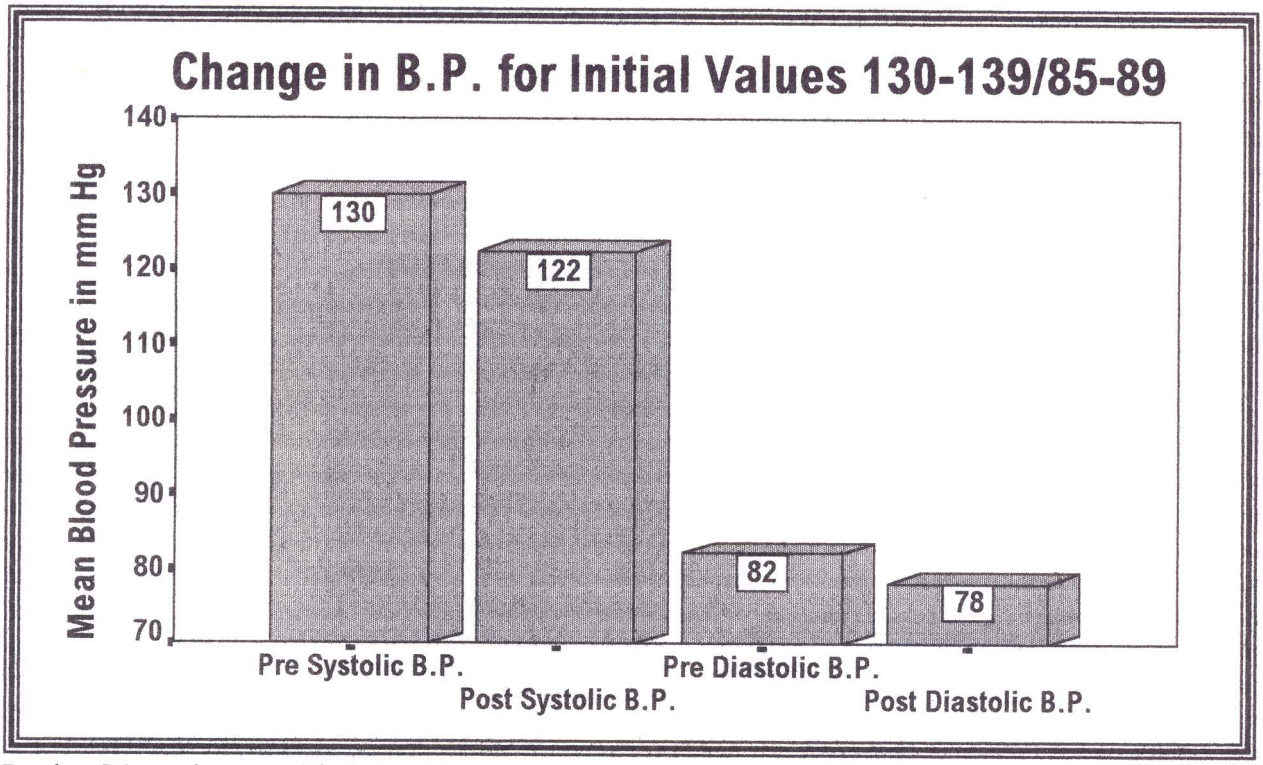
CHANGE IN BLOOD PRESSURE FOR PATIENTS WITH INITIAL LEVELS >= 140/90 mm Hg

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_BP_S	Pre Blood Pressure, Systolic	43	.109	.486	150.5581	13.693	2.088
PST_BP_S	Post Blood Pressure, Systoli				133.5814	15.084	2.300

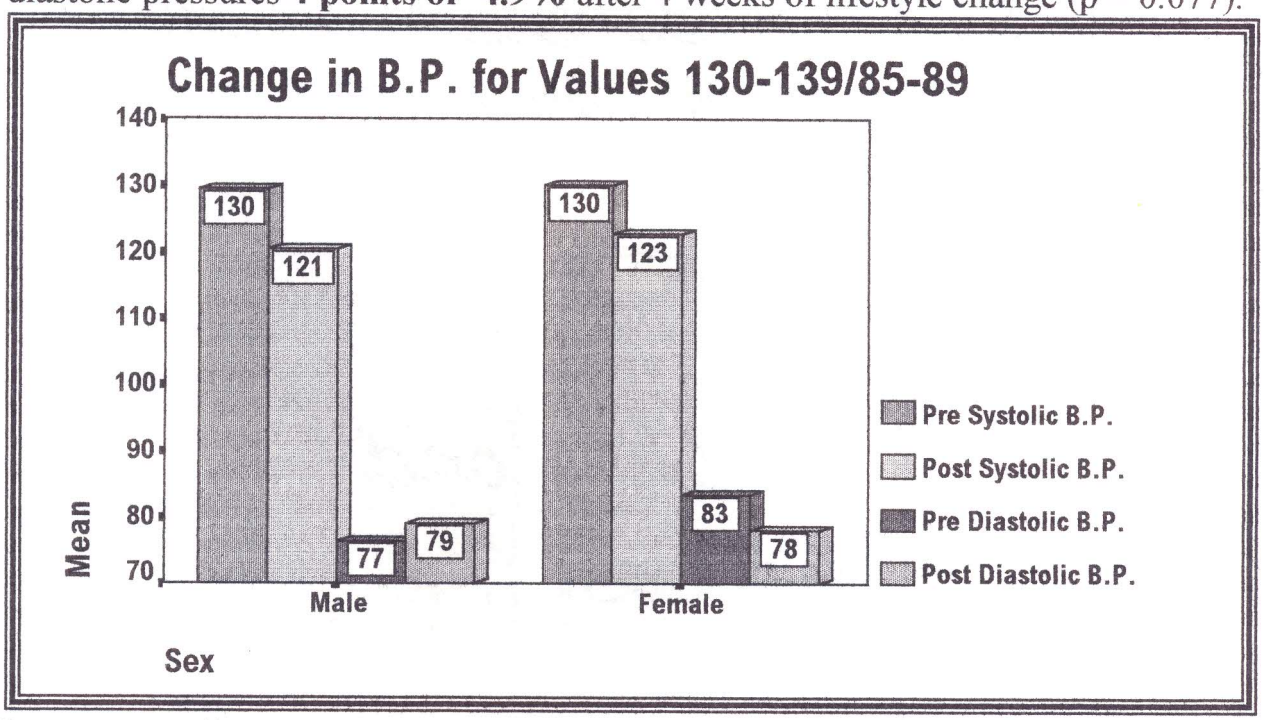
Paired Differences					
Mean	SD	SE of Mean	t-value	df	2-tail Sig
16.9767	19.235	2.933	5.79	42	.000
95% CI (11.056, 22.898)					

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_BP_D	Pre Blood Pressure, Diastoli	43	.497	.001	87.4419	8.611	1.313
PST_BP_D	Post Blood Pressure, Diastol				80.6744	7.593	1.158

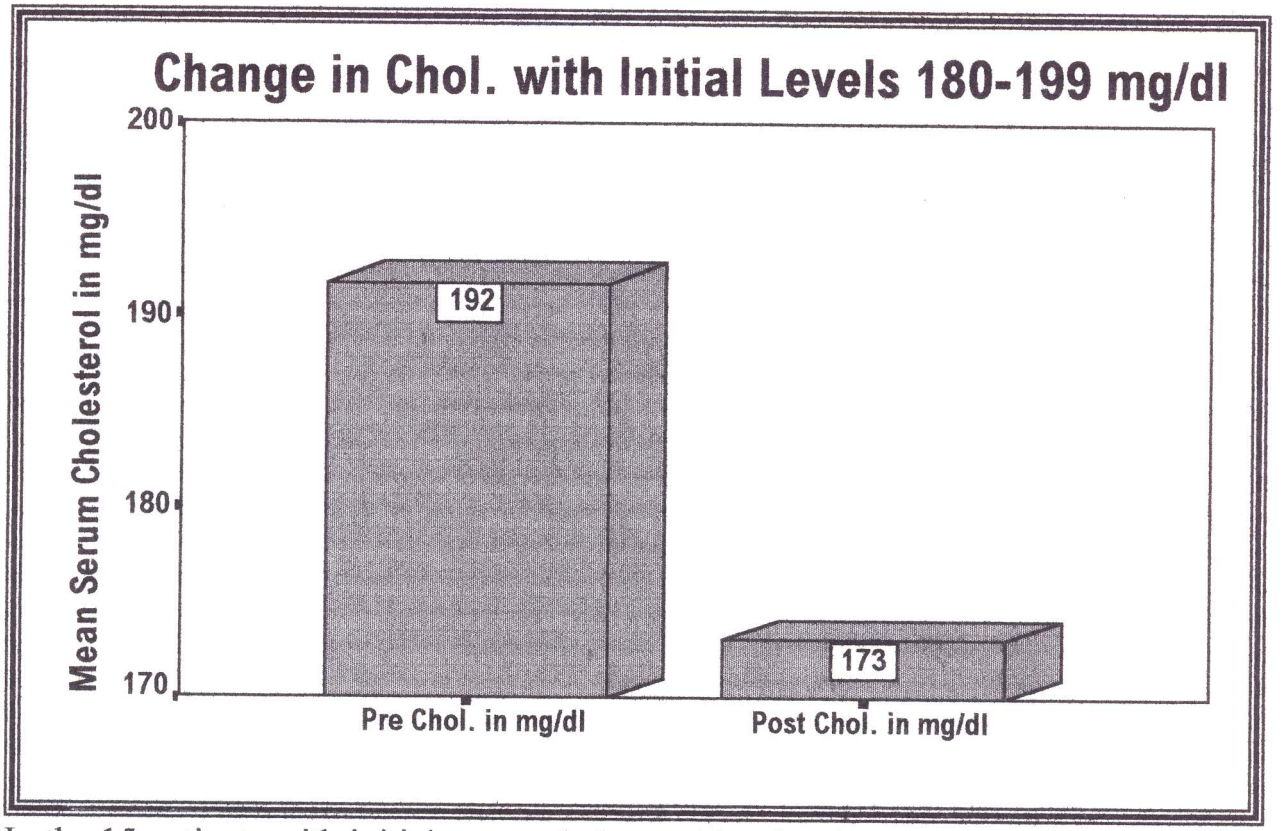
Paired Differences					
Mean	SD	SE of Mean	t-value	df	2-tail Sig
6.7674	8.170	1.246	5.43	42	.000
95% CI (4.252, 9.282)					



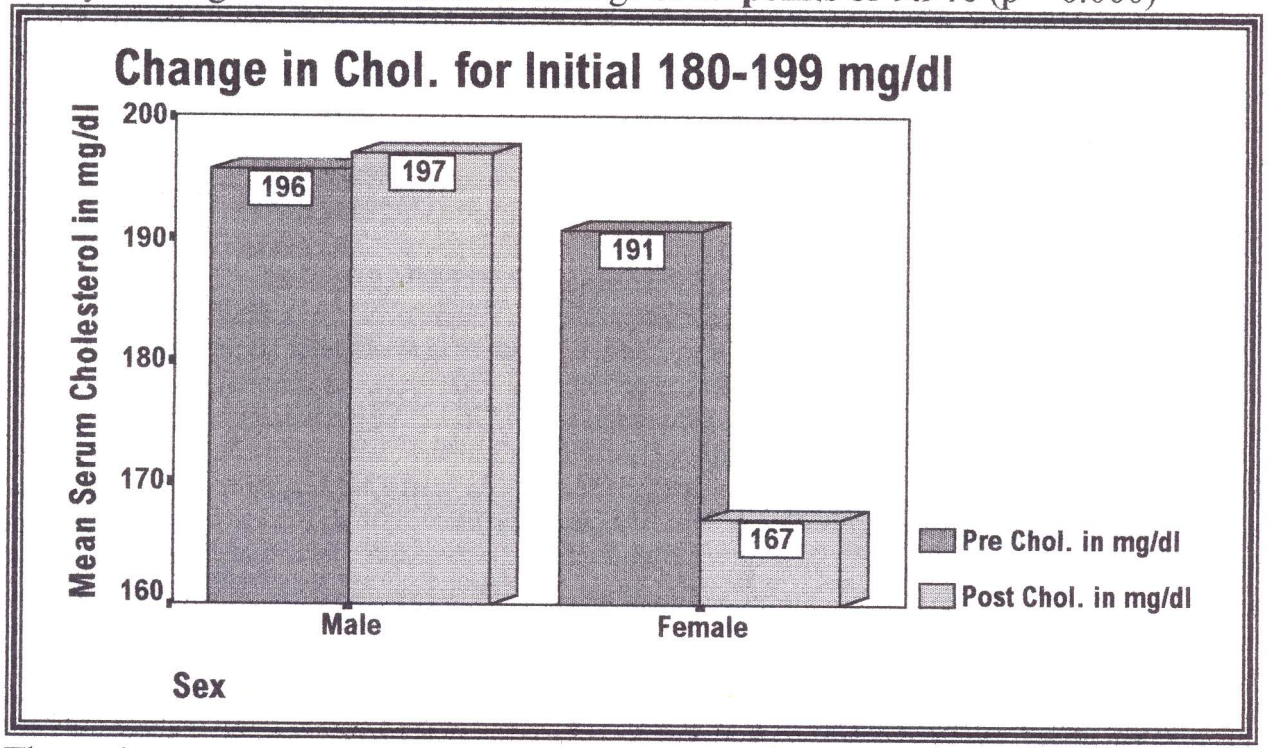
In the 21 patients with initial blood pressures 130-139/85-89 mm Hg, average resting systolic blood pressures were lowered **8 points or 6.2%** ($p = 0.020$) and diastolic pressures **4 points or 4.9%** after 4 weeks of lifestyle change ($p = 0.077$).



In men, systolic pressure was lowered **9 points or 6.9%** and diastolic pressure increased **2 points or 2.6%** (although only 3 men were in this subset). In women, blood pressures dropped **7 points or 5.4% and 5 points or 6.0%** respectively.



In the 15 patients with initial serum cholesterol levels of 180-199 mg/dl, 4 weeks of lifestyle change lowered values an average of **19 points or 9.9%** ($p = 0.000$)



These changes were seen as a **1 point or 1%** increase for men (only 3 men were in this subset), and a **24 point or 12.6%** drop in women.

CHANGE IN SERUM CHOLESTEROL FOR PATIENTS WITH INITIAL LEVELS 180-199 mg/dl

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_CHOL	Pre 12-hr Fasting Serum Chol	15	.037	.895	191.6667	5.287	1.365
PST_CHOL	Post Cholesterol				173.0667	27.533	7.109

Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
18.6000	27.841	7.188	2.59	14	.021
95% CI (3.178, 34.022)					

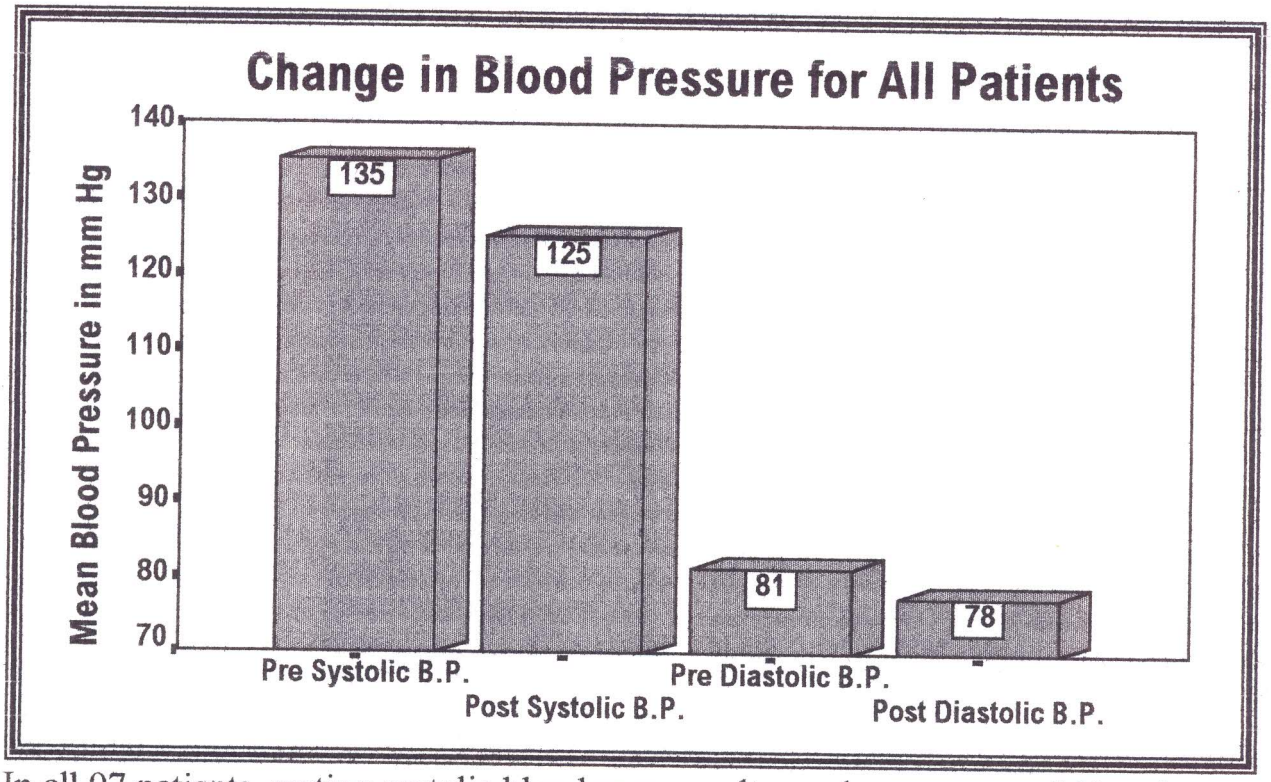
CHANGE IN BLOOD PRESSURE FOR PATIENTS WITH INITIAL LVLS. 130-139/85-89 mm Hg

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_BP_S	Pre Systolic B.P.	21	.161	.486	130.0000	5.692	1.242
PST_BP_S	Post Systolic B.P.				122.2857	13.774	3.006

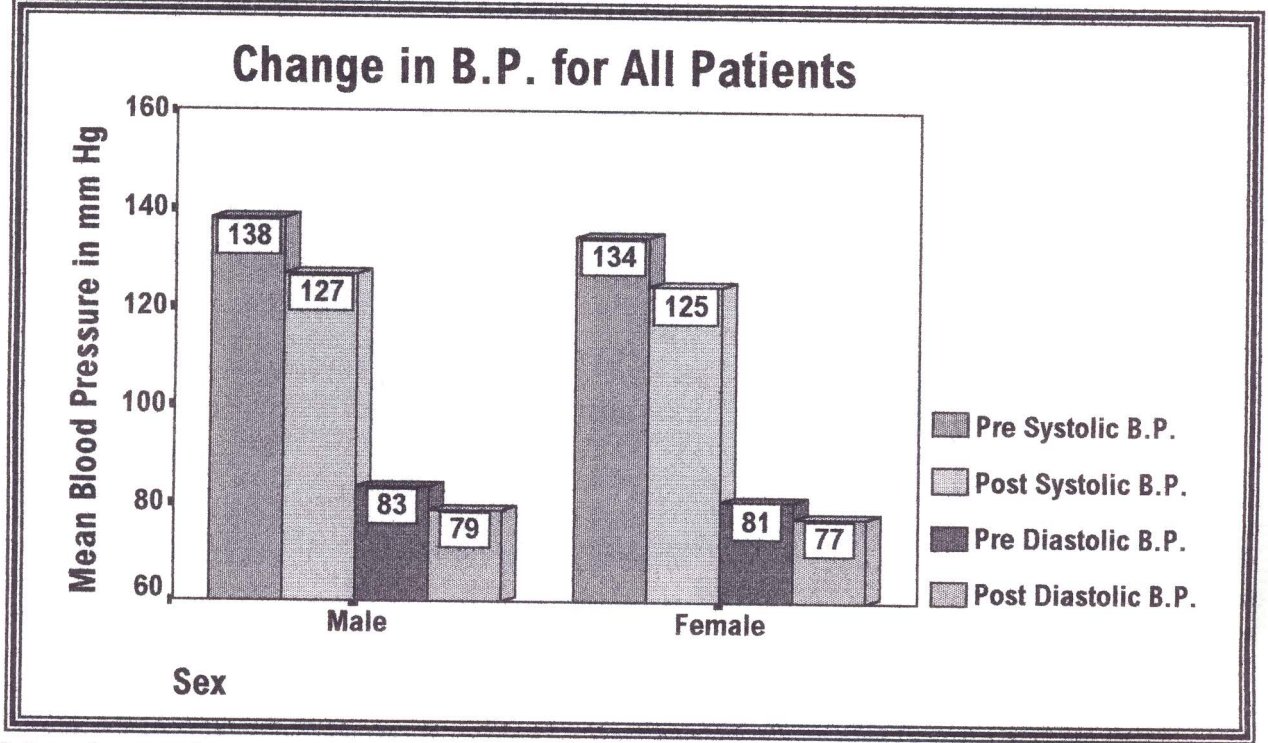
Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
7.7143	14.033	3.062	2.52	20	.020
95% CI (1.325, 14.103)					

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_BP_D	Pre Diastolic B.P.	21	.004	.986	82.0952	7.056	1.540
PST_BP_D	Post Diastolic B.P.				78.1905	6.509	1.420

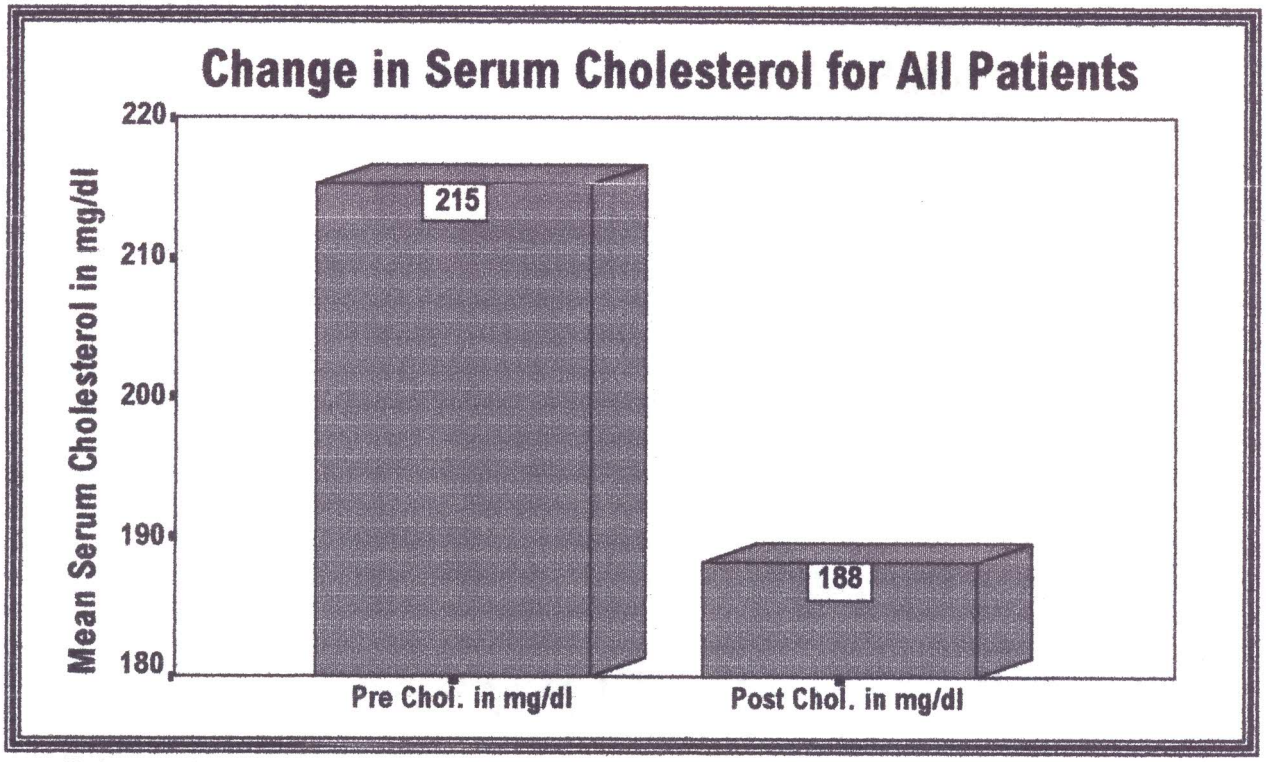
Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
3.9048	9.581	2.091	1.87	20	.077
95% CI (-.457, 8.267)					



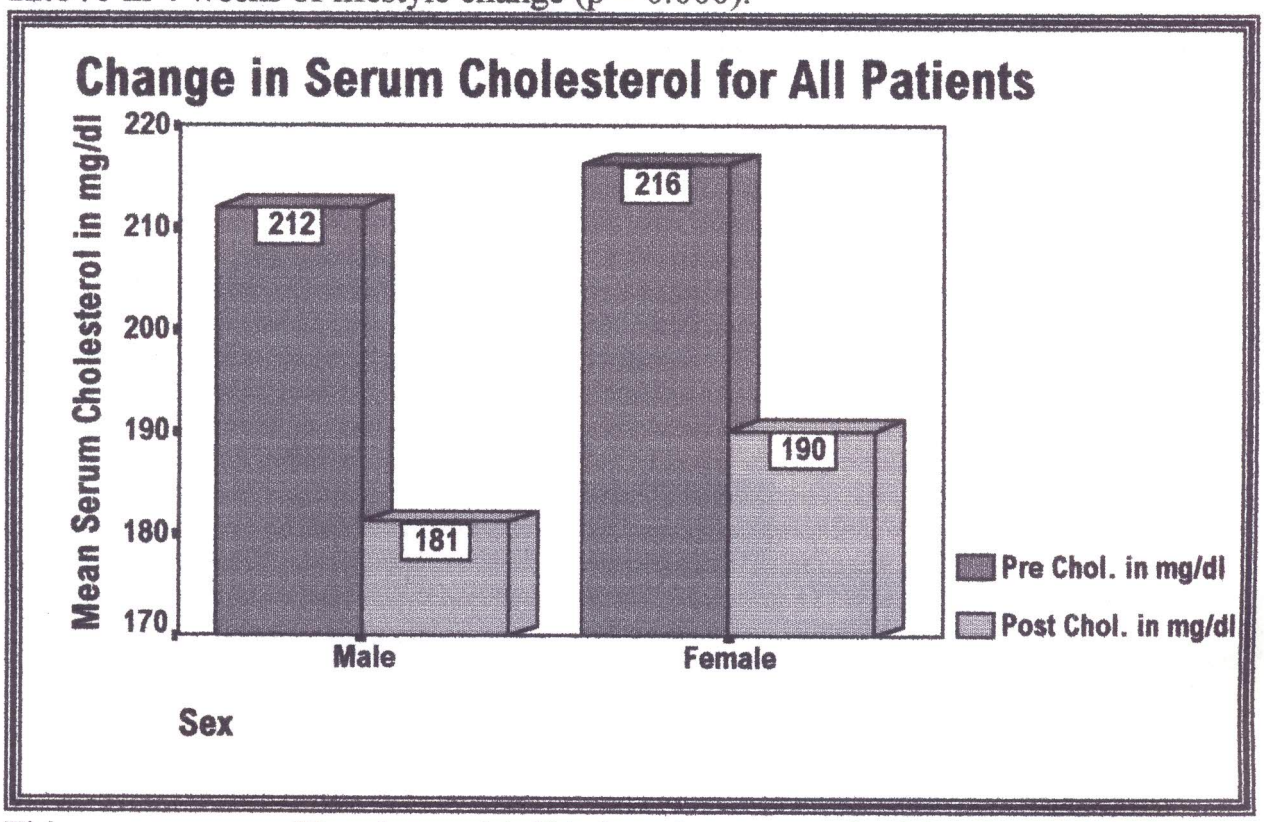
In all 97 patients, resting systolic blood pressure dropped an average of **10 points or 7.4%** ($p = 0.000$) and diastolic pressures dropped **3 points or 3.7%** ($p = 0.000$) after 4 weeks of lifestyle therapy.



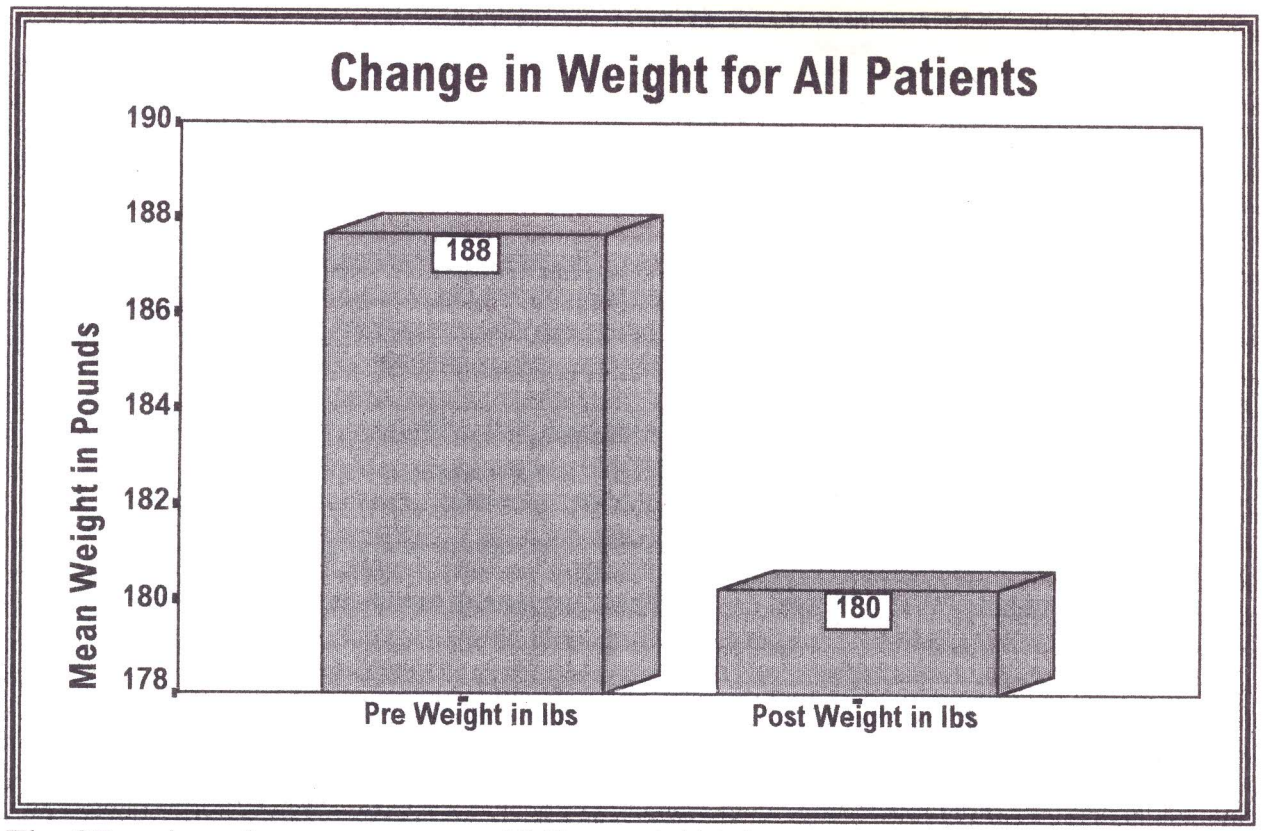
Men dropped an average of **11 points or 8.0%** in systolic pressures and **4 points or 4.8%** in diastolic. Women dropped **9 points or 6.7% and 4 points or 4.9%.**



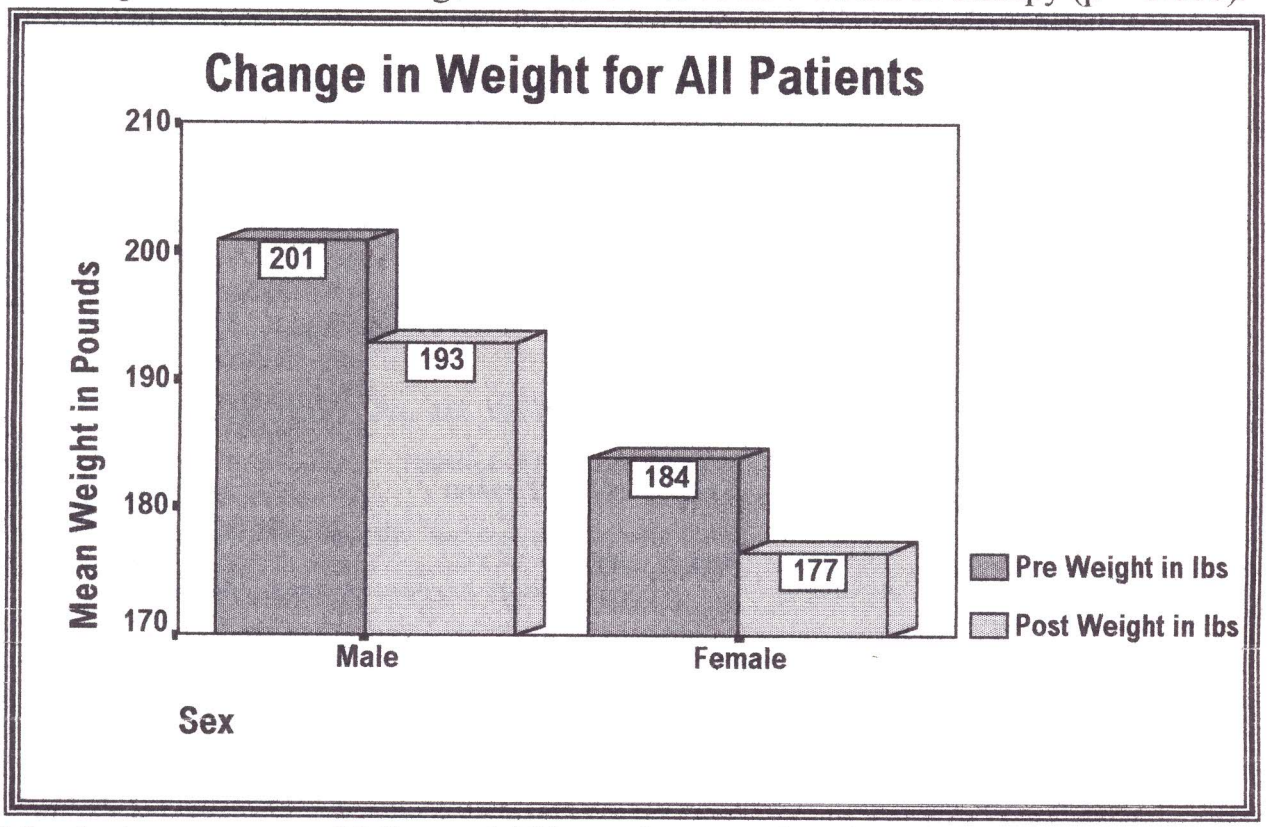
In all 97 patients, serum cholesterol levels dropped an average of **27 points or 12.6%** in 4 weeks of lifestyle change ($p = 0.000$).



This was seen as a **31 point or 14.6%** drop for men, and a **26 point or 12.0%** drop for women.



The 97 patients lost an average of **8 lbs or 4.3%** in 4 weeks of therapy (p = 0.000).



Men lost an average of **8 lbs or 4.0%** , and women lost **7 lbs or 3.8%.**

CHANGE IN SERUM CHOLESTEROL FOR ALL PATIENTS

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_CHOL	Pre 12-hr Fasting Serum Chol	97	.785	.000	215.3402	47.023	4.774
PST_CHOL	Post Cholesterol				188.2887	41.979	4.262

Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
27.0515	29.582	3.004	9.01	96	.000
95% CI (21.088, 33.015)					

CHANGE IN BLOOD PRESSURE FOR ALL PATIENTS

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_BP_S	Pre Blood Pressure, Systolic	97	.500	.000	135.2371	17.735	1.801
PST_BP_S	Post Blood Pressure, Systoli				125.0722	15.819	1.606

Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
10.1649	16.866	1.713	5.94	96	.000
95% CI (6.765, 13.565)					

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_BP_D	Pre Blood Pressure, Diastoli	97	.395	.000	81.3608	10.031	1.019
PST_BP_D	Post Blood Pressure, Diastol				77.5979	8.003	.813

Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
3.7629	10.064	1.022	3.68	96	.000
95% CI (1.734, 5.792)					

CHANGE IN WEIGHT FOR ALL PATIENTS

Variable		Number of pairs	Corr	2-tail Sig	Mean	SD	SE of Mean
PRE_WT	Pre Weight in Pounds	97	.994	.000	187.6701	39.847	4.046
PST_WT	Post Weight in Pounds				180.1959	38.093	3.868

Paired Differences			t-value	df	2-tail Sig
Mean	SD	SE of Mean			
7.4742	4.519	.459	16.29	96	.000
95% CI (6.563, 8.385)					