A One-Year Observational Study to Determine the Efficacy and Safety of Strauss Heartdrops® in Reducing Risk to Coronary Heart Disease

Study

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Appendices and References

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Confidential

ABSTRACT

Strauss Heartdrops, an all-herbal medication, was tested for safety and efficacy in a year long study enrolling fourteen volunteers. Over the one year period subjects ingested label-recommended dosages of the Heartdrops, three times per day, while maintaining their usual life-style and habits. Blood lipids and pressure were monitored, monthly, throughout the entire period. Blood pressure was taken using a standard gauge and cuff device and lipids were determined with a Polymer Technology Systems CardioChek portable whole blood test system.

Reduction in diastolic blood pressure was statistically significant and an important contribution to managing heart disease.

All 'at risk' blood lipids, including total cholesterol, LDL cholesterol, triglycerides and the total cholesterol/HDL were calculated using the CardioChek device. Over the one- year period, the subject group moved from blood lipid values that indicated increased risk to one of 'normal' or reduced risk of coronary heart disease. None of the subjects expressed undesirable side effects or discomfort from using the Heartdrops.

Reduction in blood lipid values was statistically significant.

From the results of this study, we conclude that Strauss Heartdrops are a safe and efficacious supplement for people at increased risk for heart disease.

INTRODUCTION

There are many medications available for lowering blood pressure, lowering cholesterol, and treating heart disease, both pharmaceutical and herbal. A number of the pharmaceutical preparations have proven to be dangerous with harmful side effects that can cause further illness and even death. (See Appendix B and C.) Clinical experience and long term use of herbal preparations indicate that they do not pose such a risk but few have been put through the rigors of scientific investigation to prove their safety and efficacy.

Heart disease remains the number one killer in North America. Elevated blood lipids such as total cholesterol, LDL cholesterol and triglycerides are used as indicators of increased risk to this illness. In addition, blood pressure, HDL and the ratio of total cholesterol to HDL are also used to determine persons at elevated risk to this illness.

Total cholesterol is the sum of all the cholesterol in an individual's blood. The higher the total cholesterol, the greater the risk for heart disease. Less than 5.2 mmol/L is a desirable level that lowers a person's risk for heart disease. A cholesterol level of 5.2 mmol/L or greater increases risk. A level of 5.2 to 6.2 mmol/L is called 'Borderline-high' and 6.2 mmol/L and above are called 'High' blood cholesterol. A person with a high level has more than twice the risk of heart disease compared to someone whose cholesterol is below 5.2 mmol/L. Similarly, other blood lipids such as LDL and Triglycerides indicate increased risk as their levels rise.

Means of lowering these parameters have normally focused on diet and exercise or various medications, however long term lowering of blood lipids often proves extremely difficult, particularly with the elderly.

In this study we demonstrate how Strauss Heartdrops, an herbal preparation lowers blood lipids that elevate a persons risk to heart disease and does this in a safe and efficacious manner, and lowers diastolic blood pressure.

We not only show that Strauss Heartdrops are efficacious for circulatory support but by using this safe herbal preparation people are avoiding the numerous side effects that are commonly found with cholesterol lowering drugs and antihypertensive drugs. (See Appendix B and C.)

MATERIALS AND METHODS

Twenty volunteers were chosen for the study. These were selected from respondents to a newspaper advertisement made prior to the study. The original 20 were selected based upon certain criteria around personal organization and note taking habits relating to their illness. The persons selected for the study all had elevated blood cholesterol and elevated blood pressure. Other blood lipids such as HDL, LDL and Triglycerides were also elevated and out of the normal range as determined by standard medical guidelines. Therefore, all of the subjects chosen for the study were at an increased risk to heart disease based upon accepted values for blood lipids and pressure.

The age of the study group was between 58 and 67 years. Fourteen of the original 20 finished the study. Of the 6 that dropped out 2 moved away and 4 lost interest or did not show up for meetings. Of the 14 completing volunteers, 9 were male and 5 female. All were currently on or had been taking some sort of prescribed heart medication prior to the study. None of the clients was asked to interrupt their usual medications, but were instructed to take the Heartdrops as suggested along with their routine medications.

Subjects were asked to provide medical records regarding their heart condition prior to the study. Values for total cholesterol, HDL, LDL and triglycerides were accumulated for each subject as a baseline before beginning the study. The first two samplings completed in January and February 2004 were included with this data to encompass the 'before' part of the study. The subjects were then sampled monthly for 12 months with the last three samplings used to calculate the 'after' part of the study.

On a monthly basis, blood pressure was recorded for each patient. This was done using a Welch Allyn Tycos blood pressure cuff and gauge. Because of the variability in blood pressure readings, care was taken to make sure that the subject was positioned in a similar fashion each time a reading was made and that the subject was relaxed and at rest. Finger prick blood samples were also taken at this time and blood lipid parameters such as total cholesterol, HDL, LDL and triglycerides were determined using a Polymer Technology Systems Cardio Chek portable whole blood test system. This device has been tested and employed in a number of clinical trials.

Dietary information was also collected for each of the study volunteers. On each sampling date, the client was asked to write down *everything* he or she ate and drank

(consumed) for the 24 hr period, prior to the sampling. In this way, we were able to accumulate data on the food intake habits of the client and thus shed light on the nutritional status of the individual.

Subjects were asked to not eat 12 hours before blood lipid and pressure samplings.

All volunteers were asked to administer the Heartdrops in the same fashion. A full dropper, approximately 1 ml pipetted under the tongue, held for a short time and swallowed, 3 times per day. Subjects were instructed to keep the Heartdrops bottle in a convenient place that they routinely visit during their day such as their desk or kitchen allowing better compliance for taking the medication.

RESULTS AND DISCUSSION

Accumulated data for each subject was averaged for the first two samplings, plus data from medical records collected before the study. This data provided the 'before' section of the study and acts as the control for the experiment. The 'after' data is an average of two samplings. Since the Heartdrops is a complex mixture of herbs with a distinct odour and taste, a placebo for this study would have been impossible or lacking in suitability. This study was carried out in such a fashion as to demonstrate efficacy and safety of the Heartdrops by achieving a statistically significant difference between the levels of the blood parameters tested before and at the end of the supplementation period, with no observable side-effects.

Results for total cholesterol showed that the test group moved from a range of high risk (greater than 6.2) to a normal range between 2.0 and 5.2, a statistically significant finding ($P<0.005$). A similar effect was seen with LDL cholesterol where the study group's average LDL was also lowered significantly ($P< 0.005$) from an at risk range (>3.5) to a normal range between 1.5 and 3.4.

A highly significant change was observed with the ratio of total cholesterol to HDL wherein a ratio greater than 5 is associated with increased risk to heart disease. The group went from an average of 6.1 to 5.1 with a P value less than 0.05.

The average blood triglyceride levels of the group were at the low end of the at risk range when beginning the study the mean value was decreased into the normal range (<2.3) over the one year period. This difference was significant, with a P value of 0.03.

In this study significant decrease in diastolic blood pressure ($P<0.005$) was observed, however the systolic pressure decrease was not significant ($P=0.14$).

Overall the data fit the working hypothesis that Strauss Heartdrops could significantly lower a persons risk to heart disease by reducing blood lipids from an 'at risk' range to a normal range and reduce diastolic blood pressure over a one year period. By significantly reducing total cholesterol and LDL and by also reducing the blood levels of triglycerides plus the ratio of cholesterol to HDL without a significant reduction in HDL, it appears that the Heartdrops are beneficial to anyone suffering from elevated levels of these risk factors.

FIGURE 1: REDUCTION IN TOTAL CHOLESTEROL OVER ONE YEAR PERIOD: 31% (ACTUAL MONTHLY RESULTS AVERAGED FOR 14 PARTICIPANTS)

(Above 5 mmol/L indicates increased risk to Coronary Heart Disease)

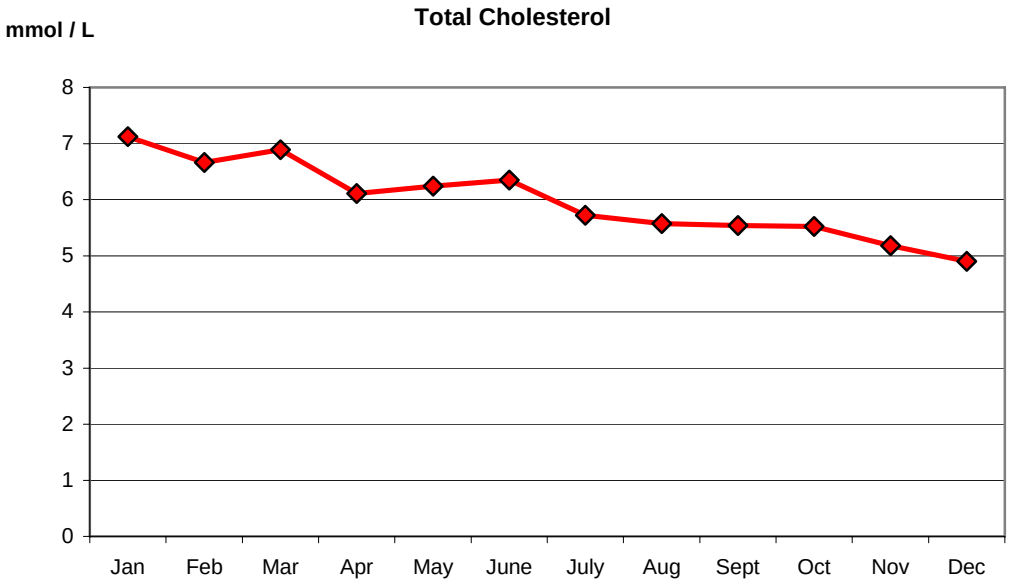


FIGURE 2: REDUCTION IN LDL CHOLESTEROL OVER ONE YEAR PERIOD: 38%

(Above 3.5 mmol/L indicates increased risk to Coronary Heart Disease)

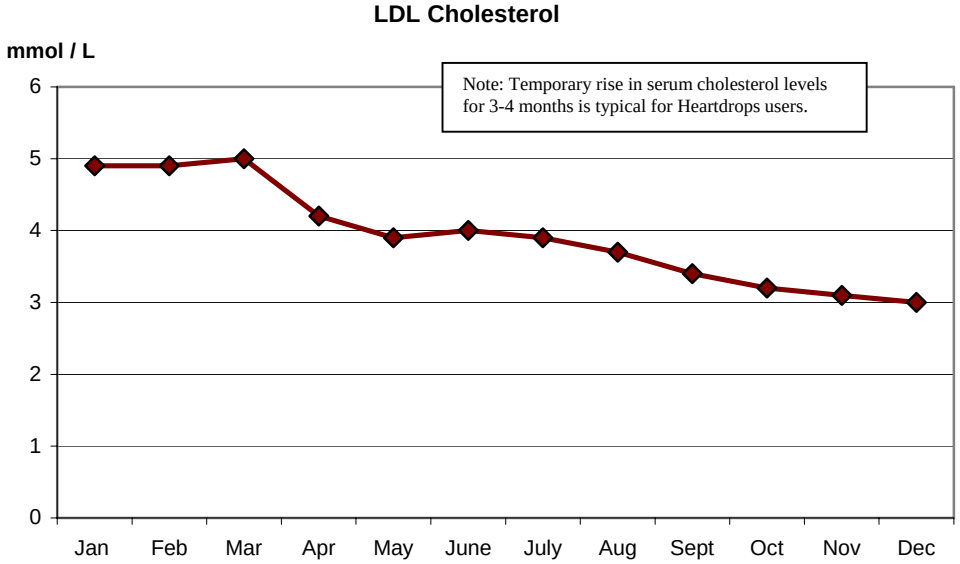
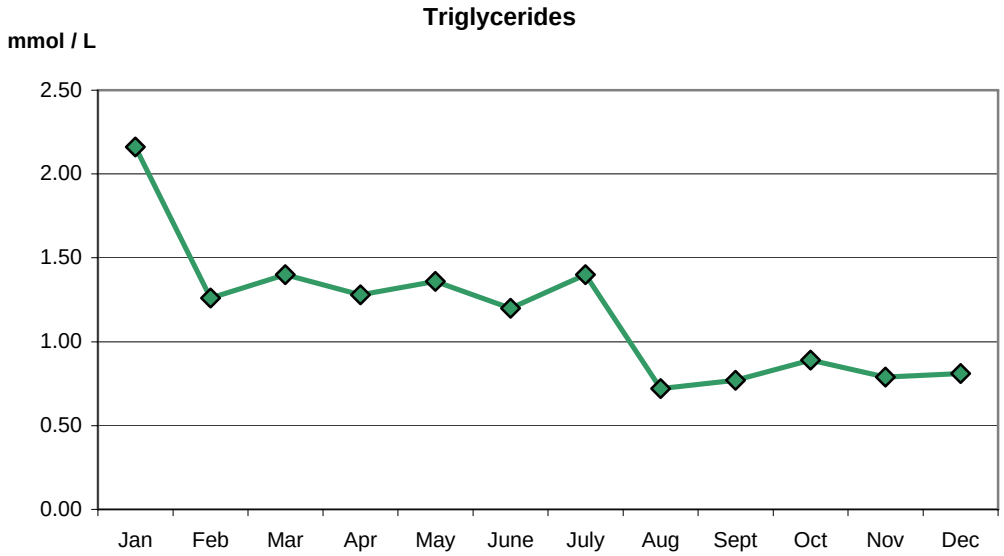


FIGURE 3: REDUCTION IN TRIGLYCERIDES OVER ONE YEAR PERIOD: 63%

(Above 2.0 mmol / L indicates increased risk to Coronary Heart Disease)



Total Cholesterol/HDL ratio and Blood Pressure charts not shown. Refer to statistical data.

CONCLUSIONS

This study has demonstrated the effectiveness of Strauss Heartdrops in lowering both blood lipids and diastolic blood pressure that are associated with increased risk to heart disease. The Heartdrops appear to lower blood lipids and diastolic blood pressure in a safe and relatively gentle manner. Since all of the blood lipids measured showed a decrease from an at risk range to a normal range with no side effects detected, the Heartdrops present an excellent supplement for circulatory system support.

STATISTICAL DATA (ALL SUBJECTS)

Total Cholesterol

Subject #	Before	After
1	8.18	5.91
2	6.42	4.48
3	5.8	5.34
4	7.25	5.72
5	6.97	6.06
6	6.45	7.02
7	6.11	4.53
8	7.87	7.46
9	6.50	5.05
10	5.13	4.25
11	5.98	3.24
12	7.54	6.89
13	5.46	4.22
14	6.06	5.65
Total	**91.72**	**75.82**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	6.55143	5.41571
Variance	0.81544	1.46578
Observations	14	14
Pearson Correlation	0.70059	
Hypothesized Mean Difference	0	
df	13	
t Stat	4.90903	
P(T<=t) one-tail	0.00014	
t Critical one-tail	1.77093	
P(T<=t) two-tail	0.00029	
t Critical two-tail	2.16037	

HDL

Subject #	Before	After
1	1.09	1.24
2	1.66	1.06
3	1.53	1.09
4	0.80	0.88
5	1.37	1.5
6	1.01	0.93
7	0.93	0.91
8	2.33	1.76
9	1.22	1.5
10	0.67	0.57
11	0.73	0.65
12	1.42	1.81
13	0.85	0.73
14	1.01	1.04
	16.62	**15.67**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	1.1871	1.11929
Variance	0.2011	0.15485
Observations	14	14
Pearson Correlation	0.7625	
Hypothesized Mean Difference	0	
df	13	
t Stat	0.8616	
P(T<=t) one-tail	0.2023	
t Critical one-tail	1.7709	
P(T<=t) two-tail	0.4045	
t Critical two-tail	2.1604	

LDL

Subject #	Before	After
1	6.1	4
2	4.1	2.9
3	3.8	3.6
4	4.4	4.2
5	5.3	4.1
6	4.4	4.7
7	4.6	3.0
8	4.3	4.5
9	4.7	3.1
10	2.9	2.8
11	3.3	1.1
12	5.8	4.7
13	3.6	2.9
14	4.6	4.0
Total	**61.9**	**49.6**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	4.42143	3.54286
Variance	0.79874	0.96879
Observations	14	14
Pearson Correlation	0.62324	
Hypothesized Mean Difference	0	
df	13	
t Stat	4.01297	
P(T<=t) one-tail	0.00074	
t Critical one-tail	1.77093	
P(T<=t) two-tail	0.00148	
t Critical two-tail	2.16037	

Ratio

Subject #	Before	After
1	7.6	4.8
2	3.9	4.2
3	3.4	4.9
4	9.1	6.6
5	5.1	4.0
6	6.4	7.4
7	6.0	5.0
8	3.4	4.2
9	5.3	3.3
10	9.8	7.5
11	8.3	5.0
12	5.3	3.8
13	6.3	5.9
14	6.7	5.5
	86.6	**72.1**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	6.1857	5.15
Variance	3.9905	1.69038
Observations	14	14
Pearson Correlation	0.6599	
Hypothesized Mean Difference	0	
df	13	
t Stat	2.5817	
P(T<=t) one-tail	0.0114	
t Critical one-tail	1.7709	
P(T<=t) two-tail	0.0228	
t Critical two-tail	2.1604	

Trig

Subject #	Before	After
1	2.11	1.40
2	1.28	1.20
3	1.11	1.40
4	4.32	1.47
5	0.59	0.87
6	2.37	2.84
7	1.05	1.22
8	2.52	2.38
9	1.08	1.03
10	3.75	1.84
11	4.14	3.08
12	0.68	0.78
13	2.23	1.19
14	2.18	1.21
Total	**29.48**	**19.16**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	2.1007143	1.565
Variance	1.5520841	0.509888
Observations	14	14
Pearson Correlation	0.6464394	
Hypothesized Mean Difference	0	
df	13	
t Stat	2.0991389	
P(T<=t) one-tail	0.0279499	
t Critical one-tail	1.7709317	
P(T<=t) two-tail	0.0558999	
t Critical two-tail	2.1603682	

BP (S)

Subject #	Before	After
1	120	120
2	126	160
3	154	140
4	150	160
5	160	130
6	150	130
7	138	140
8	130	128
9	150	140
10	180	180
11	160	150
12	140	140
13	185	180
14	178	160
Total	**2121**	**2058**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	151.5	147
Variance	401.03846	350.6154
Observations	14	14
Pearson Correlation	0.6919337	
Hypothesized Mean Difference	0	
df	13	
t Stat	1.1036964	
P(T<=t) one-tail	0.1448681	
t Critical one-tail	1.7709317	
P(T<=t) two-tail	0.2897363	
t Critical two-tail	2.1603682	

BP (D)

Subject #	Before	After
1	83	80
2	80	80
3	90	90
4	80	80
5	90	80
6	88	85
7	82	70
8	80	70
9	83	80
10	90	80
11	84	84
12	85	80
13	80	80
14	82	78
	1177	**1117**

t-Test: Paired Two Sample for Means

	Variable 1	Variable 2
Mean	84.071429	79.785714
Variance	15.302198	26.489011
Observations	14	14
Pearson Correlation	0.5510045	
Hypothesized Mean Difference	0	
df	13	
t Stat	3.616836	
P(T<=t) one-tail	0.0015503	
t Critical one-tail	1.7709317	
P(T<=t) two-tail	0.0031006	
t Critical two-tail	2.1603682	

APPENDIX A

SYSTOLIC BLOOD PRESSURE

Taken from The National Heart, Lung, and Blood Institute of the NIH:

Both numbers in a blood pressure test are important, but for people who are 50 or older, systolic pressure gives the most accurate diagnosis of high blood pressure. Systolic pressure is the top number in a blood pressure reading. It is high if it is 140 mmHg or above.

What is systolic blood pressure?

Systolic pressure is the force of blood in the arteries as the heart beats. It is shown as the top number in a blood pressure reading. High blood pressure is 140 and higher for systolic pressure. Diastolic pressure does not need to be high for you to have high blood pressure. When that happens, the condition is called "isolated systolic hypertension," or ISH.

Is isolated systolic high blood pressure common?

Yes. It is the most common form of high blood pressure for older Americans. For most Americans, systolic blood pressure increases with age, while diastolic increases until about age 55 and then declines. About 65 percent of hypertensives over age 60 have ISH. You may have ISH and feel fine. As with other types of high blood pressure, ISH often causes no symptoms. To find out if you have ISH — or any type of high blood pressure — see your doctor and have a blood pressure test. The test is quick and painless.

Is isolated systolic high blood pressure dangerous?

Any form of high blood pressure is dangerous if not properly treated. Both numbers in a blood pressure test are important, but, for some, the systolic is especially meaningful. That's because, for those persons middle aged and older, systolic pressure gives a better diagnosis of high blood pressure.

If left uncontrolled, high systolic pressure can lead to stroke, heart attack, congestive heart failure, kidney damage, blindness, or other conditions. While it cannot be cured once it has developed, ISH can be controlled.

Clinical studies have proven that treating a high systolic pressure saves lives, greatly reduces illness, and improves the quality of life. Yet, most Americans do not have their high systolic pressure under control.

APPENDIX B
ANTIHYPERTENSIVE DRUGS AND THEIR SIDE EFFECTS

DIURETICS

Diuretics are the first line of drug treatment for hypertension. They are prescribed to lower the fluid level in the body in order to take pressure off the blood vessels. Fluid is eliminated but essential minerals such as sodium, potassium, magnesium, and zinc are also flushed out. Most people know that potassium is lost due to diuretics and are either prescribed potassium pills or advised to eat bananas as a source of potassium. However, the loss of magnesium, which is required for proper heart function, goes unchecked. The daily loss of magnesium can also affect bone health and lead to osteoporosis. Diuretics cause a loss of the water-soluble vitamins, especially the B-vitamins, which are flushed out with the urine.

Dehydration is a common side effect of diuretics, which can be devastating for an elderly person leading to heat stroke, undiagnosed low-grade fever, and constipation. And according to the Canadian Compendium of Pharmaceuticals and Specialties (CPS) and the U.S. Physician's Desk Reference (PDR)[1] diuretics, in general, worsen left ventricular heart function and glucose tolerance, and exert detrimental effects on the lipid profile.

THIAZIDE DIURETICS

Thiazides are the most commonly prescribed diuretics. By an unknown mechanism, thiazide diuretics increase the excretion of sodium, chloride, and water by inhibiting sodium ion transport across the renal tubular epithelium. Unfortunately, the mechanism of action and effectiveness of the drug requires that sodium be eliminated, but sodium deficiency causes electrolyte disturbances and upsets many metabolic functions in the cells of the body. Thiazides also increase the excretion of potassium, magnesium, and bicarbonate, and decrease the elimination of calcium and uric acid. The retention of calcium can lead to a further deficiency of magnesium. An increased calcium-to-magnesium ratio is a cause of heart disease. Elevation of uric acid can lead to gout, which requires anti-inflammatory drugs and Allopurinol for relief.

If proper blood tests are not done to determine electrolyte and mineral deficiency and if deficient minerals and electrolytes are not replaced, the side effects manifesting as dry mouth, weakness, dizziness, muscle pains or cramping muscles, constipation, confusion, drowsiness, and elevated heart rate are not attributed to their rightful cause. Patients with undiagnosed mineral and electrolyte deficiencies are often given medications to relieve constipation, muscle spasms, and an elevated heart rate while the original cause goes untreated.

The PDR and CPS list about 40 serious adverse reactions to hydrochlorothiazide, the most common thiazide diuretic. These range from agranulocytosis (destruction of white blood cells) to xanthopsia (a visual defect in which colored objects appear unnaturally yellow and colorless objects appear tinged with yellow). Loss of appetite, headaches, skin rashes and impotence are the most common side effects. If neither the patient nor doctor suspect the diuretic as the cause, pain medications are given for the headaches, cortisone creams for skin rash, and Viagra for impotence. All these additional drugs have their own side effects adding to the body's toxic burden.

Thiazide diuretics have measurable interactions with other medications. They may raise the levels or prolong the effects of the following drugs: Allopurinol, anesthetics, calcium supplements, chemotherapy drugs, digoxin, lithium, and loop diuretics. Other drugs may be reduced in potency or flushed out of the body. These include anticoagulants, insulin, muscle relaxants, and sulfonylureas (antidiabetic drugs). There are other drugs that increase the potency of thiazide drugs: Amphotericin B, anticholinergics, corticosteroids, and laxatives; whereas others reduce the effectiveness of the thiazides: cholystyramine (cholesterol-lowering drug), colestipol (cholesterol-lowering drug), methenamines (for bladder infections), and NSAIDs (non-steroidal anti-inflammatory drugs).

BETA BLOCKERS

Beta blockers, like propranolol, approved by the FDA in 1967, lower blood pressure by reducing the strength of the heart muscle contraction, slowing the heart rate, and relaxing the arteries. They act to stop certain catecholamine adrenal hormones from stimulating the heart. They are usually not used alone but given in combination with a thiazide diuretic. They are not just anti-hypertensives but are prescribed for angina, irregular heart rhythm, migraines, and anxiety. However, the side effects of these medications include myocardial infarction—due to serious arrhythmias—congestive heart failure, stroke, worsening of diabetic peripheral circulation, and asthma.

With a less forceful heart action the very real side effects of beta blockers are dizziness and fatigue, which interfere with quality of life. They also cause sexual dysfunction. Dizziness, fatigue, and sexual dysfunction can lead to depression. Patients are offered Viagra and Prozac to cope with these side effects. Dozens of other side effects include alopecia (hair loss), heart block, high blood sugar, and chronic itching.

Almost fifty drugs are affected by interaction with beta blockers, including blood thinners, digoxen, diabetic drugs, calcium channel blockers, other diuretics, MAO inhibitors, aspirin, and thyroid hormones.

ACE INHIBITORS

ACE inhibitors are angiotensin antagonists. They exert their effect by blocking production of several natural body chemicals, especially angiotensin. When the blood pressure drops - for whatever reason (for example, it could be due to suddenly jumping out of bed), the kidney releases a hormone called renin. Renin immediately stimulates angiotensin to constrict the arteries, which raises blood pressure. Angiotensin further stimulates the adrenal hormone aldosterone, which signals the kidney to hold onto sodium and eliminate potassium, which causes fluid to build up and raises blood pressure. ACE inhibitors interfere with this crucial activity. They are prescribed for high blood pressure, usually in combination with thiazide diuretics. Some types of ACE inhibitors may be used for congestive heart failure and for post-MI (myocardial infarction) patients.

The most common side effect of ACE inhibitors is a nagging cough. Most patients misinterpret the cough as a cold, or even bronchitis. If they smoke, then it's an aggravation of their smoker's cough. Patients self-medicate but get no relief and often end up asking their doctor for stronger cough suppressants and sleeping pills because the cough keeps them awake. Other side effects range from breast

enlargement in men to reduction in white blood cell count, kidney and liver damage, teratogenesis (fetal damage) as well as the more common allergic reactions, dizziness, headaches, irregular heart beat, nausea, sexual dysfunction, jaundice, metallic taste, and flushing.

As for drug interactions, ACE inhibitors increase the potency of loop diuretics and potassium-sparing diuretics and if used together should be done so with caution. But they lose their potency when used with antacids and NSAIDs. Tetracycline loses its effectiveness in the presence of ACE inhibitors but the effects of erythromycin are increased.

CALCIUM CHANNEL BLOCKERS

Calcium channel blockers are the fourth major class of antihypertensives. Calcium channel blockers are also prescribed for angina, arrhythmias, rapid heart rate, and congestive heart failure. Beyond heart disease, they are also prescribed for Raynaud's syndrome and migraine headaches.

Proper amounts of calcium, under the guidance of magnesium, move in and out of the cells in the body; magnesium is a natural calcium channel blocker. Especially when there is a existing magnesium deficiency, too much calcium can enter the smooth muscle cells of blood vessels leading to constriction and hypertension. Blocking calcium from entering these cells results in less constricted blood vessels, lowering of blood pressure, and increased blood flow through the coronary arteries. However, when a synthetic calcium channel blocker drug is used, there is no guarantee that just the right amount of calcium will be blocked.

The list of side effects and drug contraindications for calcium channel blockers is even longer than the other antihypertensive medications. Increasing numbers of studies that inventoried these side effects have appeared over the last several years. A review article in the British Medical Journal in 1998 found that the suicide risk for patients on calcium channel blockers was 5.4 times higher than in users of other heart drugs. The study was initiated because there were a number of earlier reports associating calcium channel blockers with depression.[2] Another study in 1998 reported that heart attacks occur ten times more often in hypertensive diabetics who take calcium channel blocker drugs than non-hypertensive diabetics.[3]

Posicor (mibefradil) was taken off the market in 1998 for several reasons. It caused life-threatening arrhythmias and worsened congestive heart failure that it was meant to treat. It was also found to have potentially dangerous interactions with more than 25 other medications because it inhibits liver enzymes used to break down other medications. Therefore, drugs taken simultaneously may build up to dangerous levels in the body.

In 2000, calcium channel blockers were found to be associated with an elevated risk of both upper and lower gastrointestinal tract bleeding. GI tract bleeding can be very dangerous, especially for heart patients who are on blood-thinning medication.[4] At the August 2000 Meeting of the European Society of Cardiology in Amsterdam, Dr. Alderman commented that calcium channel blockers may be "a drug of rather last resort." Dr. Alderman was commenting on an analysis of nine clinical trials that included more than 27,000 patients. Compared to patients on other blood pressure medications, patients treated with calcium channel blockers had a 27% increased risk of heart attacks; the risk of heart failure was 26% higher;

and the risk of any major cardiovascular event was 11% higher. The report also stated that calcium channel blockers are significantly more expensive, costing up to 15 times more than some diuretics.

REFERENCES

1. All side effects and drug interactions noted in the introduction are listed in the Physician's Desk Reference PDR and the Canadian Compendium of Pharmaceuticals and Specialties (CPS) which is available in any doctor's office, pharmacy, library, or online.

2. Lindberg G, et al. Use of calcium channel blockers and risk of suicide: ecological findings confirmed in population based cohort study. British Medical Journal 1998;316:741-745.

3. Estacio RO. The effect of nisoldipine as compared with enalapril on cardiovascular outcomes in patients with non-insulin-dependent diabetes and hypertension. N Engl J Med. 1998;338:645-652.

4. Kaplan RC. Use of calcium channel blockers and risk of hospitalized gastrointestinal tract bleeding. Archives of Internal Medicine 2000;160:1849-1855.

APPENDIX C
CHOLESTEROL LOWERING DRUGS AND THEIR SIDE EFFECTS

HMG- CoA Reductase Inhibitors (Statins)

Atorvastatin (Lipitor) is probably the most well known statin drug but there is a long list of others that can be prescribed when the side effects of one become too pronounced. Baycol (cerivastatin-now taken off the market) Fluvastatin (Lescol), Lovastatin (Mevacor), Pravastatin (Pravachol), and Simvastatin (Zocor).

These drugs are direct cholesterol-blockers. They block an enzyme that is essential for making cholesterol in the body. Even though cholesterol made in the body is a precursor to all our hormones: progesterone, estrogen, testosterone, cortisone, and DHEA, cholesterol-blocking drugs are used on a daily basis by millions of people. The hormone-related side effects from these drugs include breast growth in men, impotence, female baldness, insomnia, and fatigue. The complete list of side effects in the Canadian Compendium of Pharmaceuticals and Specialties (CPS) and the U.S. Physician's Desk Reference (PDR) includes abdominal pain, joint pain, lack of energy, back pain, gall stones, cirrhosis, constipation, diarrhea, drowsiness, dyspepsia, elevated liver enzymes, fatigue, fever, flatulence, headache, liver necrosis, hepatitis, malaise, myalgia, myasthenia, myoglubinuria, myopathy, nausea and vomiting, pancreatitis, pharyngitis, renal tubular obstruction, rhabdomyolysis, sinusitis, weakness.

Before putting patients on a statin drug they should be screened for alcohol problems, hormone disorders (such as diabetes and under-active thyroid), blood salt imbalance, infection, kidney disease, liver disease, low blood pressure, muscle disorder or condition, recent surgery, seizures (convulsions), severe injury, allergic reaction to the statins, other medicines, foods, dyes, or preservatives, pregnancy or a patient trying to get pregnant, or breast-feeding.

One serious side effect of the statin drugs is the depletion of Coenzyme Q-10. But most doctors are not aware of this side effect. Coenzyme Q-10 is an extremely important antioxidant and essential for the heart pumping action. Statin medications can actually increase the risk of heart disease in some patients by depleting Coenzyme Q-10. What is apparent from studying how statins work is that the same mechanism that blocks cholesterol production shuts down Coenzyme Q-10 production. The side effects of muscle pain and weakness are a direct result of lack of Coenzyme Q-10.

In a group of about two hundred patients, half took atorvastatin and the other half took simvastatin to determine what effect statin therapy had on cholesterol absorption from the diet. They concluded that effective inhibition of cholesterol synthesis by the statins reduced serum cholesterol but triggered a mechanism by the body to increase dietary cholesterol absorption.[1]

A research database of the province of Manitoba was used to identify users of statins between January 1, 1995, and March 31, 1998. A total of 28,705 statin users were identified. During the study period, 24,496 (85.3%) individuals took 1 statin, 3751 (13.1%) took 2 statins, and 458 (1.6%) took 3 to 5 statins at any one time. Patients on more than one statin were found to utilize significantly more health care resources than those on one statin. The study makes much of the fact that

pravastatin users taking interacting drugs had significantly fewer hospitalizations (1.3); fewer physician visits (24.2); and lower health care costs ($5,526) compared with users of lovastatin (1.7, 28.0, and $6,925, respectively) and fewer physician visits than simvastatin users (25.6). But when you look closely at the data, pravastatin and lovastatin users had 1.3 versus 1.7 hospitalizations, 24.2 versus 28 doctors visits, and $5,526 versus $6,925. There seems to be no great difference and an incredible assumption that hospitalizations, doctors' visits, and dangerous side effects are somehow normal. [2]

In a Russian study, 90 patients with mild or moderately high cholesterol and heart disease were given a 12-week trial of lovastatin after 6 weeks of diet alone. We weren't told what the cholesterol levels were after the diet phase. According to protocol, if after six weeks on 20 mg/day of the drug the cholesterol levels were not reduced, the dosage was increased to 40 mg/day. Only 47% of patients reached the target level and by doubling the dosage, which the layperson might assume would double the results, there were only 11% more patients who reached the target levels of cholesterol.[3] Noting the side effects of the statin drugs, a double dosage seems a high price to pay for only an 11% gain. However, that is the way doctors are advised to treat cholesterol. Those who don't respond, the other 42%, will be offered one or two or even three more statin drugs.

In 1996 a review of lipid-lowering drugs in JAMA proved the cancer-causing potential of the statins and fibrates—two of the most popular types. [4] The authors found that every drug in the statin and fibrate classifications caused cancer in rodents. They conceded that longer-term clinical trials and careful post-marketing surveillance during the next several decades are needed to determine whether cholesterol-lowering drugs cause cancer in humans. The authors suggest that the fibrates and statins should be avoided except in patients at high short-term risk of coronary heart disease. That review was in 1996 but there was no sign of abatement in the use of the statin drugs. And it is widely recommended that people go on lipid-lowering drugs for long-term and even life-long management of their lipids. The emphasis is on long-term use especially since there is a rebound cholesterol elevation when a person goes off statin drugs that can triple the risk of heart disease. [5]

The cancer-causing potential of the statin drug simvastatin (Zocor) is now more understood. The drug seems to mimic the action of a particular vascular growth factor and by promoting the growth of new blood vessels it could increase the growth of blood vessels in cancer tumors.[6-8] Vascular endothelial growth factor VEGF also plays a role in diabetic retinopathy.[9] Since many people with diabetes have heart disease there is a tremendous risk to all diabetics who takes statin medications.

The cholesterol-lowering statin drug Baycol was pulled from the market in 2001 after it was linked to at least 31 American deaths in its less than four years.[10] However, physicians and the makers of Baycol, a German company, Bayer AG, have known since 1987 of this fatal side effect. About 700,000 Americans were taking Baycol and had to switch to another drug but the article speculated that about twelve million people taking the other half-dozen statin drugs must be wondering whether theirs is safe. Baycol causes a muscle weakness called myositis (inflammation of the muscles) and muscle destruction in about 1 in 1,000 statin users, according the National Cholesterol Education Program at the National

Heart, Lung, and Blood Institute. In the unfortunate 31 their muscles broke down completely flooding the kidney with protein and causing kidney failure—a condition called rhabdomyolysis. Muscle pain is an extremely common finding in patients on statin drugs. This pain is often medicated with the NSAID's such as the ill-fated Vioxx, which Dr. David Graham of the FDA found to be responsible for as many as 139,000 fatal heart attacks in it's four year history.[11]

A 2001 report of a population of 29,000 patients from Brigham Women's Hospital in Boston found that 1,575 patients were taking statin drugs in 1996.[12] Only about one third were taking statins for heart disease. Another 1,080 patients without documented heart disease were prescribed this medication for "primary" prevention of heart disease. Only one in three patients met the National Cholesterol Education Program guidelines for who should take the drugs. Half of the more than 500 individuals who were receiving statin therapy were being overtreated. Dr. Susan Abookire, the Harvard-affiliated lead researcher, from Partners Healthcare in Boston, Massachusetts said that doctors are, "influenced by what their patients want, which is influenced by advertising."

Research from Switzerland shows that several statin drugs, Lipitor, Mevacor and Pravachol suppress certain immune system cells known as helper T-cells. The researchers put a bizarre positive spin on their results saying, "This unexpected effect provides a scientific rationale for using statins as immunosuppressors, not only in organ transplantation, but in numerous other pathologies as well".[13,14]

Cholesterol-lowering statin drugs may also increase the risk of peripheral nerve damage or neuropathy. Researchers in this study confirm the link between statins and neuropathy in at least 166 patients.[15,16]

A group of Chinese investigators studied the relationship between four different lipid-lowering drug therapies and successful patient outcome. The success rates, defined as reaching specific total cholesterol and LDL cholesterol target levels, were 51.8% for simvastatin, 42.9% for pravastatin, 31.6% for fluvastatin, 12.5% for other drugs respectively. When it became obvious that only 36% of the patients had reached target levels, instead of questioning the form of treatment the researchers recommended more aggressive treatment.[17]

Statin drugs are being recommended to treat patients after an acute MI. but one study showed no effect of fluvastatin on heart ischaemia and no major clinical events in the first year after acute MI could be detected. The authors conclude that their present data do not confirm other reports, which support widespread use of statin treatment early after acute myocardial infarction.[18]

Dr. Uffe Ravnskov, a highly regarded critic of statin medications remarks that the studies on statin drugs, mostly done with drug company funding, may draw dramatic conclusions from very minor differences between drug treatment groups and placebo groups.[19]

In the CARE trial Prevastatin was studied in the treatment of lipids. The final conclusion was that statin treatment improved a heart patient's risk of escaping death from a heart attack over five years by the very low amount of 1.1 %. Dr. Ravnskov analyzed the data and found that the chances of avoiding death for a patient with heart disease was 94.3 %, which only improved to 95.4 % with statin treatment.[20]

Dr. Ravnskov looked at another trial called WOSCOPS, which studied healthy people with high cholesterol to determine whether statin therapy would improve their life expectancy. The effect is even smaller with 98.4 % survival rate off statins and 98.8 % on statins. Patients are being asked to pay upwards of $150 dollars per month for a patented statin drug to obtain a 0.4% difference in mortality. Meanwhile the morbidity and long-term mortality of the statins continues to rise.[21]

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